Health Benefits of Reducing Particulate Air Pollution from Heavy Duty Vehicles

Bryan J. Hubbell, Ph.D., U.S. EPA (Corresponding Author), Trish Koman, M.S., U.S. EPA, Tyler J. Fox, M.S., U.S. EPA, Norman Possiel, M.S., U.S. EPA, Gregory Stella, M.S., U.S. EPA, and Brian Timin, M.S., U.S. EPA

Correspondence to: Dr. Bryan J. Hubbell

Innovative Strategies and Economics Group

Office of Air and Radiation

U.S. Environmental Protection Agency

Mail Code C339-01

RTP, NC 27711

Ph: (919)541-0621

Fax: (919)541-0839

E-mail: hubbell.bryan@epa.gov

Bio: Dr. Bryan J. Hubbell, Senior Economist, U.S. Environmental Protection Agency, Office of Air and Radiation.

--

Health Benefits of Reducing Particulate Air Pollution from Heavy Duty Vehicles

Abstract:

The U.S. Environmental Protection Agency (U.S.EPA) recently promulgated regulations to reduce air pollution from heavy-duty vehicles. This article reports the estimated health benefits of reductions in ambient particulate matter (PM) concentrations associated with those regulations based on the best available methods of benefits analysis. The results suggest that when heavy-duty vehicle emission reductions from the regulation are fully realized in 2030, they will result in substantial, broad scale reductions in ambient particulate matter. This will reduce the incidence of premature mortality by 8,300, chronic bronchitis by 5,500, and respiratory and cardiovascular hospital admissions by 7,500. In addition, over 175,000 asthma attacks and millions of respiratory symptoms will be avoided in 2030. The economic value of these health benefits is estimated at over $65 billion.

Keywords: air pollution, health benefits, diesel

Acknowledgements:

The authors would like to acknowledge Ellen Post, Kenneth Davidson, Don McCubbin, Brad Firlie, and Leland Deck at Abt Associates for their work in developing and running CAPMS. We also acknowledge Lisa Conner for her work on the income adjustment procedure. Thanks also to the many reviewers who contributed valuable comments on earlier drafts, including Mary Ross, Sam Napolitano, Jim Democker, and Ron Evans.

INTRODUCTION

Kunzli et al. (2000) recently estimated the health effects and costs of traffic-related air pollution in Austria, France, and Switzerland. The authors estimated that around 22,000 deaths (out of a total of 40,000 estimated air pollution related deaths) were due to traffic-related air pollution. While their analysis focused on attributing health effects to total traffic-related air pollution, they did not examine the health effects of specific air pollution controls on vehicles. The U.S. Environmental Protection Agency (EPA) recently issued a rule to reduce emissions of diesel particles, non-methane hydrocarbons (NMHC), nitrogen oxides (NOx), sulfur dioxide (SO_2), carbon monoxide (CO), and air toxics from heavy-duty vehicles. To support this rule, we estimated the human health and welfare benefits associated with these emission reductions. Due to the complexity of the overall multi-pollutant analysis, this article focuses on reporting the health benefits of reductions in ambient particulate matter (PM) concentrations. However, health improvements also come from reductions in ozone, CO, and air toxics. The full analysis, including PM and ozone-related health benefits as well as non-health related benefits such as improved visibility, is available in the Regulatory Impact Analysis for the Heavy-duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements, which is available along with other supporting documentation at http://www.epa.gov/otaq/diesel.htm. (U.S. Environmental Protection Agency, 2000a)

The reductions in emissions of NOx, SO_2, and PM from heavy-duty vehicles in the United States are expected to result in wide-spread reductions in ambient concentrations of fine particulate matter ($PM_{2.5}$). These improvements in air quality are expected to result in substantial health benefits, based on the body of epidemiological evidence linking PM with health effects

such as premature mortality, chronic lung disease, hospital admissions, and acute respiratory symptoms.. Based on modeled changes in ambient concentrations of $PM_{2.5}$ and PM_{10}, we estimate changes in the incidence of each health effect using concentration-response (C-R) functions derived from the epidemiological literature with appropriate baseline populations and incidence rates. We then apply estimates of the dollar value of each health effect to obtain a monetary estimate of the total PM-related health benefits of the rule. Additional details on the analysis are available in the technical support document for the benefits analysis, available on the internet at http://www.epa.gov/ttn/ecas/regdata/tsdhddv8.pdf. (Abt Associates. 2000).

OVERVIEW OF ANALYTICAL METHODS

This section summarizes the four steps involved in our analysis: 1) Project the impact that the regulation will have on nationwide emissions of NOx, NMHC, SO_2, and PM in 2030; 2) Model air quality in 2030 to determine the changes in ambient concentrations of PM that will result from the changes in emissions of precursor pollutants; 3) Estimate the changes in human health effects that result from the changes in ambient concentrations of PM; and 4) Compute the economic value of changes in human health effects.

Time and resource constraints prevented us from performing extensive new research to measure either the health outcomes or their values for this analysis. Thus, similar to Kunzli et al (2000), our estimates are based on the best available methods of benefits transfer. Benefits transfer is the science and art of adapting primary research from similar contexts to obtain the most accurate measure of benefits for the environmental quality change under analysis.

There are significant categories of benefits that can not be monetized (or in many cases

even quantified), and thus are not included in our accounting of health benefits. These unquantified effects include infant mortality, low birth weight, changes in pulmonary function, chronic respiratory diseases other than chronic bronchitis, morphological changes, altered host defense mechanisms, non-fatal cancers, and non-asthma respiratory emergency room visits. A complete discussion of PM related health effects can be found in the PM Criteria Document (US Environmental Protection Agency, 1996). Since many health effects overlap, such as minor restricted activity days and asthma symptoms, we made assumptions intended to reduce the chances of "double-counting he alth benefits. These assumptions may have lead to an underestimate of the total health benefits of the pollution controls.

Emissions and Air Quality Models

Prior to modeling changes in particulate matter, it was necessary to first develop a national emission inventory. A 1996 base year (a past year where data can be collected and models can be evaluated against observed data) emission inventory was prepared based on Federal Highway estimates of vehicle operation and activity, the estimated distribution of fuel type and weight class of vehicles, and adjusted MOBILE5b and PART5 emission factors. MOBILE5 is a computer program that estimates year-by-year hydrocarbon (HC), carbon monoxide (CO), and oxides of nitrogen (NOx) emission factors for gasoline-fueled and diesel highway motor vehicles. Various modifications were applied to MOBILE5b and PART5 emissions factors, including vehicle type and control combination adjustments, air conditioning usage factors, and emission factor updates to heavy-duty diesel vehicles. Information on the U.S. EPA's highway vehicle emissions models can be found on the internet at

http://www.epa.gov/otaq/models.htm.

