Air Pollution Health Effects: Toward an Integrated Assessment

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Abstract

Scientists and policy makers have become increasingly aware of the need to jointly study climate change and air pollution because of the interactions among policy measures and in the atmospheric chemistry that creates the constituents of smog and affects the lifetimes of important greenhouse gases such as methane. Tropospheric ozone and aerosols, recognized constituents of air pollution, have important effects on the radiative balance of the atmosphere. Existing methods for estimating the economic implications of environmental damage do not provide an immediate approach to assess the economic and policy interactions. Towards that end, we develop a methodology for integrating the health effects from exposure to air pollution into the MIT Emissions Prediction and Policy Analysis (EPPA) model, a computable general equilibrium economic model of the economy that has been widely used to study climate change policy. The approach incorporates market and non-market effects of air pollution on human health, and is readily applicable to other environmental damages including those from climate change. The estimate of economic damages depends, of course, on the validity of the underlying epidemiological relationships and direct estimates of the consequences of health effects such as lost work and non-work time and increased medical expenses. We apply the model to the US for the historical period 1970 to 2000, and reevaluate estimates of the benefits of US air pollution regulations originally made by the US Environmental Protection Agency. We also estimate the economic burden of uncontrolled levels of air pollution over that period. Our estimated benefits of regulation are somewhat lower than the original EPA estimates, and we trace that result to our development of a stock model of pollutant exposure that predicts that the benefits from reduced chronic air pollution exposure will only be gradually realized. As modeled, only population cohorts born under lower air pollution levels fully realize the benefits. While other assumptions about the nature of health effects of chronic exposure are possible, some version of a stock model of this type is needed to accurately estimate the timing of benefits of reduced pollution.

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Introduction

Scientists and policy makers have become increasingly aware of the need to jointly study climate change and air pollution because of the interactions among policy measures and in the atmospheric chemistry that creates the constituents of smog and affect the lifetimes of important greenhouse gases such as methane. Tropospheric ozone and aerosols, recognized constituents of air pollution, have important effects on the radiative balance of the atmosphere. Existing methods for estimating the economic implications of environmental damage do not provide an immediate approach to assess the economic and policy interactions. Most economic analyses of environmental damages are aimed solely at valuation, often using current values of critical economic data such as wages or medical expenses.

Integrated assessment seeks to understand the feedbacks and interactions among complex systems. For integrated assessment of global environmental change we are interested in impacts in different regions of the world and over long time horizons. Estimates of economic impact of environmental damage, where the value of key economic variables often are drawn mostly from a few countries circa the 1990's, are unlikely to be constant over time or across regions. These values may be difficult to predict with accuracy but models that estimate mitigation costs have not shied away from making estimates. In then comparing an estimate of the benefits of avoided environmental damage with the cost of mitigation one would like to use similar assumptions about key economic variables on both the benefit and cost side of the equation. A reason for integrating these effects is thus simply a consistency of valuing them with mitigation costs.

The ultimate goal is a fully integrated model of anthropogenic emissions and mitigation costs, the relevant earth system responses to these forcings, and the feedback on the economy of environmental effects with potential implications for economic activity and emissions. Thus, we are concerned not just with the valuation of impacts, but on how climate or air pollution affect the economy, and thus potentially the emissions of pollutants. As a first step toward that end, we develop a methodology for integrating the health effects from exposure to air pollution into the

MIT Emissions Prediction and Policy Analysis (EPPA) model, a computable general equilibrium economic model of the economy that has been widely used to study climate change policy (Babiker, et al., 2001; Paltsev, et al., 2003, 2004). In that regard, the EPPA model is representative of a large number of economic models that provide a detailed representation of economic activity that contributes to emissions of polluting substances. We are focused here on the largely neglected part of the problem: how to provide an equally detailed and consistent representation of the economic impact of environmental damage within such a modeling framework. To identify this new version of the model, we refer to it as EPPA-HE (EPPA-Health Effects).

The approach we develop incorporates market and non-market effects of air pollution on human health, and is readily applicable to other environmental damages including those from climate change. We begin with the basic data that supports CGE models, the Social Accounting Matrix (SAM) that includes the input-output tables of an economy, the use and supply of factors, and the disposition of goods in final consumption. We identify where environmental damage appears in these accounts, estimate the physical loss, and value the loss within this accounting structure.

Our approach is first and foremost an exercise in environmental accounting, augmenting the standard national income and product accounts to include environmental damage. Our estimate of economic damages stemming from the health effects of urban air pollution depends, of course, on the validity of the underlying epidemiological relationships and direct estimates of the consequences of these health endpoints such as lost work and non-work time, and increased medical expenses. For this purpose we have used estimated relationships drawn from a large body of work on the epidemiological effects of air pollution and economic valuation of them. We make no claim of creating better estimates of these relationships that in the end are crucial to any economic analysis. Our contribution is to introduce these relationships in a dynamic economic model so that economic valuation of damage over time is consistent with the projected economy.

We apply the model to the US for the historical period 1970 to 2000. To do this, we simulate the economy with air pollution damages we estimate to have occurred because of the existing level of air pollution during that period. This is an effort in benchmarking the economic model so that the macroeconomic performance of the economy matches the actual historical performance. Once we have the model benchmarked in this manner, we are able to then re-simulate it over the period (or into the future) with other levels of air pollution.

We evaluate estimates of the benefits of US air pollution regulations in the US and compare them to a set of benefit estimates originally made by the US Environmental Protection Agency

(US EPA, 1989, 1999). For this purpose, we use the counterfactual level of air pollution (what it would have been without regulation) estimated by the US EPA in their study. This allows us to focus more specifically on how our endogenous valuation approach compares with the more traditional method used by the US EPA. We also estimate the economic burden of uncontrolled levels of air pollution over that period. Here we simulate the counterfactual case of what the economy would have been like if pollution levels had been at their background or 'natural' levels, without any contribution from human activity. This, we argue, is the environmental accounting exercise—comparing the actual economic performance over the period to what it might have been without the high and changing levels of air pollution.

We begin with a description of the EPPA-HE model, identifying the additions we made to the standard EPPA. We next turn to the problem of developing the basic data needed for the model. We then provide the estimates of benefit and burden of air pollution in the US from 1970-2000. We finally offer some conclusions.

MIT EPPA-HE

The MIT EPPA-HE model is built on the standard EPPA 4 model extended to include health effects. The EPPA model is a recursive-dynamic multi-regional general equilibrium model of the world economy, which is built on the GTAP dataset (Hertel, 1997; Dimaranan and McDougall, 2002) and additional data for greenhouse gas (CO_2, CH_4, N_2O, HFCs, PFCs and SF_6) and urban gas emissions (Mayer, et al., 2000). The version of EPPA used here (EPPA 4) has been updated in a number of ways from the model described in Babiker et al. (2001). Most of the updates are presented in Paltsev et al. (2003, 2004). The various versions of the EPPA model have been used in a wide variety of policy applications (e.g., Jacoby et al., 1997; Jacoby and Sue Wing, 1999; Reilly et al., 1999; Paltsev et al., 2003). EPPA 4 includes (1) greater regional and sectoral disaggregation, (2) the addition of new advanced technology options, (3) updating of the base data to the GTAP 5 data set (Dimaranan and McDougall, 2002) including newly updated input-output tables for Japan, the US, and the EU countries and rebasing of the data to 1997, and (4) a general revision of projected economic growth and inventories of non-CO_2 greenhouse gases and urban pollutants (Table 1).

The base year for the EPPA 4 model is 1997. From 2000 onward, it is solved recursively at 5-year intervals. All production sectors and final consumption are modeled using nested Constant Elasticity of Substitution (CES) production functions (or Cobb-Douglas and Leontief forms, which are special cases of the CES). The model is written in the GAMS software system and solved using the MPSGE modeling language.

Extending the model to included health effects involves valuation of non-wage time (leisure) and inclusion of a household production of health services, which we represent in a simplified diagram of a Social Accounting Matrix (SAM) as shown in Figure 1. The extensions of model are highlighted in italic bold. This simplified SAM ignores government, investment, and exports and imports as they are not directly affected by the extensions for EPPA-HE (but are part of the model, and are indirectly affected in simulations). The basic SAM includes the inter-industry flows (input-output tables) of intermediate goods and services among industries, delivery of goods and services to final consumption, and the use of factors (capital, labor and resources) in production. EPPA 4 contains a household production sector for personal transportation that delivers transportation services to final consumption (Paltsev, et al, 2004).

