Control of Hazardous Air Pollutants from Mobile Sources

Summary and Analysis of Comments

Chapter 6
Cost-Benefit Analysis and EIA

Assessment and Standards Division
Office of Transportation and Air Quality
U.S. Environmental Protection Agency

SUMMARY AND ANALYSIS OF COMMENTS:
CHAPTER 6
COST-BENEFIT ANALYSIS

6. COST-BENEFIT ANALYSIS

What We Proposed:

The comments in this chapter correspond to Section IX of the NPRM, and therefore are targeted at the cost-benefit analysis. A summary of the comments received, as well as our response to those comments, are located below. For the full text of comments summarized here, please refer to the public record for this rulemaking.

6.1 Predicted Health Benefits of the Rule

What Commenters Said:

The commenters stated that they believe that EPA does not justify the benefit of each programs contained in MSAT2 separately; instead, they claimed the three programs are combined to assess costs and benefits. The commenters stated that this approach makes it difficult to assess the individual contribution of each program under the MSAT2 proposal. If evaluated independently, the commenters stated, the fuels program is likely to provide the lowest potential incremental benefit, yet most costly element of the proposed rule.

The commenters also noted that the quantified economic benefits used by EPA to justify the MSAT2 Proposal are based entirely on reduction of tailpipe emissions of particulate matter. They stated that they believe it is not appropriate to use benefits from one part of one regulatory initiative, namely, the Cold Temperature Vehicle Standard, to justify what essentially constitutes two other separate regulatory initiatives, new limits on benzene content in gasoline and a hydrocarbon emission standard for gas cans.

The commenters stated that they believe EPA's cost/benefit analysis completely fails to monetize the benefits of its gasoline benzene reduction proposal, focusing instead on the particulate matter (PM) related benefits associated with its proposed cold temperature vehicle standards. Hence, the commenters believe that EPA has not justified the need for its proposed reduction in benzene content of gasoline to 0.62 vol% on a cost/benefit basis.

Letters:
American Petroleum Institute (API)   OAR-2005-0036-0884
ExxonMobil Refining & Supply Company   OAR-2005-0036-0772, -1013
Marathon Petroleum Company   OAR-2005-0036-0946, -1008

Our Response:

We found, and continue to find, that each of the three major aspects of the rule are separately justifiable under either Clean Air Act section 202(l) or (for portable fuel containers) section 183(e). Furthermore, standards under sections 202(l) and 183(e) are not established or justified on a cost-benefit basis, and we did not justify any of the rule's programs on that basis.

The statement that the programs are combined to assess costs and benefits is incorrect. We present the emission reductions associated with each of the three programs in this rule. Likewise, we have separately characterized the costs of each program. EPA does not combine the benefits of each of the rule provisions. Chapter 12 of the Regulatory Impact Analysis (RIA) presents only the PM-related benefits associated with emission reductions attributed to the cold temperature vehicle standards. Benefits for all other rule provisions are described qualitatively due to analytical constraints and limitations discussed in the RIA. We do, however, present a comparison between the benefits of the cold temperature vehicle standards versus the costs of that program, as well as a comparison of the benefits of the cold temperature standards versus costs across all programs.

What Commenters Said:

Both API and ExxonMobil commented that they believe the quantified benefits of reducing particulate matter are based on a series of highly uncertain and questionable scientific assumptions and represent extreme overestimates. These include use of overly conservative and inaccurate concentration response functions (CRFs) for mortality and morbidity health endpoints, and monetizing health endpoints for which a causal relationship has not been established. Specifically, the commenters believe: 1) a lower CRF should be used to assess the critical endpoint of chronic mortality; 2) infant mortality should be removed from the cost-benefit analysis (CBA) since causality has not been established; and 3) the morbidity endpoints of bronchitis and restricted activity days cannot be clearly linked to exposure to fine PM nor quantified with any degree of accuracy and should also be removed from the CBA. Our scientific concerns with the EPA benefits assessment regarding particulate matter are further detailed below.

Letters:
American Petroleum Institute   OAR-2005-0036-0884
ExxonMobil Refining & Supply Company   OAR-2005-0036-0772, -1013

Our Response:

We rely on the published scientific literature to ascertain the relationship between PM and adverse human health effects. We evaluate the epidemiological studies using a well-established set of selection criteria. These criteria include consideration of whether the study was peer-reviewed, the match between the pollutant studies and the pollutant of interest, the study design and location, and characteristics of the study population, among other considerations. The selection of concentration-response functions for all of EPA's

benefits analyses is guided by the goal of achieving comprehensiveness and scientific defensibility.

