Evaluating the Health Benefits of Air Pollution Reductions: Recent Developments at the U.S. EPA

Bryan J. Hubbell*

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*U.S. EPA, Office of Air Quality Planning and Standards
Innovative Strategies and Economics Group

Background and Introduction

The analysis of expected health benefits of reductions in air pollution is of great importance both in Europe and the United States. The emergence of strong evidence that air pollution is contributing to significant increases in premature mortality has provided support for a number of recent efforts to reduce air pollution through regulation of both mobile and stationary sources. Dr. Pearce has provided an overview of recent European experiences with health benefits analysis. The purpose of this paper is to describe recent developments in the science and art of health benefits analysis of air pollution regulations at the U.S. Environmental Protection Agency.

EPA is required under Executive Order 12866, signed by President Clinton, to evaluate the costs and benefits of major regulations, defined as those expected to have a cost of at least $100 million dollars. This Executive Order (E.O.) directs federal agencies to "include both quantifiable measures and qualitative measures of costs and benefits that are difficult to quantify, but nevertheless essential to consider." In analyzing pollution regulations dealing with criteria air pollutants, EPA seeks to comply with this E.O. by providing the most comprehensive evaluation of both health and welfare related benefits associated with changes in ambient levels of air pollution resulting from regulation induced changes in emissions of precursor pollutants, such as NOx, SO_2, VOC, and directly emitted particulate matter (PM).

Over the past several years, EPA has conducted a number of analyses of the projected health and environmental benefits of air pollution regulations, including the Tier 2 and Heavy Duty Engine rules aimed at reducing mobile source pollution and the NOx SIP call rule aimed at reducing interstate transport of NOx from stationary sources. As indicated by Dr. Pearce, in addition to regulatory analyses of costs and benefits, EPA has also recently completed its first Prospective Analysis of the Costs and Benefits of the Clean Air Act from 1990 to 2010 (U.S. EPA, 1999). Many of the methods used in the Prospective Analysis are also used in EPA's regulatory analyses, although several advances were incorporated into the analysis of the benefits of the Heavy Duty Engine rule (U.S. EPA, 2000a). During the course of these analyses, EPA has worked consistently to improve the estimation of health benefits, both in terms of quantifying health effects, and in valuing those quantified health effects. EPA has worked with its Science Advisory Board to ensure that our methods represent the best available science[1].

While EPA uses the best tools available, there are inevitably many uncertainties

[1] In recent years, EPA has increased its reliance on guidance provided by its independent Science Advisory Board, both from the Advisory Council on Clean Air Compliance Analysis (the review body for the Section 812 analyses) and the Environmental Economics Advisory Committee (EEAC). The 812 Advisory Council provides guidance on all aspects of benefit analysis for the Section 812 studies, from air quality modeling to epidemiology to economic valuation. The EEAC addresses only economics issues, but provides broader advice to the agency on these issues. These committees are composed of top scientists and economists from a variety of backgrounds. The meetings of these committees are public and comments from the public are accepted at these meetings. The minutes of the meetings and advisories from the committees are available on the Internet (http://www.epa.gov/sab).

embodied in estimates of the health benefits of regulations. In addition, there are many health and environmental effects which we are unable to quantify or value, such as changes in ecosystem function or chronic changes in lung function. Because of the lack of available epidemiological studies on many hazardous air pollutants (HAP) or air toxics, such as benzene or formaldehyde, our quantitative analyses tend to focus on so called criteria pollutants, such as ozone and PM. This is true even for HAP regulations, where HAP pollution controls lead to reductions in both HAPs and criteria pollutant precursors. Many important issues remain unresolved, especially in the area of the quantifying and valuing the effects of reductions in mortality risk from exposure to ambient levels of air pollution. Just as important, however, is the lack of methods to quantify and value the full morbidity effects of reductions in air pollution. The purpose of this paper is to present an overview of EPA's current directions in understanding these issues. The paper is divided into two main sections. The first section deals with quantifying mortality and morbidity impacts. The second deals with valuing those impacts. The paper ends with a discussion of the increased need for communication between epidemiologists and economists.

The Tip of the Iceberg

In his presentation, Dr. Pearce asks the question "Why are health benefits high?" While this is one question that may be relevant, another, equally relevant question is "How high should health benefits be?" In a recent analysis of the benefits of reducing emissions from diesel engines[2] (U.S. EPA, 2000a), we found that by 2030 (when the rule is to be fully implemented) reductions in ambient PM are estimated to annually result in thousands of premature mortalities avoided, thousands cases of chronic bronchitis avoided, thousands of hospital admissions avoided, hundreds of thousands of respiratory symptoms avoided, and millions of lost work days avoided. The reductions in ambient ozone are estimated to result in thousands of hospital admissions and hundreds of thousands of asthma attacks and other respiratory symptoms avoided. These reductions in health effects, along with improvements in visibility and increases in agricultural productivity are valued at more than $70 billion (1999 US$)[3]. When viewed on a per capita basis, this amounts to around $200 per person, or less than half of a percent of 2030 per capita income.

