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Health Based Cost Effectiveness of Ambient PM2.5 Reductions

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Abstract

Health-based cost-effectiveness analysis (CEA) and cost-utility analysis (CUA) have been used to analyze numerous health interventions but have not been widely adopted as tools to analyze environmental policies. This analysis estimates changes in health-based effectiveness measures associated with a one microgram reduction in ambient $PM_{2.5}$ across the United States, and introduces a new aggregate effectiveness metric, Morbidity Inclusive Life Years (MILY), to address some of the concerns about aggregation of life extension and quality-of-life impacts. The analysis uses health impact analysis methods to estimate reductions in life years lost and incidence of chronic bronchitis and non-fatal acute myocardial infarctions. These changes in health are then valued using published estimates of quality of life scores for each condition. The analysis suggests that for current populations, each microgram of ambient $PM_{2.5}$ reduced will result in 13,600 (95% CI: 4,700 - 22,600) premature deaths avoided, 160,000 (95% CI: 56,000 - 260,000) discounted life years gained, or 220,000 (95% CI: 61,000 - 400,000) discounted MILY gained. Taking into account avoided medical costs of $2.5 billion, current $PM_{2.5}$ control costs of up to $13 billion (95% CI: $9.4 billion - $18 billion) per microgram are cost effective relative to a benchmark of $50,000 per QALY.

JEL Classification Codes: I10, I18, Q53

Key Words: air pollution, cost-effectiveness, QALY, particulate matter, chronic disease

I. Introduction

Analyses of environmental regulations have typically used cost-benefit analysis to characterize impacts on social welfare, because it provides a comparative framework which can consistently combine human mortality and morbidity and other non-health benefits such as improved visibility. One of the great advantages of the benefit-cost paradigm is that a wide range of quantifiable benefits can be compared to costs to evaluate the economic efficiency of particular actions. However, alternative paradigms such as cost-effectiveness and cost-utility analyses may also provide useful insights. QALY-based cost-utility analysis has been widely adopted within the health economics literature [1,2] and in the analysis of public health interventions [3]. QALY based analyses have not been as accepted in the environmental economics literature due to concerns about the theoretical consistency of QALYs with individual preferences [4], treatment of non-human health benefits, aggregation of acute and long-term health impacts [5], aggregation of life extensions and quality-of-life improvements in different populations and a number of other factors [6,7]. The appropriateness of health-based CEA should be evaluated on a case-by case basis subject to the availability of appropriate data and models, among other factors. Recently several academic analyses have proposed the use of life-years based cost-benefit or cost-effectiveness analyses of air pollution regulations [8,9,10,11]. In addition, the World Health Organization has adopted the use of disability adjusted life years, a variant on QALYs, to assess the global burden of disease due to different causes, including environmental pollution [12,13].

Recently, interest has grown in providing alternative analytical perspectives on the

impacts of air pollution regulations. The U.S. Office of Management and Budget [14] has issued new guidance requiring federal agencies to provide both cost-effectiveness and cost-benefit analyses for major regulations. The OMB Circular A-4 directs agencies to "prepare a CEA for all major rulemakings for which the primary benefits are improved public health and safety to the extent that a valid effectiveness measure can be developed to represent expected health and safety outcomes." This paper proposes methods for conducting cost-effectiveness analyses for reductions in ambient fine particulate matter (defined as particulate matter with a diameter of 2.5 microns or less, and often denoted as $PM_{2.5}$), focusing on effectiveness measured by improvements in life expectancy and reductions in incidence of two diseases with chronic impacts on quality of life, chronic bronchitis and nonfatal acute myocardial infarctions. The focus of this paper is not a specific regulation, and as such the cost-effectiveness is presented in terms of the implied costs necessary to exceed a particular cost-effectiveness threshold.

This paper develops cost-effectiveness and cost-utility methodologies for evaluating programs to reduce ambient $PM_{2.5}$, starting from the standard QALY literature and seeking a parallel structure to cost-benefit analysis in the use of air quality and health inputs (see [15] for a discussion of some of the issues that arise in comparing QALY and cost-benefit frameworks in analyzing air pollution impacts). This analysis provides estimates of commonly used health-based effectiveness measures, including lives saved, life years saved (from reductions in mortality risk), and QALYs saved (from reductions in morbidity risk) associated with the reduction of ambient $PM_{2.5}$. In addition, a new aggregate effectiveness metric, Morbidity Inclusive Life Years (MILY) is introduced to address some of the concerns about aggregation of life extension and quality-of-life impacts. It represents the sum of life years gained due to reductions in premature mortality and the QALY gained due to reductions in chronic morbidity.

This measure may be preferred to existing QALY aggregation approaches because it does not devalue life extensions in individuals with preexisting illnesses that reduce quality of life. However, the MILY measure is still based on life years and thus still inherently gives more weight to interventions that reduce mortality and morbidity impacts for younger populations with higher remaining life expectancy.

For the life years and QALY approaches, I use life table methods to calculate the change in life expectancy expected to result from changes in mortality risk from particulate matter. I use existing estimates of preferences for different health states to obtain QALY weights for morbidity endpoints associated with air pollution. In general, consistent with the Gold et al [2] recommendations, I use weights obtained from a societal perspective when available. I explore several different sources for these weights to characterize some of the potential uncertainty in the QALY estimates. I follow many of the principles of the Reference Case analysis as defined in [2], although in some cases I depart from the Reference Case approach when data limitations require me to do so. I also depart from the Reference Case in my method of combining life expectancy and quality of life gains.

Monte Carlo simulation methods are used to propagate uncertainty in the model parameters throughout the analysis. I characterize overall uncertainty in the results with 95 percent confidence intervals based on the Monte Carlo simulations. In addition I examine the impacts of changing key parameters, such as the discount rate, on the effectiveness measures and the cost-effectiveness metrics.

The remainder of this paper provides an overview of the key issues involved in life year and QALY based approaches for evaluating the health impacts of air pollution regulations and provided detailed discussions of the steps required for each type of effectiveness calculation.

Section 2 introduces the various effectiveness measures and discusses some of the assumptions required for each. Section 3 details the methodology used to calculate changes in life years and quality adjustments for mortality and morbidity endpoints. Section 4 provides the results and discussion of their implications for cost-effectiveness of $PM_{2.5}$ controls.

II. Effectiveness Measures

There are three major classes of benefits associated with reductions in air pollution: mortality, morbidity, and non-health (welfare). For the purposes of benefit-cost analysis, the U.S. EPA has presented mortality-related benefits using estimates of avoided premature mortalities, representing the cumulative result of reducing the risk of premature mortality from long term exposure to $PM_{2.5}$ for a large portion of the U.S. population [16]. Morbidity benefits have been characterized by numbers of new incidences avoided for chronic diseases such as chronic bronchitis, avoided admissions for hospitalizations, and avoided days with symptoms for minor illnesses. Non-health benefits are characterized by the monetary value of reducing the impact, e.g. the dollar value of improvements in visibility at national parks.

For the purposes of cost-effectiveness analysis, I will be focusing the effectiveness measure on the quantifiable health impacts of the reduction in $PM_{2.5}$. Treatment of non-health benefits is important and will be discussed later in this section. If the main impact of interest is reductions in mortality risk from air pollution, the effectiveness measures are relatively straightforward to develop. Mortality impacts can be characterized similar to the benefits analysis, by counting the number of premature mortalities avoided, or can be characterized in terms of increases in life expectancy or life years.

Life expectancy is an ex ante concept, indicating the impact on an entire population's expectation of the number of life years they have remaining, before knowing which individuals

will be affected. Life expectancy thus incorporates both the probability of an effect and the impact of the effect if realized. Life years is an ex post concept, indicating the impact on individuals who actually die from exposure to air pollution. Changes in population life expectancy will always be substantially smaller than changes in life years per premature mortality avoided, although the total life years gained in the population will be the same. This is due to the fact that life expectancy gains average expected life years gained over the entire population, while life years gained measures life years gained only for those experiencing the life extension.

Estimates of premature mortality have the benefit of being relatively simple to calculate, are consistent with benefit-cost analysis, and do not impose additional assumptions on the degree of life shortening. However, some have argued that counts of premature mortalities avoided are problematic because a gain in life of only a few months would be considered equivalent to a gain of many life years, and the true effectiveness of an intervention is the gain in life expectancy [10,17].

Calculations of changes in life years and life expectancy can be accomplished using standard life table methods [17]. However, the calculations require assumptions about the baseline mortality risks for each age cohort affected by air pollution. A general assumption may be that air pollution mortality risks affect the general mortality risk of the population in a proportional manner. However, some concerns have been raised that air pollution affects mainly those individuals with preexisting cardiovascular and respiratory disease, who may have reduced life expectancy relative to the general population. This issue is explored in more detail below.

Air pollution is also associated with a number of significant chronic and acute morbidity endpoints. Failure to consider these morbidity effects may understate the cost-effectiveness of

air pollution regulations, or give too little weight to reductions in particular pollutants that have large morbidity impacts but no effect on life expectancy. One measure that has been widely used to evaluate medical interventions that affect both life expectancy and morbidity is the quality adjusted life year (QALY). The QALY approach explicitly incorporates morbidity impacts into measures of life years gained and is often used in health economics to assess the cost effectiveness of medical spending programs [2]. Using a QALY rating system, health quality ranges from 0 to 1, where 1 may represent full health, 0 death, and some number in between (e.g., 0.8) an impaired condition. QALYs thus measure morbidity as a reduction in quality of life over a period of life. QALYs assume that duration and quality of life are equivalent, so that one year spent in perfect health is equivalent to two years spent with quality of life half that of perfect health. While there are some very strong assumptions (detailed below) associated with QALYs, they can be used to evaluate environmental rules under certain circumstances. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommended the use of QALYs when evaluating medical and public health programs that primarily reduce both mortality and morbidity [2]. While there are significant non-health benefits associated with air pollution regulations, over 90 percent of quantifiable monetized benefits are health-related (See for example the benefit-cost analysis of the recently issued Clean Air Interstate Rule [16]). Thus, it can be argued that QALYs are applicable for these types of regulations. However, the value of non-health benefits should not be ignored, and as discussed below, should at least be subtracted from the costs in the numerator of the cost-effectiveness ratio.

Also, it is important to note that while used extensively in the economic evaluation of medical interventions [2], QALYs have not been widely used in evaluating environmental health regulations. A number of specific issues arise with the use of QALYs in evaluating

environmental programs that affect a broad and heterogeneous population and that provide both health and nonhealth benefits. The U.S. Public Health Service report on cost-effectiveness in health and medicine notes the following:

> "For decisions that involve greater diversity in interventions and the people to whom they apply, cost-effectiveness ratios continue to provide essential information, but that information must, to a greater degree, be evaluated in light of circumstances and values that cannot be included in the analysis. Individuals in the population will differ widely in their health and disability before the intervention, or in age, wealth, or other characteristics, raising questions about how society values gains for the more and less health, for young and old, for rich and poor, and so on. The assumption that all QALYs are of equal value is less likely to be reasonable in this context." ([2], p. 11)

Use of QALYs as a measure of effectiveness for environmental regulations is still developing, and while this analysis provides one framework for using QALYs to evaluate environmental regulations, there are clearly many issues, both scientific and ethical, that need to be addressed with additional research. The Institute of Medicine panel evaluating QALYs and other effectiveness measures (see http://www.iom.edu/project.asp?id=19739) will develop criteria for choosing among the measures that potentially are useful for regulatory impact analysis and will make recommendations regarding measures appropriate for assessing the health benefits of regulatory interventions and propose criteria for identifying regulations for which CEA is appropriate and informative.