In addition to the above selection criteria, EPA relies on the guidance provided by internal and external review panels, comprised of distinguished scientists, engineers, and economists who are recognized, non-governmental experts in their respective fields. EPA consults with the Science Advisory Board's Health Effects Subcommittee (SAB-HES) and Clean Air Science Advisory Committee (CASAC) in the development and improvement of methods we use to estimate and value the potential reductions in health effects associated with air quality improvements. All of EPA's regulatory analyses also are reviewed extensively by the Office of Management and Budget (OMB). EPA also looks to recommendations provided by panels such as those convened by the National Academy of Sciences (NAS) to specifically address facets of our cost and benefits analyses.

In regard to PM-related adult mortality, the SAB-HES panel recommended using long-term prospective cohort studies in estimating mortality risk reduction.[1] This recommendation has been confirmed by a recent report from the National Research Council (NRC), which stated that "it is essential to use the cohort studies in benefits analysis to capture all important effects from air pollution exposure" (NRC, 2002, p. 108).[2] More specifically, the SAB recommended emphasis on the American Cancer Society (ACS) study because it includes a much larger sample size and longer exposure interval and covers more locations (e.g., 50 cities compared to the Six-Cities Study) than other studies of its kind. Because of the refinements in the extended follow-up analysis, the SAB-HES recommends using the Pope et al. (2002) study[3] as the basis for the primary mortality estimate for adults and suggests that alternate estimates of mortality generated using other cohort and time-series studies could be included as part of the sensitivity analysis (U.S. EPA-SAB, 2004b).[4]

The SAB-HES also recommended using the specific estimated relative risks from the Pope et al. (2002) study based on the average exposure to $PM_{2.5}$, measured by the average of two $PM_{2.5}$ measurements, over the periods 1979–1983 and 1999–2000. In addition to relative risks for all-cause mortality, the Pope et al. (2002) study provides relative risks for cardiopulmonary, lung cancer, and all-other cause mortality. Because of concerns regarding the statistical reliability of the all-other cause mortality relative risk estimates, we calculated mortality impacts for the primary analysis based on the all-cause relative risk. Based on our most recently available SAB guidance, we provide mortality impacts based on the ACS study as the best estimate for comparing across the current and previous RIAs.

The NRC (2002) also recommended that EPA use formally elicited expert judgments as a means of characterizing uncertainty in the concentration-response relationship between $PM_{2.5}$ exposures and mortality. EPA therefore convened a panel of experts to elicit probabilistic distributions describing uncertainty in estimates of the reduction in mortality among the adult U.S. population resulting from reduction in ambient annual average $PM_{2.5}$ levels. The results of this study, completed in 2006

(Industrial Economics, 2006),[5] found that the majority of expert opinion (11 out of 12 experts) believed that the PM2.5-mortality effect was stronger than a comparable result derived from the Pope et al. (2002) ACS study. This leads the Agency to expect that our estimates of mortality derived from the ACS study understate the benefits associated with the final cold temperature vehicle standard.

Regarding infant mortality, recently published studies have strengthened the case for an association between PM exposure and respiratory inflammation and infection leading to premature mortality in children under 5 years of age. Specifically, the SAB-HES noted the release of the WHO Global Burden of Disease Study focusing on ambient air, which cites several recently published time-series studies relating daily PM exposure to mortality in children.[6] The SAB-HES also cites the study by Belanger et al. (2003)[7] as corroborating findings linking PM exposure to increased respiratory inflammation and infections in children. Recently, a study by Chay and Greenstone (2003)[8] found that reductions in TSP caused by the recession of 1981–1982 were related to reductions in infant mortality at the county level. With regard to the cohort study conducted by Woodruff et al. (1997),[9] the SAB-HES notes several strengths of the study, including the use of a larger cohort drawn from a large number of metropolitan areas and efforts to control for a variety of individual risk factors in infants (e.g., maternal educational level, maternal ethnicity, parental marital status, and maternal smoking status). Based on these findings, the SAB-HES recommends that EPA incorporate infant mortality into the primary benefits estimate and that infant mortality be evaluated using an impact function developed from the Woodruff et al. (1997) study.[10] A more recent study by Woodruff et al. (2006)[11] continues to find associations between $PM_{2.5}$ and infant mortality. The study also found the most significant relationships with respiratory-related causes of death. We have not yet sought comment from the SAB on this more recent study and as such continue to rely on the earlier 1997 analysis.