As with the European studies cited by Dr. Pearce, health benefits account for over 90 percent of total monetized benefits. However, many non-health benefits, including ecosystem impacts, urban visibility, nitrogen deposition, and materials damage, could not be monetized with enough reliability to be included in the primary estimate of benefits. In addition, no

[2]For a complete discussion of the HD Engine rule benefits analysis, please see the *Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements* (available on the internet at http://www.epa.gov/otaq/diesel.htm).

[3]Following the advice of the SAB-EEAC, we adjusted the value of health benefits to account for growth in willingness-to-pay due to growth in real income out to 2030. Details of this adjustment procedure are provided in the RIA for the Heavy Duty Engine/Diesel Fuel rule (U.S. EPA, 2000a).

benefits associated with reductions in CO or diesel toxics (including benzene, 1,3-butadiene, formaldehyde, and others) were estimated, due to a lack of appropriate air quality and health effect models.

Health benefits are dominated by the value of PM-related premature mortality benefits. The absolute size of mortality benefits is driven by two factors, the relatively strong concentration-response function, which leads to a large number of premature deaths predicted to be avoided per microgram of ambient $PM_{2.5}$ reduced, and the value of a statistical life, estimated to be about $8 million (1999$, adjusted for income growth to 2030). I will discuss each of these factors in more detail in later sections. The relative size of mortality benefits, i.e. the share of total health benefits accounted for by mortality, is driven by both the large absolute magnitude of mortality benefits and by the relatively low values placed on non-mortality effects. Many of these effects are valued using proxies for willingness-to-pay (WTP), such as cost-of-illness, or approximations to WTP, such as the combination of WTP for symptom clusters to approximate the WTP for an episode of upper respiratory symptoms. And, the full morbidity effects of air pollution reductions may be understated in our analyses. A more complete accounting for the true WTP for morbidity effects may reduce to some extent the wide discrepancy between the value of mortality and the value of morbidity effects.

There are several factors that suggest EPA is providing a low estimate of health benefits, at the very least those health benefits related to reduced morbidity. First, EPA has chosen to use the lowest of the available C-R functions from recent chronic exposure studies[4]. Several other estimates from recent chronic exposure studies suggest that the mortality risks from chronic $PM_{2.5}$ exposure may be much higher than that used in our analyses. Sensitivity analyses suggest that choosing a different C-R function could increase the estimate of reduced premature mortality by 10 to 290 percent[5].

[4]In its recent benefits analyses, EPA has chosen to use only C-R functions based on cohort-type studies, such as the C-R function derived from the American Cancer Society (ACS) Study, as reported in Pope, et al. (1995). A recent extensive reanalysis of the ACS data and data from the Harvard Six Cities Study (Dockery et al., 1993) has been conducted by the Health Effects Institute (HEI). EPA has chosen to use a C-R function from the HEI reanalysis (Krewski et al, 2000) of the ACS study in place of the original C-R function.

[5]Applying the C-R function based on the regional adjustment model from the HEI reanalysis increases the predicted avoided mortality from 8,300 to 9,400. Applying the C-R function from the HEI reanalysis of the Harvard Six-Cities study increases the predicted avoided mortality from 8,300 to 24,200.

Second, our analysis focuses on first-order endpoints, such as mortality and hospital admissions directly related to PM levels. However, several of the endpoints associated with PM, such as hospital admissions for cardiac diseases, are known risk factors for premature mortality, regardless of future exposures to PM. And, admission to a hospital for one of these diseases is often followed by changes in lifestyle including restrictions in activity levels and increased frailty. Therefore the full economic impact of these endpoints may be severely underestimated by only examining the costs of the hospital stay for the PM related episode. In terms of lost lifespan, these second order "ripple" effects may be more important than the first order effects. Carrothers, Graham, and Evans (1999) suggest that a shift from Coronary Heart Disease to Diagnosed Coronary Heart Failure can reduce life expectancy by 4 years for a 70 year old male. They also note that "if air pollution is causing population-wide decreases in lung function, as some evidence suggests, these changes could have substantial impacts on life-expectancy. Even though air pollution would not be the direct cause of death, it would accelerate the process by which people become sick and die from other causes."

