MIT Joint Program on the Science and Policy of Global Change

Economic Benefits of Air Pollution Regulation in the USA: *An Integrated Approach*

Trent Yang, Kira Matus, Sergey Paltsev and John Reilly

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To inform processes of policy development and implementation, climate change research needs to focus on improving the prediction of those variables that are most relevant to economic, social, and environmental effects. In turn, the greenhouse gas and atmospheric aerosol assumptions underlying climate analysis need to be related to the economic, technological, and political forces that drive emissions, and to the results of international agreements and mitigation. Further, assessments of possible societal and ecosystem impacts, and analysis of mitigation strategies, need to be based on realistic evaluation of the uncertainties of climate science.

This report is one of a series intended to communicate research results and improve public understanding of climate issues, thereby contributing to informed debate about the climate issue, the uncertainties, and the economic and social implications of policy alternatives. Titles in the Report Series to date are listed on the inside back cover.

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Abstract

Market and non-market effects of air pollution on human health are estimated for the U.S. for the period from 1970 to 2000. The pollutants include tropospheric ozone, nitrogen dioxide, sulfur dioxide, carbon monoxide, and particulate matter. 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 model of the economy that has been widely used to study climate change policy. Benefits of air pollution regulations in USA rose steadily from 1975 to 2000 from $50 billion to $400 billion (from 2.1% to 7.6% of market consumption). Our estimated benefits of regulation are somewhat lower than the original estimates made by the U.S. Environmental Protection Agency, 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. We also estimate the economic burden of uncontrolled levels of air pollution over that period. The estimate of economic benefits and damages depends 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.

Contents

1. INTRODUCTION

In 1970 the U.S. Environmental Protection Agency introduced the Clean Air Act Amendment (42 U.S.C. s/s 7401 et. seq (1970)) to limit the amount of air pollution and to establish limits on "criteria pollutants" to protect public health and welfare. The "criteria pollutants" include ozone (O_3), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon monoxide (CO), particulate matter (PM), and lead. The task of quantifying the benefits of the Clean Air Act is complicated because the benefits do not fall on a particular person or a group but on the entire population through decreased morbidity and mortality levels. In addition, different population groups react differently to reductions in air pollution and will acquire different benefits. The valuation of air pollution effects should be consistent with wage, total income, labor supply, and other economic

variables over time. These economic variables produce a feedback on pollution levels. Existing methods for estimating the economic implications of environmental damage do not provide an immediate approach to assess the economic and policy interactions. Several studies (*e.g.*, U.S. EPA, 1989, 1999; Holland *et al.*, 1998) use current values of critical economic data such as wages or medical expenses, but they do not fully incorporate the economic valuation of air pollution in an integrated economic model.

The goal of this paper is to present a methodology for integrating the health effects from exposure to air pollutants to examine economic and policy interactions. We apply our methodology to estimate the benefits of air pollution regulation in the U.S. in 1970-2000, and we also estimate the economic burden of uncontrolled levels of pollution over that period. 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.

We integrate the health effects from exposure to the following air pollutants: ozone (O_3), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon monoxide (CO), particulate matter (PM 2.5 and PM 10) 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 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 U.S. 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 the U.S. air pollution regulations and compare them to a set of benefit estimates originally made by the U.S. Environmental Protection Agency (U.S. EPA, 1989, 1999). For this purpose, we use the counterfactual level of air pollution (*i.e.*, what it would have been without regulation) estimated by the U.S. 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 U.S. 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.

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 U.S. from 1970-2000. A sensitivity analysis with respect to the different values of economic growth, assumptions about the age structure of mortality due to PM, and cost shares of health impacts. We finally offer some conclusions.

2. MIT EPPA-HE

The MIT EPPA-HE model is built on the standard EPPA4 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 (EPPA4) 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). EPPA4 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 U.S., 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 EPPA4 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

Table 1. Dimensions of 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	REFOIL
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 Combined Cycle	NGCC
Rest of World[e]	ROW	Electric: NGCC with Sequestration	NGCAP
		Electric: Integrated Gasification with Combined Cycle and Sequestration	IGCAP
		Oil from Shale	SYNO
		Synthetic Gas	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] Hungary, Poland, Bulgaria, Czech Republic, Romania, Slovakia, Slovenia.

[d] South Korea, Malaysia, Phillipines, Singapore, Taiwan, Thailand.

[e] All countries not included elsewhere: Turkey, and mostly Asian countries.

	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***

Figure 1. Expanded Social Accounts Matrix for EPPA-HE. Newly added components in bold italics.

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. EPPA4 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 require proportionally more of all inputs to deliver the same level of health service, or lower levels require proportionally less.[1] **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.

[1] 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.

