

# Economic and Policy Implications of Urban Air Pollution in the United States: 1970 to 2000

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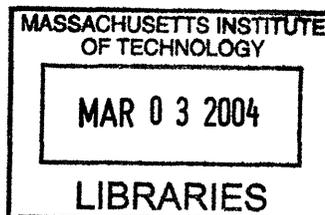
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**Abstract**

In the last 30 to 40 years, an increasing awareness of the link between urban air pollutant levels and negative health effects have led to numerous studies and policies that are targeted towards both understanding the linkage and mitigating its effects. In 1970, the Environmental Protection Agency (EPA) created the Clean Air Act directed at reducing harmful emissions that cause high pollution levels in urban areas. Ever since then, environmental economists and policy makers have attempted to better understand the economic impacts of these regulations through cost and benefit analysis.

Towards that end, we have developed a methodology for fully integrating the health effects from exposure to air pollution into a computable general equilibrium economic model. This model represents the first attempt at fully incorporating the economic valuation of air pollution in an integrated economic model that has endogenously built-in consumer demand and preference curves to accurately represent the demand for air pollution health. This framework provides a way to consistently value effects with commonly used approaches for valuing costs of mitigation and to explore uncertainties in these estimates. Furthermore, we also describe a new stock and flow model to track the extra mortalities from chronic exposure to particulate matters. Past frameworks have assumed an immediate relationship between pollution levels and mortality levels. While this is true for mortality due to acute exposure, changes in mortalities from chronic exposure due to a change in pollution levels are only gradually realized and so the full effects on the economy are observed for many years. This new framework allows the tracking of total pollution in-take and its effect on mortality levels over time as pollution levels change.

Using these new models, we valued the economic benefit of reduced air pollution due to the Clean Air Act regulations to be over \$7 trillion from 1970 to 2000, or 2.1% of aggregate US economic welfare over the period. This does not include the benefits into the future (after 2000) from reduction in mortality due chronic exposure during these years. The economic benefit of those saved mortalities is another \$7 trillion using a 3% discount rate. Another calculation is the remaining economic burden of unmitigated pollution levels (actual historical pollution). We estimate this to be approximately \$9 trillion over the same period. The \$9 trillion burden includes the early mortalities due to chronic exposure to PM before and during this period.

While these economic benefits of air pollution regulation are large (\$7 trillion), they are considerably less than the \$27.6 trillion estimated in EPA's own analysis of the

benefits of air pollution regulation. The main difference for our lower estimate is the stock-flow accounting of mortality due to chronic exposure. There are considerable uncertainties in these estimates both because of uncertainties in the relationship between air pollution exposure and the health effects, and in the assumptions needed to value these effects.

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# Chapter 1

## Introduction

### 1.1 Climate Change, Air Pollution and Health

While global climate change studies and policies have received much recent news coverage and attention (Kyoto), health effects from high emission levels of air pollutants are of much more concern to the local economies and welfare of the citizens in the immediate time frame. Furthermore, it has also come to the attention of scientists and policy makers that climate changing green house gases (GHGs) are also related to urban air pollutants through complicated atmospheric chemistry that may have counterintuitive results if these policies are considered separately. For example, if both the emissions of VOCs (volatile organic compounds) and NO<sub>x</sub> (these are usual precursors GHGs) are held at a low enough level, it could actually lead to the increase of Ozone (both an urban air pollutant and a GHG) levels in the atmosphere.

In the last 30 to 40 years, an increasing awareness of the link between urban air pollutant levels and negative health effects have led to numerous studies and policies that are targeted towards both understanding the linkage and mitigating its effects. In this thesis, I focus on studying air pollution health effects within and economic model that has been used to study climate change mitigation. It is a contribution to economic analysis of air pollution effects, and the first step toward an integration of these two important, and inter-related policy questions.

On the policy front, EPA in 1970 introduced the Clean Air Act Amendment in hopes of limiting the amount of air pollutants being released into the ambient air and to establish limits in "criteria pollutants" to protect public health, and welfare. The "criteria pollutants" include Ozone, Particulate Matter (PM), Carbon Monoxide (CO), Nitrogen Dioxide (NO<sub>2</sub>), Sulfur Dioxide and Lead. At the same

time, EPA launched and sponsored multiple studies to better understand the direct and indirect links between air pollution and human health effects.

The range of health studies includes chamber, epidemiological cohort and population-level ecological studies. Chamber studies of air pollution involve exposing human subjects to various levels of air pollution in a carefully controlled and monitored laboratory situation. Epidemiological studies take real-world health and pollution data and assess the relationship between population-wide health information (such as daily mortality, hospital admissions, etc) and ambient levels of air pollution using statistical methods. Cohort-based studies track individuals over a certain period of time, with periodic evaluation of the individuals' exposure and health status.

After thirty years of regulations, there is natural tendency among policy analysts and economists to analyze the value of the policies through cost and benefit analyses. The task of quantifying the benefits of the Clean Air policies are difficult because they do not fall on a particular person or a group of people but rather the entire population through decreased morbidity and mortality levels. Furthermore, different populations groups (such as kids vs. adults vs. elderly and those who have asthma) react differently to increased air pollution and will acquire different benefits at varying levels.

Several improvements have been made in the past decades to set a proper framework for assessing air pollution effects and costs. Hohmeyer (ref, 1988) was the first to use a top down approach, multiplying an inventory of emissions by toxicity factors and then by costs of damages. However, the toxicity factors used were derived from government regulations for maximum permissible concentrations at workplace instead of being absolute intrinsic toxicity factors. Additionally, the results were averaged to cost per kWh of electricity produced without considering the effect of population density on the number of people exposed to air pollution. In recent years, several investigators and agencies have taken this idea forward and made many meaningful contributions (ref 1, 5, 6, 9, 10, 15, 16, and 21). Most recently, Laurianne Curtil

of MIT's Global Climate Change Program (ref 21) utilized the ExternE (Externalities of Energy) compilation of air pollution and human health effects in a large-scale macroeconomics model for economic valuation of air pollution across multiple regions of the world and over a long (100 yr) time frame. Other similar studies include the ExternE's own economic valuation of air pollution, European Agency's study on traffic pollution and human health effects and valuation, EPA's cost and benefit analysis of the clean air act and Kerry Smith's recent studies on incorporating individual preferences in valuating the economic costs of air pollution (ref. 15 and 16)

## **1.2 Research Contribution of Thesis**

While all of the above approaches have their own merits in valuing the economic consequences of air pollution, to this date no study has fully incorporated the economic valuation of air pollution in an integrated economic model that has endogenously built-in consumer demand and preference curves to accurately represent the demand for air pollution health. Dr. Kerry Smith was one of the first to suggest this approach to value air pollution costs (ref 15, 16) but this thesis represents the first time that this approach has been utilized within a full economic model. Benefits of such an approach are that the valuation of air pollution effects is directly calculated in welfare terms measured as equivalent variation, and the valuation that is consistent with wage, income, and other price differences as they vary across different regions of the world and over time.

Furthermore, the effects of air pollution, including sick days, bronchitis, and early mortality among others, redirect resources in the economy toward medical expenditures or result in lost labor or non-labor time with consequent effects on all other economic activities. All of these effects may in turn produce potential feedback on emissions levels. Study of these effects ultimately requires a fully coupled economic and pollution system model.

Towards that end, we have developed a methodology for fully integrating the effects of the air pollution into the Emissions Prediction and Policy Analysis

(EPPA) model. EPPA is a computable general equilibrium (CGE) model that represent the circular flow of goods and services in the economy. Not only does it endogenously model a full set of inter-industry transactions, consumer demand for goods and services but also savings as well as taxes to provide funds for investment and government purchases. Furthermore, EPPA is a component of the MIT Integrated Global Systems Model (IGSM) a coupled economics-chemistry-atmosphere-ocean-terrestrial biosphere model of earth systems including an air pollution model resolving the urban scale. IGSM is at the forefront of modeling climate changes through the integration of economics, policy and atmospheric policy. We hope that by endogenizing the air pollution data and its consequent economic effects into this model, a fuller and more complete understanding of the economic and social costs of air pollution can be understood in the full context of global climate change.

The new model within EPPA integrates the epidemiological relationships on health damages within the overall CGE model and explicitly considers stock-flow aspects of the pollution, exposure, and health impact relationship. From this model, not only are we better able to describe the time profile over which the health benefits of a change in pollution would be realized but we are also then able to estimate, for example, the value of lost wages that are consistent with the wage rates in different regions of the world, and changing wages and productivity of the labor force overtime.

This framework is then applied to the US for the period 1970 – 2000 for two different analysis. The first study is a benefit analysis of the Environment Protection Agency's (EPA) Clean Air Act. We compare the economic growth with the Clean Air Act in place (i.e. historical US economic growth record from 1970-2000 – the "control" scenario) with the hypothetical case of "no-control" where urban air pollution is allowed to grow without the strict controls put in place by the local, state and national governments. In the "no-control" case, we utilize EPA's predictions on the growth of urban air pollution without control in the EPPA economic model to determine its economic state. The difference between the two scenarios is the economic benefit of having the Clean Air Act regulations.

This benefit value can then be compared to the actual costs of implementing the Clean Air Acts for a true cost-benefit analysis of the policies.

The second study analyzes the burden of historical urban air pollution on the US economy. The “control” scenario is used as a comparison, this time to the hypothetical “green” scenario that has urban air pollution at “natural” levels observed in the non-polluted parts of the world such as the ocean. The economic differences between the two scenarios are the cost of urban air pollution on the US economy even with all the policies of the last thirty years that are designed to curb these effects.

### **1.3 Simulation Result Overview**

Simulation of the different scenarios reveals that the economic benefits of the Clean Air Act regulations far outweigh the costs of implementation. From 1970 to 2000, the total economic benefit in the United States from reduced urban air pollution levels had an economic benefit of over \$7 trillion. Furthermore, due to the reduced particulate matter levels from that period, population mortality levels will also be lower even beyond year 2000. Chronic exposure to particulate matters has been shown to cause earlier mortality but these deaths only come about after prolonged exposure. With the clean air regulations, and the reduced particulate matter levels in urban areas, fewer people will develop the lung and cardiopulmonary diseases that are associated with chronic exposure to these pollutants. In all, over half a million more people in the next 60 years will live to their normal life expectancy because of the lowered air pollution levels from 1970-2000 than if pollution levels had not been controlled. Over the years, the reduced mortality levels also increase the overall economic welfare of the country. If a 3% discount is used, the economic value of reduced mortalities is approximately another \$7 trillion in year 2000 value.

The second study on the economic burden of the unmitigated air pollution levels show that even with the tough Clean Air Act regulations, significant air pollution remain in our cities and it has a high economic impact on the society. In the same 1970-2000 period, unmitigated pollution levels created a \$9 trillion burden on the society. On average, this translates to 3% of the annual total economic welfare of the United States including the non-market (leisure values) or 5.8% of the GDP.

## **1.4 Thesis Organization**

There are three main sections in this thesis. The first section analyzes all the past health impact studies. We pay attention to the most controversial studies such as Pope's particulate matter study (ref 2 and 3), and understand the current expert consensus. As part of this discussion, we also describe how the results of these epidemiological studies can be used in a CGE economic valuation framework.

In the second section, we outline the basic framework for endogenizing the air pollution results into EPPA. Both the underlying economic theories and the practical modeling techniques will be discussed in detail. A key issue is the difference between chronic and acute exposures and the implications of these differences for the economic models. Recent epidemiological studies [ref 9 and 10] have suggested that mortality from chronic exposure to particulate matters account for over 75% of the economic costs of air pollution. Many past modeling methods have been particularly inadequate in dealing with these effects, failing to adequately differentiate between a one time illness that occurs from acute exposure and the long-term effect of exposure to pollution over many years. We introduce an explicit stock-flow accounting of both the exposure and the loss of life in the model.