The 2030 base case (a future year projection of conditions without the policy) inventory was then prepared by applying area and vehicle-type specific growth and control assumptions to the 1996 base year inventory. The 2030 control case (a future year projection of conditions with the policy in place) was then modeled by applying reduction percentages to the 2030 base case emissions from heavy-duty gasoline vehicles and heavy-duty diesel vehicles as well as additional SO_2 reductions from non-road diesel vehicles. Details of the final comprehensive emission inventories used for the PM air quality modeling and their development are provided in a separate report (U.S. Environmental Protection Agency, 2000b)

We used a national-scale version of the Regulatory Modeling System for Aerosols and Deposition (REMSAD) to estimate PM air quality in the contiguous United States. REMSAD was appropriate for evaluating the impacts of the Heavy-duty Engine and Diesel Fuel rule on U.S. PM concentrations, because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions. The annual county level emission inventory data was speciated, temporally allocated and gridded to the REMSAD modeling domain to simulate PM concentrations for the 1996 base year and 2030 base and control scenarios. Peer-reviewed for the EPA, REMSAD is a three-dimensional grid-based Eulerian air quality model designed to estimate annual particulate concentrations and deposition over large spatial scales, based on inputs of emission inventories of PM precursors (Seigneur et al., 1999). Each of the future scenarios was simulated using 1996 meteorological data to provide daily averages and annual mean PM concentrations required for input to the concentration-response functions of the benefits analysis. Details regarding the application of REMSAD for this analysis are provided in

US EPA (2000c) The peer review recommended several updates to the nitrate, sulfate, and organic aerosol formation processes in the model in order to bring these components up to the current "state-of-the-science". EPA is currently completing these improvements to REMSAD.

Concentration-Response Functions

Benefits for this analysis are based on health effect incidence changes due to predicted air quality improvements in the year 2030. Integral to the estimation of such benefits is a reasonable estimate of future population projections. The underlying data used to create county-level 2030 population projections is based on 1990 county-level population statistics for all U.S. counties collected by the U.S. Census, and future-year state and metropolitan area population estimates provided by the Bureau of Economic Analysis (U.S. Department of Commerce, 1995). Growth factors are calculated using the BEA data and are applied to the 1990 county-level populations.

Fundamental to the estimation of health benefits was our utilization of the PM epidemiology literature. We rely upon C-R functions derived from published epidemiological studies that relate health effects to ambient concentrations of $PM_{2.5}$ and PM_{10}. Most of the reductions in ambient PM are in the fine fraction ($PM_{2.5}$ or less). However, only PM_{10} based C-R functions are available for many health effects. The specific studies from which C-R functions are drawn are listed in Table 1. While a broad range of serious health effects have been associated with exposure to elevated PM levels, we include only a subset of health effects in this benefit analysis due to limitations in available C-R functions and concerns about double-counting of overlapping effects (U.S. Environmental Protection Agency, 1996).

To generate health outcomes, projected changes in ambient PM concentrations were input

to the Criteria Air Pollutant Modeling System (CAPMS), a customized GIS-based program. CAPMS assigns pollutant concentrations to 8 kilometer square population grid cells for input into concentration-response functions. CAPMS uses census block population data and changes in pollutant concentrations to estimate changes in health outcomes for each grid cell. Details on the application of CAPMS for this analysis are provided in a separate report (Abt Associates. 2000).

The baseline incidences for health outcomes used in our analyses are selected and adapted to match the specific populations studied. For example, we use age and county-specific baseline total mortality rates in the estimation of PM-related premature mortality. County-level incidence rates are not available for other endpoints. We used national incidence rates whenever possible, because these data are most applicable to a national assessment of benefits. However, for some studies, the only available incidence information comes from the studies themselves; in these cases, incidence in the study population is assumed to represent typical incidence at the national level. Sources of baseline incidence rates are reported in Table 1.

In this assessment we made analytical judgements affecting both the selection of C-R functions and the application of those functions in estimating impacts on health outcomes. Some of the more important of these are discussed below. Alternative assumptions about these judgements may lead to substantially different results and they are explored using appropriate sensitivity analyses in a later section.

As in the Kunzli et al. (2000) analysis, we focus on the prospective cohort long-term exposure studies in deriving the C-R function for premature mortality. Cohort analyses are better able to capture the full public health impact of exposure to air pollution over time (Kunzli, 2001).

We selected a C-R function from the reanalysis of the American Cancer Society (ACS) study conducted for the Health Effects Institute (Pope et al., 1995; Krewski et al; 2000) The selected C-R function relates premature mortality and mean $PM_{2.5}$ levels rather than median levels as used in the original ACS analysis. For policy analysis purposes, functions based on the mean air quality levels may be preferable to functions based on the median air quality levels because changes in the mean more accurately reflect the changes in peak values targeted by many policies than do changes in the median.

We pooled the results of two chronic bronchitis studies by Schwartz (1993) and Abbey et al. (1995) to obtain the primary estimate of avoided incidences of chronic bronchitis. Based on the Abbey et al. study, we estimate the number of new chronic bronchitis cases that will "reverse over time and subtract these reversals from the estimate of avoided chronic bronchitis incidences. Reversals refer to those cases of chronic bronchitis that were reported at the start of the Abbey et al. survey, but were subsequently not reported at the end of the survey. Since we assume that chronic bronchitis is a permanent condition, we subtract these reversals. Given the relatively high value assigned to chronic bronchitis, this ensures that we do not overstate the economic value of this health effect.

Most emergency room (ER) visits do not result in an admission to the hospital. Therefore we estimate both hospital admissions and ER visits and treat them separately, taking account of the fraction of patients admitted to inpatient care (Lipfert, 1993; Smith, 1997) To avoid double-counting, the baseline incidence rate for ER visits is adjusted by subtracting the percentage of patients that are admitted into the hospital. The reported incidence rates suggest that ER visits for asthma occur 2.7 times as frequently as hospital admissions for asthma (Smith, 1997) To

avoid double-counting, however, only 63 percent of the resulting change in asthma ER visits associated with a given change in pollutant concentrations is counted in the ER visit incidence change.

The minor restricted activity days (MRAD) outcome is estimated using a C-R function derived from Ostro and Rothschild (1989). MRADs are characterized by many of the same symptoms as those which define an asthma attack. The study population in Ostro and Rothschild did not exclude asthmatics, so we reduce the estimated number of avoided MRAD incidences by the estimated number of avoided asthma attacks to prevent double-counting of asthma attacks. This simple subtraction may result in an underestimate of non-asthma attack related MRADs, since asthma attacks are estimated for asthmatics of all ages and MRADs are estimated only for ages 18 to 65.