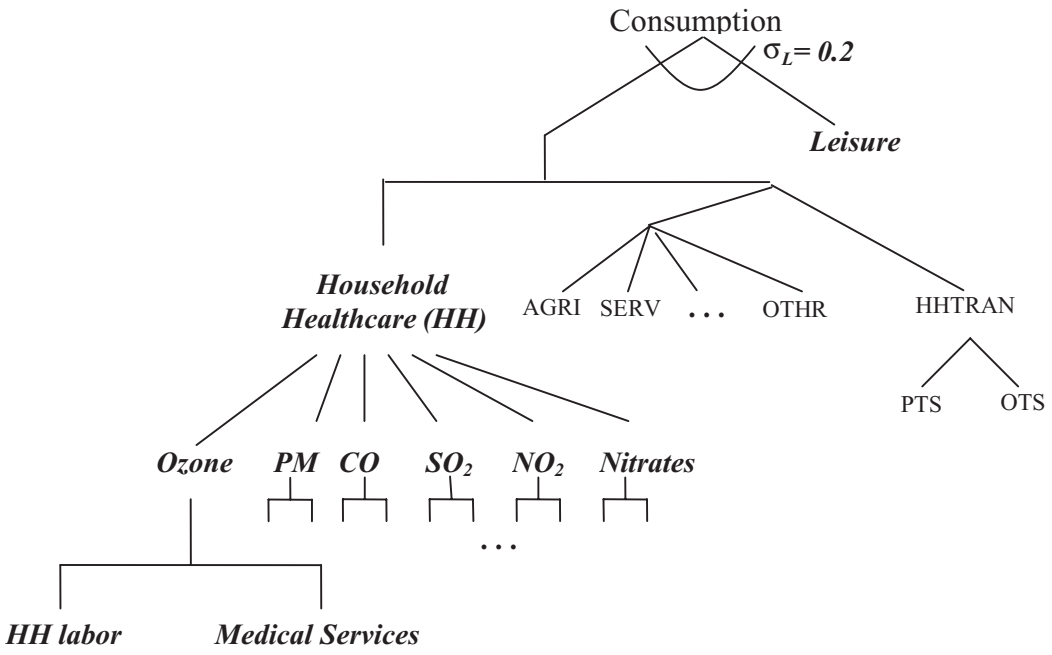

Figure 2. Household and Consumption Structure for EPPA-HE. New household activities in EPPA-HE are in bold italic. Pollutant labels (Ozone, PM, CO, SO_2, NO_2, Nitrates) are used as shorthand reference to health services used to combat various health effects from the pollutant.

3. 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.*, U.S. EPA, 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 of labor endowment in the economy and the population's exposure to pollution. 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.[2]

[2] 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.

3.1 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**, **3** and **4** are adapted from the Holland *et al*. (1998) in an extensive study for the European Commission. The reported relationships summarize the known health effects of exposure to these pollutants, building on a data compilation originally started in the U.S. Tables 2 and 3 contain relationships estimated for a general healthy population, and reflects the fact that some of the relationships

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

Receptor	Impact Category	Pollutant	ER fct[†]	Reference
Entire Population	Respiratory hospital admissions	PM 10	2.07E-6	Dab *et al*., 1996
		Nitrates	2.07E-6	
		SO_2	2.04E-6	
		O_3	7.09E-6	Ponce de Leon,1996
	Cerebrovascular hospital admissions	PM 10	5.04E-6	Wordley *et al*., 2004
		Nitrates	5.04E-6	
	Symptoms days	O_3	3.30E-2	Krupnick *et al*., 1990
Children	Chronic bronchitis	PM 10	1.61E-3	Dockery *et al*., 1989
		Nitrates	1.61E-3	
	Chronic cough	PM 10	2.07E-3	
		Nitrates	2.07E-3	
Adults	Restricted activity day	PM 10	2.50E-2	Ostro, 1987
		Nitrates	2.50E-2	
	Minor restricted activity day	O_3	9.76E-3	Ostro & Rothschild, 1989
		PM 10	4.90E-5	
	Chronic bronchitis	Nitrates	4.90E-5	Abbey *et al*., 1995
		PM 10	1.85E-5	
Elderly 65+	Congestive heart failure	CO	5.55E-7	Schwartz & Morris, 1995
		Nitrates	1.85E-5	
		PM 2.5	3.09E-5	

Source: Adapted from Table 8.1 in Holland *et al*. (1998).
[†] Units of exposure factor are [cases/(yr-person-μg/m^3)].