Finally, the third major section contains the simulation results of the new model in the EPPA. We model the US economy from 1970 to 2000 under the

three different conditions – historical (control), no-control, and green – and present two sets of estimates of the urban air pollution effects on the US economy. The first set uses EPA’s own estimates of air quality conditions without the Clean Air Act regulations of the last 30 years (no-control scenario) and determines the benefits in monetary terms of having the improved air quality (no-control vs. control). This result can be compared to EPA’s own estimate of the economic benefits derived from improved air qualities due to its regulations as part of its major cost-benefit analysis [ref 9, 10]. This is a typical benefit calculation – how much damage was avoided because of controls that kept pollution below what they would have been without controls. Apart from uncertainties in the epidemiological relationships (past studies) and economic valuation of these effects (this thesis), such calculations also depend upon the correct forward propagation of what pollution would have been without regulation. Those values are taken directly from EPA’s own projections [ref 9, 10] and will not be repeated in this thesis.

The second model estimates the historical costs (economic burden) in the US from 1970 to 2000 that remain from incompletely controlled urban air pollution even with all the regulations of the last thirty years. For these calculations we use historical urban air pollution levels from 1970-2000 to estimate the cost to the society compared with the hypothetical case (green scenario) of urban air conditions equivalent to “natural” levels observed in the other parts of the world such as over the ocean or non-populated land.

Lastly, I draw conclusions and suggest what additional research and data would be needed to extend these estimates to other economies of the world.

## Chapter 2

### Air Pollution and Health Studies

#### 2.1 Introduction

Estimates of health costs due to air pollution are based upon known relationships between air pollution intakes and the associated mortality and morbidity. In the last 20-30 years, a large number of health studies have been used to establish the correlation between a particular air pollutant and the corresponding health effects. Past and recent epidemiological research has found consistent and coherent associations between air pollution and various outcomes (eg, respiratory symptoms, reduced lung function, chronic bronchitis and mortality). [ref 1 - 5] While it would be most ideal to back these studies with actual understanding of the body chemistry interaction with these pollutants, we have neither the scientific means for a detailed chemistry and biological analysis nor the resources and human capital for long-term controlled chamber studies that are representative of the entire population. With this in mind, we have selected a set of epidemiological results that are widely accepted within the scientific and policy arena and show a consistent statistical relationship between its ambient air pollution level and the exposed population's health. In addition, we have also a smaller set of health relationships that are not as widely accepted but have strong epidemiological results that will serve as a sensitivity analysis to our studies. In a sense, the additional "uncertain" health effects will provide a ceiling as the maximum level of health and economic effects of urban air pollution.

In this chapter, a survey of the current health studies are presented along with the selection of results that will be used to calculate the economic effects of air pollution in a general equilibrium economic model – the Emissions Prediction and Policy Analysis (EPPA), developed at MIT. The air pollutants considered for this study include particulate matters (PM<sub>10</sub>), nitrogen dioxide (NO<sub>2</sub>), sulfur dioxide (SO<sub>2</sub>), ozone (O<sub>3</sub>), carbon monoxide (CO) and nitrates. These are, for

the most part, “criteria” air pollutants as identified by the EPA. Criteria air pollutants are designated by EPA as the most common air pollutants and its effects are of particular concern to the population and the environment at large. In addition, nitrate is also added for sensitivity analysis due to recent epidemiological studies [ref 1 and 5] that show strong linkage between it and several health effects. The only “criteria” pollutant not included is lead which has been virtually eliminated from the air since the late 1980s.

## **2.2 Epidemiological Studies**

### **2.2.1 Introduction and Types of Health Studies**

Health studies aimed at understanding the correlation between exposure to air pollutants and human health can be placed into two major categories: chamber and epidemiological studies. Chamber studies involve exposing humans and laboratory animals to carefully controlled closed environments where exact dosages of air pollutants are known. Physiological conditions of human subjects including pulse rate, altered lung function, and coughing among others are carefully recorded along with the air pollutant levels. Animal studies allow for longer and larger dosages, and more invasive investigation of physiological effects. The obvious advantage of chamber studies is that the exact correlation between air pollution intake and physiological change can be correlated using rigorous laboratory and scientific analysis. But several factors preclude chamber studies from being the de-facto choice in understanding the relationship between human health and air pollution exposure. Due to the potential harmfulness of the study, it is extremely difficult (if not impossible) to get enough subjects to fully represent the entire population and expose them to an extended period that would simulate chronic exposure health conditions. Scaling from animal studies to human exposures also introduce uncertainties. These concerns limit the usefulness of these studies and in fact they are mostly used to verify specific physiological effects of pollution and for comparison purposes against the second type of study – epidemiological analysis.

Epidemiological studies estimate a statistical relationship between health endpoints and pollution levels as measured across different populations or individuals. The health end-point data is usually gathered through hospital records, health statistics and questionnaires to study participants. Among epidemiological studies, there are two different types of analysis – cohort-based and population-level ecological studies.

The cohort study can follow a set of prospective cohorts and track their health and exposure to ambient air pollution forward in time or study a set of retrospective cohorts and gather historical data on health impacts and past air pollution levels. Studies can involve tracking multiple sets of population with each exposed to different levels of pollution. These studies can also use data from individuals, including health status (where available), individual exposure (not usually available), and individual covariates or risk factors, observed over time. Studying the statistical differences between the populations' health endpoints and pollution level can give scientists a good estimate of the relationship between the two. This set of tests is most commonly used for detecting health effects from long-term or chronic exposures to ambient air pollution.

The second type of epidemiological study is a population-level ecological study. These studies follow the relationship between population-wide health statistics and ambient levels of air pollution. Most of these studies look at the daily variations in human health reports vs. any changes in ambient air pollution measurements. This the most common type of study relating one time health effects with acute exposure to local air pollution.

Chamber studies are valuable for establishing the specific mechanisms or physiological effects of the pollutants but it is very difficult to use these to estimate affects on pollution on a population. The rest of the thesis concentrates on the results of epidemiological studies.

### **2.2.2 Epidemiological Study Results**

Ever since EPA's clean air act, a large number of studies have been done to try to quantify the relationship of various air pollutants to health effects. Many of these studies address a limited set of pollutants or examine limited local population (ref 21). Because this report aims at producing a comprehensive framework for studying air pollution valuation, a full set of compatible studies for all the air pollutants and effects is needed.

Fortunately, there have been several attempts to synthesize the disparate epidemiological study results. One of the most comprehensive studies of this kind is the European Externality of Energy (ExternE) assessment. At the time of its second release (1998), it had reviewed and synthesized all relevant health studies into one coherent set of air pollutants and end health effects that were statistically relevant. The following tables summarize the ExternE assessment. The first table lists the pollutants and the health effects that are most accepted by the scientific field, while the second table shows the more uncertain relationships which will be used in the sensitivity analysis.

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 <sub>2</sub>	2.04E-06	Ponce de Leon 1996
		O <sub>3</sub>	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 <sub>3</sub>	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 <sub>2</sub>	0.072%	Anderson et al 1996, Touloumi et al 1996
		O <sub>3</sub>	0.059%	Sunyer et al 1996
	Chronic Mortality	PM2.5	0.64%	Pope et all 2002
		PM 10	0.40%	

Receptor	Impact Category	Pollutant	E-R fct	Reference
Children				
	Chronic Bronchitis	PM 10	1.61E-03	Dockery et al 1989
		Nitrates	1.61E-03	
		PM 2.5	2.69E-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 <sub>3</sub>	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	
		CO	5.55E-07	

Receptor	Impact Category	Pollutant	E-R fct	Reference
Asthmatics				
All				
	Asthma attacks	O <sub>3</sub>	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
		Nitrates	1.68E-01	
		PM 2.5	2.80E-01	
	Lower respiratory symptoms (wheeze)	PM 10	6.10E-02	Dusseldrop et al
		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 respiratory symptoms	PM 10	1.03E-01	Dusseldrop et al 1995

	(wheeze)	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	

Table 1 Extern-E compilation of health effects

Receptor	Impact Category	Pollutant	E-R fct	Reference
Entire Population				
	Respiratory hospital admissions	NO <sub>2</sub>	1.40E-06	Ponce de Leon, 1996
	Emergency Room Visit for Chronic Obstructive Pulmonary Disease	PM 10	7.20E-06	Sunyer et al, 1993
		Nitrates	7.20E-06	
		PM 2.5	1.20E-05	
	Emergency Room Visit for asthma	PM 10	6.45E-06	Schwartz, 1993 and Bates, 1990
		Nitrates	6.45E-06	
		PM 2.5	1.08E-05	
		O <sub>3</sub>	1.32E-05	Cody, 1992 and Bates, 1990
	Acute Mortality	CO	0.00%	Touloumi et al, 1994
		NO <sub>2</sub>	0.03%	Sunyer et al, 1996, Anderson et al, 1996

Table 2 Extern-E compilation of uncertain health effects – sensitivity analysis

In general, the health effects are divided into two major categories: morbidity and mortality. For morbidity, the studies found that the air pollutants were significantly linked to the following types of health impacts: hospital admissions, emergency room visits, restricted activity days, and increased cases of bronchitis, wheezing, and coughing. Hospital admissions were largely due to air pollution's negative effect on lung, heart and brain functions which resulted in respiratory, cardiovascular and cerebrovascular illnesses. Emergency room visits were mainly from respiratory problems caused by sudden high exposures of PM and Ozone. Another health end point is a restricted activity day (RAD) is defined as a day when a study subject was forced to alter his or her normal activity. RADs include days off work for employed adults and days off school for children, whether or not the subjects were confined to bed on those days. A separate category of minor RAD was created to cover cases where there was no work or school loss but other limitations on the general livelihood of the person was noted. In general health studies have correlated levels of air pollution to levels of health effects through the exposure rate (ER) term. Exposure Rates (ER) values for morbidity are interpreted as following:

Morbidity: # of cases = ER \* concentration level \* population

where ER here has the units of cases / (person year ug/m<sup>3</sup>)

Mortality effects can be further broken down as those due to acute or chronic exposure to pollution. Acute exposure mortality effects refer to the cases of sudden death when exposed to higher pollution level. In general, these cases resulting from exposure to higher ambient urban levels of pollution are limited to people who are near to their "expected" death age. In most literature, the lost time estimated to be approximately 0.25 to 0.5 years. The second category of mortality effect includes cases resulting from chronic exposure of air pollution where a person's life is shortened gradually over its entire course due to a constant intake of these harmful pollutants. Examples are fatal heart or lung diseases that develop only after years of exposure and eventually cause an early

death. The relationship between mortalities and exposure to pollution levels are defined as:

$$\text{Mortality: } \% \text{ increase / year} = \text{ER} * \text{concentration level}$$

where ER here has the units of % change in annual mortality rate / ( $\text{ug}/\text{m}^3$ ).

While cases of mortality related to acute exposure to severe air pollution were well documented by the 1970s, it was not until the early nineties that epidemiological studies began to surface that suggested increased mortality levels even with low concentration levels of air pollution over a long period of exposure[ref 2]. Several recent studies [ref 2,3,4], suggest that long-term exposure of even low level air pollution is not only hazardous but may be of larger consequence than short-term acute effects [ref 2]. Most long-term mortality effects from air pollution are based upon cross-sectional studies or more recently, prospective cohort studies.

For example, the largest of these studies, the American Cancer Study by Pope [ref 2,3], had 1.2 million adult (>30 yrs of age) volunteer participants residing in all 50 states, District of Columbia and Puerto Rico. Vital statistics including age, sex, weight, height, smoking history, alcohol use, occupational exposures, diet, education, marital status and other characteristics were collected and used in future statistical analysis as either indicators or control variables [ref 2]. The 1.2 million volunteers were then restricted to those who lived within an urban area with available pollution data. Mean concentrations of air pollution for the metropolitan areas were compiled from various data sources [ref 2] and calculated for each metropolitan area during the 1 to 2 years prior to enrollment. In the 16 years of study, death certificates were gathered for 98% of the mortalities within the cohort. The baseline analysis of this study used these mortality levels in all the metropolitan cities with different ambient PM levels to estimate the adjusted relative risk (RR) ratios (similar to the exposure-rate value described above) for chronic exposure to PM. Any person(s) who moved or were still alive at the end of the study were not considered in the statistical analysis. The study found that mortality levels were positively correlated with different PM

levels even after controlling for cigarette smoking, BMI, diet, occupational exposure, other individual risk factors and any regional or other spatial differences.