Third, for some endpoints, estimated relative risks may incorporate any behavioral responses of individuals that reduce individual exposure to ambient pollution, such as minimizing time spent outdoors. It may be that at high levels of ambient pollution, individuals take averting actions to reduce ambient exposure, flattening the C-R function at high ambient concentrations. Bresnahan, Dickie, and Gerking (1997) find that symptomatic individuals spend less time outdoors and engage in other forms of averting behavior at high ozone concentrations. Thus, if those individuals were to not engage in averting behaviors, estimated C-R functions for ozone-related symptoms would be higher. This also affects the economic valuation because we value only the reduction in measured risk, and not the averting behavior that led to the reduced estimate of risk. Depending on the types of averting behavior, this could lead to a significant underestimation of the benefits of reducing ambient PM levels. It can be argued that in some cases, substitution of public risk reductions for private risk reductions may lead to greater efficiency in achieving desired risk reductions. There may also be longer term behavioral adjustments in response to chronic pollution levels, such as permanently altering exercise patterns or relocation to less polluted areas. These longer term adjustments may have significant impacts on welfare, either by restricting the individual's choice set or by substituting reductions in air pollution risk for other activities.

Fourth, there are few air toxic related endpoints for which adequate C-R functions are available. Thus, any benefits associated with reductions in air toxics are left unquantified, which essentially assigns them a value of zero for the purposes of comparison with other monetized benefits. To fill this gap in the benefits analysis framework, economists need to broaden the ability to value reductions in very uncertain risks. In addition to specific reductions in known health effects, a major component of the regulation may be provision of an insurance policy against risks discovered in the future or an increase in peace of mind from knowing that any potential unknown risks have been eliminated. This would require a significant change from our current damage function based approach, and it is not clear whether current stated preference methods could provide a plausible choice alternative for respondents. It would also make difficult any incremental analysis of individual air toxic regulations, which have tended to be

focused on reducing air toxic emissions from specific source categories.

Epidemiologists and toxicologists also need to work towards measuring health effects in terms economists can value. For example, manganese emissions have been linked to increased involuntary finger tapping and other indicators of impaired motor function (Beuter et al. 1999, Mergler et al. 1999). Clearly, these are not effects for which economists can easily assign a value. However, finger tapping is a bioindicator for increased risk of nerve damage similar to that associated with Parkinson's disease (Pal, Samii, and Calne, 1999), which is normally found in much older individuals and should be able to be valued using traditional valuation methods. The endpoint which is suitable for economic valuation may then be the early onset of chronic nerve damage. By examining the shift in the relationship between prevalence of a disease and age conditional on exposure to an air toxic, it may be possible to determine the increase in costs of a disease due to premature onset. However, this requires that economists understand the linkage between the bioindicator and the ultimate health impact.

Based on the four points outlined above, it seems clear that there is not enough information, at least in terms of physical effects estimation, to state that estimated health benefits are too high. More research into the full impact of air pollution related morbidity is required, both for criteria and toxic air pollutants. It may very well turn out that health benefits of air pollution reductions are in fact higher, or at least more far reaching, then our current estimates suggest.

Valuing Reductions in the Risk of Premature Mortality from Air Pollution

The value of reductions in premature mortality risk is still highly uncertain, especially in applications to mortality risk associated with air pollution. There are several differences between the labor market studies EPA uses to derive a value of statistical life[6] (VSL) estimate and the air pollution context to which that VSL is applied, including age, attitudes towards risk, voluntariness of the risk, and the nature of the death, i.e. immediate vs. after a long illness. These differences may imply both upward and downward adjustments to the VSL. As indicated by Dr. Pearce, age may be particularly important in the air pollution context since many of those most affected by increased mortality risk from air pollution are elderly.

As part of the regulatory impact analysis for the Radon in Drinking Water rule, EPA asked the EEAC of the SAB to review a white paper on valuation of reduced risks of cancer fatalities. In this white paper, EPA laid out several possible adjustments to the $6.1 million standard VSL for factors such as age, voluntariness, dread, etc. The conclusions of the SAB were that while such adjustments may be theoretically appropriate, sufficient reliable information does not exist at this time to make quantitative adjustments to the primary estimate of VSL,

[6]The Value of Statistical Life (VSL) approach is a convenient short-hand method for describing the effect of small changes in the risk of premature death for a large population of potentially exposed people. The VSL is "built" by dividing the *value* of a small risk change by the actual change in risk.

including adjustments for age (EPA-SAB-EEAC-00-013, 2000). The only adjustments suggested by the SAB were for growth in income over time and for discounting over latency periods.