In the following sections, I lay out a phased approach to describing effectiveness. I begin by discussing how the life extending benefits of air pollution reductions are calculated, and then

incorporate morbidity effects using the QALY approach. I also introduce an alternative aggregated health metric, Morbidity Inclusive Life Years (MILY), to address some of the ethical concerns about aggregation of life extension and quality of life impacts in populations with preexisting disabling conditions.

III. Changes in Premature Death, Life Years and Quality of Life

To generate health outcomes, a one microgram (:g) change in ambient PM concentrations was entered into BenMAP, a customized geographic information system based program. BenMAP has been used by EPA to assess the benefits of reducing air pollution in several recent analyses. More details on BenMAP can be found on the EPA Innovative Strategies and Economics Group website, http://www.epa.gov/ttn/ecas . BenMAP uses 2000 census population data and changes in pollutant concentrations to estimate changes in health outcomes for each grid cell. Details on the BenMAP program can be found in the BenMAP User's Manual [18].

BenMAP uses health impact functions to generate changes in the incidence of health effects. Health impact functions are derived from the epidemiology literature. A standard health impact function has four components: an effect estimate from a particular epidemiological study, a baseline incidence rate for the health effect (obtained from either the epidemiology study or a source of public health statistics like the Centers for Disease Control), the affected population, and the estimated change in the relevant PM summary measure.

A typical health impact function might look like:

$$\Delta y = y_0 \cdot (e^{\beta \cdot \Delta x} - 1), \quad (1)$$

where y_0 is the baseline incidence, equal to the baseline incidence rate times the potentially affected population, ∃ is the effect estimate, and)x is the estimated change in $PM_{2.5}$. There are

other functional forms, but the basic elements remain the same.

A. Calculating reductions in premature deaths

As in several recent air pollution health impact assessments [16,19,20], I focus on the prospective cohort long-term exposure studies in deriving the health impact function for our estimate of premature mortality. Cohort analyses are better able to capture the full public health impact of exposure to air pollution over time [21,22]. I selected an effect estimate from the extended analysis of the American Cancer Society (ACS) cohort [23]. The effect estimate from [23] quantifies the relationship between annual mean $PM_{2.5}$ levels and all-cause mortality in adults 30 and older. I selected the effect estimate estimated using the measure of PM representing average exposure over the follow-up period, calculated as the average of 1979-1984 and 1999-2000 $PM_{2.5}$ levels. The effect estimate from this study is 0.0058, which is equivalent to a relative risk of 1.06 for a 10 :g change in $PM_{2.5}$.

While there are other cohort-based studies of the relationship between $PM_{2.5}$ and mortality, none provide the same level of population and geographic coverage as the ACS study. Use of the ACS study also provides for comparability with recent cost-benefit analyses conducted by EPA [16,20]. The reductions in incidence of premature mortality within each age group for a one :g reduction in $PM_{2.5}$, based on 2000 Census populations, are summarized in Table 1.

B. Calculating changes in life years from direct reductions in $PM_{2.5}$ related mortality risk

In order to calculate changes in life years associated with a given change in air pollution, I use a life table approach coupled with age-specific estimates of reductions in premature mortality. Table 2 presents the abridged life table for 10 year age intervals for adults over 30 (to match the Pope et al. 2002 study population), derived from the complete unabridged life table for the United States in 2000 [24,25]. Note that the abridgement actually includes one five year interval, covering adults 30 to 34, with the remaining age intervals covering 10 years each. This is to provide conformity with the age range covered by the ACS study.

From the abridged life table (Table 2) I obtain the remaining life expectancy for each age cohort, conditional on surviving to that age. This is then the number of life years lost for an individual in the general population dying during that age interval. This information can then be combined with the estimated number of premature deaths in each age interval calculated with BenMAP (see previous subsection). Total life years gained will then be the sum of life years gained in each age interval:

$$Total\ Life\ Years = \sum_{i=1}^{N} LE_i \times M_i , \qquad (2)$$

where LE_i is the remaining life expectancy for age interval i, M_i is the change in incidence of mortality in age interval i, and N is the number of age intervals.

Following standard practice, I discount gains in future life years using a 3 percent discount rate, reflecting empirical evidence on the social rate of time preference. Selection of a 3

percent discount rate is also consistent with recommendations from the NAS Panel on Cost Effectiveness in Health and Medicine [2]. Impacts of selecting an alternative 7 percent discount rate are explored in a sensitivity analysis. Discounted total life years gained is calculated as:

$$Discounted\ LY = \int_0^{LE} e^{-rt}\,dt\,, \qquad (3)$$

where r is the discount rate, equal to 0.03 in this case, t indicates time, and LE is the life expectancy at the time when the premature death would have occurred.

Use of all-cause mortality is appropriate if there are no differences in the life expectancy of individuals dying from air pollution related causes and those dying from other causes. The argument that long term exposure to $PM_{2.5}$ may affect mainly individuals with serious preexisting illnesses is not supported by current empirical studies. The U.S. EPA Science Advisory Board Health Effects Subcommittee (SAB-HES) suggests using average life expectancy for matching age groups, based on evidence from the ACS reanalysis [26] which suggests that the mortality risk is no greater for those with pre-existing illness at time of enrollment in the study. Life expectancy for the general population in fact includes individuals with serious chronic illness. Mortality rates for the general population then reflect prevalence of chronic disease, and as populations age the prevalence of chronic disease increases. The only reason one might use a lower life expectancy is if the population at risk from air pollution was limited solely to those with preexisting disease. I examine the impacts of assumptions regarding preexisting conditions in a sensitivity analysis provided in the appendix to this article.

Should Life Years Gained Be Adjusted for Initial Health Status?

The methods outlined above provide estimates of the total number of life years gained in a population, regardless of the quality of those life years, or equivalently, assuming that all life

years gained are in perfect health. In some cost-effectiveness analyses [8,9], analysts have adjusted the number of life years gained to reflect the fact that 1) the general public is not in perfect health and thus "healthy" life years are less than total life years gained, and 2) those affected by air pollution may be in a worse health state than the general population and therefore will not gain as many "healthy" life years from an air pollution reduction. This adjustment, which converts life years gained into QALYs, raises a number of serious ethical issues pertaining to the context of environmental public health impact assessment. Proponents of QALYs have promoted the nondiscriminatory nature of QALYs in evaluating improvements in quality of life, e.g. an improvement from a score of 0.2 to 0.4 is equivalent to an improvement from 0.8 to 1.0, so the starting health status does not affect the evaluation of interventions that improve quality of life. However, for life extending interventions, the gains in QALY will be directly proportional to the baseline health state, e.g. an individual with a 30 year life expectancy and a starting health status of 0.5 will gain exactly half the QALYs of an individual with the same life expectancy and a starting health status of 1.0 for a similar life extending intervention. This is troubling, as it imposes an additional penalty for those already suffering from disabling conditions. Brock (2002) notes that "the problem of disability discrimination represents a deep and unresolved problem for resource prioritization." [27]

For the purpose of this analysis, I do not reduce the number of life years gained to reflect any differences in underlying health status that might reduce quality of life in remaining years. I maintain the assumption that all direct gains in life years resulting from mortality risk reductions will be assigned a weight of 1.0. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommends that "since lives saved or extended by an intervention will not be in perfect health, a saved life year will count as less than 1 full QALY" [2]. However, for the

purposes of this analysis, we propose an alternative to the traditional aggregate QALY metric that keeps separate quality adjustments to life expectancy and gains in life expectancy. As such, we do not make any adjustments to life years gained to reflect the less than perfect health of the general population. Gains in quality of life will be addressed as they accrue because of reductions in the incidence of chronic diseases. This is an explicit equity choice in the treatment of issues associated with quality-of-life adjustments for increases in life expectancy that still capitalizes on the ability of QALYs to capture both morbidity and mortality impacts in a single effectiveness measure.

IV. Calculating Changes in the Quality of Life Years

In addition to directly measuring the quantity of life gained, measured by life years, it may also be informative to measure gains in the quality of life. Reducing air pollution also leads to reductions in serious illnesses that affect quality of life. These include chronic bronchitis and cardiovascular disease, for which I am able to quantify changes in the incidence of non-fatal heart attacks. In order to capture these important benefits in the measure of effectiveness, they must first be converted into a life year equivalent so that they can be combined with the direct gains in life expectancy.

For this analysis, I develop estimates of the QALY gained from reductions in incidence of chronic bronchitis and non-fatal heart attacks associated with reductions in ambient $PM_{2.5}$. In general, QALY calculations require four elements:

1) The estimated change in incidence of the health condition,

2) The duration of the health condition,

3) The quality of life weight with the health condition, and

4) The quality of life weight without the health condition (i.e. the baseline health state)

The first element is derived using the health impact function approach. The second element is based on the medical literature for each health condition. The third and fourth elements are derived from the medical cost-effectiveness and cost-utility literature. In the following two subsections, I discuss the choices of elements for chronic bronchitis and non-fatal heart attacks. All of the elements of the analysis are summarized in Table 3.

The preferred source of quality of life weights are those based on community preferences, rather than patient or clinician ratings [2]. There are several methods used to estimate quality of life weights. These include rating scale, standard gamble, time tradeoff, and person tradeoff approaches [28]. Only the standard gamble approach is completely consistent with utility theory. However, the time tradeoff method has also been widely applied in eliciting community preferences [28].

Quality of life weights can be directly elicited for individual specific health states, or for a more general set of activity restrictions and health states which can then be used to construct QALY weights for specific conditions [29,30,31]. For this analysis, I use weights based on community based preferences, using time-tradeoff or standard gamble when available. In some cases, I use patient or clinician ratings when no community preference based weights are available. I have chosen not to use weights constructed from generic utility instruments due to concerns about mapping the specific conditions considered in this analysis with the health states for which preferences were elicited. Weights used in the analysis are summarized in Table 3. Sources for weights are discussed in more detail below.

A. Calculating QALYs Associated with Reductions in the Incidence of Chronic Bronchitis

Chronic bronchitis is characterized by mucus in the lungs and a persistent wet cough for at least 3 months a year for several years in a row. Chronic bronchitis affects an estimated 5 percent of the U.S. population [32]. For gains in quality of life resulting from reduced incidences of PM-induced chronic bronchitis, QALYs are calculated as

$$QALY\,GAINED = \sum_i \Delta CB_i \times D_i \times \left(w_i - w_i^{CB}\right), \qquad (6)$$

where $)CB_i$ is the number of incidences of chronic bronchitis avoided in age interval i, D_i is the duration of life with chronic bronchitis for individuals with onset of disease in age interval i, w_i is the average QALY weight for age interval i, and

w_i^{CB} is the QALY weight associated with chronic bronchitis.