<b>Causes of Mortality</b>	<b>Adjusted RR associated with a 10 ug/m<sup>3</sup> difference in PM<sub>2.5</sub> (95% CI)</b>
All-causes	1.04 (1.01-1.08)
Cardiopulmonary	1.06 (1.02-1.10)
Lung Cancer	1.08 (1.01-1.16)
All other causes	1.01 (0.97-1.05)

Table 3 Mortality RR levels from extended Pope's study [ref 2]

The RR levels shown in Table 3 are the increase in mortality for that specific cause due to an increase in chronic exposure of PM<sub>2.5</sub> of 10 ug/m<sup>3</sup>. Using those RR values, the number of deaths at different pollution levels can be calculated. Table 4, shows the mortality effects in Boston at various pollution levels using Pope's ACS study results. These results are based upon a population of 1 million with an annual nominal mortality rate of 5% or 50,000 and a zero threshold assumption.

<b>PM<sub>2.5</sub> level (µg/m<sup>3</sup>)</b>	<b>Increase in mortality rate</b>	<b>Total Mortality</b>	<b>Extra Mortality from Exposure to PM</b>
0	0	50,000	0
10	5%*4% = 0.2%	52,000	2,000
50	1%	60,000	10,000
100	2%	70,000	20,000

Table 4 Example of Linearity Assumption with Pope's Mortality Study

An important assumption present in the Pope et al's cohort study is that each participant has been exposed to the same relative pollution levels throughout their entire life and that the exposure is directly related to the measured ambient pollution levels in the urban area.

Studies on particulate matter (PM) health effects, in particular its effect on increased mortality, have come under heavy scrutiny due to both its relative high impact values and the underlying assumptions and statistical work involved. Several experts have argued that the association between fine particulate matters and increased mortality rates were due to inadequate control of important individual risk factors. After extensive independent audit and reanalysis of the original study [ref 2], Pope published a new set of results in 2002 [ref 3] that confirms earlier study results and contained more data and controlled for new factors such as smoking, education, marital status, BMI, alcohol consumption, occupational exposure, diet variables, other pollutant levels and regional and spatial differences. Although the new analysis confirms the previous study results, there still remain some controversies. Unlike a substance such as CO that is clearly toxic at high enough levels, particulate matter is a mix of substances, many inert, some toxic (heavy metal) and the exact mix varies across different regions. Thus, the underlying epidemiological analysis for the exact amount of effects remains hard to verify through other means such as chamber studies.

Other assumptions used in the rest of the study include linearity and no threshold for health effects from exposure to ambient air pollution.

The linearity assumption states that the total number of health impacts (regardless of the type) is linearly proportional to the level of pollution regardless of a baseline threshold for the pollution to affect a population. While different people respond differently to varying levels of pollution exposure, on the whole, most studies have not found strong evidence for a non-linear relationship between concentration level and health effects is an accurate representation. In

fact, the linearity assumption is used widely in almost all health cost studies these days [ref 5, 21].

One form of non-linear relationship that has been much debated is whether there is a threshold below which no damages occur, and then a positive relationship (possibly linear at higher levels). Realistically, most people these days do not become sick from normal exposure to the ambient background air pollution. However, there is no good evidence of a threshold at the population level (ref. 5). For large population groups, there may always be people who become adversely affected by even the smallest amount of air pollution exposure. In fact, as of last check, the World Health Organization (WHO) has also adopted a “no threshold” position for particulate matters and ozone.

Furthermore, even with the adoption of a threshold level, these are usually so low that they do not have a meaningful contribution to any cost and benefit studies involving air pollution control policy. I.e. the difference between policy and no policy usually does not ever involve levels as low as any reasonable threshold. However, the existence of a threshold would be important in our “green” scenario estimates.

### **2.2.3 Past Valuation of Health Impacts**

In order to gain an aggregate understanding of the costs of the health impacts from urban air pollution and to compare different endpoints on a similar basis, environmental economists seek to monetize these health endpoints. Assigning a monetary value to each of the health impacts allows policy advisors to compare expected benefits of reducing pollution to the costs of doing so. While an important task, it generates additional controversies and uncertainties [ref 9]. The costs of the health effects are complicated and involve both market valued goods (such as lost work time and increased purchase of medical services) and non-market goods (such as lost leisure time and the agony and suffering of the individual). Both the methodologies used in valuing these costs

and its application are among some of the more heated debates in environmental economics and policy [ref 9].

The two most commonly accepted valuation methods for both morbidity and mortality are the contingent valuation method (CVM) and the cost of illness (COI). The total value of any illness is composed of: the value of the time lost because of the illness, the value of the lost utility because of the pain and suffering and the costs of any expenditure on averting and/or mitigating the effects of the illness [ref 5].

The costs of illness (COI) is the easiest to measure and are usually based on the actual expenditures associated with different illnesses or on the expected frequency of the use of different services for different illnesses. These values can normally be obtained through hospital and/or insurance records. Furthermore, if the illness involves some loss of performance at work either due to loss of ability or work time, COI would include those costs as well. The COI, in general, can be thought of as all the market values of any illness and would have a direct effect on the GDP of any country. The biggest drawback of this method is that it does not value non-market costs such as pain and suffering. For those valuations, the CVM is currently the only accepted methodology. Loss of leisure (non-work time) is often valued as part of a CVM study, but, in principle, the time lost can be estimated and then valued at a prevailing market wage.

Contingent valuation is mostly based upon questionnaires that are designed to tease out the values that individuals place upon these non-market values in the context of the illness through either willingness-to-pay (WTP) or willingness-to-accept (WTA) values. As their names imply, WTP means the amount of money a person is willing to pay to reduce their chance (risk) of getting an illness, and WTA is the amount of money it takes for a person to willingly accept a risk increase in getting the illness. Because the WTA can be unbounded, most CVMs are designed to find the WTP value. This exercise is extremely difficult and usually results in very controversial solutions that span a wide range.

For this thesis, the costs for each of the illness (apart from mortality – see section 3.3.2) are taken directly from the ExternE study [ref 5]. The ExternE team utilized the extensive US literature on this subject to come up with a comprehensive and systematic set of values for each of its health endpoints:

<b>Health impacts</b>	<b>Costs in US dollars 2000</b>
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	\$106

Table 5 Morbidity Valuation from Extern-E

The costs stated in Table 5 represents both the market and non-market costs of these illnesses. While this collection is the best point estimate of the health effects, in order to truly gain a sound economic valuation over time, these costs need to be folded into preference and utility functions of an economic model. This is the topic of the next chapter.

## Chapter 3

### Economic Framework and Integration into EPPA

#### 3.1 Introduction

Health impact valuations up to now have been limited to using a mixed set of market and non-market valuation approaches. Mostly, analysts have relied upon the use of point estimates (see Table 5) derived from gross production/consumption loss or willingness to pay surveys. These methodologies are based upon partial equilibrium concepts and while such estimates are sound under some circumstances, they do not take into consideration the potential economic response and interactions that may occur in the economy as represented in general equilibrium models. Further, the reason for valuing damages in the first place is for cost-benefit analysis, where the marginal value of avoided damage (i.e. the policy benefit) is compared with the marginal cost of the policy. But with marginal cost derived from one analysis exercise and benefit from another it is difficult to insure consistency in underlying data on wage rates or other economic values that, if not treated explicitly in the analysis, are at least implicit in the answers of survey respondents.

In this section, we describe an approach for directly incorporating pollution health damages within a computable general equilibrium economic model – MIT's emission projection and policy analysis (EPPA). Once the health impacts are introduced in the model the valuation of policy benefits is endogenous, and reflects responses and interactions represented in the model. Moreover, EPPA was designed specifically to simulate pollution emissions that are commensurate with the overall economic growth in different parts of the world. This provides an ideal solution to incorporate feedbacks on economic growth and its effects on emission. Other benefits of such an approach include valuations of air pollution effects that are directly calculated in welfare terms measured as equivalent

variation. The approach draws from and builds on efforts to include non-market environmental effects in national income and product accounts (NIPA) (ref 17) in that we begin by identifying where health effects lie in the underlying Social Accounting Matrix (SAM) for an economy, the underlying data for CGE modeling. The SAM is built directly from the NIPA accounts. A related approach is that of Kerry who incorporated pollution health damages in the utility or preference functions of consumers to capture these effects (ref 15, 16).

### 3.2 MIT Emissions Prediction and Policy Analysis (EPPA)

#### 3.2.1 General Overview

The Emissions Prediction and Policy Analysis (EPPA) model is a component of an integrated framework of natural and social science models being developed by the MIT Joint Program on the Science and Policy of Global Change. It is a detailed, global, computable general equilibrium (CGE) model with a long time horizon and regional as well as sectoral detail (ref. 19).

The EPPA model is a multi-region, multi-sector, recursive-dynamic computable general equilibrium (CGE) model. The world is divided into the sixteen economic regions shown in Table 6, which are linked by international trade. The economic structure in each region consists of eleven production sectors and four consumption sectors, all shown in Table 6, plus one government and one investment sector.

Regions:		Production Sectors:	
USA	United States	AGRI	Agriculture
CAN	Canada	COAL	Coal
MEX	Mexico	OIL	Crude Oil
JPN	Japan	ROIL	Refined Oil
ANZ	Australia - New Zealand	GAS	Gas
EUR	Europe	ELEC	Electricity
EET	Eastern Europe	EINT	Energy-intensive Industries
FSU	Russia Plus	OTHR	Other Industries
ASI	East Asia	SERV	Services
CHN	China	TRAN	Transport
IND	India	CGD	Savings Good

IDZ    Indonesia AFR    Africa MES    Middle East LAM    Latin America ROW    Rest of the World	
<p style="text-align: center;"><b>Primary Factors</b></p> Labor Capital (by vintage) Sector-specific fixed factors for each fuel Land in agricultural	<p style="text-align: center;"><b>Consumer Sectors:</b></p> All production sectors + PTRN      personal transport

Table 6 EPPA model's key dimensions

The EPPA model is calibrated with the 1997 base year data. The data set consists of Social Accounting Matrices for each of the 16 regions, and a international trade matrix. Figure 1 shows a simplified example of a SAM leaving out government, investment and trade. The elements of the SAM are the input-output relationships among production sectors, the factors (labor, capital, land, and energy resources) supplied by households and used by the production sectors, and the final consumption goods. In this simplified representation, final consumption must equal total income. In the fuller development of the SAM (as modeled in EPPA), government is treated as another end user and consumers have the option of investment as another final use of goods. The rest of the thesis will only refer to this simplified representation of the SAM table as all the variables that we need are shown here.

	Production sectors	Final Consumption
Production sectors	Input / Output	Goods
Factors	Labor, capital, resources	Total consumption = Total income (factor payments)

Fig. 1 Example SAM table layout

Each of the eleven production sectors are described by a nested constant elasticity of substitution (CES) production functions, see figure 2, that combines the output of other sectors as material or energy inputs, and uses labor and capital as primary factors. Various natural resources (such as oil, coal, gas, land) constitute an additional primary "fixed factors" input that enter relevant production sectors. The consumption equation is also described as a CES function.