EPA disagrees with the statement that "the morbidity endpoints of bronchitis and restricted activity days cannot be clearly linked to exposure to fine PM nor quantified with any degree of accuracy and should also be removed from the CBA." Regarding chronic bronchitis, Abbey et al. (1995)[12] examined the relationship between estimated $PM_{2.5}$ (annual mean from 1966 to 1977), PM_{10} (annual mean from 1973 to 1977) and TSP (annual mean from 1973 to 1977) and the same chronic respiratory symptoms in a sample population of 1,868 Californian Seventh Day Adventists. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. In single-pollutant models, there was a statistically significant $PM_{2.5}$ relationship with development of chronic bronchitis, but not for airway obstructive disease (AOD) or asthma; PM_{10} was significantly associated with chronic bronchitis and AOD; and total suspended particulates (TSP) was significantly associated with all cases of all three chronic symptoms. Other pollutants were not examined. Because the cold temperature vehicle standards control direct $PM_{2.5}$, this analysis uses only the Abbey et al. (1995) C-R function based on the results of the $PM_{2.5}$ single pollutant model.

Ostro and Rothschild (1989)[13] estimated the impact of $PM_{2.5}$ and ozone on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for ozone, two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years. The C-R function for PM is based on this co-pollutant model. The study is based on a "convenience" sample of non-elderly individuals. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals under 65. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994, p. 30)[14] and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994; Schwartz, 1994).[15,16]

The Agency would also like to point out that MRADs and other morbidity endpoints have been a standard part of recent, peer-reviewed benefits assessments. These include Ostro et al. (2006),[17] Levy et al. (2003),[18] Cifuentes et al. (2001),[19] Levy et al., (2001)[20] and Hubbell et al., (2005).[21]

What Commenters Said:

Both API and ExxonMobil noted that, for the fuels program separately, EPA states it cannot quantify benefits since the NATA assessments do not take full account of the exposure ranges of the population. The commenters stated that EPA instead listed "unquantified" and nonmonetized effects. The commenters noted that these effects are categorized as "ozone health", "ozone welfare", "PM health", "PM welfare", "MSAT Health", and "MSAT welfare" (Table IX.E1, page 15908). The commenters believe that the only benefits from this list that are applicable to the fuels program are "MSAT Health". The commenters also noted that under "MSAT Health", EPA listed the following as applicable to benzene: cancer, anemia, disruption of production of blood components, reduction in the number of blood platelets, excessive bone marrow formation, and depression of lymphocyte counts. The commenters stated that it appears that EPA simply listed all outcomes that have ever been associated with benzene, regardless of whether they could possibly occur at ambient levels. Lastly, the commenters stated that EPA's own reference concentration (RfC) of 30 µg/m3 is in fact regarded as the safe level to protect against all noncancer health effects. The commenters believe that since this level is above present day ambient concentrations, noncancer effect should be referenced in this table.

Letters:
American Petroleum Institute (API)   OAR-2005-0036-0884
ExxonMobil Refining & Supply Company   OAR-2005-0036-0772, -1013

Our Response:

As discussed in Chapter 3 of the RIA, there are numerous observations of personal exposure and indoor air concentrations of benzene in excess of the RfC. Also, as discussed in the RIA for the proposal, estimated average population cancer risks from inhalation exposure to benzene are likely to be substantial underestimates. In addition to the potential for the current unit risk range to be a substantial underestimate, inventories used in risk modeling for the proposal did not include elevated cold start emissions for gasoline vehicles or portable fuel container emissions. Moreover, the exposure modeling did not adequately capture near road impacts on risk, or impacts of emissions in attached garages to exposure and risk. Modeling done for the final rule did account for the additional sources of exposure described above.

What Commenters Said:

The commenters stated that ambient levels of sulfur dioxide in the U.S. are much lower today than those present when the ACS study was conducted. The commenters further stated that clinical and toxicology studies clearly demonstrate that sulfur dioxide (SO_2) enhances the toxicity of PM, particulate matter (Costa, 2001). The commenters believe this supports the concept that CRFs reported by Pope et al. and used by EPA in the benefits analysis for particulate matter should be adjusted downward to account for the reduced impact of SO_2 between past and current conditions.

The commenters believe that whether ambient exposure to SO_2 produces an independent risk for mortality, as suggested by Krewski et al., acts as a surrogate for other pollutants in the air pollution mix (as suggested by some authors) or actually increases the risk of PM (as suggested by clinical and toxicology studies) is arguable. The commenters state that since SO_2 increases the toxicity of particulate matter, use of the 6% value – without adjustment – does not provide an accurate estimate of PM risk.