EPA followed this advice in the HD Engine benefits analysis, presenting only the unadjusted VSL in the primary estimate. However, EPA provided a prominent sensitivity analysis examining the effects of using age-specific estimates of VSL obtained by applying the ratios of VSL at different ages to the VSL of a 40 year old. There are several potential sources for these ratios. Estimates from two Jones-Lee studies provide a reasonable low and high end for this type of adjustment. The ratios based on Jones-Lee (1989), as summarized in U.S. EPA (2000b), suggest a steep inverted U shape between age and VSL, with the VSL for a 70 year old at 63 percent of that for a 40 year old, and the VSL for an 85 year old at 7 percent of that for 40 year old. The ratios based on Jones-Lee (1993) and summarized in U.S. EPA (2000b), suggest a much flatter inverted U shape, with the VSL for a 70 year old at 92 percent of that for a 40 year old, and the VSL for an 85 year old at 82 percent of that for a 40 year old. The general U shaped relationship is supported by recent analyses conducted in Canada and the U.S. by Krupnick et al. (2000). Their results suggest a curvature somewhere between the two Jones-Lee estimates.

The wide range of age-adjustment ratios, especially at older ages demonstrates the difficulty in making these kinds of adjustments. 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. Figure 1 shows the estimated ratios of age-specific VSL to the VSL of a 40 year old worker based on the two Jones-Lee articles (extrapolated ratios are indicated by dotted lines). Figure 1 also shows the distribution of avoided premature mortalities in each of the corresponding age categories. 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. Since the estimated quadratic is not as severe in the Jones-Lee (1993) study, the bias is not likely to be as great. However, EPA chose to present both calculations in order to demonstrate both the sensitivity of total benefits to this specification and the high degree of uncertainty involved in this assumption. Figure 2 shows the dollar impact of extrapolating the Jones-Lee results to the very elderly. While theoretically, the VSL may actually be close to zero as individuals approach 90 years of age, additional research on the actual WTP of these individuals should be conducted, rather than just assuming that there is such a steep drop-off in VSL

Why It Is Difficult to Value Morbidity More Completely

With good reason, a great deal of effort has been focused on the value of reducing mortality risks. However, one area that is often overlooked is the lack of suitable WTP studies for morbidity effects. EPA relies on benefits transfer to provide WTP estimates for many respiratory effects, such as chronic bronchitis, and has no accepted values for asthma attacks and other asthma related symptoms. EPA uses cost-of-illness estimates to value reductions in hospital admissions, which are known to underestimate the WTP for these endpoints. And, as discussed earlier, even the use of hospital admissions as a health endpoint may understate the health impact of a PM health event that leads to hospitalization. EPA is currently considering better ways of characterizing morbidity impacts, both in terms of health effects and economic values. While some existing endpoints, such as chronic bronchitis, merely need focused research by economists, others may require a rethinking of the relationship between the disciplines of epidemiology and economics.

Asthma provides an excellent example of the difficulties in valuing morbidity endpoints. While asthma attacks are a serious concern to asthma sufferers, an asthma attack is a relative ill-defined health endpoint from a clinical standpoint. Although it is easily understood by individuals with asthma who might respond to a WTP question, the definition of an asthma attack may be completely subjective and specific to each individual. Epidemiologists have focused on separating asthma attacks into their component symptoms, however, while making consistent measurement of health effects easier, this makes valuation more difficult and can potentially lead to double-counting of benefits. A number of recent epidemiology studies show a relationship between PM and/or ozone levels and asthma-related respiratory symptoms such as wheezing, coughing, acute bronchitis and shortness of breath. Many of these studies use "asthma symptom scores" as an endpoint. This may be a difficult endpoint to translate into a choice variable that can be offered to respondents in a WTP survey.

Only one early study (Whittemore and Korn, 1980) estimated the relationship between asthma attacks and photochemical air pollutant concentrations. The likely reason for the emphasis of most studies on particular asthma symptoms is the subjective definition of an asthma attack and the subsequent lack of specificity in measuring an asthma attack occurrence. In this analysis, the endpoint "asthma attack" is a better match for the available economic valuation studies and avoids potential over-prediction (as one attack may involve some combination of symptoms). Accordingly, an asthma attack is an endpoint that summarizes the collection of symptoms, so potential double-counting may occur if individual asthma symptoms estimated from other studies are summed. An asthma attack, as measured by Whittemore and Korn (1980), is based on subjective reporting by study participants and likely consisting of one or more of the respiratory symptoms listed above occurring at varying levels of severity. For example, a subject reporting an asthma attack in the Whittemore and Korn (1980) study may have shortness of breath and wheezing. This is accounted for as one attack, while using individual symptom studies would record this as two separate symptom occurrences. Conversely, a participant may experience symptoms but not consider the symptoms to be "an attack." Thus, the use of "asthma attacks" as an indicator may understate symptoms.