For EPPA-HE we add a household production sector that provides a 'pollution health service' to final consumption to capture economic effects of morbidity and mortality from acute exposure. This household production sector is shown as 'household mitigation of pollution health effects.' It uses 'health services' (i.e. hospital care and physician services) from the SERV sector of EPPA and household labor to produce a health service. The household labor is drawn from labor and leisure and thus reduces the amount available for other uses; i.e. an illness results in purchase of medical services and/or patient time to recover when they cannot work or participate in other household activities. We use data from traditional valuation work to estimate the amount of each of these inputs for each health endpoint as discussed in the following sections. Changed pollution levels are modeled as a Hick's neutral technical change: higher pollution levels requires proportionally more of all inputs to deliver the same level of health service, or lower levels require proportionally less.[2] Figure 2 shows the household production structure with the added components for EPPA-HE in bold italics. The key new additions are (1) leisure as a component of consumption and (2) the Household Healthcare (HH) sector that includes separate production relationships for health effects of each pollutant. The elasticity, σ_L, is parameterized to represent a labor own-price supply elasticity typical of the literature, as discussed in more detail later. The HH sector is Leontief in relationship to other goods and services and among pollutant health endpoints. Mortality effects simply result in a loss of labor and leisure, and thus are equivalent to a negative labor productivity shock.

[2] Modeled here as a negative technical change, greater expenditure *due to more pollution* draws resources from other uses and thus reduces consumption of other goods and leisure—more pollution is thus bad. The increased expenditures combat the pollution effects, and do not increase consumption and welfare. Of course, greater expenditure for *a fixed level of pollution* will generate more health benefits.

Data and Stock-Flow Accounting

Impacts on health are usually estimated to be the largest air pollution effects when measured in economic terms using conventional valuation approaches, dominating other losses such as damage to physical infrastructure, crops, ecosystems and loss of visibility (e.g. US Environmental Protection Agency, 1999). The health effects of air pollution present themselves as both a loss of current well-being (an illness brought on by acute exposure to air pollution that results in temporary hospitalization or restricted activity) and as an effect that lasts through many periods (years of exposure that eventually lead to illness, and deaths where losses to society and the economy extend from the point of premature death forward until that person would have died of other causes had they not been exposed to pollution.) Thus, we are faced with accounting both for stocks and flows. Health effects also present themselves as both market and non-market effects. Death or illness of someone in the labor force means that person's income is no longer part of the economy, clearly a market effect. Illness also often involves expenditure on medical services, counted as part of the market economy. Death and illness also involve loss of non-paid work time, a non-market impact. This likely involves a loss of time for household chores or a loss of time spent on leisure activities. The health effects area thus is both a large component of total air pollution damages and provides an opportunity to develop methods to handle a variety of issues faced in valuing changes in environmental conditions.[3]

Epidemiological Relationships

Epidemiological relationships have been estimated for many pollutants, as they relate to a variety of health impacts. The work has been focused on a set of substances often referred to as 'criteria pollutants,' so-called because the U.S. EPA developed health-based *criteria* as the basis for setting permissible levels. These same pollutants are regulated in many countries. Tables 2 and 3 are adapted from the Holland et al. (1998) in an extensive study for the European Commisssion. The reported relationships summarize the known health effects of exposure to these pollutants, building on a data compilation originally started in the US. Table 2 contains relationships estimated for a general healthy population, and reflects the fact that some of the relationships differ for children or the elderly as compared with the general adult population. Table 3 contains estimated relationships for the population of asthmatics, a group that is more vulnerable to air pollution. Holland et al. (1998) also include a set of estimates for effects they considered less certain. They report studies that have found a statistically significant relationship

[3] Health effects raise other issues as well, such as non-use value, and interdependency of welfare among individuals, that we do not attempt to address here.

but there is not, in the view of the experts consulted in the Holland et al. (1998) study, general agreement that there is a significant relationship. We did not include these, but Yang (2004) conducted a sensitivity analysis where he included them. He found these could be quite important, doubling estimates of the damage. Much of this comes from a suspected relationship between elevated CO and mortality.

All of the relationships including those in Tables 2 and 3 are, of course, subject to uncertainty as to the magnitude of the relationship. The relationships reported in these tables are linear, but there remains considerable debate about whether the relationships may be non-linear in some way. One aspect of this is whether there is a threshold below which pollution has no effect. Another is whether the effects are independent as these simple relationships imply, or instead whether exposure to multiple pollutants might be more or less harmful than the sum of each independent effect. There is not strong evidence supporting a particularly non-linear relationship, although this should be probably understood as just that: absence of evidence for non-linearity rather than evidence that the relationship is linear. An aspect of these estimated relationships is that they cover a broad population. Any relationship thus reflects to some degree both individual response to varying dose levels and varying vulnerability within the population.

The effects in Tables 2 and 3 range from hospital admissions due to respiratory problems and restricted activity days (the normal activities of individuals are impaired but no medical care is required) to death due to acute or chronic exposure. The pollutants include tropospheric ozone (O_3), nitrates, SO_2, CO, and particulate matter (PM 10, PM 2.5). The Holland, et al. (1998) study does not identify PM as among the highly uncertain relationships, but subsequent to their review controversy developed around the relationship of mortality and chronic exposure to PM. An earlier study by Pope et al. (1995) cited in the Holland et al. (1998) review was found to suffer from an error introduced by statistical package used to produce the estimates. We have included in Table 2 a revised study (Pope et al., 2002) that corrected that error.

The PM relationship has been the subject of contentious debate in the United States as the US EPA moved to strengthen regulations governing fine particulates. Particulate matter, unlike other substances such as CO or O_3, is not a chemically well-defined substance. It is dust or soot, and is variously composed of organic carbon, black or elemental carbon, and other materials such as sulfur or nitrogen compounds and heavy metals. Thus, while the widely used work by Pope and colleagues finds a relationship between chronic exposure to PM and death rates, particular constituents of PM may be the real culprit. In any case, whereas a pollutant such as carbon monoxide is clearly toxic at high enough levels and has measurable physiological effects at lower levels, clearly establishing the physiological effects of PM on the human body has been more

difficult. Since the composition of PM can vary widely, a statistical relationship estimated across different locations with different PM composition may then not hold if one changes the level of PM in a particular location if one changes PM levels or if one tries to use the relationship for other locations not in the original sample.

We have not tried in any way to resolve these uncertainties in the epidemiological relationships, but simply use the set reported in Tables 2 and 3, noting that this the basis for evaluation of air pollution benefits in Europe and similar assessments by the US EPA draw on these same studies. We separate effects by pollutant and the mortality effects of exposure to PM, to help understand which uncertainties are potentially important for the results.

Accounting for Health Effects in the SAM

The next step is to turn these health endpoints into units relevant to our economic model. An economy's SAM, constructed from national income and product accounts and input-output tables, is the base data for a computable general equilibrium model such as EPPA. The data in these tables are interpreted as physical quantities of the goods or factors in the economy. As economic aggregates, however, they must be reported in common units, and currency units (i.e. US dollars) are used in these aggregations. For example, national economic accounting values labor contributions at the wage rate. Thus, the labor force contribution of a high-wage individual working 40 hours per week will be a bigger than a low-wage individual working the same number of hours. Similarly, agricultural output or output of the steel industry is simply the total value of sales of the industry rather than in tons of output. This weights products by their value rather than tonnage or some other unit that would obviously make comparison of computer chips and cement, or haircuts and surgery problematic.

In a similar way, we make use of the traditional economic valuation literature to interpret the components of value as a measure of the quantity of labor or leisure lost, or of the quantity of medical services required to treat the health effect. Often this literature constructs the valuation estimates in exactly this manner, identifying a hospitalization day as the medical service and then valuing it at the average cost of a day in the hospital to treat the endpoint, or identifying lost work time, and valuing it at the average wage rate. Other valuation estimates have tried to estimate the total value of the health endpoint including 'non-market' effects. These are based on methods such as contingent value surveys, asking people their willingness to pay to avoid the health endpoint. Normally, one would expect this to include market effects (lost wages or expenditures on health care) plus some valuation of the non-market effects of illness—pain and suffering and

associated loss of enjoyment or attention to household activities because of the illness. We have exploited the components of these valuation estimates: costs related to hospital costs we treat as a demand for medical services, lost work time we treat as a reduction in the labor force (in dollar equivalents), and damages beyond these market effects we treat as a loss of leisure.