For the critical health effect of chronic PM mortality, the commenters stated tht they believe the authors of the CBA used an overly conservative, scientifically invalid and inflated value of 6% change in mortality per 10 µg/m3 of PM2.5, as derived from the ACS Study as reported by Pope et al. (2002). The commenters stated that they believe the results of the thorough reanalysis of this study by Krewski et al. (2000, 2003) clearly demonstrate effect modification by education and other factors such as temperature variation and population change, attenuation of particle effect when spatial correlation was considered, and most importantly, strong attenuation of the particle effect when sulfur dioxide was simultaneously considered in the model. The commenters therefore suggested using a coefficient of 1% rather than 6%, based on data by Krewski et al. which they believe provides a more complete adjustment for the effects of SO_2.

Letters:
American Petroleum Institute (API) OAR-2005-0036-0884
ExxonMobil Refining & Supply Company   OAR-2005-0036-0772, -1013
Alliance of Automobile Manufacturers   OAR-2005-0036-0881

Our Response:

We agree with the need to address co-pollutants when employing epidemiologic models. The Health Effects Institute (HEI) reanalyses generally confirmed the original investigators' findings of associations between mortality and long-term exposure to PM, while recognizing that increased mortality may be attributable to more than one ambient air pollution component. Regarding the validity of the published ACS Studies, the HEI Reanalysis Report concluded that overall, the reanalyses assured the quality of the original data, replicated the original results, and tested those results against alternative risk models and analytic approaches without substantively altering the original findings of an association between indicators of particulate matter air pollution and mortality.

The most recent external review draft of the PM criteria document reaches similar conclusions.

While the Agency recognizes the ongoing need to research the issue of copollutants, including SO_2, and their role in quantifying the relationship between long-term exposure to PM2.5 and mortality, we disagree with the commenter's interpretation of the HEI reanalysis and their assertion that we are using an overly conservative, scientifically invalid and inflated coefficient. Although the HEI reanalysis did find a robust association between mortality and SO_2, such an association was also reported for fine particles and sulfate. In addition, the study points out that efforts to address spatial autocorrelation for ecologic-scale variables such as fine particles and sulfate may have over-adjusted estimated effects for these regional pollutants compared with effect estimates generated for local copollutants including SO_2. This could partially account for the higher effect estimate generated for SO_2 relative to fine particles and for sulfate. In addition, SO_2 is associated with sulfate formation and consequently, SO_2 concentrations are likely surrogates for sulfate concentrations, which could explain their statistical association with PM2.5-related mortality.

In considering this issue of SO_2 as a copollutant and its impact on the association between mortality and long-term exposure to PM2.5, it is also important to consider the wider literature. Two recent studies examining the relationship between gaseous copollutants (including SO_2) and PM-related health effects including mortality (Samet et al., 2000),[22] conclude that SO_2 is likely to represent a surrogate for ambient PM2.5 concentrations and may in certain circumstances represent a surrogate for personal exposure to PM2.5. Furthermore, both studies conclude that SO_2 is unlikely to be a confounder for PM2.5-related health effects (i.e., it is unlikely to be associated directly with these health effects while being correlated with PM2.5 exposure). Further evidence against SO_2 as a confounder specifically for mortality effects involves biological plausibility. While SO_2 is recognized as effecting airways causing difficulty in breathing, especially for asthmatics, there is little evidence of a causal link between SO_2 exposure and cardiovascular- or lung cancer-related mortality. This argues against SO_2 as a confounder for PM2.5-related mortality effects.

Following recommendations from the National Academy of Sciences and SAB-HES, we have continued to update our methods for benefits estimation to reflect the latest research and are now using the Pope et al, (2002) reanalysis of the ACS study data. This latest reanalysis has a number of advantages over prior studies in evaluating the role of SO_2 in the relationship between PM2.5 exposure and mortality. The ACS reanalysis includes 8 additional years of follow up data, including data on fine particulates and gaseous copollutant exposure. The ACS reanalysis also considers a variety of additional covariates believed to be associated with mortality and uses the latest statistical methods (e.g., non-parametric spatial smoothing) for addressing key issues such as spatial autocorrelation. While the ACS reanalysis continues to show a strong correlation between SO_2 and all cause and cardio-vascular mortality, suggesting that it is likely a surrogate for particulate fine and more likely sulfate exposure, the study also provides the strongest evidence yet for an association between long-term exposure to PM2.5 and mortality.

The NRC (2002) also recommended that EPA use formally elicited expert judgments as a means of characterizing uncertainty in the concentration-response relationship between $PM_{2.5}$ exposures and mortality. EPA therefore convened a panel of experts to elicit probabilistic distributions describing uncertainty in estimates of the reduction in mortality among the adult U.S. population resulting from reduction in ambient annual average $PM_{2.5}$ levels. The results of this study, completed in 2006 (Industrial Economics, 2006),[23] found that the majority of expert opinion (11 out of 12 experts) believed that the PM2.5-mortality effect was stronger than a comparable result derived from the Pope et al. (2002) ACS study. This leads the Agency to expect that our estimates of mortality derived from the ACS study understate the benefits associated with the final cold temperature vehicle standard.