Other recent analyses of the impacts of air pollution reductions on asthma symptoms have used collections of asthma symptom studies (Kunzli et al., 2000). This may be a useful approach in the future if suitable indices of asthma symptoms can be developed for the U.S. However, care must be taken in pooling effect estimates from studies with widely varying symptom definitions, as standard inverse-variance pooling methods tend to bias the pooled effect estimate toward the lowest estimated coefficient[7].

Only a limited number of economic studies have been conducted on the value of reduced asthma symptoms. One valuation study by Rowe and Chestnut (1986) calculated the value of reduction in "bad asthma days," which we interpret as equivalent to a day with an asthma attack. By using the Whittemore and Korn (1980) asthma attack C-R function in combination with the Rowe and Chestnut (1986) valuation study, we are able to provide a quantified and monetized estimate of asthma-related symptoms that is representative of the full spectrum of impacts of air pollution reductions on asthma sufferers. However, concerns raised by reviewers about the reliability of the WTP estimates from Rowe and Chestnut (1986) led us to leave asthma attacks as an unmonetized benefit category in the HD Engine/Diesel Fuel analysis.

Several other important issues arise when evaluating the impacts of air pollution reductions on symptoms in populations with chronic respiratory diseases. Note that in estimating the number of avoided asthma attacks one must consider the total change over the full population of asthmatics, potentially including multiple avoided attacks for a single individual. Also, because the estimate of asthma attacks is based on both the incidence of asthma attacks and the prevalence of asthma in the population, to the extent that asthma incidence rates are increasing (or decreasing), the number of asthma attacks avoided will also be increasing (or decreasing). The prevalence of asthma in the U.S., especially among children, has been increasing over the past two decades (Pew Environmental Health Commission, 2000), suggesting that if current trends continue, the impact on asthma symptoms of reductions in air pollution in the future will be greater than current estimates would suggest.

Collaboration between economists and epidemiologists in the future can potentially provide definitions of asthma symptoms or asthma attacks that can then be carefully linked to valuation studies for the same endpoint definitions. This can lead to improved estimates of the value of reducing symptoms/attacks in asthmatics. In addition, careful measurement of baseline incidence and prevalence rates will be essential to ensure accurate estimation of avoided symptoms in affected populations.

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[7]For example, consider two estimated relative risks, one for shortness of breath, and one for wheezing. For the shortness of breath study, the pollution coefficient is 0.0006, with a standard deviation of 0.0003, and for the wheezing study, the pollution coefficient is 0.006, with a standard deviation of 0.003. Even though both have the same relative variance (the standard deviation in each case is 0.5 of the estimate), the standard pooling methodology would assign a weight of 0.99 to the shortness of breath coefficient and a weight of 0.01 to the wheezing coefficient, because the inverse variance weighting uses absolute variances rather than a measure of relative precision, like t-values. When studies are measuring exactly the same outcome, inverse variance weights are appropriate. They may not be appropriate in developing combined estimates for dissimilar endpoints.

The Need for Better Communication Between Economists and Epidemiologists

Economists have tended to be end users of epidemiology studies, rather than collaborators in the research process. As greater demand for health benefits analyses develops, benefits analysts must play a greater role in the design of epidemiological analyses, to ensure that the outputs of such analyses are suitable for use as inputs to benefits analyses. For example, designing an asthma symptoms study that provides a clear link between air pollution and asthma symptoms as understood by asthma sufferers, and with a clear measurement of baseline incidences and prevalence, would provide an endpoint that can more easily be valued by economists with current valuation tools.

In addition to defining endpoints appropriately, perhaps one of the most important design issues is that, in a benefits analysis framework, we are interested in the C-R function, i.e. the relationship between changes in PM and changes in the risk of premature mortality or morbidity. In many epidemiology studies, the focus often seems to be on the significance of a relationship between observed air pollution levels and mortality or morbidity, and how consistent that finding is across locations, time, and model specification (inclusion of potential confounders, control for weather, etc.). Economists would prefer that the design of an epidemiology study for use in a benefits analysis include appropriate controls for behavioral adjustments and protocols for examining the shape of the C-R function, rather than just the significance of the pollution parameter.