Table 3. Mortality Health Effects of Air Pollutants on the General Population

Receptor	Impact Category	Pollutant	ER fct[†]	Reference
Entire Population	Acute Mortality	O_3	0.06%	Sunyer *et al*., 1996; Anderson *et al*., 1996; Touloumi, 1996
		SO_2	0.07%	Verhoeff *et al*., 1996
		PM 10	0.04%	Spix & Wichmann 1996
		Nitrates	0.04%	Verhoeff *et al*., 1996
	Chronic Mortality	PM 10	0.25%	Pope *et al*., 2002

Source: Adapted from Table 8.1 in Holland *et al*. (1998) and Pope et al. (2002).
[†] Units of exposure factors are [% change in annual mortality rate/μg/m^3].

Table 4. Morbidity Air Pollution Health Effects on Asthmatics

Receptor	Impact Category	Pollutant	ER fct[†]	Reference
All	Asthma attacks	O_3	4.29E-3	Whittemore & Korn,1980
Adults	Bronchodilator usage	PM 10	1.63E-1	Dusseldrop *et al*., 1995
		Nitrates	1.63E-1	
	Cough	PM 10	1.68E-1	
		Nitrates	1.68E-1	
	Lower respiratory symptoms (wheeze)	PM 10	6.10E-2	
		Nitrates	6.10E-2	
Children	Bronchodilator usage	PM 10	7.80E-2	Dusseldrop *et al*., 1995
		Nitrates	7.80E-2	
	Cough	PM 10	1.33E-1	
		Nitrates	1.33E-1	
	Lower respiratory symptoms (wheeze)	PM 10	1.03E-1	
		Nitrates	1.03E-1	
Elderly	Ischaemic heart disease	PM 10	1.75E-5	Schwartz & Morris, 1995
		Nitrates	1.75E-5	
		CO	4.17E-7	

Source: Adapted from Table 8.1 in Holland *et al*. (1998).
[†] Units of exposure factors are [cases/(yr-person-μg/m^3)].

differ for children or the elderly as compared with the general adult population. Table 4 contains estimated relationships for the population of asthmatics, a group that is more vulnerable to air pollution. Exposure Factors (ERfct) presented in Tables 2 through 4 are defined as a number of cases due to exposure to a pollutant (μg/m^3) over a year for morbidity health impacts, and as a percent change in the annual mortality rate due to exposure (μg/m^3) for mortality health impacts. Holland *et al*. (1998) also include a set of estimates for effects they considered less certain. These relationships between health and air pollution have been found to be statistically significant in some studies. However, these were studies of small populations or the relationships have been found statistically insignificant in other studies. 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. Most of his results come from a suspected relationship between elevated CO and mortality.

All of the relationships including those in Tables 2 to 4 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 in Tables 2

and 3 is that they cover the entire population. Any relationship thus reflects to some degree both individual response to varying dose levels and varying vulnerability within the population.

The health effects presented in Tables 2 to 4 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 3 results from a revised study (Pope *et al*., 2002) that corrected the previous error.

The PM relationship has been the subject of contentious debate in the United States as the U.S. 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 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 through 4, noting that this the basis for evaluation of air pollution benefits in Europe and similar assessments by the U.S. 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.

3.2 Accounting for Health Effects in the SAM

The next step is to turn the impact categories (often referred to as “health endpoints” in epidemiological literature) 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.*, U.S.

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 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 estimates 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 U.S. dollars are shown **Table 5**. For each health impact category related to each pollutant (*e.g.*, respiratory hospital visit due to exposure to ozone), we allocated a

Table 5. Morbidity Valuation Estimates

Health Impacts	Cost in US$
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 U.S. dollars in year 2000.

share of the total cost to demand for medical service, lost labor, or lost leisure. As one can see from Tables 2 to 4, not all pollutants are associated with all impact categories, but we end up with 50 separate combinations. Based on Yang (2004) and Holland *et al*. (1998), 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. 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.

3.3 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 of potential labor force 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. For example, Babiker *et al*. (2003) assumed a value of an additional potential labor force of 25% to the recorded payments to labor endowment. 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:

$$\varepsilon = \frac{\alpha}{1-\alpha}\sigma \tag{1}$$

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, the supply of labor in response to a change in wages will be approximately 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 an estimate of 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 U.S. 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.[3]

[3] 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.