What Commenters Said:

Commenters stated that they believe that EPA's continued use of the Value of a Statistical Life (VSL) approach, with a cost of $6 million per hypothesized mortality event, markedly inflates the benefits in this proposal. They suggested that EPA consider the more scientifically valid approach based on life years lost.

<u>Letters:</u>
American Petroleum Institute (API)   OAR-2005-0036-0884
ExxonMobil Refining & Supply Company   OAR-2005-0036-0772, -1013

Our Response:

EPA agrees that there is a large amount of uncertainty in the VSL for application to environmental policy analysis. However, as noted in the RIA, the SAB Environmental Economics Advisory Committee has advised that the EPA "continue to use a wage-risk-based VSL as its primary estimate, including appropriate sensitivity analyses to reflect the uncertainty of these estimates," and that "the only risk characteristic for which

adjustments to the VSL can be made is the timing of the risk"(EPA-SAB-EEAC-00-013).[24] In response to concerns about the range of estimates included in the VSL distribution, we have modified the value of life distribution. The mean value of avoiding one statistical death is now assumed to be $5.5 million in 1999 dollars. This represents a central value consistent with the range of values suggested by recent meta-analyses of the wage-risk VSL literature. The distribution of VSL is characterized by a confidence interval from $1 to $10 million, based on two meta-analyses of the wage-risk VSL literature. The $1 million lower confidence limit represents the lower end of the interquartile range from the Mrozek and Taylor (2000) meta-analysis.[25] The $10 million upper confidence limit represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis.[26] The mean estimate of $5.5 million is consistent with the mean VSL of $5.4 million estimated in the Kochi et al. (2006) meta-analysis.[27]

In developing our estimate of the benefits of premature mortality reductions, we have discounted over the lag period between exposure and premature mortality. However, in accordance with the SAB advice, we use the VSL in our primary estimate. Consistent with the SAB advice and in accordance with the provisions contained in the FY04 Appropriations bill, we do not adjust the VSL to reflect any differences across age groups.

What Commenters Said:

API and ExxonMobil commented that they have concerns regarding EPA's evaluation of bronchitis as a health endpoint. First, contribution of particulate matter to the incidence of bronchitis is entirely attributed to fine particle exposure, which is scientifically incorrect. They commented that this invalid attribution contrasts with the etiology of bronchitis, and highlights the need for clinical input to the EPA CBA. The commenters noted that bronchitis is primarily a disease of the upper respiratory tract; and coarse particles, which deposit in the upper respiratory tract, are much more likely to contribute to the etiology of this disease. The commenters stated that fine particles deposit primarily in the lower respiratory tract, and are not expected to significantly contribute to the incidence of bronchitis. The commenters stated that they believe it is biologically inappropriate to convert the morbidity function from a study using coarse PM10 to fine PM2.5; rather, for bronchitis, a separate benefits analysis for PM10 or other coarse-particle metric – such as total PM2.5, 10 or TSP – should be provided. The commenters noted that in the study by Abbey et al. (1995a,b), a stronger relationship was observed for TSP – the actual metric of particle exposure used – than for either PM10 or PM2.5.

The commenters also commented that they are concerned that concentration response functions used were incorrectly applied to the air pollutant under consideration. Since monitoring both PM10 and PM2.5 was very limited in California before 1986, Abbey et al. (1995a,b) used data for TSP to estimate PM10, and airport visibility records to derive an estimate for PM2.5. The commenters stated that they believe this approach

is awkward for estimating exposures, and seriously jeopardized the findings from this study.

The commenters further commented that they are concerned that the assessment of bronchitis is based on a single study (Abbey et al., 1995a,b), for which the result was not statistically significant – even at the 5% level. They do not believe that causality can be established based on the results of a single ecological epidemiology study. Further, they questioned the accuracy of a concentration response function based on a single study result. In particular, they stated that they are concerned with the accuracy of the adjustment for smoking in this study, which they believe is a major contributor to the incidence of bronchitis. The commenters stated that they believe the magnitude of calculated risk due to PM air pollution, which is essentially the same as the background rate attributed to all other factors, raises more suspicion and illustrates the need for reality checks. Finally, the commenters stated that EPA's development of a concentration response function based on findings from a single study (here, results that were not statistically significant at the 5% level) begs the question as to whether are sufficiently robust data set to conclude in a CBA.