The number of cases of chronic bronchitis in each age interval is derived from application of the impact function from [33], to the population in each age interval with the appropriate baseline incidence rate. Prevalence rates for chronic bronchitis were obtained from the 1999 National Health Interview Survey [34]. Prevalence rates were available for three age groups, 18-44, 45-64, and 65 and older. Prevalence rates per person for these groups were 0.0367 for 18-44, 0.0505 for 45-64, and 0.0587 for 65 and older. The incidence rate for new cases of chronic bronchitis (0.00378 per person) was taken directly from [33]. The effect estimate from [33] is 0.0137, which, based on the logistic specification of the model, is equivalent to a relative risk of

1.15 for a 10 :g change in $PM_{2.5}$. Table 1 presents the estimated reduction in new incidences of chronic bronchitis associated with a one microgram reduction in ambient $PM_{2.5}$.

Chronic bronchitis is assumed to persist for the remainder of an affected individual's lifespan. Duration of chronic bronchitis will thus equal life expectancy conditioned on having chronic bronchitis. The CDC has estimated that COPD (of which chronic bronchitis is one element) results in an average loss of life years equal to 4.26 per COPD death, relative to a reference life expectancy of 75 years [35]. As such, I subtract 4.26 from the remaining life expectancy for each age group, up to age 75. For age groups over 75, I apply the ratio of 4.26 to the life expectancy for the 65 to 74 year group (0.237) to the life expectancy for the 75 to 84 and 85 and up age groups to estimate potential life years lost and then subtract that value from the base life expectancy.

Quality of life with chronic lung diseases has been examined in several studies. In an analysis of the impacts of environmental exposures to contaminants, de Hollander et al. [13] assigned a weight of 0.69 to years lived with chronic bronchitis. This weight was based on physicians' evaluations of health states similar to chronic bronchitis. Salomon and Murray [36] estimated a pooled weight of 0.77 based on visual analogue scale, time trade-off, standard gamble, and person trade-off techniques applied to a convenience sample of health professionals. The Harvard Center for Risk Analysis catalog of preference scores reports a weight of 0.40 for severe chronic obstructive pulmonary disease, with a range from 0.2 to 0.8, based on the judgments of the study's authors [37, 38]. The Victoria Burden of Disease (BoD) study used a weight of 0.47 for severe COPD and 0.83 for mild to moderate COPD, based on an analysis by Stouthard et al. [39] of chronic diseases in Dutch populations [40]. Based on the recommendations of Gold, et al. [2], quality of life weights based on community preferences are

preferred for cost-effectiveness analysis of interventions affected broad populations. Use of weights based on health professionals is not recommended. It is not clear from the Victoria BoD Study whether the weights used for COPD are based on community preferences or judgments of health professionals. The Harvard catalog score is clearly identified as based on author judgment. Given the lack of a clear preferred weight, I select a triangular distribution centered at 0.7 with upper bound at 0.9 (slightly better than a mild/moderate case defined by the Victoria BoD Study) and a lower bound at 0.5 based on the Victoria BoD study. Additional empirical data on quality of life with chronic respiratory diseases based on community preferences will be needed to improve these estimates.

Selection of a reference weight for the general population without chronic bronchitis is somewhat uncertain. It is clear that the general population is not in perfect health; however, there is some uncertainty as to whether individuals ratings of health states are in reference to a perfect health state or to a generally achievable "normal" health state given age and general health status. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommends that "since lives saved or extended by an intervention will not be in perfect health, a saved life year will count as less than 1 full QALY" [2]. Available population level estimates of age specific quality of life suggest a decline in overall quality of life scores with age [29]. However, these population level estimates are based on a population where the prevalence of chronic diseases such as COPD and heart disease increase with age. As such, the appropriate baseline for comparing individuals with a chronic disease to those without is not the population average, but rather the population without the chronic disease. In the absence of appropriate baseline weights, and following Carrothers, Evans and Graham [11], I assume that the reference weight for the general population without chronic bronchitis is 0.95. To allow for

uncertainty in this parameter, I assign a triangular distribution around this weight, bounded by 0.9 and 1.0. Note that the reference weight for the general population is used solely to determine the incremental quality-of-life improvement applied to the duration of life that would have been lived with the chronic disease. For example, if CB has a quality-of-life weight of 0.7 relative to a reference quality-of-life weight of 0.9, then the incremental quality-of-life improvement in 0.2. If the reference quality-of-life weight is 0.95, then the incremental quality-of-life improvement is 0.25. As noted above, the population is assumed to have a reference weight of 1.0 for all life years gained due to mortality risk reductions.

Following the literature, I discount QALYs over the duration of the lifespan with chronic bronchitis using a 3 percent discount rate [2]. Based on the assumptions defined above, I use Monte Carlo simulation methods as implemented in the Crystal Ball™ software program to develop the distribution of QALY gained per incidence of chronic bronchitis for each age interval. Monte Carlo simulation uses random sampling from distributions of parameters to characterize the effects of uncertainty on output variables. For more details, see [41]. Based on the assumptions defined above, the mean QALY gained per incidence of chronic bronchitis for each age interval along with the 95 percent confidence interval resulting from the Monte Carlo simulation is presented in Table 4. Table 4 presents both the undiscounted and discounted QALY gained per incidence.

B. Calculating QALYs Associated with Reductions in the Incidence of Non-fatal Myocardial Infarctions

Non-fatal heart attacks, or acute myocardial infarctions, require more complicated calculations to derive estimates of QALY impacts. The actual heart attack, which results when an area of the heart muscle dies or is permanently damaged due to oxygen deprivation, and subsequent emergency care are of relatively short duration. Many heart attacks result in sudden death. However, for survivors, the long term impacts of advanced coronary heart disease are potentially of long duration and can result in significant losses in quality of life and life expectancy.

Our approach adapts a coronary heart disease model developed for the Victoria Burden of Disease study [42]. This model accounts for the lost quality of life during the heart attack and the possible health states following the heart attack. Figure 1 shows the heart attack QALY model in diagrammatic form. The total gain in QALYs is calculated as:

$$AMI\ QALY\ GAINED = \sum_i \Delta AMI_i \times D_i^{AMI} \times \left(w_i - w_i^{AMI}\right) + \sum_i \sum_{j=1}^{4} \Delta AMI_i \times p_j D_{ij}^{PostAMI} \times \left(w_i - w_{ij}^{postAMI}\right) \quad (7)$$

where)AMI$_i$ is the number of non-fatal acute myocardial infarctions avoided in age interval *i*, D_i^{AMI} is the duration of the acute phase of the AMI,w$_i$ is the average QALY weight for age interval i, w_i^{AMI} is the QALY weight associated with the acute phase of the AMI, p$_j$ is the probability of being in the *j*th post-AMI status, $D_{ij}^{PostAMI}$ is the duration of post-AMI health status *j*, and $w_{ij}^{postAMI}$ is the QALY weight associated with post-AMI health status *j*.

Nonfatal heart attacks have been linked with short-term exposures to $PM_{2.5}$ in the United States [43] and other countries [44]. I use a recent study [43] as the basis for the impact function

estimating the relationship between $PM_{2.5}$ and nonfatal heart attacks. This study is the only available U.S. study to provide a specific estimate for heart attacks. Other studies [45,46] show a consistent relationship between all cardiovascular hospital admissions, including for nonfatal heart attacks, and PM.

The number of avoided nonfatal AMI in each age interval is derived from application of the impact function to the population in each age interval with the appropriate baseline incidence rate. Daily nonfatal myocardial infarction incidence rates per person were obtained from the 1999 National Hospital Discharge Survey (assuming all diagnosed nonfatal AMI visit the hospital). Age specific rates for 4 regions are used in the analysis. Regional averages for populations 18 and older are 0.0000159 for the Northeast, 0.0000135 for the Midwest, 0.0000111 for the South, and 0.0000100 for the West. The effect estimate from the Peters et al. (2001) study is 0.0241, which, based on the logistic specification of the model, is equivalent to a relative risk of 1.27 for a 10 :g change in $PM_{2.5}$. Table 1 presents the estimated reduction in nonfatal AMI associated with a one microgram reduction in ambient $PM_{2.5}$.

Acute myocardial infarction results in significant loss of quality of life for a relatively short duration. The WHO Global Burden of Disease study, as reported in [42], assumes that the acute phase of an acute myocardial infarction lasts for 0.06 years, or around 22 days. An alternative assumption is the acute phase is characterized by the average length of hospital stay for an AMI in the U.S., which is 5.5 days, based on data from the Agency for Healthcare Research and Quality's Healthcare Cost and Utilization Project (HCUP) [47]. Note that the average length of stay estimated from the HCUP data includes all discharges, including those due to death. As such, the 5.5 day average length of stay is likely an underestimate of the average length of stay for AMI admissions where the patient is discharged alive. I assume a

distribution of acute phase duration characterized by a uniform distribution between 5.5 and 22 days, noting that due to earlier discharges and inhome therapy available in the U.S., duration of reduced quality of life may continue after discharge from the hospital. In the period during and directly following an AMI (the acute phase), I assign a quality of life weight equal to 0.605, consistent with the weight for the period in treatment during and immediately after an attack [42].

During the post-AMI period, there are a number of different health states that can determine the loss in quality of life. I have chosen to classify post-AMI health status into four states defined by the presence or absence of angina and congestive heart failure (CHF). Probabilities for the four post-AMI health states sum to one.

Given the occurrence of a non-fatal AMI, the probability of congestive heart failure is set at 0.2, following the heart disease model developed in [42]. The probability is based on a study by Cowie et al [48], which estimated that 20 percent of those surviving AMI develop heart failure, based on an analysis of the results of the Framingham Heart Study.

The probability of angina is based on the prevalence rate of angina in the U.S. population. Using data from the American Heart Association [49], I calculated the prevalence rate for angina by dividing the estimated number of people with angina (6.6 million) by the estimated number of people with coronary heart disease of all types (12.9 million). I then assume that the prevalence of angina in the population surviving an AMI is similar to the prevalence of angina in the total population with CHD. The estimated prevalence rate is 51%, so the probability of angina is 0.51.

Combining these factors leads to the probabilities for each of the four health states as follows:

I. Post AMI with CHF and angina = 0.102

II. Post AMI with CHF without angina = 0.098

III. Post AMI with angina without CHF = 0.408

IV. Post AMI without angina or CHF = 0.392

Duration of post-AMI health states varies, based in part on assumptions regarding life expectancy with post-AMI complicating health conditions. Based on the model used for established market economies (EME) in the WHO Global Burden of Disease study, as reported in [42], I assume that individuals with CHF have a relatively short remaining life expectancy, and thus a relatively short period with reduced quality of life (recall that gains in life expectancy are assumed to be captured by the cohort estimates of reduced mortality risk). Columns two and three of Table 5 provide the duration (both discounted and undiscounted) of CHF assumed for post-AMI cases by age interval.