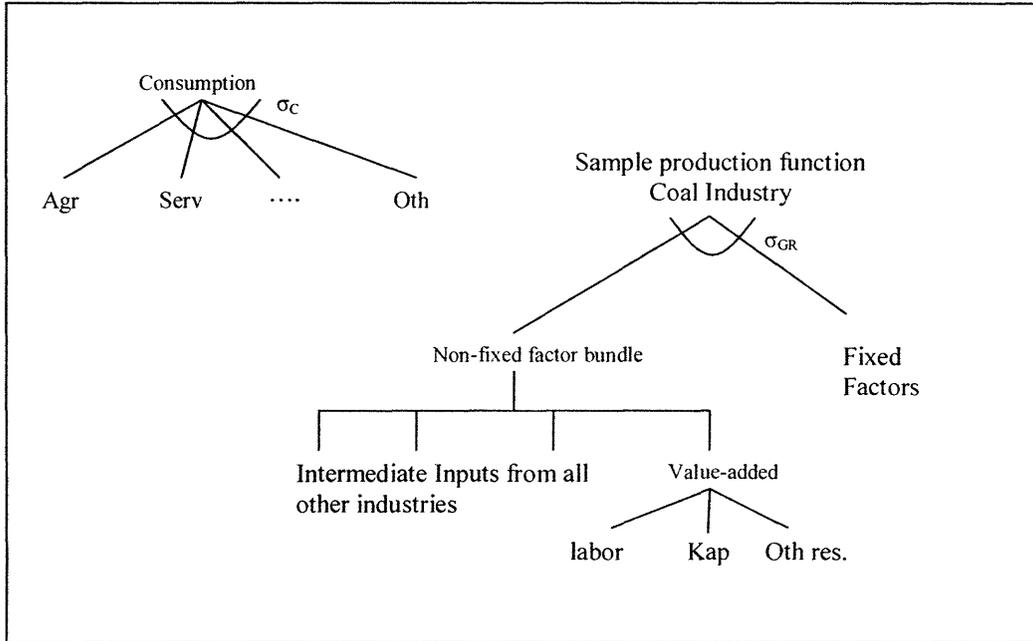


Fig. 2 Sample consumption and production structure in EPPA

The key parameters are the elasticity of substitution (shown as  $\sigma_C$  and  $\sigma_{GR}$  in the figure above). The consumption elasticity represents the willingness of consumers to substitute their consumption of one good for another. In production, the elasticity is the technical capability to substitute one input for another. In most cases, these elasticity values are ascertained from expert solicitations. More detailed discussion of the model along with the general equilibrium equations can be found in references 13 and 19.

### 3.3 Framework for Adding Pollution Effects Into Economic Model

In this section, we discuss the basic underlying ideas and frameworks associated with adding the effects of urban air pollution into the CGE model. The two main areas of focus are: an expansion of the SAM table and the CGE model to include a household production of health to combat the effects of the urban air

pollution and the development of a new stock-and-flow model for dealing with mortality from chronic exposure to particulate matters.

### **3.3.1 Expansion of CGE / SAM**

Health impacts from urban air pollution affect the economy, through lost labor, greater demand for health care, and lost leisure. From past health studies (detailed in Chapter 2), exposure to higher levels of air pollutants can cause responses ranging from increased coughing and asthma attacks to hospital visits for respiratory and cerebrovascular symptoms to even higher levels of mortality. All of these health impacts affect the economy differently. For mild symptoms such as coughing and asthma, the majority of lost value is on the suffering and loss of leisure for the patients (non-market effect). These symptoms are measured in number of occurrences and last for a short period of time (less than a day or two). For more serious health consequences such as chronic bronchitis, heart disease and hospital visits due to respiratory and cerebrovascular problems, the economic impacts may include lost leisure (non-market), lost labor (market) and an increase in hospital and medical services (market). To recover from these more serious health implications, individuals not only need to rest and give up both work and play but also pay for more and better medical care.

The basic SAM tables for economic analysis normally include only market related goods. As discussed above, health effects include not only these market related labor time but also a loss of non-work time enjoyment for working adults and children and elderly time as well. In order to capture all of these effects, the basic SAM table as shown in figure 1 is updated to include a production/consumption sector called “household production of health” (figure 3) and non-working time (i.e. leisure) into EPPA. Leisure is added into the EPPA economic model in an effort to capture the “non-market” effects of the health endpoints while “household” production sector is setup to capture all the necessary inputs for keeping the population healthy from exposure to urban air pollution. We refer to this household production sector as ‘household production

of healthcare' but note that rather than breakout the entire household health production activity and medical services sector we focus just on demands associated with environmental health damages. These demands are then drawn from the aggregate 'Service (SERV)' sector in EPPA where the medical service sector is included. The new household healthcare sector takes as input the amount of lost labor (both leisure and work labor) and healthcare services that are required by the affected population to regain their health. Figure 3 illustrates this process through the expansion of SAM table to include both the "household production of health for air pollution" and the non-work time.

	Production Sectors	Household Production	Final Consumption
Production Sectors	Medical Services Input Output	Household Mitigation of Pollution Health Effects	Increased demand for medical services to maintain health Goods  Less other goods for consumption
Factors	Labor, Capital, Resources	Household labor	Leisure / non-work time

Fig. 3 The additional of Household Healthcare production sector

Within the SAM table, the new household health production sector takes as input both medical services from the original production sectors (for full list of production sectors in EPPA see Table 6) and both workforce labor and the new leisure (non-work) labor. Different illnesses will have different inputs. For example, the illness of restricted activity day (RAD) is significantly different than hospital visits for cerebrovascular disease. For RAD, in order to recover, the patient mostly stays home from work and as a further consequence of the illness, does not fully enjoy his/her non-work (leisure) time either. For the more serious

cerebrovascular disease, not only are lost work and leisure time part of the equation, the recovery from such an illness would also require large medical expenditures as well. Furthermore, some of the health effects only affect a small subset of the population. For example, quite a few of the health effects only affect elderly folks which means besides medical services, only non-work time will be lost. For a complete listing of all the different health effects from exposure to urban air pollution and the inputs (lost work time, lost leisure time and/or medical services) that are necessary to make the patient feel better see Table 7 in the next section.

Overall, as total pollution increases (decreases) over time, the “household pollution effect mitigation” sector will require more (less) of the two inputs, medical service and labor. This will have a ripple effect on other goods and prices. As more medical service is needed, more of the factors of production (land, capital, natural resources) will be redirected towards that sector and away from other sectors. Thus if medical services is labor intensive then labor prices will go up relatively vs. capital and resources. Furthermore, labor will be diverted from producing other goods towards keeping the population at the same level of healthiness. In the end, the total welfare of the economy will be lower as the total amount of goods produced is decreased from both the loss of production factors (mainly labor) and redistribution of the remaining factors to produce more of the medical services that are needed to maintain health in the face of air pollution.

Several issues arise regarding the inclusion of non-work (leisure) time. Often when leisure is added to CGE models, the reason for doing so is to create a labor force response to the changing wage ratio [ref 23]. In those cases, a benchmark quantity of leisure might be chosen to reflect a maximum amount of additional labor that could ever be supplied. For example, Babiker, Metcalf and Reilly [ref 23] chose a quantity of 20% of the labor force. This amount can be viewed as the maximum extra labor that might be injected into the workforce depending upon the labor-leisure elasticity. However, our interest is not focused on leisure as a potential supply of labor to the economy but on its value to households as either leisure or as household labor. In this thesis, we follow the

economic theory that on the margin, people value their leisure time just as much as their working time (i.e. the value of an extra leisure minute is the same as how much they are being paid for work). This makes the leisure value of the working adults to be equal to its labor value and we can get an estimate of the children and elderly hours though the wage distribution of the US. For details of this value and actual implementation into the CGE EPPA model see section 3.4.

### **3.3.2 Framework for Modeling Mortality in the CGE Model**

The economic effects of mortality cases persist in time. For example, the economic effect of someone who passes away at the age of 60 from air pollution exposure, who otherwise would have lived until 75, will persist over that remaining 15 years. This differs from effects such as RADs or hospitalizations that are events where the direct effect is seen only in the period in which the event occurs. The difference is one of stock and flows. Mortality is a change in the stock of available labor (and leisure) whereas a morbidity illness is an interruption in the flow of services. In the example above where the person passed away at age sixty instead of 75, he/she would have lost both work labor time from 60 to the normal retirement age of 65 and leisure (non-work) time from 60 until her normal death age of 75. It is necessary to keep track of this prolonged economic effect to gain a complete understanding of the effects on the overall economy from mortality due to exposure to air pollution. The exact modeling techniques as they apply to this model will be detailed in the next section.

The second stock and flow problem is the mortality due to chronic exposure to pollution. The economic characterization of the mortalities from chronic exposure to air pollution has not been adequately modeled in past assessments of health and economic damages due to air pollution. Almost all of the economic analysis of the costs of air pollution has made the simplified assumption that as Particulate Matter increase (decrease), all of the changes in mortalities occur immediately (see figure 4). This assumption is not correct.

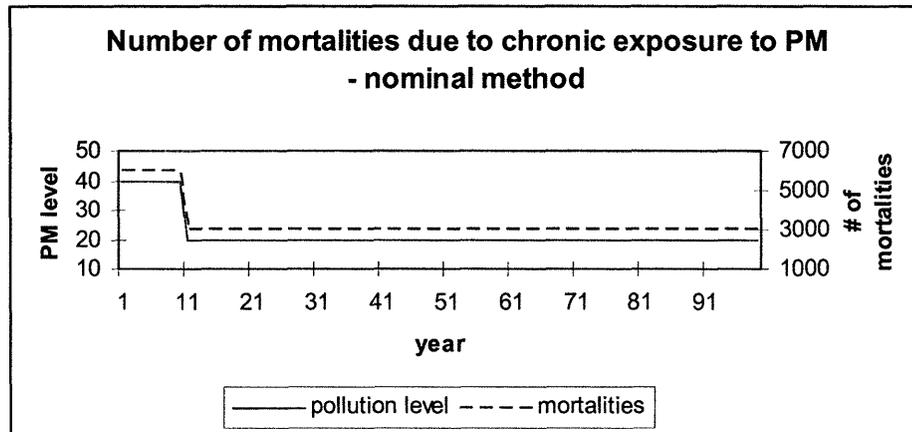


Fig. 4 Pollution level vs. mortality level due to chronic exposure to pollution – past methodologies.

While this simple assumption is true in terms of total number of people affected due to that year's increased pollution, the timing and value of the effect is not correct. From the epidemiological studies (primary Pope et al studies [ref 2 and 3]), most of these mortalities are due to long term exposures which means any mortality from increased PM levels in one year will most likely not take place until sometime in the future and will not affect overall economy for many years. In fact, the extra mortalities are only gradually realized after the pollution level change. Over time, the build up of PM in-take by the general population decreases (increases) to reflect the lower (higher) overall pollutant level and the overall mortality levels will reflect that change as time passes. In the first few years of pollution change, most of the population has only been exposed to the original pollution level for their entire life and a sudden change for a single year does not greatly alter the expected mortality for that year. This difference presents a major problem for past cost & benefit studies. First, the cost of mortality to a society is very large and will have a large affect on any cost and benefit analysis for urban air pollution policies. By incorrectly identifying when these mortalities occur, the true costs of urban air pollution will be misguided. Further, if one is trying to study the efficacy of a policy designed to reduce urban air pollution, it is even more imperative to know when the benefits (lowered

mortalities and morbidities) are likely to occur and again the timing of such matter will have a large affect on the benefit side of the story.

Here, we propose a new methodology using the stock and flow methodology to estimate the changes in mortality levels due to changes in PM concentration. The basic idea involves three steps. First, we have to determine how PM mortality affects the population. Does the age distribution of mortality from PM resemble normal (average) mortality levels or is it different? Studying the epidemiological studies (ref 2, 3), we find that the highest statistical increases in mortality levels due to higher PM concentration are lung and cardiopulmonary diseases. Figure 5 shows a comparison of the normal mortality distribution to the mortality distribution for lung and cardiopulmonary disease.