For example, the lack of data on the existence of possible thresholds in the C-R function for PM-related premature mortality is a cause of considerable uncertainty in the application of the C-R function to regulation-derived reductions in PM. While EPA's Science Advisory Board has advised EPA not to assume any particular threshold in its primary estimate of PM-related health benefits, sensitivity analyses suggest that thresholds at or above 15 $\mu g/m^3$ may significantly reduce the estimated benefits of major regulations. For the HD Engine rule, the distribution of premature mortality incidences in Figure 3 indicates that approximately 90 percent of the premature mortality related benefits of the final HD Engine/Diesel Fuel rule are due to changes in PM concentrations occurring above 10 $\mu g/m^3$, the lowest observed level in the ACS/Krewski, et al. study. However, only around 60 percent of avoided incidences are due to changes occurring above 15 $\mu g/m^3$. Thus, establishing a threshold at the 15 $\mu g/m^3$ level could reduce benefits by up to 40 percent. For other rules with more rural reductions in PM, the effect of a threshold is even more pronounced. While some recent research suggests that PM may be a non-threshold pollutant (Rossi et al, 1999; Schwartz, 2000), further research into the effects of fine particles at low levels is needed to determine whether reductions in PM at low levels will have significant health benefits.

The other area where collaboration between economists and epidemiologists may bear fruit is in discovering the relationship between air pollution and health endpoints which are easily valued. For example, an epidemiology study that examined the relationship between ozone and the number of hours an individual can maintain a given exercise level might be more easily translated into economic value than a study of the relationship between ozone and forced

expiratory volume (FEV). A corollary effort would be for epidemiologists to help economists understand the economic context of clinical measures/indicators such as FEV or heart rate variability (HRV), such as how they relate to ability to play sports, or school performance. A recent study by Fuhlbrigge et al. (2001) found a significant association between percent predicted FEV_1 and risk of an asthma attack over the subsequent year. If a suitable value for asthma attacks could be determined (see earlier section), this may provide a link from FEV to an economically valuable endpoint. As another example, we may be able to use a linkage process to be able to relate HRV to heart attacks, which can then be valued using standard methods. However, the latter approach adds additional uncertainty to the ultimate benefits estimate which would need to be taken into account

Finally, if air pollution ultimately leads to changes in non-traditional endpoints, such as early onset of chronic nerve damage, as is potentially the case with manganese exposure, economists may need to work with epidemiologists to understand how changes in exposure may shift the age distribution of risk for chronic diseases. Again, early discussions between the two disciplines may improve the ability of epidemiologists to provide results in a form that facilitates use in benefits analyses.

Concluding Remarks

Epidemiology is an essential tool and input into the process of analyzing the health benefits of federal regulations. Economists, epidemiologists, and others in the policy analysis community must begin to see it as an essential piece of the analytical puzzle in developing quantified and monetized estimates of the benefits of air pollution regulations, similar to emissions and air quality models. In order to be able to answer the fundamental question of "How high should health benefits be?", we must address some of the key gaps in our understanding of health benefits of air pollution, including a better understanding of the morbidity and mortality effects of chronic exposures to ambient pollution, "ripple" effects of acute morbidity, behavioral responses to ambient air pollution, and the lack of data on the effects of ambient air toxics. Economists must work with epidemiologists to develop studies that examine the relationship between reductions in air pollution and mortality and morbidity endpoints, not just to determine whether a relationship exists, but specifically to provide a C-R function suitable for use in a national benefits analysis, including factors such as behavioral responses of individuals and enough locational attributes to ensure the transferability of the C-R function across broad geographic regions. And, analyses should consider any possible application issues, such as possible thresholds or non-linearities in the C-R functions. Also, some consistency between epidemiology and economics journals in the reporting of statistical results would enable economists to better evaluate the studies and match the populations and endpoints examined with valuation studies. Economists would find it useful if epidemiology articles would highlight any assumptions that are of critical importance in interpreting the study results. Ideally, epidemiology studies and valuation studies should be designed together, so that the endpoints and the values applied to those endpoints are internally consistent.

It is also essential for epidemiologists working in the area of air toxics to begin

developing methods to generate best estimates of dose-response relationships suitable for use in cost-benefit analysis. Current estimates based on conservative unit risk factors and exposure models may result in over-predictions of reductions in cancer cases. The lack of non-cancer effect models severely limits the ability of health benefit analysts to provide a comprehensive assessment of the health benefits associated with reductions in ambient levels of hazardous air pollutants.