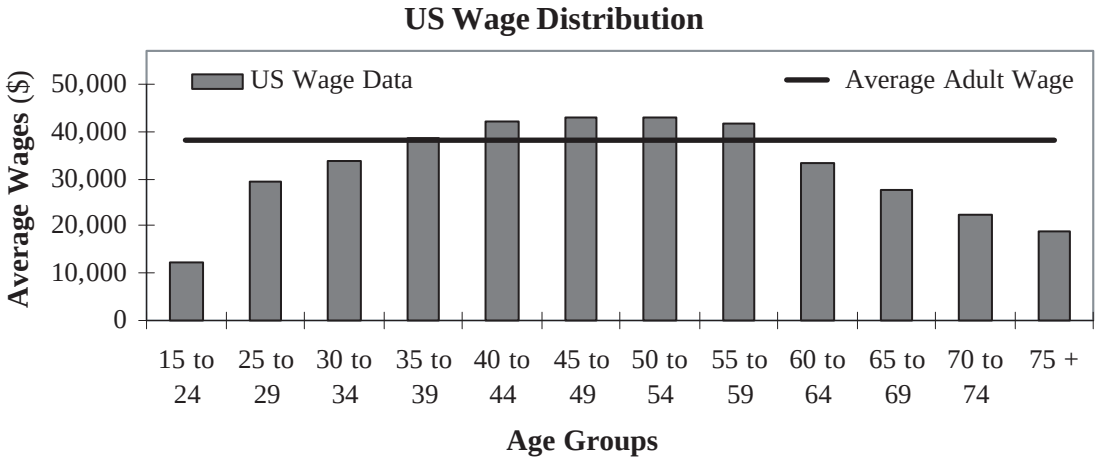

Figure 3. U.S. wage distribution, annual wages. Source: U.S. Dept. of Labor (2004).

3.4 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.

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.

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

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:

$$\%\Delta pr(d) = ERfct \times \overline{C} \tag{3}$$

where ERfct is the variable as defined in Table 3. 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 Equation 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:

$$ERfct_n = ERfct_T \times \frac{\Pr(d:cpl)_n \,/\, \Pr(d)_n}{\Pr(d:cpl)_T \,/\, \Pr(d)_T} \tag{4}$$

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 U.S., this conditioning ratio rises from about 0.75 for 30-44 to 0.9 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 on accounting of potential labor supply over the period of 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.

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.

4. ECONOMIC IMPACTS OF AIR POLLUTION: THE CASE OF THE U.S. 1970-2000

4.1 Benchmarking EPPA-HE with Historical Pollution Levels

To test EPPA-HE we apply it to the U.S. 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 U.S. EPA study (U.S. EPA, 1999). The first step in this analysis is to benchmark EPPA-HE to data for the U.S. 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. The observed economic trends (*e.g.*, GDP, consumption) occurred with damages from historic pollution levels. 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.[4] 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, salary disbursements, and other labor 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 obtained from the U.S. EPA (1989, 1999, 2003) and assumed the entire urban metropolitan population of the U.S. was exposed to these average levels. Data on the urban population are from U.S. 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, which are presented in **Table 6**. For age cohorts alive in 1970, who were born before 1923 we assumed exposure in earlier years was at the 1923 level.

Table 6. PM10 Concentrations

Year	Concentration ($\mu g\text{-}m^{-3}$)
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).

[4] We have not attempted to re-benchmark 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.

4.2 Counterfactual Simulations—Benefits and Burdens

We consider two counterfactual scenarios for the period 1970-2000. One scenario simulates the U.S. economy as if there had been no air pollution regulations over the period. The second scenario simulates the U.S. economy with pollution at background (natural) levels. We then compare 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 U.S. EPA (1989, 1999) estimates of what pollutant concentrations would have been without regulations. The actual historical urban pollution concentrations and counterfactual case with no regulations are summarized in **Figure 4**. 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 approximated these levels by assuming background levels at 1 percent of the 1970 average U.S. urban levels.