Based on a review of the recent literature on health effects of PM exposure (Daniels et al., 2000; Pope, 2000; Rossi et al., 1999; Schwartz, 2000), we chose for the purposes of this analysis to assume that PM-related health effects occur down to natural background (i.e. there is no health effects threshold). We assume that all of the C-R functions are continuous and differentiable down to background levels. However, we explore this important assumption in a sensitivity analysis described in a later section.

Economic Values for Health Outcomes

Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health affects by a fairly small amount for a large population. The appropriate economic measure is therefore willingness-to-pay for changes in risk prior to the regulation (Freeman, 1993). For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect as a primary estimate. These costs of illness (COI) estimates generally understate the true value of reductions in risk of a health effect, reflecting the direct expenditures related to treatment but not the value of avoided pain and suffering from the health effect (Harrrington and Portnoy, 1987; Berger, 1987).

It is currently unknown whether there is a delay between changes in PM exposures and changes in mortality rates. The existence of such a time lag is important for the valuation of premature mortality incidences as economic theory suggests benefits occurring in the future should be discounted relative to benefits occurring today. Although there is no specific scientific evidence of a PM effects lag, current scientific literature on adverse health effects associated with smoking and the difference in the effect size between chronic exposure studies and daily mortality studies suggest that all incidences of premature mortality reduction associated with a given incremental change in PM exposure would not occur in the same year as the exposure reduction. This literature implies that lags of a few years are plausible. For our primary estimate, we have assumed a five-year distributed lag structure, with 25 percent of premature deaths occurring in the first year, another 25 percent in the second year, and 16.7 percent in each of the remaining three years. To account for the preferences of individuals for current risk reductions

relative to future risk reductions, we discount the value of avoided premature mortalities occurring beyond 2030 using a three percent discount rate.

Our analysis accounts for expected growth in real income over time. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real incomes increase. The economics literature suggests that the severity of a health effect is a primary determinant of the strength of the relationship between changes in real income and WTP (Alberini, 1997; Miller, 2000; Viscusi, 1993). As such, we use different factors to adjust the WTP for minor health effects, severe and chronic health effects, and premature mortality. Adjustment factors used to account for projected growth in real income from 1990 to 2030 are 1.10 for minor health effects, 1.34 for severe and chronic health effects, and 1.30 for premature mortality. Details of the calculation of the income adjustment factors are provided in the full report U.S. Environmental Protection Agency, 2000a).

Treatment of Uncertainty

In any complex analysis, there are likely to be many sources of uncertainty. This analysis is no exception. Many inputs are used to derive the final estimate of economic benefits, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological estimates of C-R functions, estimates of values, population estimates, income estimates, and estimates of the future state of the world (i.e., regulations, technology, and human behavior). Some of the key uncertainties in the benefits analysis are presented in Table 2. For some parameters or inputs it may be possible to provide a statistical representation of the underlying uncertainty distribution. For other parameters or inputs, the necessary information

necessary is not available.

In addition to uncertainty, the annual benefit estimates presented in this analysis are also inherently variable due to the truly random processes that govern pollutant emissions and ambient air quality in a given year. Factors such as vehicle miles traveled and weather display constant variability regardless of our ability to accurately measure them. As such, the estimates of annual benefits should be viewed as representative of the magnitude of benefits expected, rather than the actual benefits that would occur every year.

We present a primary estimate of the benefits, based on the best available scientific literature and methods, and then provide alternative calculations to illustrate the effects of uncertainty about key analytical assumptions. We do not attempt to assign probabilities to these alternative calculations, as we believe this would only compound the uncertainty of the analysis or present a false picture about the precision of the results. Instead, the reader may examine the impact of applying different assumptions on the estimate of total benefits. While it is possible to combine all of the alternative calculations with a positive impact on benefits to form a "high estimate or all of the alternative calculations with a negative impact on benefits to form a "low estimate, this would not be appropriate because the probability of all of these alternative assumptions occurring simultaneously is likely to be very low. Instead, the alternative calculations are intended to demonstrate the sensitivity of our benefits results to key parameters which may be uncertain.

Some recent benefit-cost analyses in Canada and Europe have estimated ranges of benefits by assigning *ad hoc* probabilities to ranges of parameter values for different endpoints (Holland, Forster, and Wenborn. 1999; Lang et al., 1995). Although this does generate a

quantitative estimate of an uncertainty range, the estimated points on these distributions are themselves highly uncertain and very sensitive to the subjective judgements of the analyst. To avoid these subjective judgements, we choose to allow the reader to determine the weights they would assign to alternative estimates.

MODEL RESULTS

Full implementation of the Heavy-duty Engine and Diesel Fuel regulations in 2030 is projected to reduce heavy-duty vehicle emissions of NOx by 2.6 million tons (90 percent), SO_2 by 140,000 tons (95 percent), and direct PM by 110,000 tons (75 percent). Based on these projected emission changes, REMSAD modeling results indicate the pollution controls generate greater absolute air quality improvements in more populated, urban areas. The rule will reduce average annual mean concentrations of $PM_{2.5}$ across the U.S. by roughly 3.1 percent, or 0.27 $\mu g/m^3$. The population-weighted average annual mean concentration is projected to decline by 4.4 percent, or 0.65 $\mu g/m^3$, which is much larger in absolute terms than the spatial average.

Table 3 presents information on the distribution of modeled reductions in ambient PM concentrations across populations in the U.S. Significant populations live in areas with meaningful reductions in annual mean $PM_{2.5}$ concentrations resulting from the pollution controls. As shown, slightly over 60 percent will live in areas with reductions of greater than 0.5 $\mu g/m^3$. This information indicates how widespread the improvements in PM air quality are expected to be.

Applying the C-R functions described in Table 1 to the estimated changes in $PM_{2.5}$ and PM_{10} yields estimates of the number of avoided incidences for each health outcome. These

estimates are presented in Table 4. We also provide quantified estimates of the 90 percent confidence intervals around these estimates based solely on the standard errors of the C-R coefficients. These intervals do not account for any uncertainties in the change in air quality, population projections, baseline incidence rates, or model uncertainties. Estimates in Table 1 are for the year 2030. Similar health benefits are expected in the years following 2030, although they will be altered somewhat by changes in population and vehicle use.