Valuation estimates we use are also from the Holland et al. (1998) survey of the literature, and the estimates, converted to US dollars are shown Table 4. For each endpoint related to each pollutant (e.g. respiratory hospital visit due to exposure to ozone), we allocated a share of the total cost to demand for medical service, lost labor, or lost leisure. Not all pollutants are associated with all endpoints, but we end up with 50 separate combinations. Broadly, the allocations for morbidity endpoints are 50 to 85% for the costs of medical services, 10 to 15% for lost leisure, and the remaining for lost labor. That is, the bulk of morbidity costs are market costs. See Yang (2004) for the complete list, and allocation for each combination. We assume mortality is only lost labor and leisure, the proportion depending on the age at death, and our accounting of leisure time for those in the work force. We discuss the approach for representing these costs in the SAM, and for inclusion of leisure time in greater detail in the following sections.

Leisure

The two critical questions regarding leisure are: (1) how much, and (2) what is its value? These are intertwined as the relevant quantity for CGE modeling is a total endowment in value terms. How much non-work time to explicitly account is somewhat arbitrary. In much traditional CGE work that includes non-work time, the goal is to represent a labor supply response. An intuitive basis for an expanded accounting of non-work time in that case is an estimate of the maximum potential labor force one could imagine for a given population. Babiker, Metcalf, and Reilly (2003) assumed an additional potential labor force of 20%. The estimate is arbitrary to a large degree because the 'known' parameter is the own-price supply elasticity of labor (ε), and it with the initial non-working share (α) of the labor force one can determine elasticity of substitution between labor and leisure (σ), the critical CGE model variable, via the following relationship:

$$\varepsilon = \frac{\alpha}{1-\alpha}\sigma \qquad (1)$$

For a given estimate of ε which we take to be representative of the econometric literature studying price responsiveness of labor supply, a higher estimated α, will simply lead to a lower benchmark value of σ. If benchmarked in this way, to a first order the supply of labor in response to a change in wages will be the same regardless of the potential labor force estimate. Here, we are interested in accounting for loss of labor and leisure time, not only of the existing and potential workforce, but also of children and elderly who are not part of the workforce. We thus estimate non-work time to include all waking non-work time of the current workforce and of children and elderly. We assume the workforce values its leisure at the margin at the wage rate, however, we note (Figure 3) that the wage profile for the US rises with age, peaking in the 50-54 age group, and then falls. Based on this wage profile we value loss of children's time at 1/3 the average adult wage rate, and the loss of the elderly's time at 2/3 that of the average adult wage. Aggregating the value of time of children, elderly, non-working, and the non-work time of those in the labor force, we estimate α at 0.55, and based on central estimates the current labor price elasticity of 0.25, we arrive at a value of σ=0.2 as shown in Figure 2. [4]

Mortality and Chronic Exposure

Air pollution deaths may result from exposure to high levels of pollution experienced during a particularly bad air pollution event (acute exposure), or from exposure over many years from low levels of pollutants (chronic exposure). Death from acute exposure normally only affects those that are close to death from other causes and the commonly accepted loss of time is 0.25 to 0.5 years (Pope et al., 1995, 2002; Holland et al., 1998). We assumed the loss was 0.5 years, and for our purposes this loss can be treated purely as a loss in the current period—a flow accounting of less labor in that period. Deaths due to chronic exposure require more complex accounting. The nature of the epidemiological results is that a reduction in exposure to a given concentration level of pollution should be interpreted as a reduction by that level each year over the lifetime of the individual, i.e. a proportional reduction in cumulative exposure. Since we have a model that we wish to simulate through time, with different levels of the pollutant in each period, we need to (1) explicitly calculate the cumulative exposure over time and how the annual average cumulative exposure is changed because of each year's change in concentrations, and (2) track the change in deaths as they occur over time. The chronic exposure deaths are from PM.

[4] It is not essential that we value all waking non-work time. We could instead have created an estimate of the maximum potential loss from air pollution damages, but the intuition is clearer if we simply include all non-work time. It also automatically facilitates a further expansion of the accounting of non-work time for other household uses or damages.

For these purposes, we construct a simple age cohort population model. Mean annual cumulative exposure of cohort n at time t, $\overline{C}_{t,n}$, is the sum of average annual exposure from the birth year, a_n, of the cohort.

$$\overline{C}_{t,n} = \sum_{i=a_n}^{t} \frac{c_i}{t - a_n}, \text{ n=1,…,8} \qquad (2)$$

Cohort age groups are: 1-4, 5-14, 15-29, 30-44, 45-59, 60-69, 70-79, and 80+. The specific formulation is used to be consistent with the underlying epidemiological relationships, as in Pope et al. (2002), that relate the percentage increase in the probability of death (%Δ pr(d)) to mean annual exposure:

$$\%\Delta pr(d) = ERfct * \overline{C} \qquad (3)$$

where ERfct is the variable as defined in Table 2. And note that mean C is not defined by cohort and is simply the average over the entire time period in these studies. Chronic exposure deaths are assumed in this literature to occur only to those over 30, even though exposure accumulates from birth as in (2). The epidemiological work does not further resolve the age distribution of death. We were concerned, however, that ERfct may vary with age cohort. Since the estimated change is the increase in the probability of death from all causes, the predicted increase due to PM will depend on the death rate from all causes for each age group. Deaths due to causes such as accidents, crime, childbirth, or infectious diseases, for example, are likely unrelated to PM exposure. Instead we expect deaths from chronic exposure to PM to be from causes like cardiopulmonary disease or disease of the lungs such as emphysema or cancer because such deaths might occur as a result of breathing PM over many years. We thus make the ERfct age-cohort specific by conditioning it on the age distribution of deaths due to cardiopulmonary and lung diseases (cpl) relative to all deaths:

$$ERfct_n = ERfct_T * \frac{\Pr(d : cpl)_n \big/ \Pr(d)_n}{\Pr(d : cpl)_T \big/ \Pr(d)_T} \qquad (4)$$

Here Pr (d: cpl) and Pr(d) are, respectively, the annual probability of death from cpl and from all causes, and the n and T subscripts are, respectively, for cohort n and the total over-30 population as whole. For the US, this conditioning ratio rises from about .75 for 30-44 to .9 for 45-60 age cohorts, and then to about 1.25 for cohorts 60-69 and 70-79. It then drops to about 1.15 for the

80+ cohort, apparently as death from 'natural causes' becomes a bigger fraction of all deaths. Conditioning the ERfct in this way thus has the effect of distributing the PM deaths toward the older age groups. This adjustment more gradually phases in the rate of death, rather than assume the risk is zero at age less than 30 and then a proportional increase in the death rate for all age cohorts over 30.

A death at an early age has a continuing effect through the remaining expected life of the individual. We assumed those who died in an age cohort were at the midpoint age for the cohort, and that the expected age of death absent chronic exposure was 75. For cohorts over 75 we assume one year of life was lost.[5] To investigate this approach we conducted a model experiment to estimate a 'value of remaining life' that we could compare to more conventional estimates. The model experiment involved running EPPA-HE from 2000 to 2100, assuming 1,000,000 deaths in 2000. The deaths were distributed across age cohorts as if they were due to chronic exposure to PM as we have modeled it (i.e., using equation 4). By 2045, given an assumed lifetime of 75 and no deaths below 30, all of these individuals would have died from other causes. Economic effects continue, however, because with a lower overall level of the economy through 2045, the capital stock is lower in 2045 than it otherwise would have been. We simulate the model through 2100. We are then able to calculate the consumption plus leisure difference between this scenario and a reference without the deaths, calculate the present discounted value of the difference, and divide the result by 1,000,000 to obtain our implicit estimate of the value of a life lost to chronic PM exposure, taking into account remaining average years of life lost. Previous cost-benefit studies use such a value directly, calculating it from studies of the value of life, and assumptions regarding the remaining years of life lost. The net present value through 2100 we obtained was $0.69 million (3% discount rate) and $0.67 million (5%) discount rate.

In comparison, Holland et al. (1998) had values ranging from $.42 million (3% discount rate) to $.38 million (5% discount rate) for the EU. The Holland et al. (1998) study is most directly comparable to ours in that it attempted to explicitly account for the years of life lost. They assumed an average of 5 years of life lost for PM exposure. Our method results in an average of 3 years, but it obviously depends on the specific pattern of exposure—in our model, higher concentrations would lead to earlier death, and more years of lost life, whereas lower

[5] 75 is an approximate mean of the life expectancy at birth for the period 1970-2000. Life expectancy, given that one has survived to certain age, e.g. 65, is considerably more than 75. For example, on average those who were 65 in 2000 had a life expectancy of 83 in the US according the Center for Disease Control (2004). We used the average life expectancy on the basis that those who suffer death due to chronic exposure are likely more vulnerable to these types of diseases and in the absence of PM exposure were also more likely to have developed these chronic diseases from other environmental factors. The best assumption here is not obvious to us, and more investigation is needed.

concentrations would reduce the number deaths and also result in shifting out the age at death, and so result in fewer years of lost life. Our approach is more structural, and richer in that sense, but in extending the structure in this way the various uncertainties in any such estimate are more apparent: at what age do people die from chronic exposure and how does it depend on cumulative exposure?