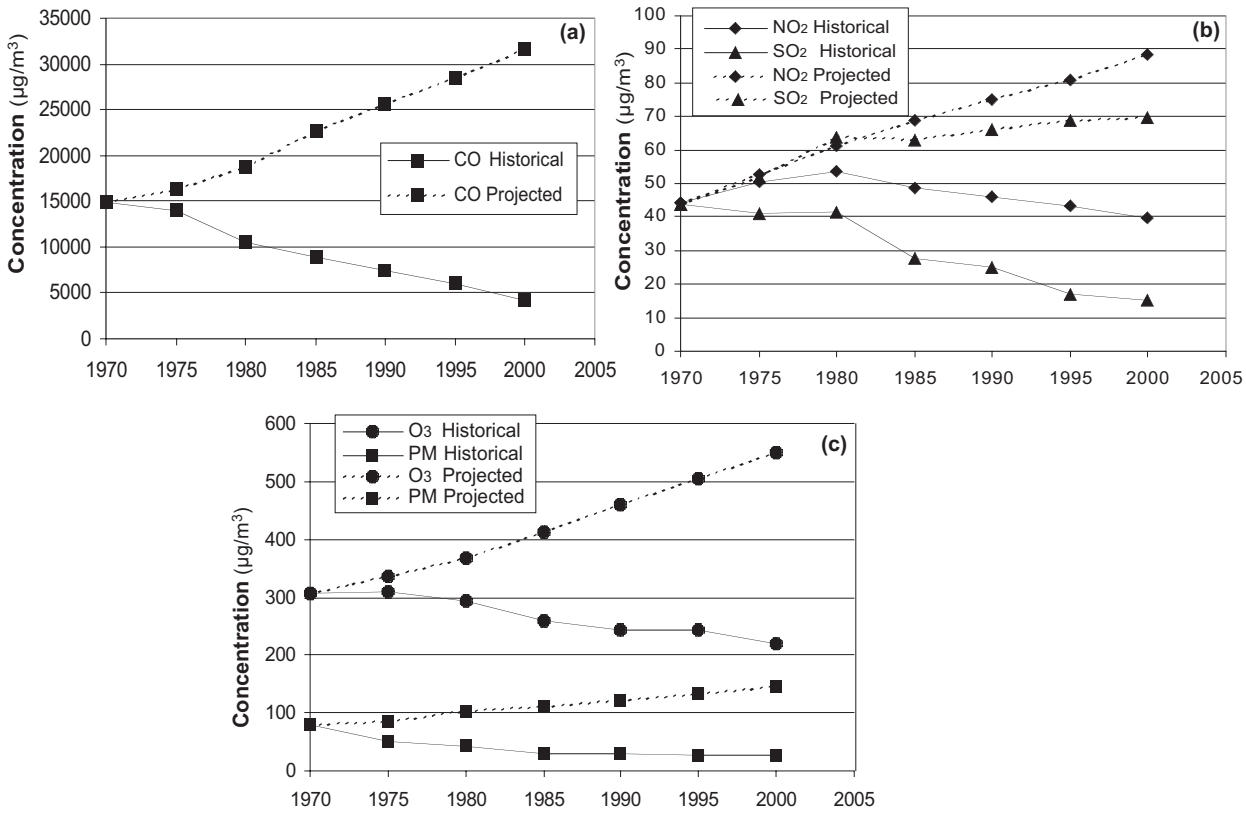

Figure 4. Historical and projected levels, without the Clean Air Act, of **(a)** CO, **(b)** NO_2 and SO_2, and **(c)** PM and O_3.

4.3 Results

As presented in **Figure 5**, the benefits from air pollution regulation rose steadily from 1975 to 2000 by our estimate. In terms of additional market consumption + leisure, the benefits rose steadily from 1975 to 2000, from around $50 billion to $400 billion (1997 USD). Ozone and PM 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.

Benefits in terms of *additional market consumption* rise to about 5.4% of *total market consumption* by 2000. *Additional market consumption + leisure* rise only to about 3.3% 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 benefits may be *additional market consumption + leisure* as a percent of *market consumption* only: this rises to 7.6% by 2000. It is worthwhile to note that in the expanded accounting 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. 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.

Figure 5 also shows the remaining costs of pollution over the period of 1975-2000. The results are less dependent on a projection of a counter-factual case. Essentially, background levels of pollution are so low that slightly different assumptions about background levels would have little effect on our estimates. Because the actual pollution levels are falling over time, due to regulations, exposure to pollution per person is falling. This alone would reduce pollution

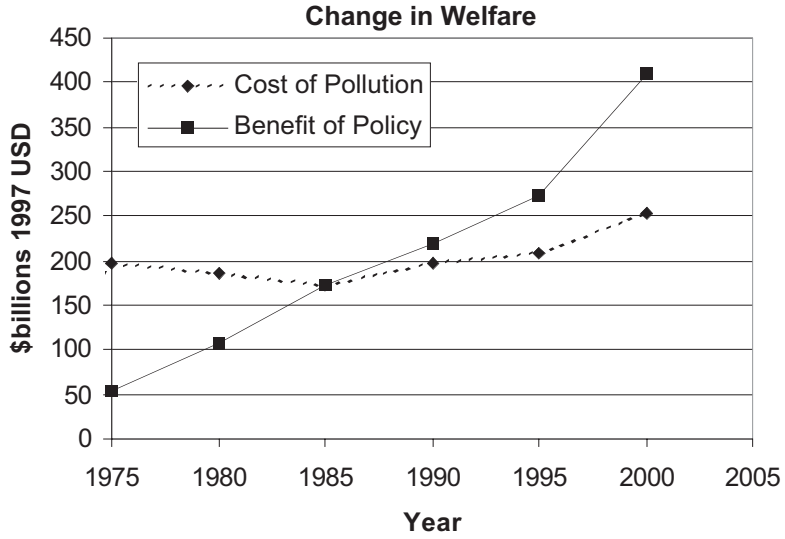

Figure 5. Benefits of air pollution regulation and costs of remaining pollution.