To provide estimates of the monetized benefits of the reductions in PM-related health outcomes described in Table 4, we multiply the point estimates of avoided incidences by unit values. The estimated value per incidence for each health outcome and for total PM-related health benefits are presented in Table 5. We also provide estimates of the 90 percent confidence intervals around these estimates using Monte Carlo techniques to combine the distributions of the health effect estimates and the valuation estimates. Again, these intervals do not account for any uncertainties in other factors. We do not calculate a confidence interval for the total economic value of all health outcomes, as this would imply a precision which is not warranted based on the gaps in information about impact of unquantified sources of uncertainty.

The largest monetized health benefit is associated with reductions in the risk of premature mortality, which accounts for over $60 billion, or over 90 percent of total monetized health benefits. The next is for chronic bronchitis reductions, although this value is more than an order of magnitude lower than for premature mortality. Minor restricted activity days and work loss days account for the majority of the remaining benefits. While the other categories account for less than $100 million each, they represent a large number of avoided incidences affecting many individuals.

SENSITIVITY ANALYSES

We explored a number of analytical judgements using sensitivity analyses. We report the results of a subset of those analyses here, but the full set of analyses are available in the full report (U.S. Environmental Protection Agency, 2000a).

Arguably, reduction in the risk of premature mortality is the most important PM-related health outcome in terms of public health significance and contribution to dollar benefits. There are four important analytical assumptions that may significantly impact the estimates of the number of avoided premature mortalities and their value. These include selection of the C-R function, structure of the lag between reduced exposure and reduced mortality risk, value of a statistical life (and the influence of age at death), and effect thresholds. The first three of these sensitivity analyses are presented in Table 6. The impact of assuming alternative effects thresholds is explored in Table 7.

Choice of C-R function can have a large impact on benefits, potentially doubling the effect estimate if the C-R function is derived from the HEI reanalysis of the Harvard Six-cities data (Krewski et al., 2000). Due to discounting of delayed benefits, the lag structure may also have a large impact on monetized benefits, reducing benefits by 30 percent if an extreme assumption that no effects occur until after 15 years is applied. If no lag is assumed, benefits are increased by around five percent. The threshold analysis indicates that approximately 90 percent of the premature mortality related benefits are due to changes in $PM_{2.5}$ concentrations occurring above 10 $\mu g/m^3$, and around 80 percent are due to changes above 12 $\mu g/m^3$, the lowest observed mean level in the ACS study. Over 60 percent of avoided incidences are due to changes occurring above 15 $\mu g/m^3$. This suggests that while the possible existence of thresholds is still

important, there would have to be a relatively high threshold to substantially affect the health benefits of the regulation. One important assumption that we adopted for the threshold sensitivity analysis is that no adjustments are made to the shape of the C-R function above the assumed threshold. Instead, thresholds were applied by simply assuming that any changes in ambient concentrations below the assumed threshold have no impacts on the incidence of premature mortality. If there were actually a threshold, then the shape of the C-R function would likely change and there would be no health benefits to reductions in PM below the threshold.

The economics literature concerning the appropriate method for valuing reductions in premature mortality risk is still developing. The U.S. EPA currently uses the value of statistical life (VSL) approach in calculating the primary estimate of mortality benefits, because we believe this calculation to provide the most reasonable single estimate of an individual's willingness to trade off money for reductions in mortality risk. The current VSL approach assigns the same value for risk reductions to all individuals, regardless of age. However, there is general agreement that the value to an individual of a reduction in mortality risk may be affected by the age of the individual, as well as other factors (Cropper and Sussman, 1990; Moore and Viscusi, 1988; Shepard and Zeckhauser, 1982). Age may be especially important in valuing air-pollution related mortality given the advanced age of many of those affected.

Several studies conducted by Jones-Lee, et al. found a significant effect of age on the value of mortality risk reductions expressed by citizens in the United Kingdom (Jones-Lee et al., 1985; Jones-Lee, 1989, Jones-Lee, 1993). We apply ratios based on two of the Jones-Lee, et al. studies (Jones-Lee, 1989 and Jones-Lee, 1993) to the estimated premature mortalities within the appropriate age groups to provide an alternative age-adjusted estimate of the value of avoided

premature mortalities. Depending on the age-WTP structure assumed, use of age-specific WTP to value avoided premature mortalities can reduce benefits by over 40 percent. One problem with both of the Jones-Lee studies is that they examine VSL for a limited age range. They then fit VSL as a function of age and extrapolate outside the range of the data to obtain ratios for the very old. Unfortunately, because VSL is specified as quadratic in age, extrapolation beyond the range of the data can lead to a very severe decline in VSL at ages beyond 75. Since many (around 40 percent) of the avoided premature mortalities from PM are estimated to occur in the 75 and older population, this leads to a potential downward bias in the mortality benefits using the age-specific VSL approach.

CONCLUSIONS

This analysis has estimated the health benefits of regulations on vehicles that lead to reductions in ambient concentrations of particulate matter. The increasing need to understand the public health impacts of air pollution regulations requires the merging of models and data from many disciplines. While necessary, this type of multi-disciplinary methodology is challenging in complexity and scope. Our approach illustrates the integration of models and data and highlights uncertainties inherent in the end results. The result suggests there will be significant health benefits arising from the regulation of emissions from heavy-duty vehicles in the U.S. This supports the findings of Kunzli et al. (2000) that traffic-related air pollution is a significant contributor to total air pollution related mortality and morbidity. While their analysis focused on total attributable risk, we examined the impact of specific pollution controls on reductions in risk. Our estimate that 8,300 premature mortalities would be avoided in 2030, when emission

reductions from the regulation are fully realized, provides additional evidence of the important role that traffic-related pollution plays in the public health impacts of air pollution.

We provide sensitivity analyses to examine key modeling assumptions. In addition, there are other uncertainties that we could not quantify, such as the importance of unquantified effects and uncertainties in the modeling of ambient air quality. Inherent in any analysis of future regulatory programs are uncertainties in projecting atmospheric conditions, source-level emissions, and vehicle miles traveled, as well as population, health baselines, incomes, technology, and other factors. The assumptions used to capture these elements are reasonable based on the available evidence. However, these data limitations prevent an overall quantitative estimate of the uncertainty associated with estimates of total economic benefits. If one is mindful of these limitations, the magnitude of the benefit estimates presented here can be useful information in expanding the understanding of the public health impacts of reducing traffic-related air pollution.