Duration of health states without CHF are assumed to be equal to the life expectancy of individuals conditional on surviving an AMI. Ganz et al [50] note that "Because patients with a history of myocardial infarction have a higher chance of dying of coronary heart disease that is unrelated to recurrent myocardial infarction (for example, arrhythmia), this cohort has a higher risk for death from causes other than myocardial infarction or stroke than does an unselected population." They go on to specify a mortality risk ratio of 1.52 for mortality from other causes for the cohort of individuals with a previous (nonfatal) AMI. The risk ratio is relative to all-cause mortality for an age-matched unselected population (i.e. general population). I adopt the same ratios and apply them to each age specific all-cause mortality rate to derive life expectancies (both discounted and undiscounted) for each age group after an AMI, presented in columns four and five of Table 5. These life expectancies are then used to represent the duration

of non-CHF post-AMI health states (III and IV).

For the four post-AMI health states, I use QALY weights based on preferences for the combined conditions characterizing each health state. There are a number of estimates of QALY weights available for post-AMI health conditions.

The first two health states are characterized by the presence of CHF, with or without angina. The Harvard Center for Risk Analysis [38] catalog of preference scores provides several specific weights for CHF with and without mild or severe angina, and one set specific to post-AMI CHF. Following the Victoria Burden of Disease model [42], I assume that most cases of angina will be treated and thus kept at a mild to moderate state. I thus focus our selection on QALY weights for mild to moderate angina. There are two sets of community preference based scores for CHF in the Harvard database [50,51]. The scores for CHF with angina range from 0.736 to 0.85. The lower of the two scores is based on angina in general with no delineation by severity. Based on the range of the scores for mild to severe cases of angina in the second study, one can infer that an average case of angina has a score around 0.96 of the score for a mild case. Applying this adjustment raises the lower end of the range of preference scores for a mild case of angina to 0.76. I select a uniform distribution over the range 0.76 to 0.85 for CHF with mild angina, with a midpoint of 0.81. The same two studies in the Harvard catalog also provide weights for CHF without angina. These scores range from 0.801 to 0.89. I select a uniform distribution over this range, with a midpoint of 0.85.

The third health state is characterized by angina, without the presence of CHF. Within the Harvard catalog, there are five sets of community preference based scores for angina, one which specifies scores for both mild and severe angina, one which specifies mild angina only, one which specifies severe angina only, and two which specify angina with no severity

classification [52,53]. The scores for the non-specific severity angina fall within the range of the two scores for mild angina specifically. As such I use the range of mild angina scores as the endpoints of a uniform distribution. The range of mild angina scores is from 0.7 to 0.89, with a midpoint of 0.80.

For the fourth health state, characterized by the absence of CHF and/or angina, there is only one relevant QALY weight available from the Harvard catalog [52]. This weight is 0.93. There is not enough information to provide a distribution for this weight; therefore it is treated as fixed value.

Similar to chronic bronchitis, I assume that the reference weight for the general population without AMI is 0.95. To allow for uncertainty in this parameter, I assign a triangular distribution around this weight, bounded by 0.9 and 1.0.

Based on the assumptions defined above, I use Monte Carlo simulation methods as implemented in the Crystal Ball™ software program to develop the distribution of QALY gained per incidence of nonfatal AMI for each age interval. For the Monte Carlo simulation, all distributions were assumed to be independent. The mean QALYs gained per incidence of nonfatal AMI for each age interval is presented in the last two columns of Table 5, along with the 95 percent confidence interval resulting from the Monte Carlo simulation. Table 5 presents both the undiscounted and discounted QALY gained per incidence.

IV. Cost effectiveness Analysis

Given the estimates of changes in life expectancy and quality of life, the next step is to aggregate life expectancy and quality of life gains to form an effectiveness measure which can be compared to costs to develop cost-effectiveness ratios. This section discusses the proper characterization of the combined effectiveness measure and the appropriate calculation of the numerator of the cost-effectiveness ratio. In addition, I calculate the implicit costs of emissions controls per microgram of $PM_{2.5}$ reduced that would be necessary for the cost-effectiveness of $PM_{2.5}$ reductions to exceed a benchmark cost per life year or QALY.

A. Aggregating Life Expectancy and Quality of Life Gains

In order to develop an integrated measure of changes in health, I simply sum together the gains in life years from reduced mortality risk in each age interval with the gains in QALYs from reductions in incidence of chronic bronchitis and acute myocardial infarctions. The resulting measure of effectiveness then forms the denominator in the cost-effectiveness ratio. What is this combined measure of effectiveness? It is not a QALY measure in a strict sense, as I have not adjusted life expectancy gains for preexisting health status (quality of life). It is however, an effectiveness measure that adds to the standard life years calculation a scaled morbidity equivalent. Thus, we term the aggregate measure morbidity inclusive life years, or MILYs. Alternatively, the combined measure could be considered as QALYs with an assumption that the community preference weight for all life expectancy gains is 1.0. If one considers that this weight might be considered to be a "fair" treatment of those with preexisting disabilities, the effectiveness measure might be termed "fair QALY" gained. However, this implies that all aspects of fairness have been addressed, and there are clearly other issues with the fairness of QALYs (or other effectiveness measures) that are not addressed in this simple adjustment. The

MILY measure violates some of the properties used in deriving QALY weights, such as linear substitution between quality of life and quantity of life. However, in aggregating life expectancy and quality-of-life gains, it merely represents an alternative social weighting that is consistent with the spirit of the recent OMB guidance on CEA. The guidance notes that "fairness is important in the choice and execution of effectiveness measures" [14]. The resulting aggregate measure of effectiveness will not be consistent with a strict utility interpretation of QALYs; however, it may still be a useful index of effectiveness.

Applying the life expectancies and distributions of QALY per incidence for chronic bronchitis and AMI to estimated distributions of incidences yields distributions of life expectancy and QALYs gained due to a nationwide one microgram reduction in ambient $PM_{2.5}$. These distributions reflect both the quantified uncertainty in incidence estimates and the quantified uncertainty in QALY gained per incidence.

Table 6 presents the mean discounted MILY gained for each age interval, broken out by life expectancy and quality of life categories, based on a 3 percent discount rate consistent with [2]. Note that quality of life gains occur from age 18 and up, while life expectancy gains accrue only after age 29. This is based on the ages of the study populations in the underlying epidemiological studies. It is unlikely that such discontinuities exist in reality, but in order to avoid overstating effectiveness, I choose to limit the life expectancy gains to those occurring in the population 30 and over and the morbidity gains to the specific adult populations examined in the studies.

It is worth noting that around a third of mortality related benefits are due to reductions in premature deaths among those 75 and older, while only 7 percent of morbidity benefits occur in this age group. This is due to two factors, 1) the relatively low baseline mortality rates in

populations under 75, and 2) the relatively constant baseline rates of chronic disease coupled with the relatively long period of life that is lived with increased quality of life without chronic bronchitis and advanced heart disease.

The relationship between age and the distribution of QALY gained from mortality and morbidity is shown in Figure 2. Because the baseline mortality rate is increasing in age at a much faster rate than the prevalence rate for chronic bronchitis, the share of QALY gained accounted for by mortality is proportional to age. At the oldest age interval, avoiding incidences of chronic bronchitis leads to only a few QALY gained, due to the lower number of years lived with chronic bronchitis. QALY gained from avoided premature mortality is low in the youngest age intervals because of the low overall mortality rates in these intervals, although the number of QALY per incidence is high. In later years, even though the QALY gained per incidence avoided is low, the number of cases is very high due to higher baseline mortality rates.

Summing over the age intervals provides estimates of total MILY gained for the nationwide one microgram reduction in ambient $PM_{2.5}$. The total number of discounted (3%) MILYs gained is 220,000 (95% CI: 61,000- 400,000). Undiscounted MILYs total 300,000 (95% CI: 83,000-560,000).

B. Cost effectiveness Ratios

Construction of cost-effectiveness ratios requires estimates of effectiveness (in this case measured by lives saved, life years gained, or MILYs gained) in the denominator and estimates of costs in the numerator. The estimate of costs in the numerator should include both the direct costs of the controls necessary to achieve the reduction in ambient $PM_{2.5}$, and the avoided costs (cost savings) associated with the reductions in morbidity [2]. In general, because reductions in air pollution do not require direct actions by the affected populations, there are no specific costs

to affected individuals (aside from the overall increases in prices that might be expected to occur as control costs are passed on by affected industries). Likewise, because individuals do not engage in any specific actions to realize the health benefit of the pollution reduction, there are no decreases in utility (as might occur from a medical intervention) that need to be adjusted for in the denominator. Thus, the elements of the numerator are direct costs of controls minus the avoided costs of illness associated with chronic bronchitis and nonfatal AMI. For the MILY aggregate effectiveness measure, the denominator is simply the sum of life years gained from increased life expectancy and the sum of QALY gained from the reductions in chronic bronchitis and nonfatal AMI.

Avoided costs for chronic bronchitis and nonfatal AMI are based on estimates of lost earnings and medical costs. Following previous cost-benefit analyses, I use age-specific annual lost earnings and medical costs [54] and a three percent discount rate to generate an estimated lifetime present discounted value (in 2000$) of costs of illness due to chronic bronchitis of $151,000 for someone between the ages of 27 and 44; $97,600 for someone between the ages of 45 and 64; and $11,100 for someone over 65 [16]. These estimates assumed that 1) lost earnings continue only until age 65, 2) medical expenditures are incurred until death, and 3) life expectancy is unchanged by chronic bronchitis.

Because the costs associated with an AMI extend beyond the initial event itself, I consider costs incurred over several years. Using age-specific annual lost earnings [54], and a three percent discount rate, I estimated a present discounted value in lost earnings (in 2000$) over 5 years due to an AMI of $8,800 for someone between the ages of 25 and 44, $12,900 for someone between the ages of 45 and 54, and $74,700 for someone between the ages of 55 and 65. Lost earnings estimates are not provided for populations under 25 or over 65. As such I do

not include lost earnings in the cost estimates for these age groups.

Two estimates of the direct medical costs of AMI are used. The first estimate is from Wittels et al. [55], which estimated expected total medical costs of AMI over 5 years to be $51,211 (in 1986$) for people who were admitted to the hospital and survived hospitalization (there does not appear to be any discounting used). Using the CPI-U for medical care, the Wittels estimate is $109,000 in year 2000$. This estimated cost is based on a medical cost model, which incorporated therapeutic options, projected outcomes and prices (using "knowledgeable cardiologists" as consultants). The model used medical data and medical decision algorithms to estimate the probabilities of certain events and/or medical procedures being used. The second estimate is from Russell et al. [56], which estimated first-year direct medical costs of treating nonfatal AMI of $15,540 (in 1995$), and $1,051 annually thereafter. Converting to year 2000$, that would be $23,000 for a 5-year period (without discounting).