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. Damages from historical pollution levels in terms of *lost market consumption* are about 4.1% of *total market consumption* in 1975 and this falls to 2.7% by 2000. *Lost market consumption + leisure,* as a percentage of *total consumption + leisure,* is somewhat lower (3.0% in 1975 falling to 2.1% in 2000). *Lost market consumption + leisure* as a percent of *market consumption* falls from 7.8% 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 6 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 1.5% of the estimate when all pollutants are changed at the same time, and so the effects are nearly linear. As noted earlier, PM and ozone give by far the largest effects. The benefits from PM reduction relative to ozone reduction decrease over time. In 1975, the benefits from PM reduction were more than 16 times the benefits to ozone reduction; by 2000, the gap had closed such that the PM benefit was 4.2 times the ozone benefit. This is because the EPA projected (Figure 4c) much faster growth in ozone levels than in PM in the absence of regulation. The relative costs of these two pollutants were nearly constant, with the relative cost of PM compared to ozone ranging between a factor of 4.2 and 4.5 over the period. CO, NO_2 and SO_2 costs are low relative to PM and ozone.

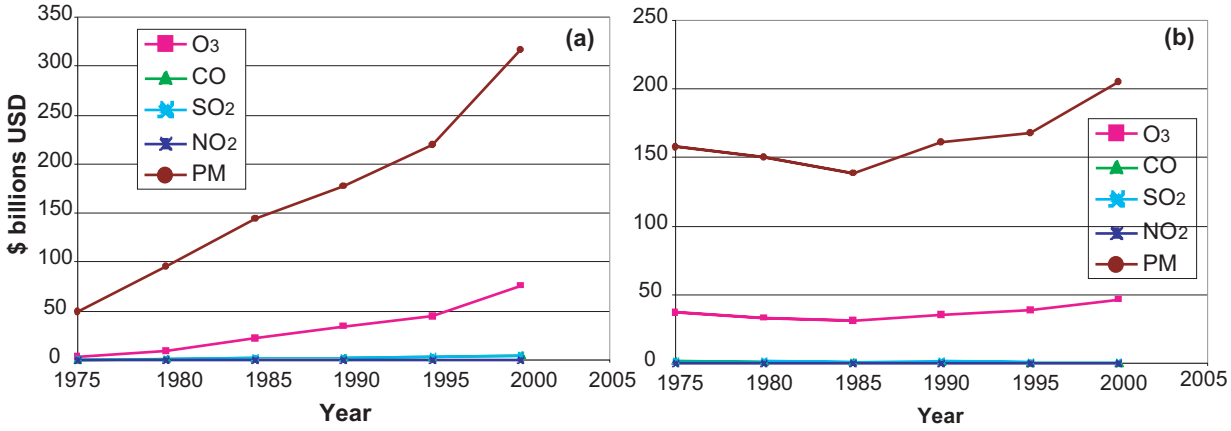

Figure 6. Welfare **(a)** benefits and **(b)** costs by pollutant.

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 in early years. 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.

4.4 Comparison to EPA Benefit Studies

This method of estimating benefits and costs is relatively novel. EPA cost-benefit studies of air pollution regulations (U.S. EPA, 1989; 1999) used a more conventional benefit valuation method. For the same set of pollutants, they estimated total benefits of $27.6 trillion (in year 2000 dollars) over the 30-year period, 1970-2000. That compares to our estimate of $5.4 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 be overcounted by EPA's approach, however, 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 $5.4 trillion was the result, however, of a model run only to the year 2000, and so it does not include the post-2000 benefits. By our estimate, the annual years of life saved peaks in 2030 at about two million, up from about 900 thousand in 2000, and there continue to be benefits until 2080 when all of those alive then are, by our assumption, born after 2000. Simulating the avoided deaths in EPPA-HE we can estimate these future economic benefits. Adding these benefits to the $5.4 trillion we still get a lower number than the EPA. If we do not discount future benefits from chronic exposure to PM, our total estimate is about half of the U.S. EPA estimate, discounted at 3% our total benefit is $7.9 trillion, and discounted at 5% is $6.9 trillion.

5. SENSITIVITY

One important question is how sensitive our results are to a set of key assumptions. We test the results of our model with respect to different values for the economic growth, the conditioning of the exposure-response function for PM, and the service/labor/leisure breakdown for each health endpoint.

As expected, the rate of economic growth affects the estimates of benefits and costs. In the low growth scenario we assume that an annual GDP growth is one percent lower than historic growth over 1970-2000. In the high growth scenario the annual GDP growth is one percent higher than historic growth. **Figures 7** and **8** present the results showing how the benefits and costs depend on the GDP growth assumptions. High growth scenario results in the estimates of benefits of air pollution regulation 30% higher in 2000 than in the case of historic growth, and