In reviews of EPA's health benefits methodologies, the scientific community as represented by the SAB, has been willing in some cases to "push the envelope" and recommend assumptions that extrapolate from the published scientific literature. Clear principles for when this type of extrapolation is appropriate need to be developed to prevent constant second guessing by both EPA and its critics as to the types of assumptions that can be supported. An excellent example of this is the SAB's advice to adopt a 5-year lag for PM related premature mortality based on similarities to smoking, but not for chronic bronchitis due to a lack of similar lags in effects in smokers [EPA-SAB-COUNCIL-ADV-00-002, 1999]. Without clear guidance, assumptions may seem arbitrary and may weaken scientific support for benefits analyses.

EPA will continue to use the best available methods and models to estimate the health benefits of regulation induced reductions in air pollution, from the emissions reduction stage through the economic valuation stage. The importance of improving benefits transfer methods, both in terms of transferring economic values and transferring estimated C-R functions, should not be underestimated. Epidemiological studies should be designed with the knowledge that they may be applied to different locations, populations, or time periods. EPA supports the development of both better models of current health endpoints and new models for endpoints related to hazardous air pollutants and additional morbidity endpoints related to criteria air pollutants. As new epidemiological approaches lead to better models of the effect of pollution reductions on human health endpoints, the reliability and usefulness of health benefits analyses will be enhanced.

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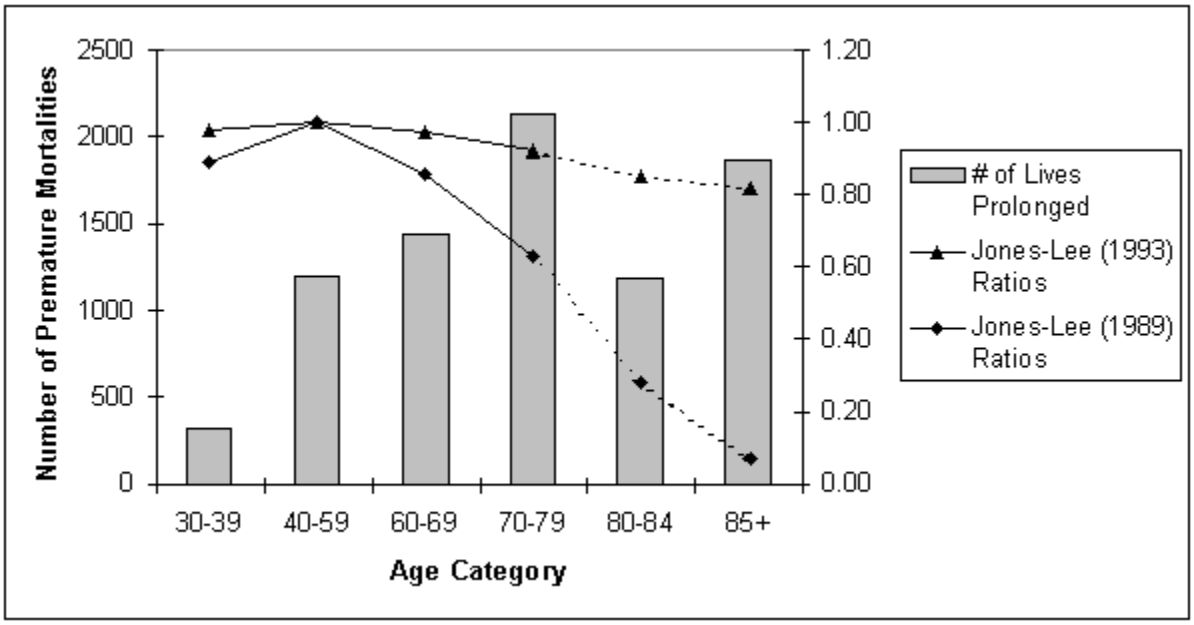