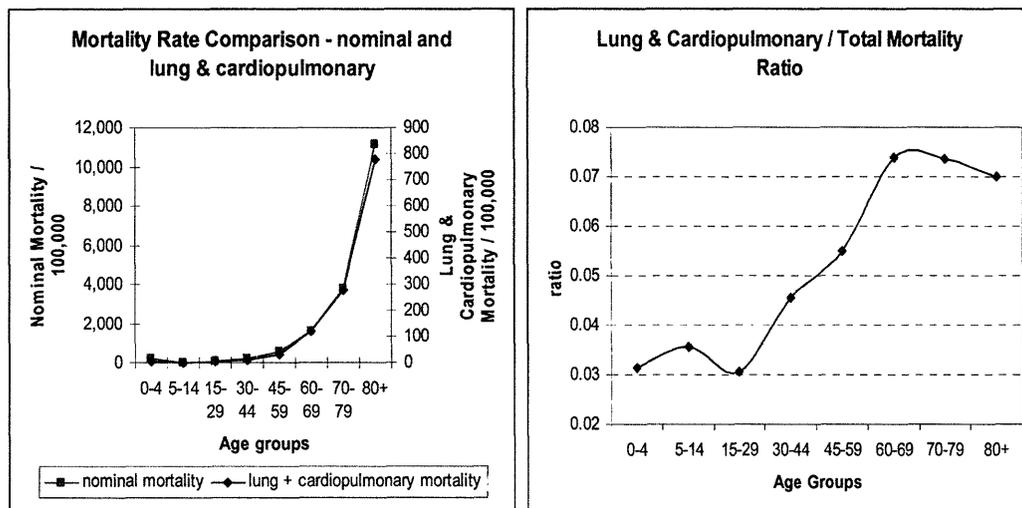


Fig. 5 Lung and Pulmonary Mortality distribution compared to nominal total mortality

Furthermore, it was noted by Pope et al that since the mortalities are due to chronic exposure, all of extra deaths occurred only in people older than 30. Using the above information along with the increased risk ratios due to PM intake, we find the percentage increase in mortality due to chronic exposure to PM if the population was exposed to the PM levels for its entire lifespan:

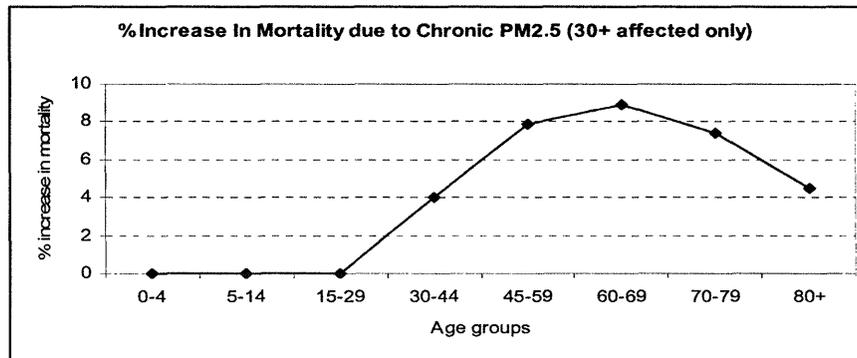


Fig. 6 Percentage increase in mortality for each age group due to 10 µg/m<sup>3</sup> of PM exposure over their lifetime.

Using the information from figure 6, one can calculate the expected increase in mortality levels of the population in a certain year if we know the past PM pollution concentration levels. By adding up all the pollution in-take of the population in the years past, an average pollution exposure level can be determined for each age group:

$$average\_PM_n = \frac{\sum_{i=1}^n PM\_level_i}{n}$$

$$\%\_inc\_mort_n = nom\_mort\_level_n * average\_PM_n * \%\_inc\_mort_n\_per\_unit\_PM$$

where the subscript *n* represents the number of years an average person has lived in that age group. Note, the *average\_PM* variable serves as a stock measure of how much PM pollution an average person in that age category has been exposed to in his or her lifetime so far.

Once we have calculated the total stock of PM pollution in-take by the population, we are now ready to deal with the changes (flow) in the PM in-take by each age group as the pollution level changes and the population becomes older. This is most easily demonstrated through an example. Lets take the case that the PM level had been constant at 10 µg/m<sup>3</sup> and now increased to 20 µg/m<sup>3</sup> for this current year, how will that affect the mortality levels in this and

future years? Let us follow the people in the age group of 60-69 year olds. We first calculate the change in average pollution in-take per year for an average person in that category:

$$(20-10)/65 = 0.154 \mu\text{g}/\text{m}^3.$$

Now the increase in mortality levels can be calculated using results from figure 6 and equation 1 above. With this example, the percentage increase in mortality is  $0.154 / 10 * 8.3 = 0.128\%$  increase in mortality for the age group ranging from 60-69 year olds for this year. The full effect of this pollution change will not be realized until 65 years later when all the people in that age group then would have been exposed to this new pollution level for their entire life. At that time, the increase in mortality rate for the 60-69 year old age group would be 16.6% instead of the current 8.3%.

The key assumption in this entire process is that the effect of changing pollution level for the current year,  $\Delta P$ , for the population is the same as changing the pollution level in-take for the entire lifespan of the population by  $\frac{\Delta P}{A_i}$  where  $A_i$  is the average age of each of the age group. Also note that  $\Delta P$  is not the difference between the current pollution level,  $P_j$ , and the previous year's,  $P_{j-1}$ , but is the difference between the current pollution level,  $P_j$ , and the first year of pollution in-take for the age group,  $P_{j-A_i}$ .

Using this stock and flow methodology, we again revisit the case as presented by figure 4 where the pollution level is constant until year 11 when it drops by 50% and then remains at that level forever. In the past, researchers have assumed that all of the gains in lost life are realized right away but with the stock-flow method, the gains in lost life is slowly realized until all of the population has only been exposed to the new pollution level:

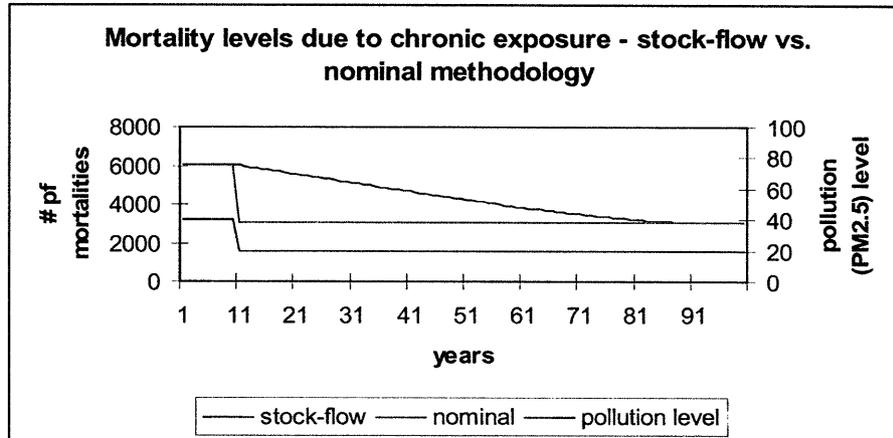


Fig. 7 Stock-flow vs. nominal method of estimating changes in mortality from chronic exposure to PM due to changes in PM level.

### 3.4 Implementation into CGE model (EPPA)

In this section, we discuss the specifics of modeling the expansion of the CGE to include “household production of health” and the inclusion of mortality from air pollution. The main issues here involve determining the new utility functions for the “household production” sector, how much non-work/leisure time to add in, how to take out the extra mortalities from the labor and non-labor market, and the calibration of the first year data (1970) to run the model.

#### 3.4.1 New utility functions for the “household production of health”

There are two factors that go into the consideration of adding new utility functions for the household production of healthcare for air pollution effects. The first is inputs necessary into the sector. In our model, there are six different pollutants which will be taken into consideration for this model. Each of them

contributes separately to the healthcare costs and do not interact directly. This makes the elasticity of substitution of between each of the pollutant to be zero. The two main inputs into each pollutant healthcare section are labor (both work time and non-work (leisure) time) and medical service. The amount of input into each of the pollutant healthcare is solely dependent upon the illnesses that are associated with each pollutant and the necessary activities to make one feel better. As discussed in section 2.2, different illnesses requires different methods of healing – some require medical service while other just take time away from work and leisure. We assume the elasticity of substitution for all the inputs into each of the pollutant healthcare cost is zero. This assumes that for each particular illness, people on average do not substitute hospital care (services) and rest (time away from work and lost leisure time). This assumption is extreme perhaps as there may well be willingness to forgo purchased healthcare in certain economic conditions and may need further examination. For the time being, this assumption is consistent with the valuation literature that would simply multiply per case costs times the number of cases [ref 1, 5, 6, 9, 10]. Figure 8 shows a picture of the new utility functions as they are implemented in EPPA. From this point forward, we will refer to this new model EPPA-HE to denote that it is different than the original EPPA model.

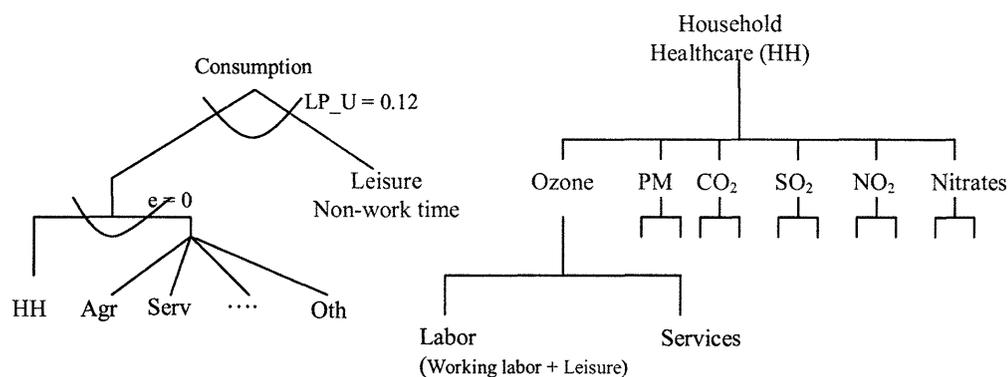


Fig. 8 New consumption functions for the implementation of household healthcare sector and non-work (leisure) time. Red are additional

With the elasticity of substitution of zero between the two inputs (labor and service), we can reformulate the basic demand function, equation 2, as Leontief equations, equation 3:

$$HH = \theta[\alpha_1 L^{\frac{1}{1-\sigma}} + \alpha_2 S^{\frac{1}{1-\sigma}}]^{\sigma} \quad (2)$$

where

- HH is the amount of household healthcare demanded for the particular pollutant
- L is the amount of labor (both work and non-work) used as input
- S is the amount of medical service (coming from the SERV sector) used as input
- $\alpha_i$  are the different shares of L, and S in factor payments
- $\theta$  is a scale parameter for the entire system

With the Leontief assumption, equation 2 reduces to the following form:

$$HH = \theta[\alpha_1 L + \alpha_2 S] \quad (3)$$

Once the year 1 parameters are determined (see section 3.4.4 below), future increases in pollution can be easily captured through the scale parameter,  $\theta$ . If pollution increases, then  $\theta$  would decrease to indicate that more labor, leisure and services will be needed to gain the same amount of health as before. This is again due to the Leontief formulation for the consumption choice between HH and the rest of the goods such as transportation, agriculture and other services and goods (see figure 8). The Leontief formulation means that in a particular case, if pollution increases, more of healthcare service and time off from work

and fun is demanded – removing these resources from other parts of the economy.

Non-work time or leisure is added into the overall consumption function with an elasticity level of 0.12. This is based on the following labor-leisure tradeoff equation:

$$\varepsilon = \frac{\alpha}{1-\alpha} \sigma \quad (4)$$

where  $\varepsilon$  is the labor price elasticity,  $\alpha$  is the fraction of total time that is spent on leisure (leisure / total\_time), and  $\sigma$  is the elasticity between leisure and consumption of goods. For the United States, the current labor price elasticity is most commonly accepted to be between 0.2 and 0.25 (ref 23).  $\varepsilon$  is estimated at 0.64 for the US (see following section for discussion on amount of leisure included in model).

### 3.4.2 Addition of leisure/non-work time

The addition of the leisure involves determining the type and the appropriate amount to add. From the consideration (see detailed discussion in section 3.3.1) that the leisure added in this context is for the valuation of the time to households as leisure or as household labor to overcome illness, the total non-work time added in this model includes all waking hours of children and elderly and the non-working time of adults.