The two estimates from these studies are substantially different, and the differences between the estimates have not been adequately resolved. Because the wage-related opportunity cost estimates from [54] cover a 5-year period, I will use estimates for medical costs that similarly cover a 5-year period. I will use a simple average of the two 5-year estimates, or $66,000, and add it to the 5-year opportunity cost estimate. The resulting range of total cost-of-illness for nonfatal AMI is from $66,000 to $141,000 depending on age.

The total avoided cost of illness by age group associated with the reductions in chronic bronchitis and nonfatal acute myocardial infarctions is provided in Table 7. The total avoided cost of illness associated with a nationwide one microgram reduction in ambient $PM_{2.5}$ exceeds $2 billion. Note that this does not include any direct avoided medical costs associated with premature mortality. Nor does it include any medical costs that occur more than 5 years from the

onset of a nonfatal AMI. As such, this is likely an underestimate of the true avoided costs of illness associated with a one microgram reduction in ambient $PM_{2.5}$.

In a traditional cost-effectiveness analysis, net costs of the intervention would be divided by the effectiveness measure to calculate a cost per life year or cost per QALY. For this illustrative analysis, there are no specific controls specified to achieve the one microgram reduction in ambient $PM_{2.5}$ and therefore no specific cost estimates. However, it is possible to calculate the costs that would be necessary for the cost-effectiveness of the reduction in ambient $PM_{2.5}$ to exceed various thresholds. Cost-effectiveness ratios are usually interpreted in a relative sense, as there is no universally agreed on cost-effectiveness cutoff for environmental health interventions. While the U.S. Public Health Service panel on cost-effectiveness did not recommend a cost-effectiveness threshold for generalized use, it may be useful to identify cost thresholds which would make controls to achieve reductions in ambient $PM_{2.5}$ cost-ineffective relative to other life-saving or quality of life improving interventions. The Harvard Cost Utility Analysis database suggests a median cost-utility ratio of $31,000 per QALY (2002$) for respiratory and cardiovascular interventions, while Tengs et al [58] report a median cost per life-year saved for live-saving interventions of $48,000 (1993$). The health economics literature often uses $50,000 per QALY as a de facto cutpoint with ratios less than $50,000 considered cost-effective. For the purposes of this analysis, I compute the costs necessary to exceed the $50,000 cost-effectiveness threshold, without endorsing $50,000 as an absolute threshold beyond which interventions should not be implemented. Decisions as to whether a specific control strategy is justified should be based on a complete comparison of benefits and costs.

Table 8 summarizes the effectiveness measures and avoided costs associated with the nationwide one microgram reduction in ambient $PM_{2.5}$ and presents the implicit cost per

microgram that would be necessary for the cost-effectiveness ratio to exceed the $50,000 threshold.

V. Sensitivity to Discount Rate

There are a large number of parameters and assumptions necessary in conducting a cost-effectiveness analysis. Where appropriate and supported by data, I have included distributions of parameter values which were used in generating the reported confidence intervals. Selection of a discount rate is an important assumption that is not easily characterized by a distribution. For this study, I felt it more appropriate to examine the impact of the assumption using a sensitivity analysis rather than through the integrated probabilistic uncertainty analysis. Another important assumption is that regarding whether air pollution primarily affects populations with disproportionate levels of preexisting heart and lung disease, however, due to the lengthy description supporting that sensitivity analysis it is included in the appendix to this article.

The choice of a discount rate, and its associated conceptual basis, is a topic of ongoing discussion within the academic community. In most cost-benefit analyses of air pollution regulations, a 3 percent discount rate has been adopted, reflecting reliance on a "social rate of time preference" discounting concept. This 3 percent discount rate is also consistent with the recommendations of the NAS panel on cost effectiveness analysis [2], which suggests that "a real annual (riskless) rate of 3% should be used in the Reference Case analysis." To examine the impact of the choice of discount rate, I have also calculated QALYs and the implicit cost thresholds using a 7 percent rate consistent with an "opportunity cost of capital" concept to reflect the time value of resources directed to meet regulatory requirements. This is the value recommended by OMB as the default for regulatory analysis. Further discussion of this topic appears in chapter 7 of [2].

Table 9 presents a summary of results using the 7 percent discount rate and the percent difference between the 7 percent results and the base case 3 percent results. More than doubling the discount rate from 3 to 7 percent decreases the estimated life years and QALY gained by reducing ambient $PM_{2.5}$. However, the reduction is not proportional to the discount rate. The estimated total MILY gained is reduced by 28 percent, while the implicit cost necessary to exceed the $50,000 cost-effectiveness threshold is reduced by 23 percent to $10 billion.

VI. Conclusions

I have calculated the effectiveness of a nationwide one microgram reduction in ambient $PM_{2.5}$ based on reductions in premature deaths and incidence of chronic disease. I measure effectiveness using several different metrics, including lives saved, life years saved, and QALYs (for improvements in quality of life due to reductions in incidence of chronic disease). I suggest a new metric for aggregating life years saved and improvements in quality of life, the MILY, which assumes that society assigns a weight of one to years of life extended regardless of preexisting disabilities or chronic health conditions.

Using the MILY metric, I estimate that air pollution regulations achieving ambient $PM_{2.5}$ reductions for less than $13 billion per microgram are likely to be cost-effective relative to other health interventions for cardiovascular and respiratory disease. In future years, this comparison will become more favorable, as populations increase and the population ages, increasing the susceptibility of the population to chronic disease impacts. Based on recent regulations proposed or promulgated by the U.S. EPA, including the Clean Air Interstate Rule and the Heavy Duty Engine and Nonroad Diesel Engine rules, costs per microgram of $PM_{2.5}$ reduced have ranged from $4 billion to $5 billion per microgram, indicating that these regulations are highly cost-effective in achieving public health improvements [16,20,59].

Cost-effectiveness analysis of environmental regulations which have substantial public health impacts may be informative in identifying programs which have achieved cost-effective reductions in health impacts and can suggest areas where additional controls may be justified. However, the overall efficiency of a regulatory action can only be judged through a complete cost-benefit analysis which takes into account all benefits and costs, including both health and non-health benefits.

VII. References

[1] Neumann, P. 2003. A Web-based Registry of Cost-utility Analyses. Risk in Perspective, 11(3).

[2] Gold, M.E., J. E. Siegel, L.B. Russell, and M.C. Weinstein, editors. 1996. Cost-effectiveness in Health and Medicine. New York: Oxford University Press.

[3] U.S. Food and Drug Administration. Federal Register 69FR9119, February 26.

[4] Hammitt, J.K. 2002. "QALYs versus WTP." *Risk Analysis* 22(5):985-1001.

[5] Bala, M.V. and G.A. Zarkin. 2000. Are QALYs an Appropriate Measure for Valuing Morbidity in Acute Diseases? Health Economics 9: 177-180.

[6] Freeman, A.M., J.K. Hammitt, P. De Civita. 2002. On Quality Adjusted Life Years (QALYs) and Environmental/Consumer Safety Valuation. AERE Newsletter, 22(1): 7-12.

[7] Johnson, F.R. 2004. When QALYs Don't Work: Measuring and Using General Time Equivalents. RTI Health Solutions.

[8] Cohen, J.T., J.K. Hammitt, and J.I. Levy. 2003. "Fuels for Urban Transit Buses: A Cost-effectiveness Analysis." *Environmental Science and Technology* 37:1477-1484.

[9] Coyle, D., D. Stieb, R.T. Burnett, P. DeCivita, D. Krewski, Y. Chen, and M.J. Thun. 2003. "Impact of Particulate Air Pollution on Quality Adjusted Life Expectancy in Canada." *Journal of Toxicology and Environmental Health, Part A* 66:1847-1863.

[10] Rabl, A. 2003. Interpretation of Air Pollution Mortality: Number of Deaths or Years of Life Lost? Journal of the Air and Waste Management Association. 53: 41-50.

[11] Carrothers, T. J., J.S. Evans, and J.D. Graham. 2002. The Lifesaving Benefits of Enhanced Air Quality. Harvard Center for Risk Analysis Working Paper, January 3.

[12] Murray, C.J.L., J.A. Salomon, C.D. Mathers and A.D. Lopez. 2002. Summary measures of population health : concepts, ethics, measurement and applications. Geneva, World Health Organization.

[13] de Hollander, A.E.M., J.M. Melse, E. Lebret, and P.G.N. Kramers. 1999. An Aggregate Public Health Indicator to Represent the Impact of Multiple Environmental Exposures. Epidemiology 10: 606-617.

[14] U.S. Office of Management and Budget. 2003. Circular A-4: Regulatory Analysis, September 17, 2003. Available at: http://www.whitehouse.gov/omb/circulars/a004/a-4.pdf

[15] Hubbell, B. 2005. Implementing QALYs in the Analysis of Air Pollution Regulations. Environmental and Resource Economics (forthcoming).

[16] U.S. Environmental Protection Agency, 2005. Regulatory Impact Analysis for the Clean Air Interstate Rule. EPA 452/-03-001. Prepared by Office of Air and Radiation. Available at: http://www.epa.gov/interstateairquality/tsd0175.pdf

[17] Miller, B.G. and J.F. Hurley. 2003. Life Table Methods for Quantitative Impact Assessments in Chronic Mortality. Journal of Epidemiology and Community Health. 57: 200-206.

[18] Abt Associates, 2003. BenMAP User Manual. Prepared for EPA Office of Air Quality Planning and Standards, Research Triangle Park, NC. Available at: http://www.epa.gov/ttn/ecas

[19] 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.

[20] U.S. Environmental Protection Agency, 2004. Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engines. EPA420-R-04-007. Prepared by Office of Air and Radiation. Available at http://www.epa.gov/nonroad-diesel/2004fr/420r04007.pdf

[21] Kunzli N., S. Medina, R. Kaiser, P. Quenel, F. Horak Jr, and M. Studnicka. 2001. Assessment of Deaths Attributable to Air Pollution: Should We Use Risk Estimates Based on Time Series or on Cohort Studies? *Am J Epidemiol* 153(11):1050-55.

[22] National Research Council (NRC). 2002. *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. Washington, DC: The National Academies Press.

[23] Pope, D.A., R.T. Burnett, M.J. Thun, E.E. Cale, D. Krewski, K. Ito, and G.D. Thurston. 2002. Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution. Journal of the American Medical Association, 287: 1132-1141.

[24] Centers for Disease Control. 2000. National Vital Statistics Reports, Vol. 49, No. 8.

[25] Centers for Disease Control. 2002. National Vital Statistics Reports, Vol. 51, No. 3.

[26] 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

[27] Brock, D.W. 2002. Chapter 14.3: Fairness and Health. In Murray, C.J.L., J.A. Salomon, C.D. Mathers and A.D. Lopez. Summary measures of population health: concepts, ethics, measurement and applications. Geneva, World Health Organization. 2002.