The more traditional approach is that of the US EPA (1999), who used a value of $4.8 million per PM mortality. Kunzli et al. (2000) in a study of externalities of transportation in the EU used $1.4 million per PM mortality. US EPA (1999) and Kunzli et al. (2000) use the value of a statistical life based on literature estimates. These are constructed in various ways. Implicitly these may reflect a personal (but unknown) discount rate. These estimates also do not directly consider the years of remaining life lost; i.e. whether the death occurred at 30 or 75 years of age. EPA (1999) identified an alternative calculation where they assumed the average years of life lost from PM was 14, considerably higher then either our estimate or that of Holland et al (1998) but the valuation estimate they used for their primary study was simply that of statistical life, and so was unrelated to this estimate of years lost.

There are of course various methods of valuing life ranging from contingent valuation and wage-risk studies to estimates of lifetime earnings. Our approach is more similar to the latter where we are not claiming to value life, but simply estimating the economic impact of a loss of someone at a particular age, including the lost leisure (household time) valued at the wage rate, assuming individuals are making this tradeoff at the margin.

Economic Impacts of Air Pollution: The Case of the US 1970-2000

Benchmarking EPPA-HE with Historical Pollution Levels

To test EPPA-HE we apply it to the US for the historical period from 1970 to 2000. This allows us to compare our estimates of economic damage from air pollution with estimates from a major US EPA study (US EPA, 1999). The first step in this analysis is to benchmark EPPA-HE to data for the US economy in 1970, with air pollution levels as they existed in 1970, and then reproduce the growth of the economy from 1970 to 2000 given the changing levels of pollution and how we estimate them to affect the economy. Given our parameterization of pollution damage functions in EPPA-HE, and given historical pollution levels, there are damages over the period. The observed economic trends (e.g. GDP, macroeconomic consumption) occurred with those damages. In this benchmarking step we match projected market GDP growth and returns to labor to the actual historical growth and returns. Because many of the damages involve lost

labor, returns to labor is a key variable in our damage estimate.[6] For the economic data we use the Council of Economic Advisors (2003) data. This includes estimates of real GDP growth and the total of wage and salary disbursements, other labor income, and proprietor income as a measure of total returns to labor. We adjusted labor productivity growth and capital accumulation to match these variables at 5-year increments, the standard EPPA resolution, starting in 1970. We used average urban pollution levels (tables 5 and 6), obtained from the US EPA (1989, 1999, 2003) and assumed the entire urban metropolitan population of the US was exposed to these average levels. Data on the urban population is from US Census Database (2004). Because deaths due to chronic exposure to PM are a function of accumulated exposure over the lifetime of individuals, we constructed an estimate of cumulative exposure of the 1970 population, using data on PM going back to 1923, the longest series we could obtain. For age cohorts alive in 1970, who were born before 1923 we assumed exposure in earlier years was that the 1923 level.

Counterfactual Simulations—Benefits and Burdens

With this revised benchmark we are then able to evaluate counterfactual scenarios with different levels of pollution. We considered two counterfactual scenarios for the period 1970-2000. One scenario simulated the US economy as if there had been no air pollution regulations over the period. The second scenario simulated the US economy with pollution at background (natural) levels. We then compared these counterfactual cases to the simulation with emissions at their actual historical levels. In the first case, we obtain an estimate of the benefits of air pollution regulations, the benefit side of a cost benefit analysis of these policies. In the second case, we are able to assess the burden on the economy of the air pollution that existed. It is an exercise in environmental accounting—what was the effect of air pollution on the economy in each year and how was growth over the period affected by changing pollution levels. For the benefit analysis we used US EPA (1989, 1999) estimates of what pollutant concentrations would have been without regulations, as summarized in Table 5. Seinfeld and Pandis (1998) report background (natural) pollution levels in ppm of CO, 0.05; Ozone, 0.01; NO_2, 0.00002; SO_2, 0.00002, and in μm^{-3} PM10, 0.001. We have assumed background levels at 1 percent of the 1970 average US urban levels.

[6] We have not attempted to rebenchmark the economy sector-by-sector, or use earlier input-output tables and predict the transition from one year's I-O tables to a later set of observed I-O relationships.

Results

The benefits from air pollution regulation rose steadily from 1975 to 2000 by our estimate (Figure 4). The rise results from reductions in emissions that were particularly large between 1975 and 1985, especially for ozone and PM. These pollutants are by far the largest sources of damage/benefit, as discussed further below. This reflects the relatively serious and numerous health effects due to exposure to these two pollutants based on existing epidemiological estimates. The EPA projected emissions do show some reduction over the period even in the absence of pollution regulations. The main sources of these pollutants are the combustion of fuels which were generally increasing. The reduction therefore reflects a general improvement of technology and other factors. If it had been assumed that emissions coefficients per unit of fuel burned would have remained at their 1970 levels without pollution control regulations, then emissions of all substances would have increased over the period and the estimated benefits would have been much larger.

Benefits in terms of *lost market consumption* rise to about 3.3% of *total market consumption* by 2000. *Lost market consumption + leisure* rise only to about 2.1% of *total consumption + leisure* value in 2000, but of course both the numerator and denominator are larger than the market consumption estimate alone. How much of leisure time to include in the expanded accounting of the economy is somewhat arbitrary, as noted previously, and so a better comparison of percentage loss may be *lost market consumption + leisure* as a percent of *market consumption* only: this rises to 5% by 2000. One aspect of the expanded accounting worth noting is how it affects the income constraint in a willingness to pay sense. A true willingness to pay estimate of benefits should be income constrained. In our approach, benefits are not necessarily constrained by market income but by the total resources available to the household including market income plus the value of leisure. This is entirely reasonable in our judgment. Faced with illness or death to a member, households will use their non-market resources as well as income to combat the disease, and thus exhibit a willingness to pay (or use) these resources.

The remaining costs of pollution over the period are less dependent on a projection of a counter-factual case. Essentially background levels of pollution are so low that little damage occurs—slightly different assumptions about background levels would thus have little effect on our estimates. In this case, we move to background pollution levels beginning in 1975, and so we see (Figure 4) high costs of pollution in 1975. Because the actual pollution levels are falling over time, due to regulations, exposure to pollution per person is falling. This alone would reduce pollution costs over time. The urban population is growing slowly, but the more important factor is that the economy and wage rates are growing over the period. As the value of lost work and

leisure rise over time, the absolute economic cost of pollution actually rises slightly over the entire period, despite a substantial decrease in the level of pollution.

Falling pollution levels are reflected in the percentage losses. Benefits in terms of *lost market consumption* are about 3.3% of *total market consumption* in 1975 and this falls to 2.5% by 2000. *Lost market consumption + leisure* rise as a percentage of *total consumption + leisure* is somewhat lower (2.7% in 1975 falling to 2.0% in 2000). *Lost market consumption + leisure* as a percent of *market consumption* only falls from 6.9% in 1975 to 4.7% in 2000. Since the total consumption and total consumption+ leisure also reflect growing population and income, we see the percentage loss decreasing even though the absolute loss is rising over time.

One aspect of the pollution calculation is worth noting with regard to chronic mortality effects in the air pollution cost burden estimate. We assumed mortality fell to 0.01% of what it was in 1970 under actual historical levels of PM. This implicitly assumes that the entire population alive in the 1970-2000 period had been exposed to "background" levels of PM their entire lives—including the pre-1970 period. This captures much of the cumulative effect of earlier exposure. In actuality, accumulated mortalities from circa 1900 to 1975 would have been avoided as well if there had been much lower PM, and so the economy would have been larger still in 1975 than in our counterfactual case. To make such a calculation would require extending our demographic model and EPPA-HE back to that much earlier date, and data limits prevented us from doing that.

Figure 4 shows the benefits and costs by pollutant. We made this calculation by running the historical case, setting each of the pollutant levels in turn to their "no control" or "background" level. Since there is the possibility of interaction effects within the economy, these separate calculations do not necessarily have to add up to the total estimates when all pollutant levels are changed at the same time. In fact, the sum of the separately calculated pollutants add up to within at most 1.2% of the estimate when all pollutants are changed at the same time, and so the effects are nearly linear and this decomposition method is quite accurate. As noted earlier, PM and ozone give by far the largest effects. In the benefits calculation ozone and PM benefits are very similar. In the costs case, however, the remaining costs of PM are higher than the remaining costs of ozone by a factor of about 2. NO_2 and SO_2 costs are so low relative to PM and ozone that the plots are indistinguishable from zero and lie nearly atop one another.