The commenter also questioned the adjustment of a concentration-response function for PM10 to one based on PM2.5 based on the simple mean ratio of these particles in urban air. The commenter stated that the authors offer no biological explanation as to why such as an adjustment is appropriate, or why fine PM would be expected to exhibit the same potency as coarse particles. The commenter noted that fine and coarse particles distribute differentially in the respiratory tract and produce a different and separate spectrum of health effects; and certain respiratory symptoms would be expected to be exacerbated more by exposure to coarse rather than fine PM, a finding consistent with the actual study, where stronger associations were observed for TSP than for PM10 or PM2.5 surrogates. The commenters stated that it is unclear why the authors of the CBA choose to attribute all RAD related effects to fine PM.

The commenters stated that they are concerned with the use of data from California during the period of 1966-1988 when air pollution was high, likely resulting in an inflated CRF. The commenters stated that the air pollution data that are the basis of the study used for the CBA are from 1966-1987—close to 30 years old. The commenters questioned if concentration response functions based on results using this air pollution data are robust enough to use in a CBA designed to project results nearly 20 years into the future. The commenters further stated that California data are dominated by photochemical smog, but this concentration response function overestimates effects of low levels of PM alone. The commenters also stated that whether or not a threshold exists for this endpoint, and whether or not the concentration response function is specific to particulate matter, photochemical pollution, other gases present in ambient air, or a combination of these, has not been evaluated.

The commenters stated that they are likewise concerned that assessment of Restricted Activity Days (RADs) and Minor Restricted Activity Days (MRADs) endpoints are solely based on the results of the Health Interview Study, as reported by

Ostro (1987) and Ostro & Rothchild (1989). They noted that the concentration-response functions derived from this study are based on air pollution data from 1976-1981, when air pollution levels were significantly greater. The commenters also questioned the exposure metrics used in this study, as PM10 and PM2.5 levels were not measured (PM2.5 levels were estimated from visibility data from airports). The commenters further stated that there has been no assessment of whether RADs or MRADs would even be triggered by lower air pollution levels—thus they believe that the issue of threshold has not been explored at all for these morbidity endpoints.

The commenters stated that it believes that the health endpoints of RAD and MRAD are highly subject to socioeconomic confounding. The commenters noted that the in study used to derive the concentration response functions, significant city-to-city differences in RAD rates were observed. The commenters believe that this was likely due to socioeconomic factors and other factors that were not adequately controlled in the selected study. The commenters also stated that they believe that many of the socioeconomic factors that need to be controlled to identify the potential effect of air pollution are likely much more important than air pollution itself in the production of RADs and MRADS.

Letters:
American Petroleum Institute (API)   OAR-2005-0036-0884
ExxonMobil Refining & Supply Company   OAR-2005-0036-0772, -1013

Our Response:

EPA relies on the guidance provided by internal and external review panels comprised of distinguished scientists, engineers, and economists who are recognized, non-governmental experts in their respective fields. EPA consults with the Science Advisory Board's Health Effects Subcommittee and Clean Air Science Advisory Committee in the development and improvement of methods we use to estimate and value the potential reductions in health effects associated with air quality improvements. All of EPA's regulatory analyses also are reviewed extensively by the Office of Management and Budget. EPA also looks to recommendations provided by panels such as those convened by the National Academy of Sciences to specifically address facets of our cost and benefits analyses. We point this out because chronic bronchitis and MRADs have been included in every major air quality-related RIA for the last 10 years.

During that time, the Agency has received much internal and external review on these, and other, morbidity endpoints. The Agency's desire to characterize a comprehensive suite of health effects associated with its rules has been noted by both the National Academy of Sciences (NRC, 2002)[28] and the SAB-HES (EPA, 2004),[29] despite our reliance on an aging literature. Though weaknesses and uncertainties in the epidemiological literature are acknowledged and described qualitatively in the RIA (see Chapter 12), our decision to include these endpoints in our cost-benefit analyses continue to be supported by Agency internal and external review.

Furthermore, the Agency's Staff Paper on the Particulate Matter Air Quality Criteria Document characterized the chronic bronchitis literature as follows,

> For respiratory effects, notable new evidence from epidemiological studies substantiates positive associations between ambient PM concentrations and not only respiratory mortality…Of much interest are emerging new findings indicative of likely increased occurrence of chronic bronchitis in association with (especially chronic) PM exposure. The biological pathways underlying such effects can include inflammatory responses, increased airway responsiveness or altered responses to infectious agents. Toxicological studies have provided evidence that supports plausible biological pathways for respiratory effects of fine particles.