The U.S. EPA will continue to evaluate new methods and models and select those most appropriate for the estimation the health benefits of reductions in air pollution. It is important to continue improving benefits transfer methods in terms of transferring economic values and transferring estimated C-R functions. Epidemiological studies should be designed with the knowledge that they may be applied to different locations, populations, or time periods. The development of both better models of current health outcomes and new models for additional health effects such as asthma and high blood pressure will be essential to future improvements in the accuracy and reliability of benefits analyses (Guo et al., 1999; Ibald-Mulli et al., 2001). Enhanced collaboration between air quality modelers, epidemiologists, and economists should

result in a more tightly integrated analytical framework for measuring health benefits of air pollution policies.

References:

Abbey, D.E., F. Petersen, P. K. Mills, and W. L. Beeson. 1993. Long-Term Ambient Concentrations of Total Suspended Particulates, Ozone, and Sulfur Dioxide and Respiratory Symptoms in a Nonsmoking Population. *Archives of Environmental Health* 48: 33-46.

Abbey, D.E., B.L. Hwang, R.J. Burchette, T. Vancuren, and P.K. Mills. 1995. Estimated Long-Term Ambient Concentrations of PM(10) and Development of Respiratory Symptoms in a Nonsmoking Population. *Archives of Environmental Health* 50(2): 139-152.

Adams, P.F. and M.A. Marano. 1995. Current Estimates from the National Health Interview Survey, 1994. National Center for Health Statistics. Hyattsville, MD. *Vital Health Statistics*, Series 10, No. 193. December.

Alberini, A., M. Cropper, T.Fu, A. Krupnick, J. Liu, D. Shaw, and W. Harrington. 1997. Valuing Health Effects of Air Pollution in Developing Countries: The Case of Taiwan. *Journal of Environmental Economics and Management*. 34: 107-126.

Berger, M.C., G.C. Blomquist, D. Kenkel, and G.S. Tolley. 1987. Valuing Changes in Health Risks: A Comparison of Alternative Measures. *The Southern Economic Journal* 53: 977-984.

Cropper, M. L. and F. G. Sussman. 1990. Valuing Future Risks to Life. *Journal of Environmental Economics and Management* 19: 160-174.

Daniels, M., F. Dominici, J.M. Samet, and S.L. Zeger. 2000. Estimating PM10-Mortality Dose-Response Curves and Threshold Levels: An Analysis of Daily Time-Series for the 20 Largest U.S. Cities. *American Journal of Epidemiology*, 152: 397-406.

Dockery, D.W., J. Cunningham, A.I. Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne and F.E. Speizer. 1996. Health Effects of Acid Aerosols On North American Children-Respiratory Symptoms. *Environmental Health Perspectives*. 104(5): 500-505.

Elixhauser, A., R.M. Andrews, and S. Fox. 1993. Clinical Classifications for Health Policy Research: Discharge Statistics by Principal Diagnosis and Procedure. Agency for Health Care Policy and Research (AHCPR), Center for General Health Services Intramural Research, US Department of Health and Human Services.

Freeman, A. M. III. 1993. *The Measurement of Environmental and Resource Values: Theory and Methods*. Resources for the Future, Washington, D.C.

Graves, E.J. and B.S. Gillum. 1997. Detailed Diagnoses and Procedures, National Hospital Discharge Survey, 1994. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 13, No. 127. March.

Guo, Y.L., Y.C. Lin, F.C. Sung, S.L. Huang, Y.C. Ko, J.S. Lai, H.J. Su, C.K. Shaw, R.S. Lin, D.W. Dockery. 1999. Climate, Traffic-Related Air Pollutants, and Asthma Prevalence in Middle-School Children in Taiwan. *Environmental Health Perspectives* 107: 1001-1006.

Harrington, W. and P. R. Portney. 1987. Valuing the Benefits of Health and Safety Regulation. *Journal of Urban Economics* 22:101-112.

Holland, M., D. Forster, and M. Wenborn. 1999. Economic Evaluation of Proposals Under the UNECE Multi-effects and Multi-pollutant Protocol. Prepared for: European Commission, DGXI, Brussels and Luxembourg. January. Report no. AEAT-4587.

Ibald-Mulli, A., J. Stieber, H.-E. Wichmann, W. Koenig, and A. Peters. 2001. Effects of Air Pollution on Blood Pressure: A Population-Based Approach. *American Journal of Public Health*. 91: 571-577.

Industrial Economics Incorporated (IEc). 1992. Review of Existing Value of Life Estimates: Valuation Document. Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. November 6.

Jones-Lee, M.W., M. Hammerton and P.R. Philips. 1985. The Value of Safety: Result of a National Sample Survey. *Economic Journal*. 95(March): 49-72.

Jones-Lee, M.W. 1989. *The Economics of Safety and Physical Risk*. Oxford: Basil Blackwell.

Jones-Lee, M.W., G. Loomes, D. O'Reilly, and P.R. Phillips. 1993. The Value of Preventing Non-fatal Road Injuries: Findings of a Willingness-to-pay National Sample Survey. TRY Working Paper, WP SRC2.

Krewski D, Burnett RT, Goldbert MS, Hoover K, Siemiatycki J, Jerrett M, Abrahamowicz M, White WH. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Special Report to the Health Effects Institute, Cambridge MA, July 2000.

Krupnick, A.J. and M.L. Cropper. 1992. "The Effect of Information on Health Risk Valuations. *Journal of Risk and Uncertainty* 5(2): 29-48.

Krupnick, A.J. 1988. An Analysis of Selected Health Benefits from Reductions in Photochemical Oxidants in the Northeastern United States: Final Report. Prepared for U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Washington, DC. EPA Contract No. 68-02-4323. September.

Kunzli N, Medina S, Kaiser R, Quenel P, Horak F Jr, Studnicka M 2001. Assessment of deaths attributable to air pollution: should we use risk estimates based on time series or on cohort studies? American Journal of Epidemiology. 153(11):1050-5.

Kunzli, N., R. Kaiser, S. Medina, M. Studnicka, O. Chanel, P. Filliger, M. Herry, F. Horak Jr., V. Puybonnieux-Texier, P. Quenel, J. Schneider, R. Seethaler, J-C Vergnaud, and H. Sommer. 2000. Public-health Impact of Outdoor and Traffic-related Air Pollution: A European Assessment. *The Lancet*, 356: 795-801.

Lang, C., G. Yarwood, F. Lalonde, and R. Bloxam. 1995. *Environmental and Health Benefits of Cleaner Vehicles and Fuels.* Prepared for: Canadian Council of Ministers of the Environment Task Force on Cleaner Vehicles and Fuels, Winnipeg, Manitoba. October.

Lipfert, F.W. 1993. A Critical Review of Studies of the Association Between Demands for Hospital Services and Air Pollution. *Environmental Health Perspectives*. 101 (Suppl 2): 229-268.

Miller, T.R. 2000. Variations between Countries in Values of Statistical Life. *Journal of Transport Economics and Policy*. 34: 169-188.