[28] Gold, M.R., D. Stevenson, and D.G. Fryback. 2002. HALYS and QALYS and DALYS,

Oh My: Similarities and Differences in Summary Measures of Population Health. Annual Review of Public Health 23:115–34.

[29] Sullivan, P.W., W.F. Lawrence, and V. Ghushchyan. 2005. A National Catalogue of Preference-Based Scores for Chronic Conditions in the U.S. Medical Care, 43(7): 736-749.

[30] Horsman, J., W. Furlong, D. Feeny, and G. Torrance. 2003. The Health Utilities Index (HUI): Concepts, Measurement Properties, and Applications. Health and Quality of Life Outcomes, 1:54.

[31] Kind, P. 1996. The EuroQoL Instrument: An Index of Health-Related Quality of Life. Quality of Life and Pharmacoeconomics in Clinical Trials, Second Edition, edited by B. Spilker. Lippincott-Raven Publishers: Philadelphia, PA, 1996, pp. 191-201.

[32] American Lung Association, 2002. *Trends in Chronic Bronchitis and Emphysema: Morbidity and Mortality*. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit.

[33] 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.

[34] American Lung Association. 2002. *Trends in Chronic Bronchitis and Emphysema: Morbidity and Mortality*. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit.

[35] U.S. Centers for Disease Control. National Center for Health Statistics. Health, United States, 2003. Table 30. Years of potential life lost before age 75 for selected causes of

death, according to sex, race, and Hispanic origin: United States, selected years 1980–2000Hyattsville, Maryland: 2003.

[36] Salomon, J.A., and C.J.L. Murray. 2003. A multi-method approach to measuring health-state valuations. Health Economics, Forthcoming (published online June 20, 2003).

[37] Bell CM, Chapman RH, Stone PW, Sandberg EA, Neumann PJ. 2001. An off-the-shelf help list: A comprehensive catalog of preference scores from published cost-utility analyses. *Med Decis Making* 2001;21:288-94.

[38] Harvard Center for Risk Analysis. 2005. Catalog of Preference Scores. Available at: http://www.hcra.harvard.edu/pdf/preferencescores.pdf

[39] Stouthard, M.E.A., J.L. Essink-Bot, G.J. Bonsel, J.J. Barendregt, P.G.N. Kramers, H.P.A. Van de Water, L.J. Gunning-Schepers. 1997. Disability Weights for Diseases in the Netherlands. Rotterdam: Department of Public Health, Erasmus University Rotterdam.

[40] Vos, T. 1999a. Final worksheet VM1 COPD.xls,prepared for Victoria Burden of Disease Study. Available at: http://www.dhs.vic.gov.au/phd/bod/daly.htm

[41] Gentile, J.E., 1998. Random Number Generation and Monte Carlo Methods, Springer Verlag.

[42] Vos, T. 1999b. Final worksheet VL2 IHD.xls, prepared for Victoria Burden of Disease Study. Available at: http://www.dhs.vic.gov.au/phd/bod/daly.htm

[43] Peters A., D.W. Dockery, J.E. Muller, and M.A. Mittleman. 2001. Increased Particulate Air Pollution and the Triggering of Myocardial Infarction. *Circulation* 103:2810-2815.

[44] Poloniecki J.D., R.W Atkinson., A.P de Leon., and H.R. Anderson. 1997. Daily Time Series for Cardiovascular Hospital Admissions and Previous Day's Air Pollution in London, UK. *Occup Environ Med* 54(8):535-40.

[45] Samet J.M., S.L. Zeger, F. Dominici, F. Curriero, I. Coursac, D.W. Dockery, J. Schwartz, and A. Zanobetti. June 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.

[46] Moolgavkar, S.H. 2000. Air Pollution and Hospital Admissions for Diseases of the Circulatory System in Three U.S. Metropolitan Areas. *J Air Waste Manag Assoc* 50:1199-206.

[47] Agency for Healthcare Research and Quality. 2000. HCUPnet, Healthcare Cost and Utilization Project.

[48] Cowie MR, Mosterd A, Wood DA, et al 1997. The epidemiology of heart failure. European Heart Journal 18:208-25.

[49] American Heart Association. Heart Disease and Stroke Statistics — 2003 Update. Dallas, Tex.: American Heart Association; 2003.

[50] Ganz, D.A., Kuntz, K.M., Jacobson, G.A., Avorn, J. 2000. Cost-Effectiveness of 3-Hydroxy-3-Methylglutaryl Coenzyme A Reductase Inhibitor Therapy in Older Patients with Myocardial Infarction. Annals of Internal Medicine. 132: 780-787.

[51] Stinnett, A.A., M.A. Mittleman, M.C. Weinstein, K.M. Kuntz, D.J. Cohen, L.W. Williams, P.A. Goldman, D.O. Staiger, M.G.M. Hunink, J. Tsevat, A.N.A. Tosteson, and L.Goldman. 1996. The Cost-effectiveness of Dietary and Parmacologic Therapy for Cholesterol Reduction in Adults. In Gold, M.E., J. E. Siegel, L.B. Russell, and M.C. Weinstein, editors. Cost-effectiveness in Health and Medicine. New York: Oxford University Press, 1996.

[52] Kuntz, K.M, J. Tsevant, L. Goldman, and M.C. Weinstein. 1996. Cost-effectiveness of

routine coronary angiography after acute myocardial infarction. Circulation. 1996 Sep 1;94(5):957-65.

[53] Pliskin, J.S., D.S. Shepard, and M.C. Weinstein. 1980. Utility Functions for Life Years and Health Status. Operations Research 28: 206-224.

[54] Cropper, M.L. and A.J. Krupnick. 1990. The Social Costs of Chronic Heart and Lung Disease. Resources for the Future. Washington, DC. Discussion Paper QE 89-16-REV.

[55] Wittels, E.H., J.W. Hay, and A.M. Gotto, Jr. 1990. Medical Costs of Coronary Artery Disease in the United States. *Am J Cardiol* 65(7):432-40.

[56] Russell, M.W., D.M. Huse, S. Drowns, E.C. Hamel, and S.C. Hartz. 1998. Direct Medical Costs of Coronary Artery Disease in the United States. Am J Cardiol. 81(9):1110-5.

[57] Harvard Center for Risk Analysis. 2005. Comprehensive Table of Cost-Utility Ratios 1976-2001. Available at http://www.hsph.harvard.edu/cearegistry/ (accessed August 8, 2005).

[58] Tengs, T.O., M.E. Adams, J.S. Pliskin, D.G. Safran, J.E. Siegel, M.C. Weinstein, and J.D. Graham. 1995. Five Hundred Life-Saving Interventions and Their Cost-Effectiveness. Risk Analysis 15: 369-390.

[59] US Environmental Protection Agency, 2000. *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.

[60] Goldberg, M.S., R.T. Burnett, J.C. Bailar III, R. Tamblyn, P. Ernst, K. Flegel, J. Brook, Y. Bonvalot, R. Singh, M. Valois, and R. Vincent. Identification of Persons with

Cardiorespiratory Conditions Who Are at Risk of Dying from the Acute Effects of Ambient Air Particles. Environmental Health Perspectives 109: 487-494.

[61] National Cancer Institute. 2001. SEER Cancer Statistics Review 1975-2000.

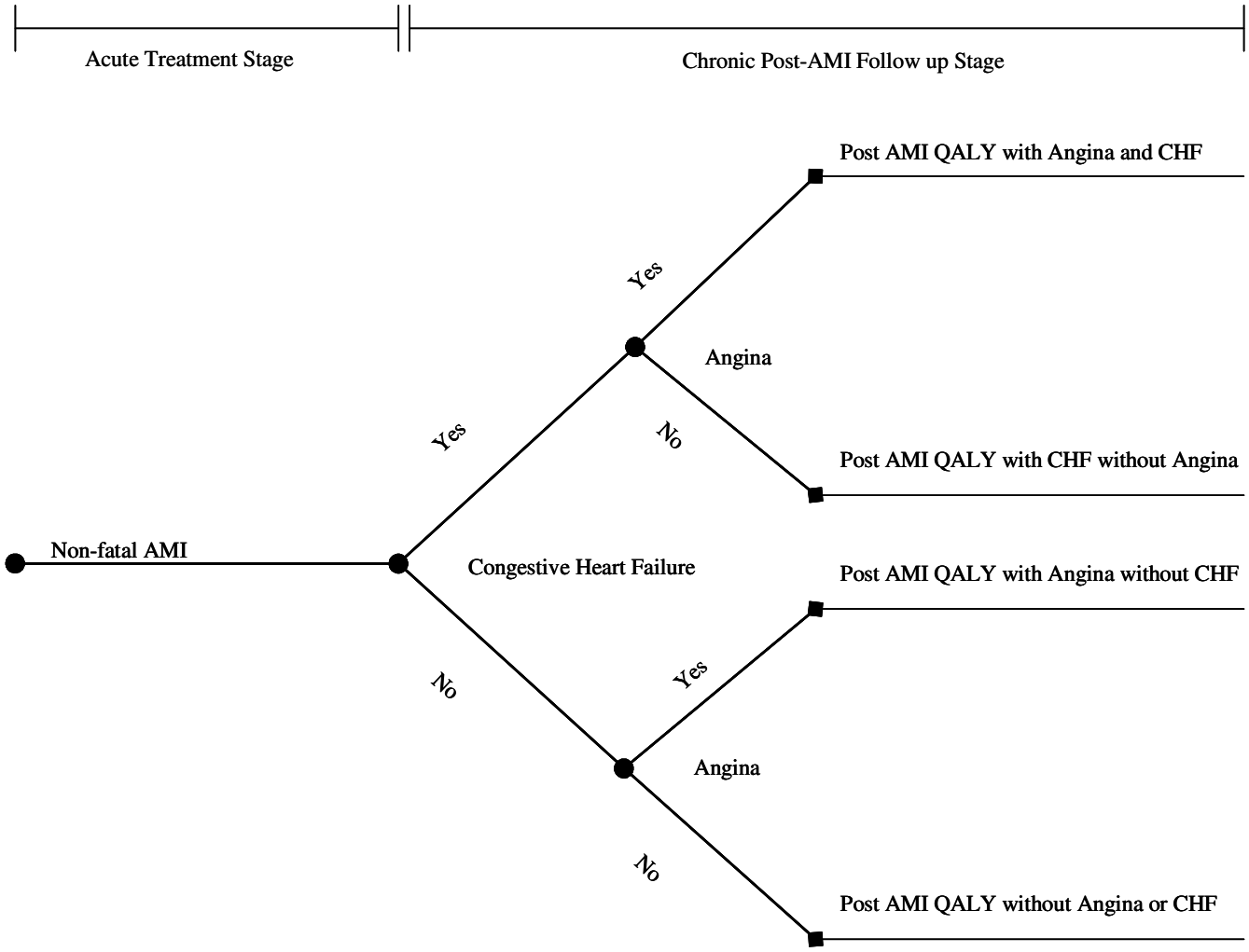

Figure 1. Decision Tree Used in Modeling Gains in QALY from Reduced Incidence of Nonfatal Acute Myocardial Infarctions