Mortality due to chronic exposure to PM remains particularly controversial. We estimated these effects separately be running the PM-only scenarios, with and without the chronic mortality effects. In the benefits calculation, mortality due to chronic exposure to PM starts out in 1975 as 5% of PM benefits and rises to just over 50% in 2000. The effects rise rapidly over the

period because of the stock nature of accumulating exposure. The small initial reductions, with substantial accumulated historical exposure, only slightly reduces the deaths due to chronic exposure. The reductions accumulate as people are exposed to lower PM levels over an increasing number of years and the benefits grow rapidly. The PM pollution costs for mortality exhibit a very different pattern, because we assume mortality drops to 0.01% of what it would have been, thereby implicitly assuming that these low levels of PM had existed over the entire lives of those alive in 1975. As already noted, if we were able to consider the current (1970-2000) economic effects of mortality in the pre-1970 period, the mortality costs would be larger.

Comparison to EPA Benefit Study

This method of estimating benefits and costs is relatively novel. EPA cost-benefit studies of air pollution regulations (US EPA, 1989; 1999) used a more conventional benefit valuation method. For the same set of pollutants, they estimated total benefits in 2000 dollars of $27.6 trillion over the 30-year period, 1970-2000. That compares to our estimate of $3.5 trillion, which we get by summing and multiplying our estimate by 5 (to interpolate for years in between our 5 year model runs). Two important factors in the difference between our estimates and EPA's are that we have (1) taken into account the gradual effects on mortality of lower levels of PM, and (2) accounted for the value of the loss of life in terms of annual loss of labor and leisure. In terms of a policy benefit calculation to be compared with costs borne in the period, our approach undercounts the total benefit of the pollution reductions, but the EPA's approach may overcount them.

Our undercounting stems from the fact that the remaining value of a saved life should be counted as part of the benefit of the policy in that period, even that part of the flow of benefits that extends beyond the accounting period. If a building or other asset is destroyed, its value is lost immediately, and a death is analogous to that situation. The number of lives saved in the period may, however, be overcounted by EPA's approach because the death rate falls as if everyone had been exposed to the new lower levels all of their lives. We track the gradual improvement over time. The $3.5 trillion was the result, however, of a model run only to the year 2000, and so it does not include the post-2000 benefits.

To get a more complete estimate of the future (post-2000) benefits of lower pollutant levels during the 1970-2000 period related to chronic exposure, we simulated the model forward to the year 2070 under the following conditions. We assumed that post-2000 pollution levels were the same in both 'actual' and the 'no control' cases. All we observe as a result is the remaining flow of benefits from the different levels of pollution in these two cases in the historical (pre-2000)

time period. The result is an additional, undiscounted, sum of $17.1 trillion for a total undiscounted cost of $20.6 trillion. This is very similar to the US EPA estimate. It is somewhat lower, and this is not surprising given that our 'average' undiscounted mortality loss was valued at $0.72 million whereas EPA valued a mortality loss at $4.8 million. We previously reported the 3% and 5% discounted value; our estimate of $0.72 is taken from that simulation, but without discounting.

While the EPA life value estimate may include an implicit discount rate, the value of lives implicitly saved years in the future should be discounted. EPA's long run equilibrium calculation does not allow that to be considered, whereas with our stock-flow accounting we identify deaths by age cohort, and when in time they occur (and would be avoided or delayed by a pollution policy). Thus, we discount the $20.6 trillion at 3% and 5%. The discounted benefit is $6.5 and $3.9 trillion, respectively. If our estimate of years of life lost is in the right range, discounting has little affect on the value per life saved because the value is only discounted 3 years on average. Thus, this big difference mainly reflects the fact that many of the lives apparently saved in EPA analysis during the 1970-2000 period would only be saved in the fairly distant future. Their benefit is that accumulated lower chronic exposure means they are less likely to develop diseases like heart and lung disease late in their lives. These values should be discounted in a proper cost benefit analysis. We also would argue that it is more appropriate to use the explicit accounting of years of lost life, rather than simply using the value of statistical life. That said, there are a number of caveats that must accompany our estimates. The years of remaining life estimate we arrive at may be low, and we had to make a variety of assumptions to generate a profile of deaths by age cohort that go beyond the underlying epidemiological estimates. Our valuation approach is not necessarily as inclusive as a contingent valuation measure that may include other 'non-use' values. Our goal was not to estimate the value of a statistical life, but instead to estimate the economic impacts of saving a life, expanded to include a value of non-work time saved. But, the difference between our results and EPA's appears less due to the fundamental value one places on saving a life, and more the result of our stock flow accounting and explicitly counting years of life saved (however valued) rather than simply a life, with no discrimination as to whether that involved saving 50 or 5 years of life.

Summary

We developed a method for endogenously calculating the economic impacts of the effects of air pollution on human health. This involved expanding the underlying economic accounts to include leisure, including a household health sector that used medical services and household

labor to mitigate the health effect of air pollution. We also developed a simple age cohort model to track cumulative exposure to particulate matter because the epidemiological literature finds increased death rates due to chronic exposure. The explicit accounting for cumulative exposure turns out to be quite important in valuing the benefits of air pollution regulation because it affects when those benefits would be realized. It also allows us to estimate how deaths in different age cohorts would change, and thus the number of years of life saved by a pollution policy. The approach was implemented in a version of the MIT EPPA model, EPPA-HE.

The ability to endogenously calculate benefits and impacts of environmental change has great promise, only partially realized in this initial exploration. Ultimately extended to other regions, it automatically values changes consistent with the economic variables for different countries and as those values change in the future under different assumptions of economic growth and policies. There are also feedbacks on emissions and other economic variables that may be important for some problems such as climate change. The methodology thus has a richer set of applications, and can assure greater consistency in economic modeling scenarios, than traditional benefit estimation.

We applied the model to the US for the period 1970-2000. This involved first re-benchmarking the model to replicate the macroeconomic performance of the economy with the air pollution health effects. We were then able to simulate counter-factual cases. One involved a "no emissions control" case—what emissions would have been had the air pollution regulations of the Clean Air Act never been put in place. A second counter-factual case involved the assumption that the urban population experienced only background levels of the pollutants that would exist if there were no emissions from industrial sources. The first scenario allowed us to estimate a benefit of air pollution regulations. We found that the benefit rose to over $250 billion per year by 2000, and equaled about 5% of total macroeconomic consumption in the year 2000. The total benefits realized over the period equaled $3.5 trillion, a large benefit but much less than the US EPA estimate of $27.6 trillion. To our estimate we must add a present value estimate the benefits from reduced cumulative exposure during 1970-2000 that will only be realized after 2000. If we do not discount this amount, our total estimate is comparable in magnitude the US EPA estimate, but discounted at 3% our total benefit is $10 trillion, and at 5% is $7.4 trillion.

The case of pollution levels at background levels allows us to estimate the remaining burden of air pollution. In absolute dollar terms this has been high and gradually rising over the entire period (from about $175 to $250 billion per year from 1975 to 2000). It has fallen as a percentage of the economy (from 6.9 to 4.7% between 1975 and 2000), however, mostly because pollution levels have fallen due to regulation. It continues to rise in absolute terms because the

wage rate and the urban population are rising and so more people are exposed and the value of lost time has risen. Properly accounting for the stock nature of chronic exposure would require us to re-simulate the economy from circa 1900, and data did not allow that. The estimate of burden to the economy during the 1970 to 2000 period does not, therefore, include an estimate of effects due to mortality that occurred prior to 1975, but would have had continuing economic effect into the study period.

In terms of both benefits and remaining burden, the effects of tropopsheric ozone and particulate matter are the most important in terms of our estimate of economic impact. CO, NO_2, and SO_2 effects were quite small in comparison. Mortality due to chronic exposure to PM is an important component of the costs, and this is one of the more controversial health effects of pollution. In the benefits calculation, much of this occurs after 2000 but it has become an important component even by 2000. In the burden calculation mortality is important over the whole period.

There remain a number of caveats that must accompany these results. We have not investigated in detail the underlying epidemiological estimates, and there remain uncertainties and controversies surrounding these. Our estimates are only as accurate as these underlying relationships. Never-the-less, our estimates are comparable to existing benefit estimates, and the differences are mostly the result of key improvements we have made in accounting for chronic exposure effects.