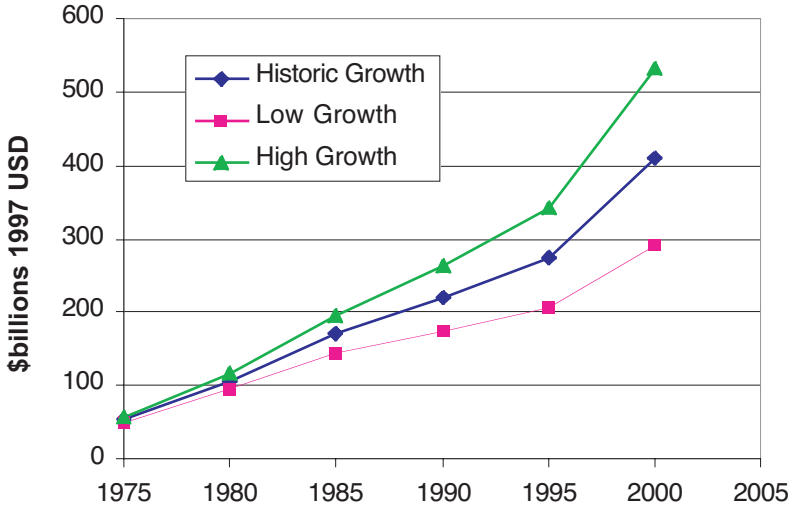

Figure 7. Benefits with different economic growth.

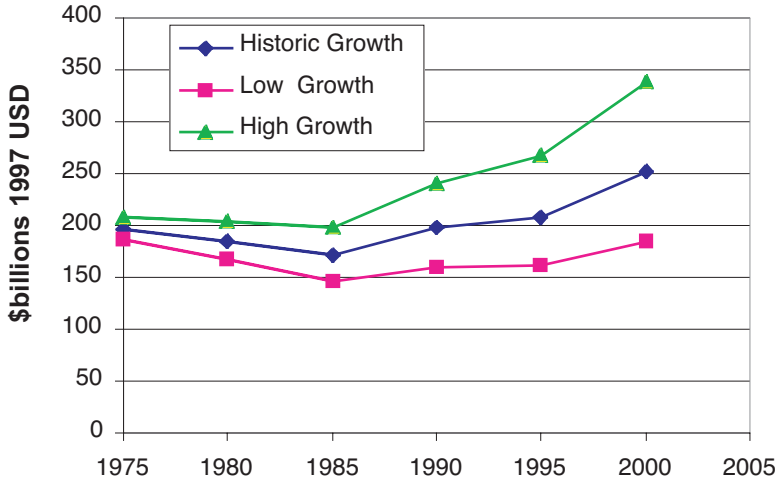

Figure 8. Costs with different economic growth.

the estimates of costs of remaining pollution are 34% higher in 2000 in comparison to a historic growth case. Low growth scenario results in 29% lower benefits estimate and 27% lower costs estimate. It shows that a change in benefits and costs are more or less proportional to a change in economic growth.

Of great importance to our exercise in environmental accounting was the development of a method for calculating the number of chronic mortalities due to PM exposure. As mentioned, for our study we conditioned the exposure-response (E-R) function reported by Pope *et al.* (2002) by the ratio of cardiopulmonary mortality to all-cause mortality for each age group. To determine whether this changes the results from the case where all age groups have the same exposure-response function, we also ran the constant E-R scenario.

Figure 9 shows that the constant (unconditioned) case has higher benefits than the conditioned case. Conditioning the E-R function increases the deaths in the older age groups, while decreasing the number of deaths in the younger age groups, as compared to using a constant value. In the unconditioned case the same reduction in the number of mortalities occurs due to the decrease in PM exposure but more of them are at younger ages and so more years of life are saved, and thus there is a greater economic impact. Deaths from chronic PM exposure are related to overall lifetime exposure and decreases in PM levels over the period only gradually effect the mortality rate. The increase in benefits in 2000 is around $14 billion.

As expected, the costs of remaining pollution to consumption + leisure are higher for the unconditioned scenario (**Figure 10**). In the unconditioned case more years of labor are lost due to the increased number of deaths in the younger age groups, which results in a higher overall cost. The magnitude of the cost increase from the use of the constant E-R function grew from $31 billion in 1975 to $63 billion in 2000.