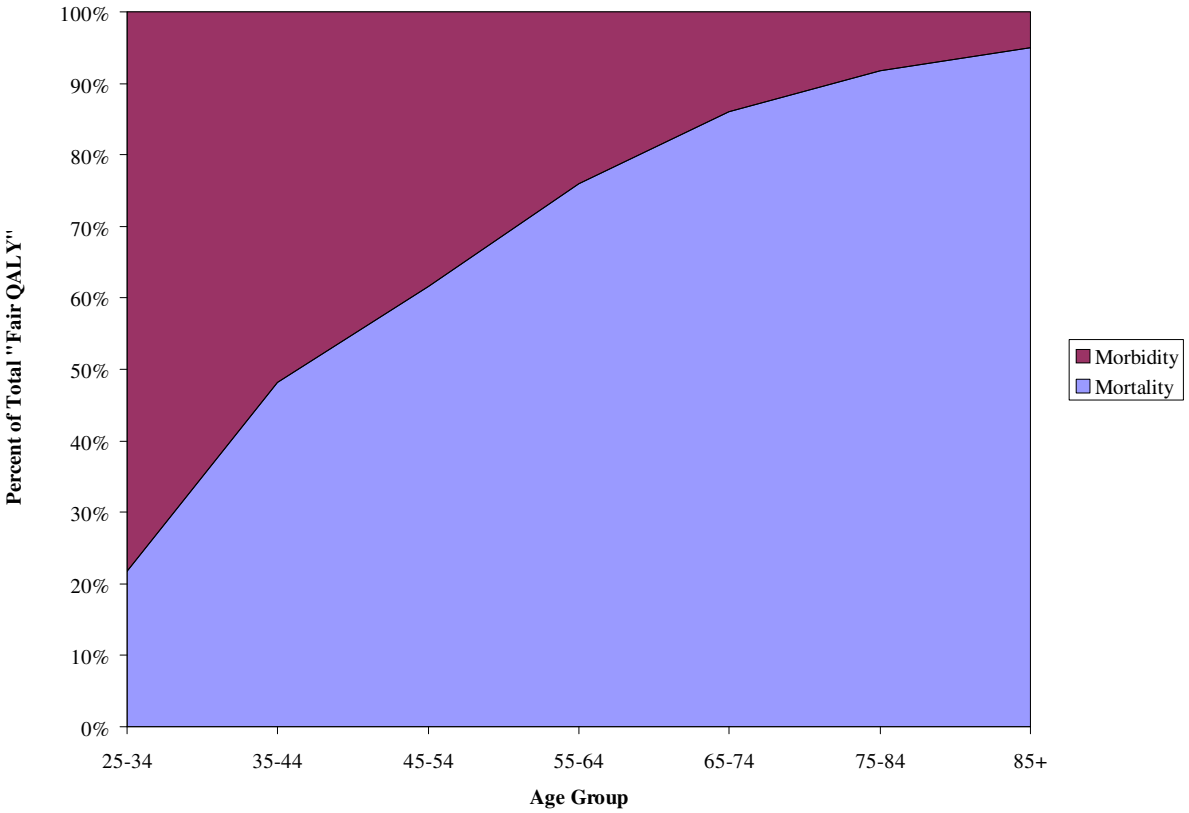

Figure 2.

Distribution of Mortality and Morbidity Related QALY Across Age Groups

(3% Discount Rate)

Table 1. Estimated Annual Reduction in Incidence of Premature Mortality, Chronic Bronchitis, and Non-fatal Acute Myocardial Infarctions Associated with a Nationwide 1 :g Reduction in Ambient $PM_{2.5}$*

Age Interval**	Reduction in All-Cause Premature Mortality (95% CI)	Reduction in Incidence of Chronic Bronchitis	Reduction in Non-fatal Acute Myocardial Infarctions
18 - 24	–	–	10
			(3 - 18)
25 - 34	140	2,000	100
	(50 - 230)	(55 - 3,800)	(25 - 180)
35 - 44	550	2,200	890
	(190 - 900)	(62 - 4,300)	(220 - 1,600)
45 - 54	950	1,800	2,500
	(320 - 1,600)	(51 - 3,600)	(630 - 4,400)
55 - 64	1,500	1,200	3,500
	(510 - 2,470)	(33 - 2,300)	(870 - 6,100)
65 - 74	2,700	880	4,500
	(910 - 4,400)	(25 - 1,700)	(1,100 - 7,800)
75 - 84	4,100	590	4,600
	(1,400 - 6,800)	(17 - 1,200)	(1,100 - 8,000)
85+	3,700	200	2,100
	(1,300 - 6,200)	(6 - 400)	(520 - 3,600)
Total	14,000	8,900	18,000
	(4,700 - 23,000)	(250 - 17,000)	(4,500 - 32,000)

* All estimtes rounded to two significant digits. Age group estimates will not sum to total due to rounding.
** Note that the lower bound age intervals do not exactly match between the different health effects. Premature mortality is estimated for ages 30 and over, chronic bronchitis for ages 27 and over, and non-fatal heart attacks for ages 18 and over.

Table 2. Abridged Life Table for the Total Population, United States, 2000

Age Interval		Probability of dying between ages x to x+1	Number surviving to age x	Number dying between ages x to x+1	Person years lived between ages x to x+1	Total number of person years lived above age x	Expectation of life at age x
Start Age	End Age	q_x	l_x	d_x	L_x	T_x	e_x
30	35	0.00577	97,696	564	487,130	4,723,539	48.3
35	45	0.01979	97,132	1,922	962,882	4,236,409	43.6
45	55	0.04303	95,210	4,097	934,026	3,273,527	34.4
55	65	0.09858	91,113	8,982	872,003	2,339,501	25.7
65	75	0.21779	82,131	17,887	740,927	1,467,498	17.9
75	85	0.45584	64,244	29,285	505,278	726,571	11.3
85	95	0.79256	34,959	27,707	196,269	221,293	6.3
95	100	0.75441	7,252	5,471	20,388	25,024	3.5
100+		1.00000	1,781	1,781	4,636	4,636	2.6

Table 3. Summary of Key Parameters Used in QALY Calculations for Chronic Disease Endpoints

Parameter	Value(s)	Source(s)
Discount rate	0.03 (0.07 sensitivity analysis)	Gold et al. (1996), U.S. EPA (2000), U.S. OMB (2003)
Quality of life preference score for chronic bronchitis	0.5 – 0.7	Triangular distribution centered at 0.7 with upper bound at 0.9 (Vos, 1999a) (slightly better than a mild/moderate case) and a lower bound at 0.5 (average weight for a severe case based on Vos [1999a] and Smith and Peske [1994])
Duration of acute phase of acute myocardial infarction (AMI)	5.5 days – 22 days	Uniform distribution with lower bound based on average length of stay for an AMI (AHRQ, 2000) and upper bound based on Vos (1999b).
Probability of CHF post AMI	0.2	Vos, 1999a (WHO Burden of Disease Study, based on Cowie et al., 1997)
Probability of angina post AMI	0.51	American Heart Association, 2003 (Calculated as the population with angina divided by the total
Quality of-life preference score for post-AMI with CHF (no angina)	0.80 – 0.89	Uniform distribution with lower bound at 0.80 (Stinnett et al., 1996) and upper bound at 0.89 (Kuntz et al., 1996). Both studies used the time trade-off elicitation method.
Quality of-life preference score for post-AMI with CHF and angina	0.76 – 0.85	Uniform distribution with lower bound at 0.76 (Stinnett et al., 1996, adjusted for severity) and upper bound at 0.85 (Kuntz et al., 1996). Both studies used the time trade-off elicitation method.
Quality-of-life preference score for post-AMI with angina (no CHF)	0.7 – 0.89	Uniform distribution with lower bound at 0.7, based on the standard gamble elicitation method (Pliskin, Stason, and Weinstein, 1981) and upper bound at 0.89, based on the time trade-off method (Kuntz et al., 1996).
Quality of-life preference score for post-AMI (no angina, no CHF)	0.93	Only one value available from the literature. Thus, no distribution is specified. Source of value is Kuntz et al. (1996).

Table 4. QALY Gained per Avoided Incidence of Chronic Bronchitis

Age Interval	QALY Gained per Incidence	
	Undiscounted	Discounted (3%)
25 - 34	12.15 (4.40-19.95)	6.52 (2.36-10.71)
35 - 44	9.91 (3.54-16.10)	5.94 (2.12-9.66)
45 - 54	7.49 (2.71-12.34)	5.03 (1.82-8.29)
55 - 64	5.36 (1.95-8.80)	4.03 (1.47-6.61)
65 - 74	3.40 (1.22-5.64)	2.84 (1.02-4.71)
75 - 84	2.15 (0.77-3.49)	1.92 (0.69-3.13)
85+	0.79 (0.27-1.29)	0.77 (0.26-1.25)

Table 5. QALY Gained per Avoided Non-Fatal Myocardial Infarction

Age Interval	Duration of Heart Failure for AMI with CHF[A]		Post-AMI Life Expectancy (non-CHF)		QALY Gained per Incidence[B]	
	Undiscounted	Discounted (3%)	Undiscounted	Discounted (3%)	Undiscounted	Discounted (3%)
18 - 24	7.11	6.51	55.5	27.68	4.18 (1.24-7.09)	2.17 (0.70-3.62)
25 - 34	6.98	6.40	46.1	25.54	3.48 (1.09-5.87)	2.00 (0.68-3.33)
35 - 44	6.49	6.00	36.8	22.76	2.81 (0.88-4.74)	1.79 (0.60-2.99)
45 - 54	5.31	4.99	27.9	19.28	2.14 (0.67-3.61)	1.52 (0.51-2.53)
55 - 64	1.96	1.93	19.8	15.21	1.49 (0.42-2.52)	1.16 (0.34-1.95)
65 - 74	1.71	1.69	12.8	10.82	0.97 (0.30-1.64)	0.83 (0.26-1.39)
75 - 84	1.52	1.50	7.4	6.75	0.59 (0.20-0.97)	0.54 (0.19-0.89)
85+	1.52	1.50	3.6	3.47	0.32 (0.13-0.50)	0.31 (0.13-0.49)

[A] The model assumes 20 percent of individuals with a non-fatal AMI develop congestive heart failure.

[B] Mean of Monte Carlo generated distribution. 95% confidence interval presented in parentheses.