Next, we need to value these non-work hours in a way that is consistent with the working labor wages in the CGE EPPA model. As discussed in section 3.3.1, the methodology of choice here is that for adults their leisure time on the margin is worth just as much as the labor time; and for children and elderly, we look towards the US wage distribution at different ages for guidance (see figure

9). Based on this, we value the time of children to be at 1/3 the wage rate of normal working adults and elderly at 2/3.

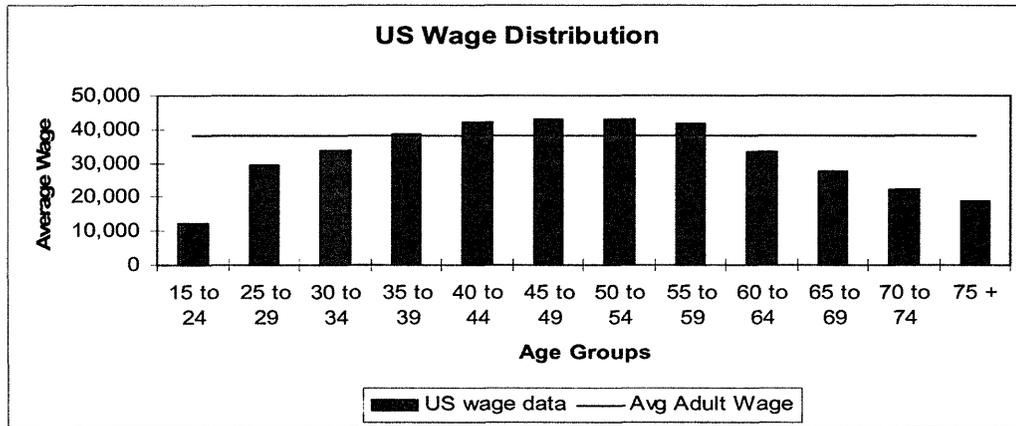


Fig. 9 US wage distribution [ref 22]

Next, to calculate the total amount of labor to add into EPPA-HE, the country's population distribution is needed. In its original setup, EPPA only considers the working population for its labor. To include children and elderly, population under 25 and above 65 must be added. The total new labor (labor + leisure) is:

$$\begin{aligned}
 new\_labor = & labor + adt\_leisure\_hrs * \frac{adt\_leisure\_val}{adt\_work\_val} + \\
 & children\_hrs * \frac{children\_leisure\_val}{adt\_work\_val} + elderly\_hrs * \frac{elderly\_leisure\_val}{adt\_work\_val}
 \end{aligned} \tag{5}$$

The total amount of labor with the addition of non-work time for all children, adults and elderly depends upon the population breakdown at each group. Using the US census reports [ref 8], we find that children and non-working young adults (<25 years old) constitute 35% of the population while the elderly (>65 years old) is another 15%. Because the old EPPA model only incorporated the working adult population (50% of total people in US), we find

(using equation 5) that the new labor is 2.8 times the original. This means that the total leisure labor in the US economy is approximately 64% of total labor.

After the successful first year calibration for the model (see section 3.4.4), all future years, both the leisure and normal working labor hours will grow at the same productivity level:

$$\text{Labor}(t+1) = \text{productivity}(t) * (\text{working\_labor}(t) + \text{leisure\_labor}(t))$$

$$\text{Labor}(1) = \text{working\_labor}(1) + \text{leisure\_labor}(1)$$

And the productivity level is set so that the total actual welfare growth for the region matches the desired growth scenario which, in this case, is the historical GDP growth levels.

### **3.4.3 Dealing with extra mortalities**

Mortalities, unlike other acute diseases, are not one time events that affect the economy at only one point. The correct model needs to carry forward those losses until their expected time of death without the presence of air pollution. The two types of mortality for exposure to air pollution are deaths from acute and chronic exposure. Mortality from acute exposure normally only affect those that are close to their time of death [ref 2, 3, 5] and the commonly accepted loss of time is 0.25 to 0.5 years. This makes the accounting of this type of mortality relatively easy in the CGE model – one needs to only reduce the overall labor force in the year of pollution change by the extra mortality number.

The second type, mortality from chronic exposure, is more complex. These are normally healthy adults who die earlier than their expected lifetime due to their lifetime of exposure to harmful air pollution. As detailed in section 3.3.2, this type of mortality affect people from the age of 30 and onward. To capture

the loss of labor force from the year of death to their normal expected lifetime of 75 years old, the following algorithm is employed:

1. If the person dies before the age of 75 then from their death till when they would have been 75, the economy losses:
  - a. One year of adult labor during every year before the person would have turned 65;
  - b. One year of adult leisure during every year before the person would have turned 65;
  - c. And one year of elderly leisure during the years that the person would have been 65-75.
2. If the person dies after the age of 75, then the economy losses:
  - a. One year of elderly leisure.

This formulation ensures that the stock-flow nature of the mortality is accounted for in the EPPA-HE model.

#### **3.4.4 Calibration of the first year data**

To run the general equilibrium model, EPPA, one must calibrate the values of the first year to match the known input/output (SAM) table to historical economic records. In this particular case, not only did we have to recalibrate the EPPA-HE for 1970, we had to add in all the new terms – leisure, household healthcare, and leisure, labor and service inputs into the healthcare sector.

The calculation of the amount of labor, leisure and services that go into Household Healthcare for the first year depends upon the number of health effects (from epidemiological study and air pollution level of that year) and the valuation of these effects from welfare studies (willingness to pay surveys, and

hospital care costs). Fortunately, the EXTERN-E study, which we are using for health end points, also has compiled a very extensive study on the costs of all these various health effects. In general, they verified willingness-to-pay values from the contingent valuation surveys, where available, to the estimated affliction time period and hospital care costs and came up with one cost for each health end point. I have then taken these values and broken it down into labor, leisure and hospital service sections. The particular steps I undertook to achieve this are:

1. Estimate the total time of affliction for each of the illnesses based upon the description of the illness.
  - a. This time then represents the number of days that the person is suffering from the illness and is neither able to work (adult) nor able to enjoy their free leisure time fully (children, adults, elderly). For example, the restricted activity day (RAD) health end point would involve a full day of lost work and leisure hours.
2. Break those times into concrete number of work hours missed and number of leisure hours not enjoyed. For RAD, this would entail eight hours of work time and eight hours of leisure time for an adult and sixteen hours of leisure time for either a child or an elderly person.
3. Using the known labor and leisure valuation for each age group, I calculate the average labor and leisure lost part of the cost. For RAD, an average person in the population (50% working adult, 35% child, 15% elderly) would loss four hours of adult work and leisure time, four hours of child leisure time and four hours of elderly leisure time value.

4. The rest of the cost is then attributed to hospital visit care (i.e. healthcare services in EPPA terms).

The following table shows the cost breakdown for each of the health end-points used in this study:

Ozone		service	labor	leisure	cost
	RHA (respiratory hospital visits)	0.85	0.04	0.11	14498.20
	Symptoms days	0.50		0.50	13.82
	Asthma attacks AA	1.00			68.20
	MRAD	0.00		1.00	13.82
	ERV for asthma	0.80	0.02	0.18	410.81
	% increase of mortality rate		0.23	0.77	46312.50
	total base cost	12727.19	11275.64	37314.47	61317.31
	total %	0.21	0.18	0.61	1.00
<b>CO</b>					
	Congestive heart failure	0.85	0.00	0.15	14498.20
	Ischaemic heart disease	0.85	0.00	0.15	14498.20
	% increase of mortality rate		0.23	0.77	46312.50
	total base cost	24646.94	10687.50	39974.46	75308.90
	total %	0.33	0.14	0.53	1.00
<b>SO2</b>					
	RHA	0.85	0.04	0.11	14498.20
	% increase of mortality rate		0.23	0.77	46312.50
	total base cost	12323.47	11267.43	37219.80	60810.70
	total %	0.20	0.19	0.61	1.00
<b>NO2</b>					
	% increase of mortality rate		0.23	0.77	46312.50
	Number of incremental deaths in million	0.00	10687.50	35625.00	46312.50
	total %	0.00	0.23	0.77	1.00
<b>Nitrates</b>					
	RHA	0.85	0.04	0.11	14498.20
	Cerebrovascular hospital admissions	0.85	0.04	0.11	14498.20
	Chronic Bronchitis	0.85		0.15	414.00
	Chronic Cough	0.85		0.15	414.00
	RAD		0.35	0.65	138.00
	Chronic bronchitis	0.30	0.20	0.50	193432.00
	Congestive heart failure	0.85		0.15	14498.20
	Cough	1.00			414.00
	Lower respiratory symptoms (wheeze)	1.00			14.00
	Cough	1.00			
	Lower respiratory symptoms (wheeze)	1.00			14.00
	ERV for COPD	0.80		0.20	411.00
	ERV for asthma	0.80		0.20	411.00
	Ischaemic heart disease	0.85	0.00	0.15	14498.20
	% increase of mortality rate		0.23	0.77	46312.50
	Number of incremental deaths in million	109126.88	50582.06	140258.36	299967.30
	total %	0.36	0.17	0.47	1.00
<b>PM10</b>					
	RHA	0.85	0.04	0.11	14498.20
	Cerebrovascular hospital admissions	0.85	0.04	0.11	14498.20
	Chronic Bronchitis	0.85		0.15	414.00
	Chronic Cough	0.85		0.15	414.00
	RAD		0.35	0.65	138.00
	Chronic bronchitis	0.85	0.04	0.11	193432.00
	Congestive heart failure	0.85		0.15	14498.20
	Cough	1.00			414.00
	Lower respiratory symptoms (wheeze)	1.00			14.00
	Cough	1.00			
	Lower respiratory symptoms (wheeze)	1.00			14.00
	ERV for COPD	0.80		0.20	411.00
	ERV for asthma	0.80		0.20	411.00
	Ischaemic heart disease	0.85	0.04	0.11	14498.20
	% increase of mortality rate		0.23	0.77	46312.50
	Number of incremental deaths in million	215514.48	20212.86	64239.96	299967.30
	total %	0.72	0.07	0.21	1.00

Table 7 table of breakdowns for each health endpoint

The table shows the percentage of the cost taken up by each of the inputs after all the division of factors.

Next, with the known historical (1970-2000) pollution concentration trend (see APPENDIX B), we can match the two important characteristics of an economy – the GDP/welfare growth and the labor wage price. To match those values, the productivity growth of labor and the capital investment values are selected. Figure 10 depicts the welfare growth of the United States from the period 1970 – 2000 (this is the historic GDP growth trend):

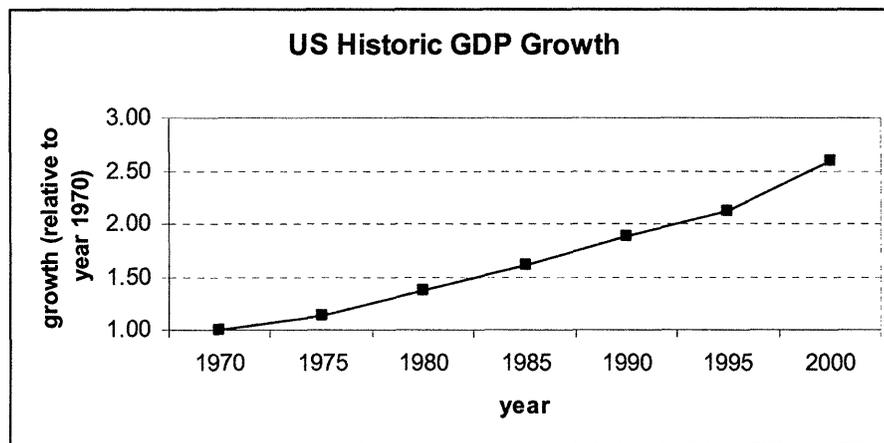


Fig. 10 historical US GDP/welfare growth

### 3.5 Value of life comparison – this model vs. past methods

One of the more unique aspects of this model is our attempt to endogenize the costs of all the air pollution including mortality in a CGE model. In the past, environmental economists have relied mostly on contingent valuation methodologies to approximate the value of lost life due to air pollution. In this model, a lost life is implicitly valued via lost labor and leisure within the CGE framework. The total lost value for mortality is the summation of each lost year of labor whether its work time or non-work/leisure time until the normal time of

retirement and death, which we assumed are 65 and 75 years old respectively for the United States. The benefits of this approach over conventional methods are:

- Consistent and integrated framework for comparing different policy effects and the economic costs/benefits.
- The ability to apply economic valuation across different countries.
- Framework for feedback to the CGE model due to lost lives from increased pollution
- No estimation required for number of lost years from each mortality.