References

Abbey D.E., M.D. Lebowitz, P.K Mills, F.F. Petersen, W. Lawrence Beeson, R.J. Burchette (1995) Long-term ambient concentrations of particulates and oxidants and development of chronic disease in a cohort of nonsmoking California residents. *Inhalation Toxicology* 7, 19-34

Anderson H.R., A. Ponce de Leon, J.M. Bland, J.S. Bower, D.P. Strachan DP (1996). Air pollution and daily mortality in London: 1987-92. *British Medical J.* 312: 665-9.

Babiker, M.H., G.E. Metcalf, J. Reilly, 2003: Tax distortions and global climate policy. *J. of Environmental Economics and Management*, **46**: 269-287

Babiker, M.H., J.M. Reilly, M. Mayer, R.S. Eckaus, I. Sue Wing & R.C. Hyman, 2001. The MIT Emissions Prediction and Policy Analysis (EPPA) Model: Revisions, Sensitivities, and Comparisons of Results, MIT Joint Program on the Science and Policy of Global Change Report 71, Cambridge, MA (Feb.)

Center for Disease Control (2004). National Center for Health Statistics, http://www.cdc.gov/nchs/fastats/lifexpec.htm

Council of Economic Advisors (2003). Economic Report of the President. US Government Printing Office, Washington DC.

Dab, W., S.M.P. Quenel, Y. Le Moullec, A. Le Tertre, B. Thelot, C. Monteil, P. Lameloise, P. Pirard, I. Momas, R. Ferry, B. Festy (1996). Short term respiratory health effects of ambient air pollution: results of the APHEA project in Paris. *J. Epidemiology Comm Health* 50 (suppl 1): S42-46.

Dimaranan B. and R. McDougall (2002). Global Trade, Assistance, and Production: The GTAP 5 Data Base. Center for Global Trade Analysis, Purdue University, West Lafayette, Indiana.

Dockery, D.W., F.E. Speizer, D.O. Stram, J.H. Ware, J.D. Spengler, B.G. Ferries (1989). Effects of inhalable particles on respiratory health of children. *American Review of Respiratory Disease* 139, 587-594.

Dusseldorp, A., H. Kruize, B. Brunekreef, P. Hofschreuder, G. de Meer, B. van Oudvorst (1995). Associations of PM10 and airborne iron with respiratory health of adults near a steel factory. *American J. of Respiratory & Critical Care Medicine* 152, 1932-9.

Holland, M., J. Berry, and D. Forster (eds.) (1998). "ExternE, Externalities of Energy", Volume 7: Methodology, 1998 update, European Commission, Directorate-General XII, Science Research and Development.

Hertel, T. (1997). *Global Trade Analysis: Modeling and Applications*. Cambridge University Press, Cambridge, UK.

Jacoby, H.D., R.S. Eckhaus, A.D. Ellerman, R.G. Prinn, D.M. Reiner Z. Yang (1997). CO_2 Emissions Limits: Economic Adjustments and the Distribution of Burdens, *The Energy Journal* 18(3): 31-58.

Jacoby, H.D., and I. Sue Wing (1999). Adjustment Time, Capital Malleability, and Policy Cost, *The Energy Journal Special Issue*: The Costs of the Kyoto Protocol: A Multi-Model Evaluation, pp. 73-92.

Krupnick A.J., W. Harrington, B. Ostro (1990). Ambient ozone and acute health effects: Evidence from daily data. *J. Environmental and Economic Management* 18, 1-18.

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, H. Sommer (2000). Public health impact of outdoor and traffic-related air pollution: a European assessment. *The Lancet* 356.

Mayer, M., R. Hyman, J. Harnisch & J. Reilly, 2000. Emissions Inventories and Time Trends for Greenhouse Gases and other Pollutants. MIT Joint Program on the Science and Policy of Global Change, *Technical Note 1*, Cambridge, MA. (July)

Mintz, D. (2003). Data on long-term historical PM concentrations, personal communication, US EPA Washington DC.

Ostro B.D. (1987). Air pollution and morbidity revisited: A specification test. *J Environmental and Economic Management* 14, 87-98.

Ostro B.D. and S. Rothschild (1989). Air pollution and acute respiratory morbidity: An observational study of multiple pollutants. *Environment and Resources* 50, 238-247.

Paltsev, S., J. Reilly, H.D. Jacoby, A.D. Ellerman and K.H. Tay (2003). Emissions trading to reduce greenhouse gas emissions in the United States: The McCain-Lieberman Proposal. MIT Joint Program on the Science and Policy of Global Change, *Report* 97, Cambridge, MA (June).

Paltsev, S., L. Viguier, J. Reilly, K.H. Tay, and, M. Babiker (2004). Disaggregating Household Transport in the MIT-EPPA Model, Mimeo, Joint Program on the Science and Policy of Global Change, Cambridge, MA.

Ponce de Leon A., H.R. Anderson, J.M. Bland, D.P. Strachan, J. Bower (1996). Effects of air pollution on daily hospital admissions for respiratory disease in London between 1987-88 and 1991-92. *J. Epidemiology Comm Health* 50 (suppl 1): S63-70.

Pope, C. A., R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, G.D. Thurston (2002). Lung Cancer, Cardiopulmonary mortality and long-term exposure to fine particulate air pollution. *J. of the American Medical Association* 287, (9), 1132-1141

Pope C.A. and D.W. Dockery (1992). Acute health effects of PM10 pollution on symptomatic and asymptomatic children. *American Review of Respiratory Disease* 145, 1123-1126.

Pope C.A., M.J. Thun , M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer F.E. and C.W. Heath (1995). Particulate air pollution as predictor of mortality in a prospective study of US adults. *American J. of Respiratory & Critical Care Medicine* 151: 669-674.

Reilly, J., Prinn, R.,J. Harnisch, J. Fitzmaurice, H. Jacoby, D. Kicklighter, J. Melillo, P. Stone, A. Sokolov, C. Wang (1999). Multi-gas assessment of the Kyoto Protocol. *Nature* **401**: 549-555.

Seinfeld, J. H. and S.N. Pandis (1998). *Atmospheric Chemistry and Physics* , John Wiley & Sons, Inc, New York.

Schwartz J. and R. Morris (1995). Air pollution and hospital admissions for cardiovascular disease in Detroit, Michigan. *American J. of Epidemiology* 142, 23-35

Spix, C. and H.E. Wichmann (1996). Daily mortality and air pollutants: findings from Köln, Germany. *J. Epidemiology Comm Health* 50 (suppl 1), S52-S58.

Sunyer J, J. Castellsague, M. Saez, A. Tobias, J.M. Anto (1996). Air pollution and mortality in Barcelona. *J. of Epidermiology Comm Health* 50 (supple 1), S76-S80.

Touloumi G., E. Samoli, K. Katsouyanni (1996). Daily mortality and 'winter type' air pollution in Athens, Greece - a time series analysis within the APHEA project. *J. of Epidemiology Comm Health* 50 (suppl 1), S47-S51.

US Census Database (2004). Population by Age Group http://www.censusscope.org/us/chart_age.html

US Department of Labor (2004). Bureau of Labor Statistics, http://www.bls.gov

US Environmental Protection Agency (1989). The Benefits and Costs of the Clean Air Act 1970 to 1990. Office of Air and Radiation, Office of Policy, Washington DC, (November).

US Environmental Protection Agency, (1999). The Benefits and Costs of the Clean Air Act 1990 to 2010. Office of Air and Radiation, Office of Policy, Washington DC, (November).

US Environmental Protection Agency (2003). Latest Findings on National Air Quality: 2002 Status and Trends, Report No. 454/K-03-001, Office of Air Quality Planning and Standards, Emissions, Monitoring and Analysis Division, Research Triangle Park, NC (August). http://www.epa.gov/airtrends.

Verhoeff, A.P., Hoek, G., Schwartz, J. and van Wijnen, J.H. (1996). Air pollution and daily mortality in Amsterdam. Epidemiology 7, 225-230.

Whittemore, A.S. and E.L. Korn (1980). Asthma and air pollution in the Los Angeles area. *American J. Public Health* 70, 687-696.

Wordley J., Walters S. and Ayres J.G.. Short term variations in hospital admissions and mortality and particulate air pollution. (In press). Carcinogenic Effects of Radionuclide Emissions.