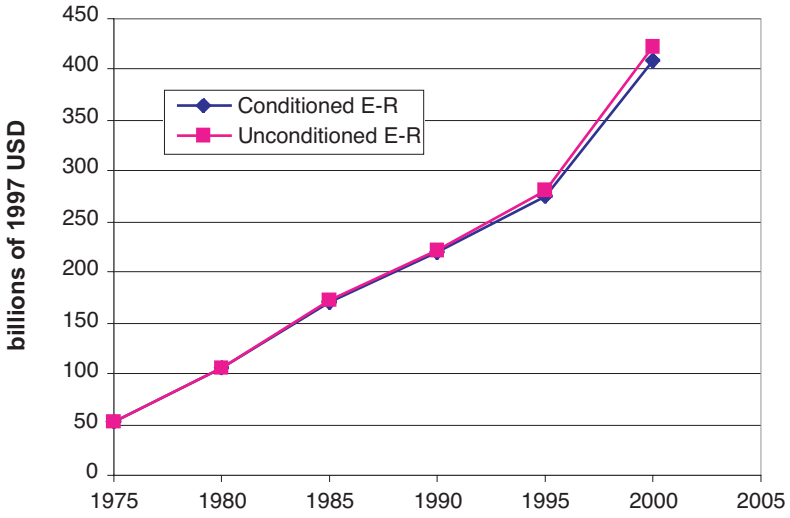

Figure 9. Benefits with different exposure-response function.

The third set of sensitivity analyses was conducted on the shares assigned to service, labor and leisure for each health endpoint. One motivation for this sensitivity, apart from uncertainty in the shares, is that some people may seek treatment (and thus reduce lost time by purchasing health care) whereas other people might not seek treatment but thereby experience more lost labor or leisure.[5] Here we report the results related to the following two health endpoints. The first, respiratory hospital admissions (RHA) is a high cost ($11,115) health endpoint, but with low incidence, with the bulk of the costs going towards the procurement of services (85%, with the remaining 15% of costs split between labor (4%) and leisure (11%). The second, restricted activity days (RAD) is a low cost ($106) health endpoint with higher incidence and no service cost (35% labor, 65% leisure). The cost/incidence differences between the two endpoints approximately offset one another so that the total cost for each is of the same order of magnitude. We consider an arbitrary scenario where the shares of costs were set to 50% service, and 50% labor/leisure. The result is that changing the shares of RAD has a much larger impact than changing the shares RHA. Changing the shares of RAD leads to an increase in welfare costs of around 15% by 2000, and a decrease in welfare benefits of around 15% as well. Manipulating the shares of RHA, however, has a much smaller impact, with a 0.5% increase in costs and a 0.36% increase in benefits in 2000. The RAD share change from 100 labor/leisure and 0 service cost to 50-50 shares is a bigger change than the RHA shift from 15-85 to 50-50 shares and this may explain the different magnitude.

[5] We do not change the size of the loss, which may be affected by whether treatment is sought or not.

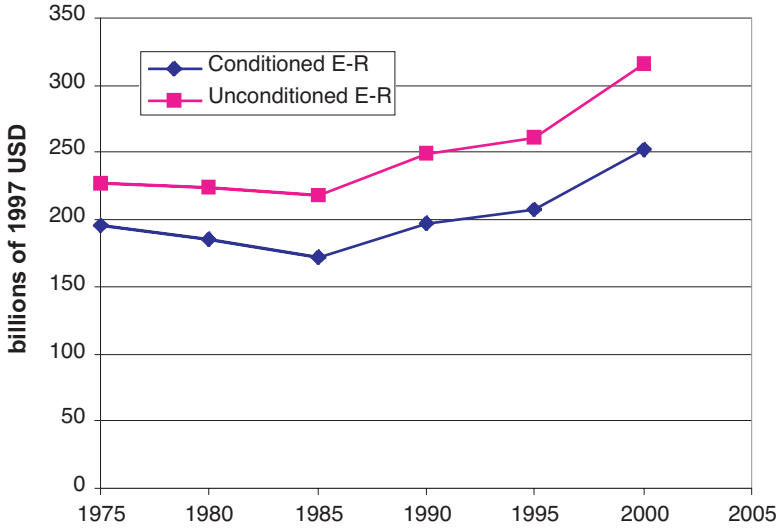

Figure 10. Costs with different exposure-response function.

6. CONCLUSION

A method for endogenously calculating the economic impacts of the effects of air pollution on human health involves 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 apply the method to the U.S. for the period 1970-2000 using the MIT Emissions Prediction and Policy Analysis (EPPA) model. This involves re-benchmarking the model to replicate the macroeconomic performance of the economy with the air pollution health effects. We then consider two counter-factual cases. One case is a "no emissions control" scenario, *i.e.*, 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 involves 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 allows us to estimate a benefit of air pollution regulations. We found that the benefits rose steadily from 1975 to 2000 from $50 billion (1997 USD) to $400 billion (1997 USD) (from 2.1% to 7.6% of market consumption). The total benefits realized over the period equaled $5.4 trillion, a large benefit but much less than the U.S. 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. Even adding this future benefit we find a lower number than the EPA—about 1/2 their estimate if the benefits are not discounted, $7.9 trillion discounted at 3%, and $6.9 trillion discounted at 5%.

The case of setting 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 $200 to $250 billion per year from 1975 to 2000). It has fallen as a percentage of total consumption (from 7.8 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. Fully 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 tropospheric 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.

Acknowledgements

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