To compare the value of lost life due to chronic exposure to particulate matter, we compare the results of past CVM studies with the valuation as calculated by this model. Since this type of mortality affects people of all ages from 30 and above, we will estimate the value of losing 1 million different people and use the average. First, we calculate the age group distribution of the 1 million people based on figure 12.

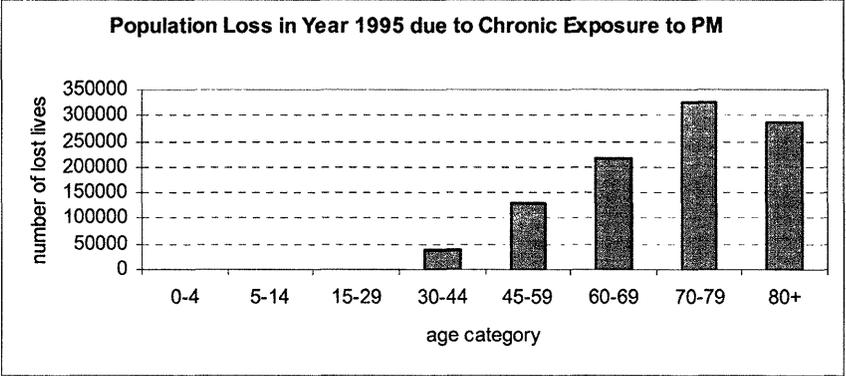


Fig. 11 Age group population loss for the 1 million mortality from chronic exposure to Particulate Matter

Out of the 1 million lost lives, there were 327,000 people who were under the age of retirement, 65. These lost laborers are taken out of the labor input for EPPA until the time when all of them would have retired. The labor losses are

then fed into the EPPA model as population losses. A loss in number of laborers will reduce the overall production capabilities and overall welfare (consumption + investment). Furthermore, everyone who is 75 or younger will be losing leisure time in 1995 and beyond until they reach the nominal mortality age of 75. For those who are in the 80+ age category, it is assumed that they only lost 1 years of their life.

Figure 12 shows the total leisure and labor cost for all one million of the mortalities. From 2025 onward, all the labor welfare losses were due to the vintaging effects of the lost consumption and investment in the first 30 years.

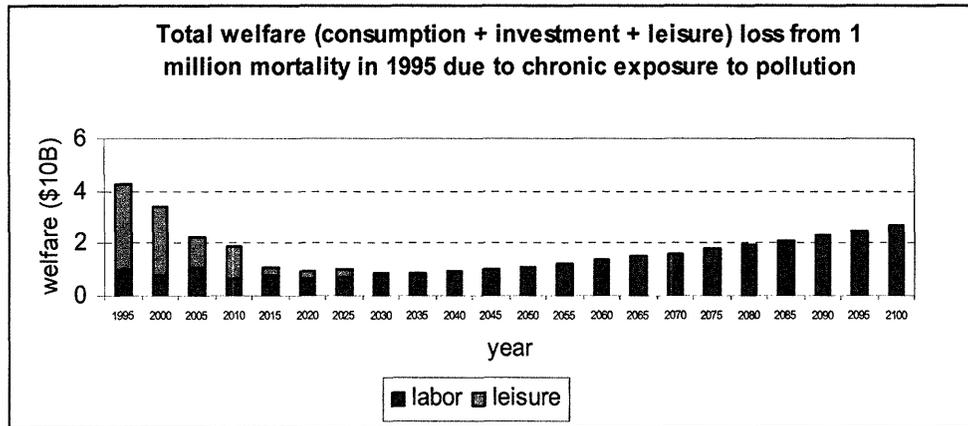


Fig. 12 labor + leisure loss for 1 million mortality in 1995

The average value of each mortality is found by combining and averaging the lost values over all years. Different discount rates are employed in this calculation and the final result is summarized and compared to past values from WTP methods:

	Our Model			Past Valuations		
	Consumption + Investment	Leisure	Total	EPA	EU	ExternE
No discount	1.47	0.46	<b>1.93</b>	<b>4.8</b>	<b>1.4</b>	<b>0.49</b>
3%	0.33	0.38	<b>0.71</b>	N/A	N/A	<b>0.42</b>
5%	0.20	0.34	<b>0.54</b>	N/A	N/A	<b>0.38</b>
10%	0.11	0.28	<b>0.39</b>	N/A	N/A	<b>0.31</b>

Table 8 Value of average mortality from chronic exposure to PM (\$M)

The three values of comparison in this table are taken from the three major cost and benefit studies in the last decade that have studied the cost of air pollution and have estimated the value of a mortality based upon past contingent and willingness-to-pay surveys. The EU\_trans refers to the European Union's transportation air pollution study [ref 1]. The ExternE refers to the comprehensive European study on externalities of energy from which we have taken the full health endpoints of urban air pollution [ref 5]. And finally EPA refers to the recent EPA's studies on the cost and benefit of having the Clean Air Acts regulations [ref 9 and 10].

It is comforting to note that the results from our methodology are similar to the results from other studies.

## Chapter 4

# Economic Valuation of Urban Air Pollution in U.S. 1970-2000

### 4.1 Introduction

This section uses the methodologies outlined in the first three chapters to estimate the economic burden of urban air pollution in the United States from 1970 to 2000 and the benefits of reductions achieved through the Clean Air Act regulations. US economic conditions under three different urban air pollution scenarios are modeled and compared in this chapter. The three scenarios are:

1. “Control” Scenario – in this scenario, the urban air pollutions are the known historical concentrations as measured by the EPA from 1970 to 2000. These pollution levels represent the average pollution level measured from over 200 different urban locations. We then use these pollution levels in EPPA-HE to benchmark the model to produce economic growth levels that are consistent with known historical US economic levels from 1970 to 2000.
2. “No Control” Scenario – in this scenario, the urban air pollutions are at levels that would have prevailed without the EPA’s Clean Air Act regulations. These values are based upon the EPA’s own projections for environmental conditions which would have prevailed without the local, state and federal programs developed under the Clean Air Acts. We then use these uncontrolled pollution levels in the EPPA-HE model to estimate what the economic conditions would have been under this pollution scenario.
3. “Green” Scenario – in this scenario, the urban air pollution concentrations are at levels that are consistent with natural

background levels that one would expect without human pollution. These values are taken from measured pollution concentrations in remote parts of the world such as the middle of the ocean. We use this pollution scenario in the EPPA-HE model to find the economic conditions in the US without urban air pollution.

The differences in the economic conditions between the “no control” scenario and the “control” scenario are the benefits of having the clean air regulations. We compare the results of this study to the benefit analysis done by the EPA in 1990 and 2000. The difference between the “green” scenario and the “control” scenario is the economic burden on the US society due to the still existing air pollution levels in urban areas despite the regulations of the last 30 years.

## **4.2 Simulation Assumptions, Data and Setup**

One set of key data for this study is the historical urban air pollution levels as compiled by the US Environmental Protection Agency [ref 9 and 10]. The focus of the study will be on the criteria pollutants sulfur dioxide, carbon monoxide, particulate matter and ozone and nitrogen dioxide. For the “control” scenario, historical urban air pollution concentration data for urban areas are collected (see APPENDIX A) and used to benchmark the US economic growth from 1970 to 2000 in EPPA-HE – the updated version of MIT’s Emission Prediction and Policy Analysis (EPPA) general equilibrium economic model that include the production of household health to counteract the effects of poor health due to exposure to high air pollution levels. The details of the model setup and the introduction of the health production sector to provide for healthcare needed from air pollution in-take can be found in Chapter 3 of this thesis.

In order to replicate the historical economic conditions with the past air pollution records, we first need to recalibrated EPPA-HE’s year one data to match the historical 1970 US records. To completely re-benchmark EPPA, we would have to reproduce the input-output (SAM) table for the US in 1970 and for

each simulated year thereafter re-benchmark to actual SAMs for that year. we instead used the already calibrated 1997 SAM data (as it existed in the original EPPA program) to include leisure and break out medical services used for air pollution damages (see chapter 3). We then scaled all production, consumption, investment and trade values by the GDP difference between 1970 and 1997. In future work it would be useful to assess effects of this simplification on the overall welfare calculations.

Second, we must calibrate the growth of the “control” scenario to the known macroeconomic growth trends as observed in the US from 1970 to 2000 (for full 1970-2000 historical economic values of interest see APPENDIX C). The key values are the economic growth from year to year and the real labor wage price. To make sure that economic growth is matched to the historical GDP growth levels and the labor wage price matches real wage earning power of the an average worker. This calibrated model is then used to run the other scenarios uder different air pollution levels.

The “no-control” scenario attempts to reflect the environmental and hence the economic conditions of the nation without the standards enacted by the 1970 Clean Air Act. The uncontrolled pollution levels are taken from EPA’s own air pollution prediction models that are based upon pollution emission conditions that would have been present without the regulations detailed by the Clean Air Acts. To find these uncontrolled emission conditions, The EPA used a general equilibrium macroeconomic model to predicted the differences in economic and industrial activity with and without the Clean Air Acts. The full set of predicted air pollution concentration levels for the “no-control” scenario can be found in APPENDIX B. Comparison of EPA’s emission modeling results for the “control” and “no-control” scenarios show that the Clean Air Act has yielded significant pollutant emission reductions. Some of the more significant results include: around 50% reduction in sulfur dioxide emissions from electric utilities due to installation of stack gas scrubbers and the use of lower sulfur content fuels; over 75% reduction in total suspended particulate emissions as a result of controls on industrial and utility smokestacks; Over 50% reduction in carbon monoxide

emissions mostly stemming from motor vehicle pollution controls; and over 40% reductions in emissions of nitrogen oxide and other volatile organic compounds that are strong precursors to the formation of particulate matters. For some of the pollutant concentration levels needed in this study (such as all years of NO<sub>2</sub>, and values for some of the other pollutants from 1990 to 2000), EPA could not provide all the needed data because its either unavailable or not measured. The assumption made for these cases are that the growth of concentration level of the pollutants in question matches the growth rates of their emission levels. This assumption was necessary for some pollutants. For example, the EPA did not have very concrete concentration data on PM levels before 1970 but did have detailed emission values. We needed the concentration data before 1970 to model the mortalities during the 1970 to 2000 year due chronic exposure to PM before and after 1970.

Scenario two represents the “green” scenario where the background ambient levels of the pollutants are at natural levels. Natural levels refer to the amount of concentration of the criteria gases that would have existed without human pollution. Overall, the ambient natural levels of these criteria gases are approximately 1% of current concentration levels [ref 20] and are detailed below in Table 9. These values are consistent with those typically seen in areas of no pollution (i.e. the Ocean) or reflect general agreement among the atmospheric chemistry experts [ref 20].

<b>Pollutant</b>	<b>2000 urban level</b>	<b>Natural level</b>
Carbon Monoxide (CO)	2200	50
Ozone (O <sub>3</sub> )	110	10
Nitrogen Dioxide (NO <sub>2</sub> )	2	0.02
Sulfur Dioxide (SO <sub>2</sub> )	2	0.02
Particulate Matter (PM <sub>10</sub> )	25	1

Table 9 Natural ambient concentration levels of criteria pollutants (note: all values in PPB except for PM which is in µg/m<sup>3</sup>)

While these “natural” concentration levels are not thought to be achievable at urban areas, this scenario allows estimation of the economic burden of the unabated urban air pollution to the society.