Moore, M. J. and W. K. Viscusi. 1988. The Quantity-Adjusted Value of Life. *Economic Inquiry* 26(3): 369-388.

Neumann, J.E., M.T. Dickie, and R.E. Unsworth. 1994. Linkage Between Health Effects Estimation and Morbidity Valuation in the Section 812 Analysis -- Draft Valuation Document. Industrial Economics Incorporated (IEc) Memorandum to Jim DeMocker, U.S. Environmental

Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. March 31.

Ostro B.D. and S. Rothschild. 1989. Air Pollution and Acute Respiratory Morbidity: An Observational Study of Multiple Pollutants. *Environmental Research* 50:238-247.

Ostro, B.D. 1987. Air Pollution and Morbidity Revisited: a Specification Test. *Journal of Environmental Economics and Management.* 14: 87-98.

Pope, C.A., III, D.W. Dockery, J.D. Spengler, and M.E. Raizenne. 1991. Respiratory Health and PM_{10} Pollution: a Daily Time Series Analysis *American Review of Respiratory Diseases* 144: 668-674.

Pope, C.A. 2000. Invited Commentary: Particulate Matter-Mortality Expsoure-Response Relations and Thresholds. *American Journal of Epidemiology*, 152: 407-412.

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

Rossi, G., M.A. Vigotti, A. Zanobetti, F. Repetto, V. Gianelle and J. Schwartz. 1999. Air

pollution and cause-specific mortality in Milan, Italy, 1980-1989. *Archives of Environmental Health*. 54(3): 158-64.

Samet JM, Zeger SL, Dominici F, Curriero F, Coursac I, Dockery DW, Schwartz J, Zanobetti A. 2000. The National Morbidity, Mortality and Air Pollution Study: Part II: Morbidity, Mortality and Air Pollution in the United States. Research Report No. 94, Part II. Health Effects Institute, Cambridge MA, June 2000.

Schwartz, J. 1993. Particulate Air Pollution and Chronic Respiratory Disease. *Environmental Research* 62: 7-13.

Schwartz, J. 2000. Assessing confounding, effect modification, and thresholds in the association between ambient particles and daily deaths. *Environmental Health Perspectives* 108(6): 563-8.

Schwartz, J., Dockery, D.W., Neas, L.M., Wypij, D., Ware, J.H., Spengler, J.D., Koutrakis, P., Speizer, F.E., and Ferris, Jr., B.G. 1994. Acute Effects of Summer Air Pollution on Respiratory Symptom Reporting in Children *American Journal of Respiratory Critical Care* Medicine 150: 1234-1242.

Schwartz, J., D. Slater, T.V. Larson, W.E. Pierson and J.Q. Koenig. 1993. Particulate air pollution and hospital emergency room visits for asthma in Seattle. Am Rev Respir Dis. Vol.

147: 826-31.

Seigneur, C., G. Hidy, I. Tombach, J. Vimont, and P. Amar. 1999. Scientific Peer Review of the Regulatory Modeling System for Aerosols and Deposition (REMSAD). Prepared for the KEVRIC Company, Inc.

Shepard, D. S. and R. J. Zeckhauser. 1982. Life-Cycle Consumption and Willingness to Pay for Increased Survival. In M.W. Jones-Lee (Editor), *The Value of Life and Safety*. Amsterdam, The Netherlands: North-Holland.

Sheppard, L., D. Levy, G. Norris, T.V. Larson and J.Q. Koenig. 1999. Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. *Epidemiology*. Vol. 10: 23-30.

Smith, D.H., D.C. Malone, K.A. Lawson, L. J. Okamoto, C. Battista, and W.B. Saunders, 1997. A National Estimate of the Economic Costs of Asthma. *American Journal of Respiratory Critical Care Medicine* 156: 787-793.

Tolley, G.S. et al. 1986. Valuation of Reductions in Human Health Symptoms and Risks. University of Chicago. Final Report for the US Environmental Protection Agency. January.

U.S. Environmental Protection Agency, 1996. Review of the National Ambient Air Quality

Standards for Particulate Matter: Assessment of Scientific and Technical Information. Office of Air Quality Planning and Standards, Research Triangle Park, N.C.; U.S. EPA report no. EPA/4521R-96-013.

U.S. Bureau of the Census. 1992. Statistical Abstract of the United States: 1992. 112 ed. Washington, DC.

U.S. Environmental Protection Agency, 2000b. *Procedures for Developing Base Year and Future Year Mass and Modeling Inventories for The Heavy-duty Engine and Vehicle Standards and Highway Diesel Fuel (HDD) Rulemaking*. Office of Air and Radiation, Washington, DC, October 2000; U.S. EPA report no. EPA420-R-00-020. Available at http://www.epa.gov/otaq/hdmodels.htm Accessed April 16, 2001.

U.S. Environmental Protection Agency, 1999. *The Benefits and Costs of the Clean Air Act, 1990-2010.* Prepared for U.S. Congress by U.S. EPA, Office of Air and Radiation/Office of Policy Analysis and Review, Washington, DC, November; U.S. EPA report no. EPA-410-R-99-001.

U.S. Environmental Protection Agency, 2000a. *Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements*. Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/otaq/diesel.htm. Accessed April 16, 2001.

Abt Associates. 2000. *Final Heavy-Duty Engine/Diesel Fuel Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods and Benefit Analysis Results*. Prepared for the Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, RTP, NC. Available at http://www.epa.gov/ttn/ecas/regdata/tsdhddv8.pdf. Accessed April 16, 2001.

U.S. Department of Commerce, Bureau of Economic Analysis. BEA Regional Projections to 2045: Vol. 1, States. Washington, DC U.S. Govt. Printing Office, July 1995.

U.S. Environmental Protection Agency, 2000c. *Technical Support Document for the Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements: Air Quality Modeling Analyses*. Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/otaq/diesel.htm. Accessed April 16, 2001.

U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics. 1999. National Vital Statistics Reports, 47(19).

Viscusi, W.K., W.A. Magat, and J. Huber. 1991. Pricing Environmental Health Risks: Survey Assessments of Risk-Risk and Risk-Dollar Trade-Offs for Chronic Bronchitis. *Journal of Environmental Economics and Management*, 21: 32-51.

Viscusi, W.K. 1992. *Fatal Tradeoffs: Public and Private Responsibilities for Risk.* (New York:

Oxford University Press).

Viscusi, W.K. and W. Evans. 1993. Income Effects and the Value of Health. *Journal of Human Resources*. 28: 497-518.