Yang, T, 2004. Economic and Policy Implications of Urban Air Pollution in the United States: 1970 to 2000, Masters Thesis, Technology Policy Program, Massachusetts Institute of Technology, Cambrdige, MA

Table 1. Countries, Regions, and Sectors in the EPPA Model

Country or Region	Sectors
Annex B	**Non-Energy**
United States (USA)	Agriculture (AGRI)
Canada (CAN)	Services (SERV)
Japan (JPN)	Energy Intensive products (EINT)
European Union+[a] (EUR)	Other Industries products (OTHR)
Australia/New Zealand (ANZ)	Transportation (TRAN)
Former Soviet Union[b] (FSU)	**Energy**
Eastern Europe[c] (EET)	Coal (COAL)
Non-Annex B	Crude Oil (OIL)
India (IND)	Refined Oil (ROIL)
China (CHN)	Natural Gas (GAS)
Indonesia (IDZ)	Electric: Fossil (ELEC)
Higher Income East Asia[d] (ASI)	Electric: Hydro (HYDR)
Mexico (MEX)	Electric: Nuclear (NUCL)
Central and South America (LAM)	Electric: Solar and Wind (SOLW)
Middle East (MES)	Electric: Biomass (BIOM)
Africa (AFR)	Electric: Natural Gas Comb.Cycle (NGCC)
Rest of World[e] (ROW)	Electric: NGCC w/ Sequestration (NGCCS)
	Electric: Integrated Coal Gasification w/ Combined Cycle and Sequestration (IGCC)
	Oil from Shale (SYNO)
	Synthetic Gas from Coal (SYNG)
	Household
	Own-Supplied Transport (OTS)
	Purchased Transport Supply (PTS)

[a]The European Union (EU-15) plus countries of the European Free Trade Area (Norway, Switzerland, Iceland).
[b]Russia and Ukraine, Latvia, Lithuania and Estonia (which are included in Annex B) and Azerbaijan, Armenia, Belarus, Georgia, Kyrgyzstan, Kazakhstan, Moldova, Tajikistan, Turkmenistan, and Uzbekistan which are not. The total carbon-equivalent emissions of these excluded regions were about 20% of those of the FSU in 1995. At COP-7 Kazakhstan, which makes up 5-10% of the FSU total joined Annex I and indicated its intention to assume an Annex B target.
[c]Includes a number of former Yugoslav republics and Albania not Part of Annex B, which contribute only a small percentage of the overall emissions of the Region.
[d]South Korea, Malaysia, Phillipines, Singapore, Taiwan, Thailand
[e]All countries not included elsewhere: Turkey, and mostly Asian countries.]

Table 2. Health Effects of Air Pollutants on the General Population

Receptor	Impact Category	Pollutant	E-R fct	Reference
Entire Population	Respiratory hospital admissions	PM 10	2.07E-06	Dab et al 1996
		Nitrates	2.07E-06	
		PM 2.5	3.46E-06	
		SO_2	2.04E-06	Ponce de Leon 1996
		O_3	7.09E-06	
	Cerebrovascular hospital admissions	PM 10	5.04E-06	Wordley et al 1997
		Nitrates	5.04E-06	
		PM 2.5	8.42E-06	
	Symptoms days	O_3	3.30E-02	Krupnick et al 1990
	Acute Mortality	PM 10	0.040%	Spix and Wichmann 1996, Verhoeff et al 1996
		Nitrates	0.040%	
		PM 2.5	0.068%	
		SO_2	0.072%	Anderson et al 1996, Touloumi et al 1996, Sunyer et al 1996
		O_3	0.059%	
	Chronic Mortality	PM2.5	0.64%	Pope et all 2002
		PM 10	0.40%	
Children	Chronic Bronchitis	PM 10	1.61E-03	Dockery et al 1989
		Nitrates	1.61E-03	
	Chronic Cough	PM 10	2.07E-03	Dockery et al 1989
		Nitrates	2.07E-03	
		PM 2.5	3.46E-03	
Adults	Restricted activity day	PM 10	2.50E-02	Ostro, 1987
		Nitrates	2.50E-02	
		PM 2.5	4.20E-02	
	Minor restricted activity day	O_3	9.76E-03	Ostro and Rothschild, 1989
	Chronic bronchitis	PM 10	4.90E-05	Abbey et al, 1995
		Nitrates	4.90E-05	
		PM 2.5	7.80E-05	
Elderly 65+	Congestive heart failure	PM 10	1.85E-05	Scwartz and Morris, 1995
		Nitrates	1.85E-05	
		PM 2.5	3.09E-05	

Source: Adapted from Table 8.1 in Holland et al. (1998).
Note: Morbidity units are in [cases/(yr-person-ug/m^3)]. Mortality are in [%Δannual mortality rate/ug/m^3]

Table 3: Air Pollution Health Effects on Asthmatics

Receptor	Impact Category	Pollutant	E-R fct	Reference
All	Asthma attacks	O_3	4.29E-03	Whittemore and Korn 1980
Adults	Bronchodilator usage	PM 10	1.63E-01	Dusseldrop et al 1995
		Nitrates	1.63E-01	
		PM 2.5	2.72E-01	
	Cough	PM 10	1.68E-01	Dusseldrop et al 1995
		Nitrates	1.68E-01	
		PM 2.5	2.80E-01	
	Lower respiratiry symptoms (wheeze)	PM 10	6.10E-02	Dusseldrop et al 1995
		Nitrates	6.10E-02	
		PM 2.5	1.01E-01	
Children	Bronchodilator usage	PM 10	7.80E-02	Dusseldrop et al 1995
		Nitrates	7.80E-02	
		PM 2.5	1.29E-01	
	Cough	PM 10	1.33E-01	Dusseldrop et al 1995
		Nitrates	1.33E-01	
		PM 2.5	2.23E-01	
	Lower respiratiry symptoms (wheeze)	PM 10	1.03E-01	Dusseldrop et al 1995
		Nitrates	1.03E-01	
		PM 2.5	1.72E-01	
Elderly	Ischaemic heart disease	PM 10	1.75E-05	Schwartz and Morris, 1995
		Nitrates	1.75E-05	
		PM 2.5	2.92E-05	
		CO	4.17E-07	

Source: Adapted from Table 8.1 in Holland et al. (1998).
Note: Morbidity units are in [cases/(yr-person-ug/m^3)]. Mortality are in [%Δannual mortality rate/ug/m^3]

Table 4. Morbidity Valuation Estimates	
Health impacts	Costs in US dollars 2000
Restricted Activity Day	$106
Respiratory Hospital Admissions	$11,115
Cerebrovascular Hospital Admissions	$11,115
Symptoms Days	$11
Chronic Bronchitis Adults	$148,296
Chronic Bronchitis Children	$318
Chronic Cough for Children	$318
Congestive Heart Failure	$11,115
Asthma attacks	$52
Cough	$318
Lower Respiratory Symptoms (wheeze)	$11
Ischaemic Heart Disease	$11,115
Minor Restricted Activity Day	$11
Emergency Room Visit	$315
Acute Mortality	$30,225
Source: Table 12.9 in Holland et al. (1998) converted to 2000 dollars.	

Table 5. US urban air pollution levels, 1970-2000: Actual and Projected Without Control Policies

	1970	1975	1980	1985	1990	1995	2000
CO-Actual	12.8	11.8	8.8	7.4	6.1	4.8	3.4
CO-No Controls	12.8	12.9	11.1	11.22	10.5	9.19	7.24
NO_2-Actual	0.0231	0.0260	0.0275	0.0246	0.0231	0.0215	0.0195
NO_2-No Controls	0.0231	0.0311	0.0382	0.0383	0.0391	0.0394	0.0391
SO_2-Actual	0.0161	0.0150	0.0150	0.0100	0.0088	0.0060	0.0053
SO_2-No Controls	0.0161	0.0179	0.0219	0.0144	0.0134	0.0094	0.0084
Ozone-Actual	0.153	0.153	0.143	0.125	0.117	0.116	0.103
Ozone-No Controls	0.153	0.168	0.172	0.169	0.175	0.191	0.185
PM10-Actual	79.0	51.3	42.8	28.9	27.0	26.6	25.0
PM10-No Controls	79.0	54.3	55.3	40.9	41.3	44.7	45.6

Concentrations in ppm, except PM10 in $\mu g\text{-}m^{-3}$

Source: Historical data and projected No-Control Emissions are from US EPA, 1988, 1999, 2003

Table 6. PM10 Concentrations

1923	94.1
1940	105.3
1945	108.6
1950	110.5
1951	111.8
1955	105.9
1960	102.0
1965	92.1
1968	85.5