> Considered together, the CD finds that the long-term exposure studies on respiratory morbidity reported positive and statistically associations between fine particles or fine particle components and lung function decrements or chronic respiratory diseases, such as chronic bronchitis (CD pp. 8-313, 8-314).[30]

The Agency would also like to point out that MRADs and other morbidity endpoints have been a standard part of recent, peer-reviewed benefits assessments. These include Ostro et al. (2006),[31] Levy et al. (2003),[32] Cifuentes et al. (2001),[33] Levy et al., (2001)[34] and Hubbell et al., (2005).[35]

What Commenters Said:

API commented that the choice of using the benefit endpoints of 2020 and 2030 and the focus on PM are clear indicators that EPA largely used the air modeling work and benefit analysis associated with the Nonroad Diesel Rule for the benefit analysis for the MSAT 2 proposal. API noted that it also commented on the Nonroad Diesel Rule proposal (68 FR 28328, May 23, 2003) that it believed that EPA's PM benefit estimates associated with the Nonroad Diesel Rule were flawed. The commenter believes that since EPA relied on that analysis to estimate PM related benefits associated with the MSAT2 proposal, the estimated PM benefits associated with the MSAT2 proposal are also flawed. API listed the following issues of concern related to the estimated PM benefits: 1) the treatment of uncertainty in cost-benefit analysis; and 2) estimates of value of statistical life.

Regarding the treatment of uncertainty in cost-benefit analysis, the commenter stated that it believes that it was incomplete, flawed and highly misleading. The commenter stated that EPA did not assume a threshold in the CR function for PM mortality, but rather reflected a background threshold assumption of 3 micrograms per cubic meter (DRIA, Ch.12, p. 1230). This, the commenter noted, despite EPA's most recent PM2.5 Criteria Document that concludes that "the available evidence does not either support or refute the existence of thresholds for the effects of PM on mortality across the range of concentrations in the studies" (DRIA, Ch.12, p. 1229). The

commenter stated that not including the uncertainties surrounding the issue of threshold values of the CR (PM mortality) function renders EPA's analysis of uncertainty to be of little or no value.

The commenter stated that it believes that another source of uncertainty is that surrounding the EPA estimates of VSL. The commenter noted that EPA used a central estimate of VSL of $6.6 million (2020 income level expressed in $2000) and $6.8 million (2030 income level expressed in $2000); however, the commenter stated that these estimates are based upon a range of values from various meta-analyses and may reflect risk preferences significantly different from the target population.

The commenter stated that it endorses the use of the best available science throughout the policy making process, and it believes that more research is needed in the derivation of defensible base estimates for the value of a statistical life. The commenter stated that estimates of VSL need to accurately reflect the risk preferences of the target population; however, it does not believe that this was the case with the use of the estimates used by EPA. The commenter noted concerns with the fact that the studies from which the estimates are derived targeted the middle-aged working population and not the most vulnerable population segments to air pollution—the elderly (in fragile health) and the very young. The commenter also stated that the type of risk being valued, typically job related risk, is very different from the risk associated with increased air pollution. The commenter further stated that it believes that little, if any, confidence can be placed in the appropriateness of the VSL estimates used by EPA in valuations of reduced mortality due to decreases in PM concentrations. The commenter stated that this is critical since VSL, along with the EPA estimate of the number of reduced mortalities due to PM reduction (also highly flawed as explained above) are overwhelmingly the predominant factors driving the benefit estimation in this RIA.

The commenter recommended that the EPA move to a comprehensive assessment of uncertainty in its benefit-cost analyses so as to reflect the true uncertainty associated with its net benefit estimates (the commenter suggested that EPA could use a Monte Carlo analysis that captured the true extent of uncertainty associated with the health impacts of PM2.5 concentrations in addition to the other major sources of uncertainty). The commenter believes that the assessment of uncertainty in the proposal is disjointed and conveys a misleading sense of certainty to its net benefit estimates, and only provides limited value to policy deliberations. The commenter also stated that it believes that EPA's unequivocal assertion that societal benefits vastly exceed societal costs in the rule is not supportable given the problems and omissions associated with its benefit estimates and uncertainty analysis.

<u>Letters:</u>
American Petroleum Institute (API)   OAR-2005-0036-0884

Our Response:

We refer the reader to the Nonroad Diesel Rule response-to-comments document for detail regarding the commenter's assertion that the Nonroad Diesel Rule's benefits were flawed (http://epa.gov/nonroad-diesel/2004fr.htm#summary).