Whittemore, A.S. and E.L. Korn. 1980. Asthma and Air Pollution in the Los Angeles Area. *American Journal of Public Health*. 70: 687-696.

.

Table 1. PM-related Health Outcomes and Studies Included in the Primary Analysis

Health Outcome	Pollutant	Applied Population	Source of Effect Estimate	Source of Baseline Incidence
Premature Mortality				
All-cause premature mortality from long-term exposure	$PM_{2.5}$	> 29 years	Krewski et al., 2000	U.S. Centers for Disease Control, 1999
Chronic Illness				
Chronic Bronchitis (pooled estimate)	$PM_{2.5}$ PM_{10}	> 26 years > 29 years	Abbey et al., 1995 Schwartz et al., 1993	Abbey et al., 1993 Abbey et al., 1993 Adams and Marano, 1995
Hospital Admissions				
COPD	PM_{10}	> 64 years	Samet et al., 2000	Graves and Gillum, 1997
Pneumonia	PM_{10}	> 64 years	Samet et al., 2000	Graves and Gillum, 1997
Asthma	$PM_{2.5}$	< 65 years	Sheppard et al., 1999	Graves and Gillum, 1997
Total Cardiovascular	PM_{10}	> 64 years	Samet et al., 2000	Graves and Gillum, 1997
Asthma-Related ER Visits	PM_{10}	All ages	Schwartz et al., 1993	Smith et al., 1997 Graves and Gillum, 1997
Other Effects				
Asthma Attacks	PM_{10}	Asthmatics, all ages	Whittemore and Korn, 1980	Krupnick, 1988 Adams and Marano, 1995
Acute Bronchitis	$PM_{2.5}$	Children, 8-12 years	Dockery et al., 1996	Adams and Marano, 1995
Upper Respiratory Symptoms	PM_{10}	Asthmatic children, 9-11	Pope et al., 1991	Pope et al., 1991
Lower Respiratory Symptoms	$PM_{2.5}$	Children, 7-14 years	Schwartz et al., 1994	Schwartz et al., 1994
Work Loss Days	$PM_{2.5}$	Adults, 18-65 years	Ostro, 1987	Adams and Marano, 1995
Minor Restricted Activity Days (minus asthma attacks)	$PM_{2.5}$	Adults, 18-65 years	Ostro and Rothschild., 1989	Ostro and Rothschild, 1989

Table 2. Sources of Uncertainty in the PM Health Benefit Analysis

1. Uncertainties Associated with Concentration-Response Functions
- The value of the PM-coefficient in each C-R function. - Application of a single C-R function to pollutant changes and populations in all locations. - Similarity of future year C-R relationships to current C-R relationships. - Correct functional form of each C-R relationship. - Extrapolation of C-R relationships beyond the range of PM concentrations observed in the study. - Application of C-R relationships only to those subpopulations matching the original study population.
2. Uncertainties Associated with PM Concentrations
- Responsiveness of the models to changes in precursor emissions resulting from the control policy. - Projections of future levels of precursor and direct emissions, especially ammonia and crustal materials. - Model chemistry for the formation of ambient nitrate concentrations. - Comparison of model predictions of particulate nitrate with observed rural monitored nitrate levels indicates that REMSAD overpredicts nitrate in some parts of the Eastern US and underpredicts nitrate in parts of the Western US.
3. Uncertainties Associated with PM Mortality Risk
- No demonstrated direct biological mechanism for observed epidemiological evidence. - Direct causal agents within the complex mixture of PM have not been identified. - The extent to which adverse health effects are associated with low level exposures that occur many times in the year versus peak exposures. - The extent to which effects reported in the long-term exposure studies are associated with historically higher levels of PM rather than the levels occurring during the period of study. - Reliability of the limited ambient $PM_{2.5}$ monitoring data in reflecting actual $PM_{2.5}$ exposures.
4. Uncertainties Associated with Possible Lagged Effects
- The portion of the long-term exposure mortality effects associated with changes in annual PM levels that would occur in a single year is uncertain as is the portion that might occur in subsequent years.
5. Uncertainties Associated with Baseline Incidence Rates
- Some baseline incidence rates are not location-specific (e.g., those taken from studies) and may therefore not accurately represent the actual location-specific rates. - Current baseline incidence rates may not approximate well baseline incidence rates in 2030. - Projected population and demographics may not represent well future-year population and demographics.
6. Uncertainties Associated with Economic Valuation
- Unit dollar values associated with health and welfare endpoints are only estimates of mean WTP and therefore have uncertainty surrounding them. - Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates due to differences in income or other factors. - Growth of income and the relationship between income and WTP are both uncertain. As such, the income adjustment factors used to adjust WTP into the future are also uncertain.
7. Uncertainties Associated with Aggregation of Monetized Benefits
- Health and welfare benefits estimates are limited to the available C-R functions. Thus, unquantified or unmonetized benefits are not included.

Table 3. Distribution of $PM_{2.5}$ Air Quality Improvements Across the 2030 U.S. Population Due to U.S. EPA Heavy-duty Engine and Diesel Fuel Standards

Reduction in Annual Mean $PM_{2.5}$ Concentrations ($\mu g/m^3$)[a]	*2030 Population*[b]		
	Number (millions)	*Percent (%)*	*Cumulative Percent*
$0.00 \leq \Delta\ PM_{2.5}\ Conc \leq 0.25$	43.0	12.4%	100.0%
$0.25 < \Delta\ PM_{2.5}\ Conc \leq 0.5$	95.0	27.5%	87.5%
$0.50 < \Delta\ PM_{2.5}\ Conc \leq 0.75$	94.9	27.4%	60.0%
$0.75 < \Delta\ PM_{2.5}\ Conc \leq 1.0$	60.5	17.5%	32.6%
$1.00 < \Delta\ PM_{2.5}\ Conc \leq 1.25$	23.4	6.8%	15.1%
$1.25 < \Delta\ PM_{2.5}\ Conc \leq 1.5$	20.9	6.0%	8.3%
$1.50 < \Delta\ PM_{2.5}\ Conc \leq 1.75$	2.9	0.8%	2.3%
$1.75 < \Delta\ PM_{2.5}\ Conc$	5.2	1.5%	1.5%

[a] The change is defined as the base case value minus the control case value.
[b] Based on projected total U.S. population of 345.8 million in 2030.