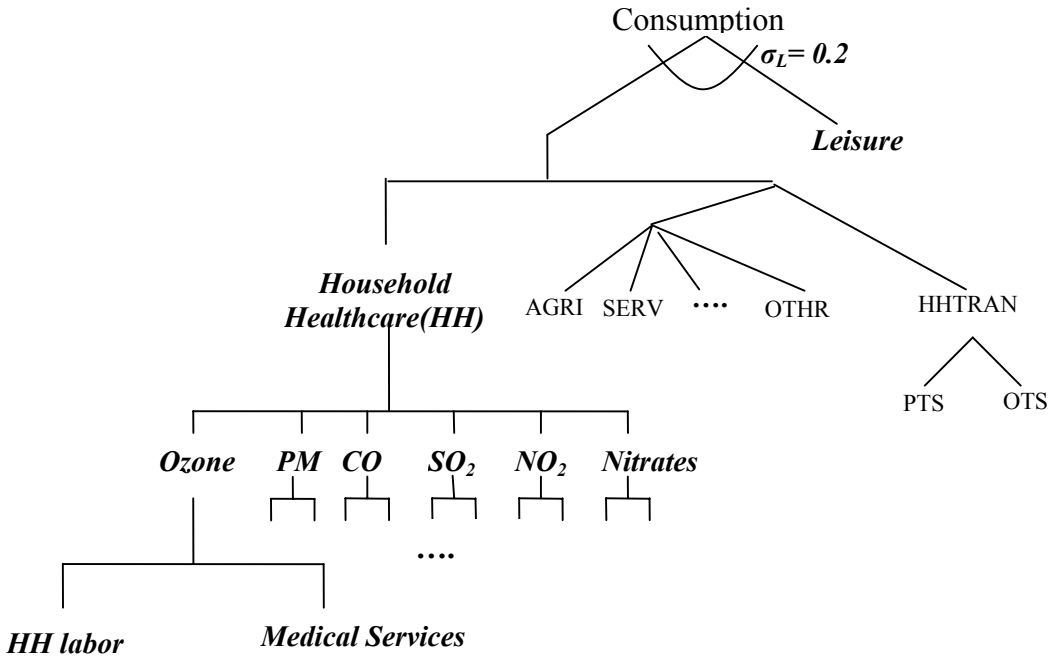
Source: Mintz, 2003, $\mu g\text{-}m^{-3}$

Figure 1: Expanded Social Accounts Matrix for EPPA-HE

	Production Sectors	***Household Production***	Final Consumption
Production Sectors	Input/Output ***Medical Services for Air Pollution***	Household Transportation ***Household Mitigation of Pollution Health Effects***	Goods and services ***Pollution Health Service*** ***Leisure***
Factors	Labor, Capital, Resources	***Household labor***	***Total Consumption =Total Factor Income***

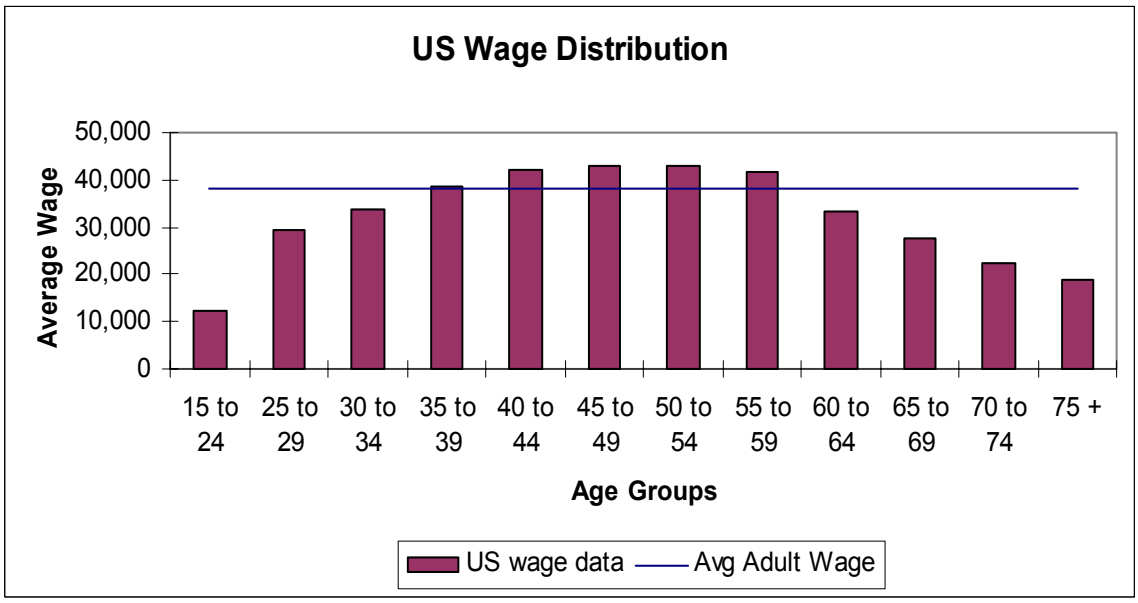
Note: Newly added components in bold italics.

Figure 2. Household and Consumption Structure for EPPA-HE



New household activties in EPPA-HE are in bold italics. Perpendicular lines are Leontief production relationships. Pollutant labels (Ozone, PM, CO, SO_2, NO_2, Nitrates) are used as shorthand reference to health services used to combat various health effects from the pollutant. Multiple health endpoints (see Tables 2-5) by assumption scale linearly with the pollutant are aggregated within EPPA-HE.

Figure 3: US Wage Distribution, Annual Wages



Source: US Dept. of Labor, 2004.

Figure 4. Remaining Costs of Pollution; Benefits of Air Pollution Regulation

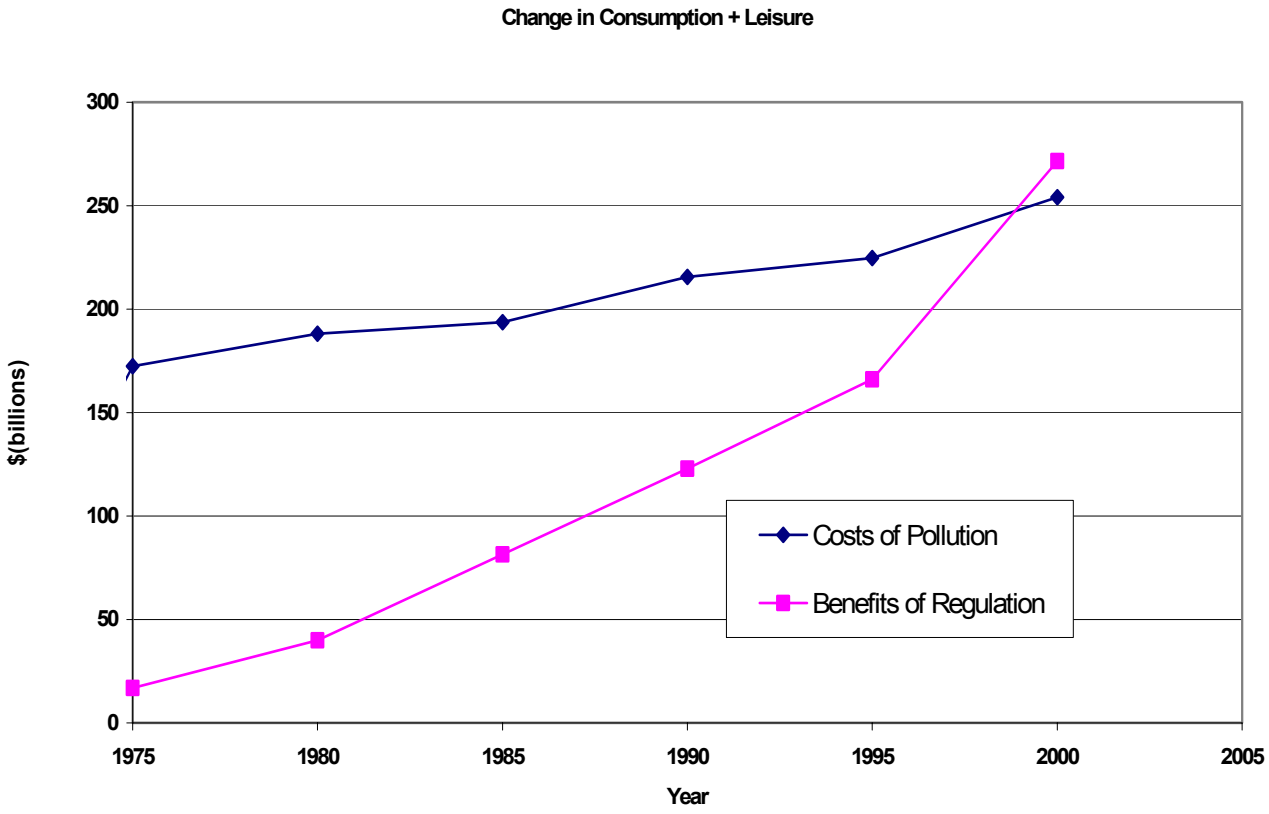


Figure 5. Benefits and Costs by Pollutant