Due to the analytical constraints associated with the benefits scaling approach, which are explained in the RIA, we are unable to conduct an analysis of the impact of alternative thresholds. We do, however, qualitatively indicate the uncertainty associated with various $PM_{2.5}$ cutpoints used in the calculation of PM-related mortality. We refer the reader to Chapter 12 of the RIA for this discussion. We also provide a scaled estimate of the Monte Carlo-based confidence interval associated with the benefits for each endpoint and for the total benefits associated with the cold temperature vehicle standards. As one can see, the statistical uncertainty associated with these estimates do not "show a distribution of benefits so disperse as to make any definitive conclusions regarding benefits and costs impossible." We acknowledge, however, that this range does not capture all sources of uncertainty, such as the impact of different thresholds. Per the recommendations of the National Research Council (2002), the Agency is moving towards a comprehensive assessment of uncertainty in its benefit-cost analyses when possible.

EPA agrees that there is a large amount of uncertainty in the VSL for application to environmental policy analysis. However, the SAB Environmental Economics Advisory Committee has advised that the EPA "continue to use a wage-risk-based VSL as its primary estimate, including appropriate sensitivity analyses to reflect the uncertainty of these estimates," and that "the only risk characteristic for which adjustments to the VSL can be made is the timing of the risk"(EPA-SAB-EEAC-00-013). In response to concerns about the range of estimates included in the VSL distribution, we have modified the value of life distribution. The mean value of avoiding one statistical death is now assumed to be $5.5 million in 1999 dollars. This represents a central value consistent with the range of values suggested by recent meta-analyses of the wage-risk VSL literature. The distribution of VSL is characterized by a confidence interval from $1 to $10 million, based on two meta-analyses of the wage-risk VSL literature. The $1 million lower confidence limit represents the lower end of the interquartile range from the Mrozek and Taylor (2000) meta-analysis. The $10 million upper confidence limit represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis. The mean estimate of $5.5 million is consistent with the mean VSL of $5.4 million estimated in the Kochi et al. (2006) meta-analysis. The modified VSL distribution is reflected in the scaled benefits estimated for this analysis.

In developing our estimate of the benefits of premature mortality reductions, we have discounted over the lag period between exposure and premature mortality. However, in accordance with the SAB advice, we use the VSL in our primary estimate. Consistent with the SAB advice and in accordance with the provisions contained in the FY04 Appropriations bill, we do not adjust the VSL to reflect any differences across age groups. The modified lag distribution is reflected in the scaled benefits estimated for this analysis.

6.2   Predicted Social Costs of the Rule

What Commenters Said:

API noted that EPA estimates costs of the fuels program to be $250 million annually in total social welfare costs in 2030 (in 2003 $) (Table IX.E4, page 15912). API believes this $250M social cost estimate is very likely to underestimate true social costs, since EPA considered only the fuels program impact on residential users in their calculations. The commenter goes on to state that the Agency focused only on impacts related to personal transportation or residential lawn/garden care and recreational use. Additional costs associated with complying with the proposed programs related to production of goods and services that use gasoline fuel as production input were not considered. EPA justifies their focus on only residential cost impacts based on 1) a Department of Energy (DOE) and California Air Resources Board (CARB) study suggesting that the commercial share of the end user market for gasoline is relatively small and 2) EPA's assumption that the share of gasoline-related costs to total production costs is small (page 15913 of proposed rule). However, the commenter believes the true costs would undoubtedly be much larger if these were taken into account. As such, a key question not answered by EPA is whether the benefits of the fuel program alone exceed the estimated $250M annual social cost of the fuels program.

Letters:
American Petroleum Institute (API)   OAR-2005-0036-0884 (p.19)

Our Response:

Our method for estimating the social costs of the program uses a partial equilibrium model that examines the impacts on directly affected stakeholders (fuel providers and users). We did not examine the impacts on application markets (goods and services produced using gasoline fuel). This is because a price change of the magnitude associated with the fuel requirements is very small and well within the normal gasoline price fluctuations experienced by such commercial entities. In addition, gasoline fuel is likely to be only a small part of the total production inputs used to produce those goods and services. For example, the gasoline used in a delivery van is likely to be small part of the operating costs of a delivery service company, with labor and other inputs constituting the main production costs. Finally, the vast majority of consumers of gasoline fuel are individual noncommercial users. This is supported by the information cited in the question as well as DOE data that indicates that only about 6 percent of gasoline fuel sold in the United States is used for commercial or industrial transportation.

For these reasons we believe that the impacts on the broader economy would be relatively small and perhaps not large enough to disturb the results had a general equilibrium model of the economy been designed and utilized in this analysis. Consequently, while there are other non-quantified social costs in addition to the social costs estimated in the EIA, these are not likely to be large.

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