Table 6. Estimated Gains in MILY Discounted at 3 Percent Associated with a Nationwide 1 :g Reduction in Ambient $PM_{2.5}$ [A]

Age	Life Years Gained from Mortality Risk Reductions (95% CI)	QALY Gained from Reductions in Chronic Bronchitis (95% CI)	QALY Gained from Reductions in Acute Myocardial Infarctions (95% CI)	Total Gain in MILY (95% CI)
18-24	-	-	22	22
			(3 - 48)	(3 - 48)
25-34	3,600	12,800	200	17,000
	(1,300 - 5,900)	(740 - 31,000)	(38 - 440)	(2,000 - 37,000)
35-44	13,600	13,000	1,600	28,000
	(4,800 - 22,000)	(720 - 32,000)	(290 - 3,500)	(5,100 - 57,000)
45-54	21,000	9,200	3,700	34,000
	(7,300 - 34,000)	(660 - 22,000)	(670 - 8,400)	(8,700 - 65,000)
55-64	27,000	4,700	3,900	36,000
	(10,000 - 44,000)	(230 - 11,000)	(680 - 8,800)	(11,000 - 64,000)
65-74	38,000	2,500	3,600	44,000
	(13,000 - 62,000)	(150 - 5,700)	(650 - 8,200)	(14,000 - 76,000)
75-84	40,000	1,100	2,400	44,000
	(14,000 - 66,000)	(46 - 2,700)	(450 - 5,400)	(15,000 - 74,000)
85+	15,000	160	620	15,000
	(5,000 - 24,000)	(3 - 370)	(130 - 1,300)	(5,400 - 26,000)
Total	160,000	44,000	16,000	220,000
	(56,000 - 260,000)	(2,500 - 100,000)	(2,900 - 36,000)	(61,000 - 400,000)

[A] Assumes a 3% discount rate, consistent with Gold et al. (1996). All estimates rounded to two significant digits. Age group estimates will not sum to total due to rounding.

Table 7. Avoided Costs of Illness Associated with Reductions in Chronic Bronchitis and Nonfatal Acute Myocardial Infarctions Associated with a Nationwide 1 :g Reduction in Ambient $PM_{2.5}$

	Avoided Cost of Illness in millions of 2000$ (95% confidence interval)[A]	
Age Range	Chronic Bronchitis	Nonfatal Acute Myocardial Infarction
18-24	NA	$0.7
		($0.1 - $1.9)
25-34	$301.4	$7.6
	($8.4 - $591.5)	($1.1 - $19.7)
35-44	$341.0	$66.7
	($9.5 - $669.3)	($9.7 - $173.5)
45-54	$181.2	$200.4
	($5.1 - $355.7)	($31.5 - $510.6)
55-64	$116.8	$499.1
	($3.3 - $229.3)	($121.0 - $1,070.7)
65-74	$9.8	$295.9
	($0.3 - $19.1)	($34.9 - $808.2)
75-84	$6.6	$304.3
	($0.2 - $12.9)	($36.0 - $831.1)
85+	$2.2	$137.6
	($0.1 - $4.4)	($16.3 - $375.8)
Total	$959.1	$1,512.3
	($26.8 - $1,882.2)	($250.7 - $3,791.5)

[A] Note that the confidence intervals for avoided costs of illness include both the uncertainty in the unit values for each health effect and the uncertainty in the estimated change in incidence for each health effect. Uncertainties are combined using Monte Carlo simulation methods.

Table 8. Summary of Results[A]

	Result Using 3% Discount Rate (95% Confidence Interval)
Life Years Gained from Mortality Risk Reductions	160,000
	(56,000 - 260,000)
QALY Gained from Reductions in Chronic Bronchitis	44,000
	(2,500 - 100,000)
QALY Gained from Reductions in Acute Myocardial Infarctions	16,000
	(2,900 - 36,000)
Total Gain in MILY	220,000
	(61,000 - 400,000)
Avoided Cost of Illness	
Chronic Bronchitis	$960 million
	($33 million - $1,900 million)
Nonfatal AMI	$1,500 million
	($250 million - $3,800 million)
Implied Cost Necessary to Exceed $50,000/QALY Threshold	$13 billion
	($9.4 billion - $18 billion)

[A] Consistent with recommendations of Gold et al (1996), all summary results are reported at a precision level of 2 significant digits to reflect limits in the precision of the underlying elements.

Table 9. Sensitivity Analysis: Impacts of Using a 7% Discount Rate

	Result Using 7% Discount Rate	% Change Relative to Result Using 7% discount rate
Life Years Gained from Mortality Risk Reductions	120,000	-25
QALY Gained from Reductions in Chronic Bronchitis	28,000	-36
QALY Gained from Reductions in Acute Myocardial Infarctions	13,000	-19
Total Gain in MILY	158,000	-28
Avoided Cost of Illness		
Chronic Bronchitis	$600 million	-38
Nonfatal AMI	$1,500 million	-3[A]
Implied Cost Necessary to Exceed $50,000/QALY Threshold	$10 billion	-23

[A] There is a 3 percent difference in estimated avoided costs of nonfatal AMI, however due to rounding, the reported cost for both 3 and 7 percent discount rates is $1,500 million.

Appendix A:

Sensitivity Analysis of Gains in Life Years in Individuals with Pre-existing Health Conditions

There is evidence that, at least for some of the mortality risks associated with short term exposure to elevated levels of air pollution, the susceptible population is comprised of individuals with chronic diseases [60]. To explore the potential impact of assumptions regarding preexisting health conditions in those at risk of death due to air pollution, we also estimate life years for separate causes of death (lung cancer, cardiopulmonary, and other causes), with assumptions about the health status prior to death. Note that using these disease conditional life expectancies assumes that no individuals contracted chronic illnesses due to air pollution. Air pollution would be assumed to only affect the risk of death for individuals who contracted chronic illness from other causes.

For lung cancer deaths, we develop conditional life expectancies using ten-year survival probabilities. Based on data obtained from the National Cancer Institute SEER Cancer Statistics Review 1975-2000 [61], we calculated the average life expectancy during the 10 years following onset of lung cancer:

$$Avg \;\; Life \;\; Expectancy = \sum_{i=1}^{10} p_i i \,, \qquad (4)$$

where p_i= probability of death in year i. For example, the probability of dying in the first year after onset of lung cancer is about 0.6. Thus, in a cohort of new lung cancer cases, 60 percent

will have a life expectancy of only one year. For individuals dying within 10 years of onset, the average life expectancy for a cohort of cancer cases is about 1.6 years. However, at the end of 10 years, about 10 percent of cases will have survived, and may be assumed at this point to have the normal life expectancy for the general population. So, average life expectancy for individuals with lung cancer is the weighted average of the 10 year life expectancy and the life expectancy for those surviving past 10 years. Again, these life expectancies will only indicate the life years lost for those dying from air pollution who already have lung cancer from other causes (e.g. smoking). However, note that the C-R function for lung cancer mortality in [23] controlled for smoking, so that the excess risk of lung cancer death associated with air pollution should be independent of that due to smoking. In fact, the relative risk of lung cancer death from $PM_{2.5}$ was higher for nonsmokers than for smokers. Table A-1 presents the conditional life expectancies with lung cancer.

For cardiopulmonary deaths, I develop conditional life expectancies based on an assumption that all individuals dying from cardiopulmonary causes related to air pollution had a preexisting diagnosis of coronary heart disease (CHD). This is clearly an overstatement but is useful for the sensitivity analysis. Carrothers, Evans, and Graham [11] report life expectancies for individuals with diagnosed CHD relative to the general population life expectancies at different ages. The reported life expectancies cover ages 40 through 85. In order to obtain conditional life expectancies for ages 30 to 40 and 85 and over, I ran a simple regression analysis predicting life expectancy with CHD as a function of age, with the intercept restricted to zero. The ordinary least squares regression with intercept constrained to zero has an R2 of 0.73, which indicates a reasonable model fit. Including a non-zero intercept results in a better fit to the observed data, but the intercept is around -2, which implies that when general population life

expectancy falls below 2 years, the life expectancy with CHD is negative. In order to constrain the life expectancy to be greater than zero, we chose to use the no intercept model. The estimated relationship is

$LE_{CHD} = 0.5878 \times LE_{General}$, where LE_{CHD} is conditional life expectancy with CHD and $LE_{General}$ is life expectancy in the general population. Table A-1 presents the conditional life expectancies with CHD. For the sensitivity analysis, these conditional life expectancies are used to represent the life expectancy of all individuals dying from cardiopulmonary causes related to air pollution.

For the sensitivity analysis, we assume that mortality from any other non-lung cancer and non-cardiopulmonary cause of death results in a loss of life expectancy equal to that of the general population. Total life years gained is calculated as the sum of life years gained for each of the three cause of death categories.

$$Total\ Life\ Years = \sum_{i=1}^{N} \sum_{j=1}^{J} LE_{ij} \times M_{ij} \quad (5)$$

where j indexes the cause of death category and J is the number of categories.

Results of the sensitivity analysis are presented in Table A-2. Assuming that individuals have preexisting health conditions reduces the estimated undiscounted life years gained by around 40 percent and discounted life years gained by around 35 percent. Cardiopulmonary deaths account for the majority of premature deaths avoided (75 percent) and life years gained (63 to 69 percent depending on whether life years are undiscounted or discounted). Lung cancer accounts for around 15 percent of premature deaths but only 5 to 7 percent of life years gained,

due to the relatively short life expectancy with diagnosed lung cancer (less than 6 years in most cases).

The implicit costs necessary to exceed the $50,000 cost-effectiveness threshold fall by 15 percent to $11 billion when preexisting diseases are assumed. This suggests that cost-effectiveness of ambient $PM_{2.5}$ reductions is not heavily influenced by assumptions regarding preexisting health status of those dying prematurely from $PM_{2.5}$ exposure.

Table A-1. Conditional Life Expectancies for Individuals with Lung Cancer and Diagnosed Coronary Heart Disease (CHD)

Age Interval	Life Expectancy with Lung Cancer at Age x	Life Expectancy with Diagnosed CHD at Age x
30 - 34	6.20	28.4
35 - 44	5.76	25.6
45 - 54	4.88	20.2
55 - 64	4.06	15.1
65 - 74	3.31	10.5
75 - 84	2.69	6.6
85 - 94	2.21	3.7
95 - 99	1.95	2.1
100+	1.86	1.5

Table A-2. Sensitivity Analysis: Life Years Gained from Reductions in Premature Mortality Assuming Preexisting Lung Cancer and Cardiopulmonary Disease.

	Age Interval	Undiscounted Life Years	Discounted Life Years (3%)
Lung Cancer	30-34	10	9
	35-44	209	195
	45-54	832	786
	55-64	1,719	1,644
	65-74	2,342	2,264
	75-84	1,581	1,543
	85+	351	345
	Total Lung Cancer	7,044	6,786
Cardiopulmonary	30-34	640	440
	35-44	4,301	3,061
	45-54	9,232	7,053
	55-64	13,239	10,837
	65-74	19,241	16,782
	75-84	22,197	20,453
	85+	13,234	12,721
	Total Cardiopulmonary	82,085	71,347
Other Causes	30-34	5,556	3,002
	35-44	14,879	8,487
	45-54	11,044	7,035
	55-64	4,859	3,455
	65-74	2,535	1,998
	75-84	1,946	1,679
	85+	-	-
	Total Other Causes	40,819	25,656
	Grand Total (Lung Cancer + Cardiopulmonary+Other Causes)	129,948	103,789