Figure 1.
Age-specific WTP Ratios and HD Engine Rule Mortality Benefits

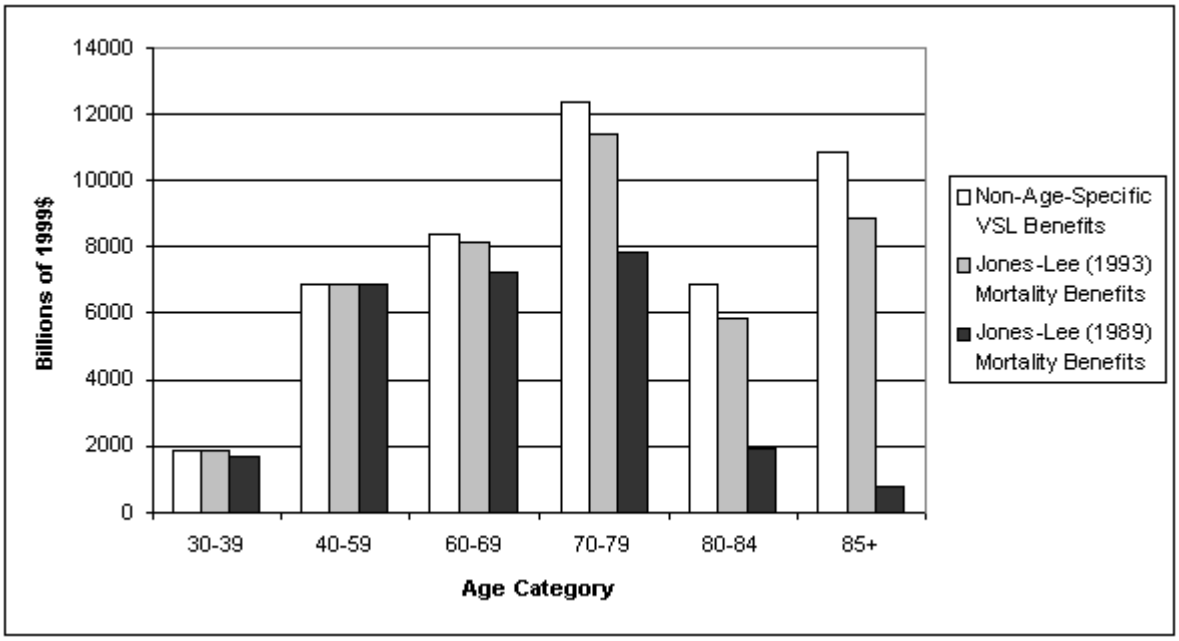

Figure 2.
Impact of Age-specific WTP on HD Engine Premature Mortality Reduction Monetized Benefits

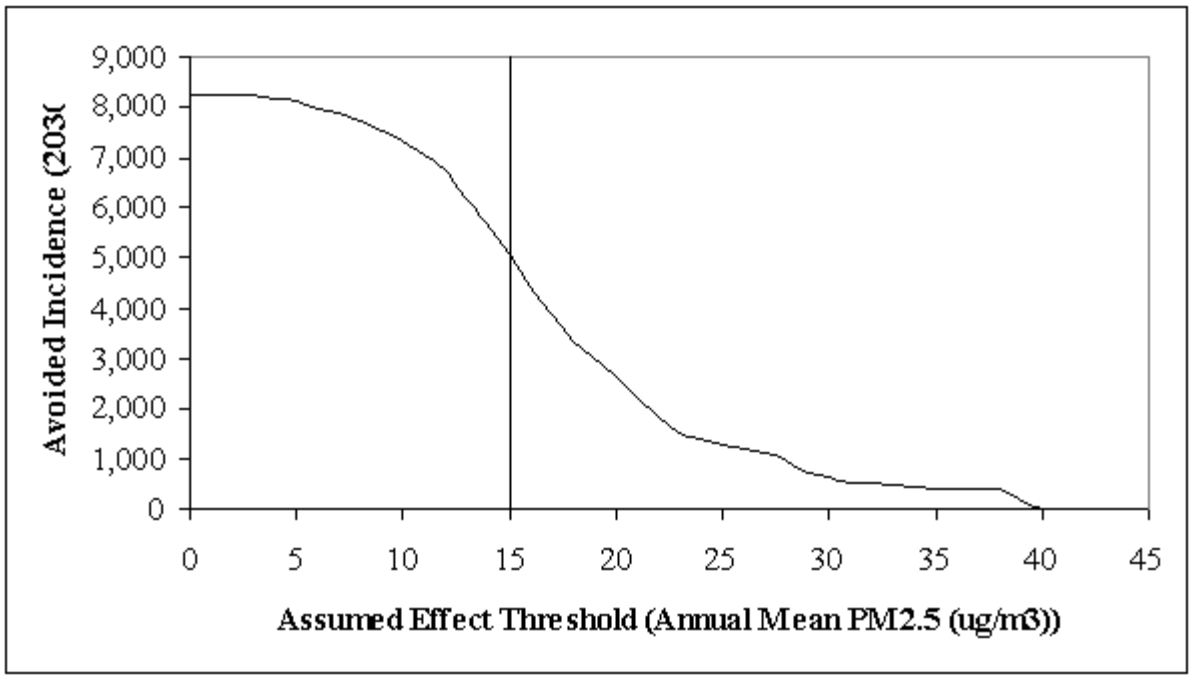

Figure 3.
Sensitivity of HD Engine/Diesel Fuel Rule Mortality Reduction Benefits to Assumed Health Effect Thresholds