Table 4. Estimated Impact of Heavy-duty Engine and Diesel Fuel Standards on Selected PM-related Health Outcomes in 2030

Health Outcome	Annual Avoided Incidences per Million Population[a] for a 1 μg/m^3 Decrease in PM	Avoided Incidence (cases/year)[b]		
		5th %ile	Mean	95th %ile
Premature Mortality				
All-cause premature mortality from long-term exposure	37	4,829	8,292	11,698
Chronic Illness				
Chronic Bronchitis (pooled estimate)	24	1,884	5,478	9,464
Hospital Admissions				
COPD	4	164	900	1,607
Pneumonia	5	610	1,106	1,601
Asthma	4	385	881	1,402
Total Cardiovascular	12	2,252	2,667	3,067
Asthma-Related ER Visits	9	864	2064	3213
Other Effects				
Asthma Attacks	774	60,984	175,931	291,914
Acute Bronchitis	80	-88	17,590	35,900
Upper Respiratory Symptoms	880	65,290	193,402	325,371
Lower Respiratory Symptoms	888	88,308	192,899	295,784
Work Loss Days	6,803	1,337,267	1,539,396	1,733,280
Minor Restricted Activity Days (minus asthma attacks)	10,767	6,806,718	7,990,406	9,104,836

[a] Based on national average incidence rates. Avoided incidences are per million base population. Rates for susceptible populations would thus be higher. For example, the rate of asthma attacks is around 13,800 per million asthmatics per microgram of PM.

[b] Based on projected total U.S. population of 345.8 million in 2030. Note that the 5th and 95th percentile interval is based solely on the standard error of the C-R function. The interval does not incorporate uncertainty about the change in air quality, population projections, or baseline incidence rates, all of which may contain significant uncertainty.

Table 5. Economic Value of Avoided PM-related Health Effects in 2030

PM-related Health Outcome	Estimated Value Per Incidence (1999$)	Total Value[b] (million 1999$)	Source of Value Estimate
Premature Mortality[a]	$8 million per statistical life	$62,580 ($8,450-$154,710)	Viscusi et al., 1992, IEc., 1993
Chronic Bronchitis[a] (CB)	$444,000	$2,430 ($230-$7,960)	Viscusi et al., 1991, Krupnick and Cropper, 1992, U.S. EPA, 1999
Hospital Admissions			
Chronic Obstructive Pulmonary Disease (COPD)	$12,378	$10 ($0-$20)	Elixhauser, 1993
Pneumonia	$14,693	$20 ($10-$20)	Elixhauser, 1993
Asthma admissions	$6,634	$10 ($0-$10)	Elixhauser, 1993
All Cardiovascular	$18,387	$50 ($40-$60)	Elixhauser, 1993
Emergency room visits for asthma[c]	$299	$0 ($0-$0)	Smith et al., 1997
Respiratory Ailments Not Requiring Hospitalization			
Upper Respiratory Symptoms[a] (URS)	$26	$10 ($0-$10)	Neumann et al., 1994
Lower Respiratory Symptoms[a,c] (LRS)	$16	$0 ($0-$10)	Neumann et al., 1994
Acute Bronchitis[a,c]	$62	$0 ($0-$0)	Neumann et al., 1994
Restricted Activity and Work Loss Days			
Work Loss Days (WLDs)	Variable	$180 ($160-$200)	Regionally adjusted median weekly wage for 1990 divided by 5 (adjusted to 1999$) US Bureau of the Census, 1992
Minor Restricted Activity Days[a] (MRADs)	$53	$430 ($260-$610)	Tolley et al., 1986
Total Economic Value of Estimated Health Effects		$65,720	

[a] Value adjusted for growth in real income between 1990 and 2030. Details of the income adjustment procedure can be found in the full report [2].

[b] Values rounded to the nearest $10 million. Values in paratheses represent the 90 percent credible interval around the central estimate. Note that this interval is based on the standard error of the C-R function and the assumed distribution of the valuation estimate. The interval does not incorporate uncertainty about the change in air quality, population projections, or baseline incidence rates, all of which may contain significant uncertainty.

[c] Note that the total values for avoided asthma-related ER visits, lower respiratory symptoms and acute bronchitis are estimated to be less than $5

million, so the rounded value is zero.

Table 6. Sensitivity of Estimates to Alternative Assumptions Regarding Quantification and Valuation of Mortality Benefits

Description of Sensitivity Analysis		Avoided Incidences	Impact on Total Benefits Estimate (billion 1999$)
Alternative Concentration-Response Functions for PM-related Premature Mortality			
1	Krewski/ACS Study Regional Adjustment Model[a]	9,400	+$8.3 (+13%)
2	Pope/ACS Study[b]	9,900	+$12.1 (+18%)
3	Krewski/Harvard Six-city Study[c]	24,200	+$119.9 (+182%)
Alternative Lag Structures for PM-related Premature Mortality			
None	Incidences all occur in the first year	8,300	+$3.2 (+5%)
8-year	Incidences all occur in the 8th year	8,300	-$9.1 (-15%)
15-year	Incidences all occur in the 15th year	8,300	-$19.1 (-31%)
Alternative Methods for Valuing Reductions in Incidences of PM-related Premature Mortality			
Value of avoided premature mortality incidences based on age-specific VSL.[d]	Jones-Lee (1989)	8,300	-$28.5 (-41%)
	Jones-Lee (1993)	8,300	-$6.8 (-10%)

[a] This C-R function is included as a reasonable specification to explore the impact of adjustments for broad regional correlations, which have been identified as important factors in correctly specifying the PM mortality C-R function..

[b] The Pope et al. C-R function was used to estimate reductions in premature mortality for several recent benefits analysis. It is included here to provide a comparable estimate for the HD Engine/Diesel Fuel rule.

[c] The Krewski et al. "Harvard Six-cities Study estimate is included because the Harvard Six-cities Study featured improved exposure estimates, a slightly broader study population (adults aged 25 and older), and a follow-up period nearly twice as long as that of Pope, et al. and as such provides a reasonable alternative to the primary estimate.

[d] Jones-Lee (1989) provides an estimate of age-adjusted VSL based on a finding that older people place a much lower value on mortality risk reductions than middle-age people. Jones-Lee (1993) provides an estimate of age-adjusted VSL based on a finding that older people value mortality risk reductions only somewhat less than middle-aged people.

Table 7. Impact of Assumed Thresholds on Estimated Reductions in PM-related Premature Mortality

Assumed Threshold ($\mu g/m^3$)	Avoided Incidences	Percent of No Threshold Estimate
No Threshold	8,300	100.0%
5	8,100	97.6%
10	7,300	88.0%
15	5,100	61.4%
20	2,600	31.3%
25	1,300	15.7%

[a] No adjustments were made to the shape of the C-R function above the assumed threshold. Instead, thresholds were applied by simply assuming that any changes in ambient concentrations below the assumed threshold have no impacts on the incidence of premature mortality.