## **4.3 Simulation Results and Policy Implications**

### **4.3.1 “Control” Scenario**

For the baseline “control” scenario, the total welfare in the United States in 1970 (in 2000 \$ value) including both market and non-market values was at \$6.69 trillion. The market portion of the welfare corresponds to that year’s GDP of \$3.55 trillion. The pollutants considered in this case (and following cases) are carbon monoxide (CO), ozone (O<sub>3</sub>), nitrogen dioxide (NO<sub>2</sub>), sulfur dioxide (SO<sub>2</sub>) and particulate matters under the size of 10 microns (PM<sub>10</sub>). All of these pollutants have been chosen based upon past epidemiological studies that show a link between it and certain health effects (see Chapter 2 for a more in-depth discussion). For particulate matters, PM<sub>10</sub> is used instead of PM<sub>2.5</sub> due to the lack of historical data before 1990 on PM<sub>2.5</sub> concentration levels in urban areas. All of the concentration data are based on EPA’s measurement stations across the nation in over 200 different urban areas [ref 22].

In the subsequent 30 years (1970 – 2000), the urban air pollution concentrations decreased due to the series of tougher Clean Air regulations. At the same time, the number of people exposed to these higher pollution levels increased due to not only the rise in general population but also more importantly urban revitalization process which saw a general trend in more people moving back into the more polluted urban areas.

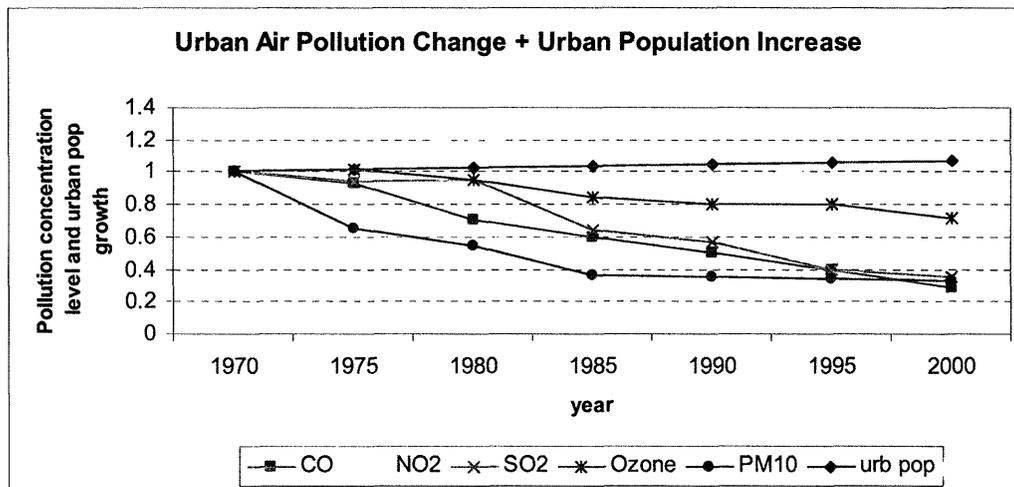


Fig. 13 Urban pollutant concentration + urban population growth

The economic growth of this scenario is matched to the historical welfare (GDP) growth and labor wage data in the United States from 1970-2000 (see figure 14):

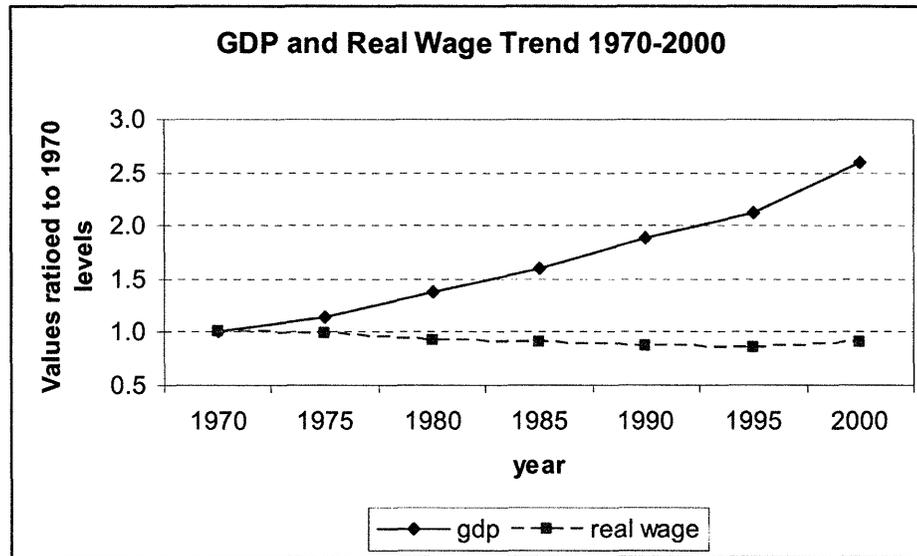


Fig. 14 US GDP and real wage trend 1970-2000

By year 2000, the total economy of the United States has risen to over two and half folds from 1970 to approximately \$9.2 trillion without leisure and \$17.6 with.

#### 4.3.2 “No control” scenario

Under the “no control” scenario, most pollutant levels would have increased instead of decreased. These values were estimated by EPA using a sequence of complex modeling and analytical procedures to predict economic and industrial activities within the US with and without Clean Air Act regulations [ref 2, 3]. The differences in economic activity were then used to model the corresponding changes in pollutant emissions which in turn provided the basis for modeling the resulting differences in air quality conditions [ref 2, 3]. The next five figures show a comparison of emission estimates for various pollutant gases with and without EPA control.

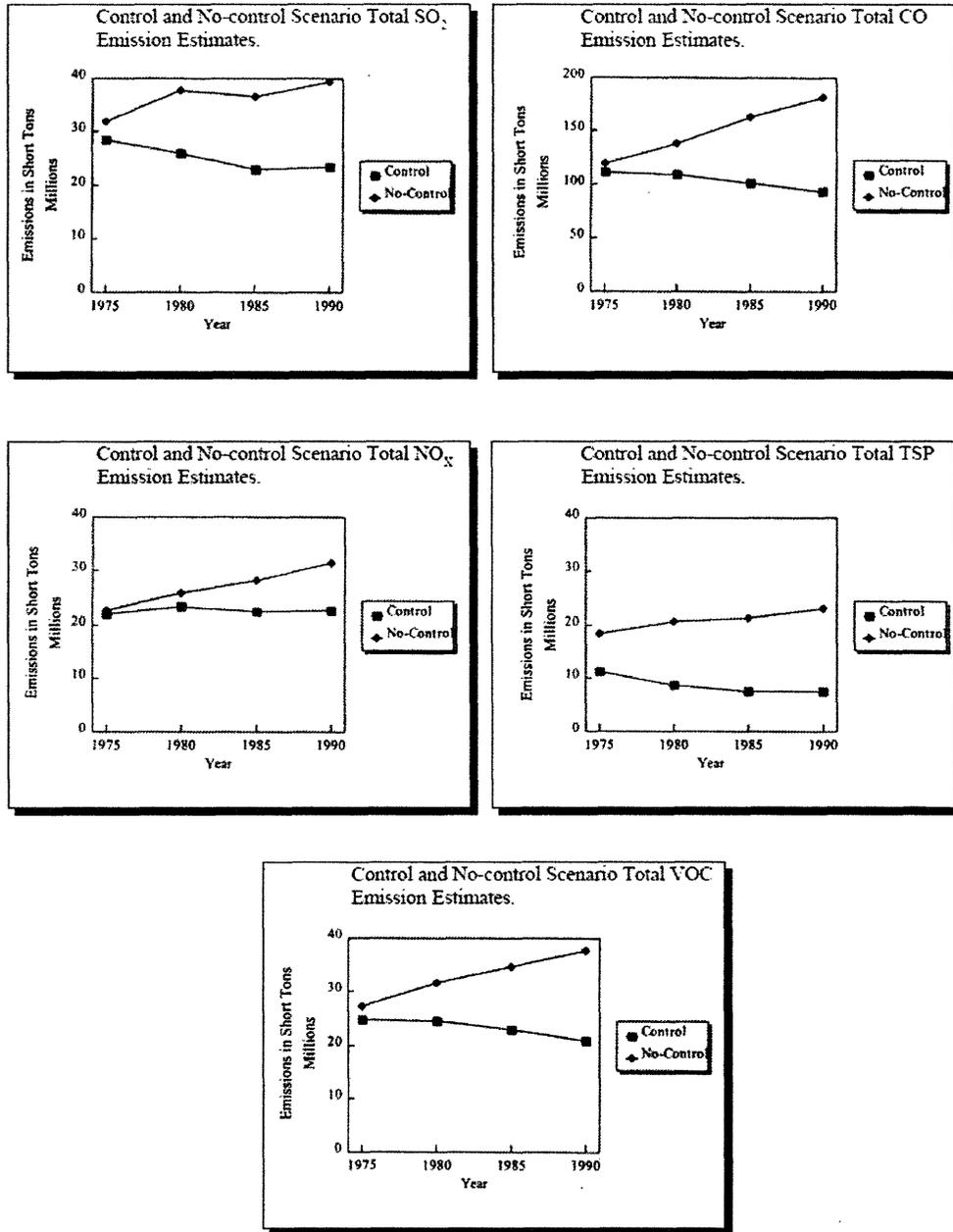


Fig. 15 EPA emission prediction for no-control scenario vs. historical records

Even by 1990, the differences between the control and no-control scenarios were almost 80% increase in emission levels. Using the resulting higher pollution concentration levels due to the higher pollution emissions, we find that the economic growth in the United States would have been lower without the tougher controls enacted by the Clean Air Acts:

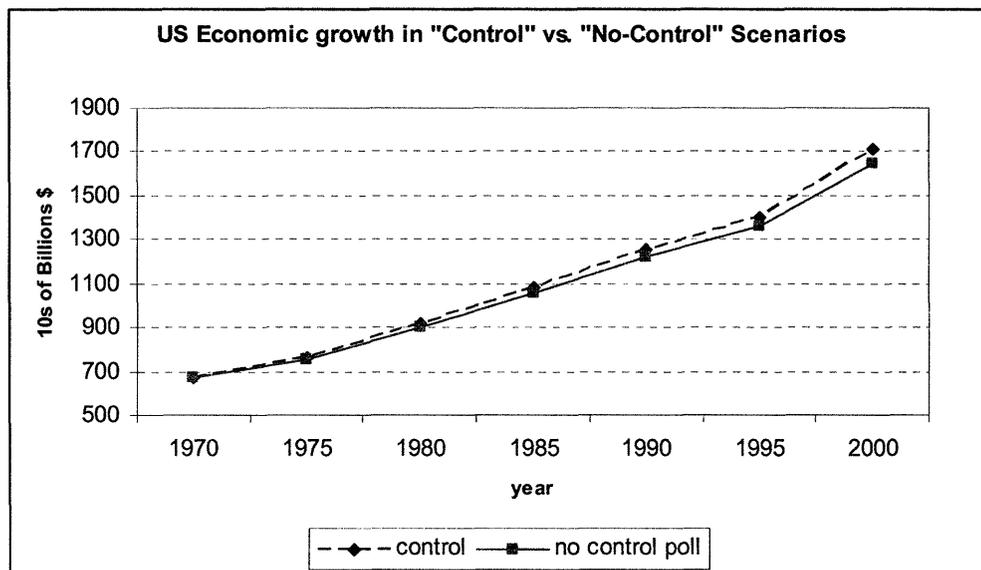


Fig. 16 Economic growth for US for "no-control" scenario compared to "control" scenario

In dollar value terms, the total economic welfare gained from having the Clean Air Act regulations are \$6.9 trillion over the last 30 years. In the year 2000 only, the economic benefits of the tougher regulations were worth almost \$600 billion (\$594 billion to be exact). In percentage terms, US economy would have been retarded by an average of over 2% a year and a high of 3.5% by year 2000 if these regulations were not in place (see figure 17):

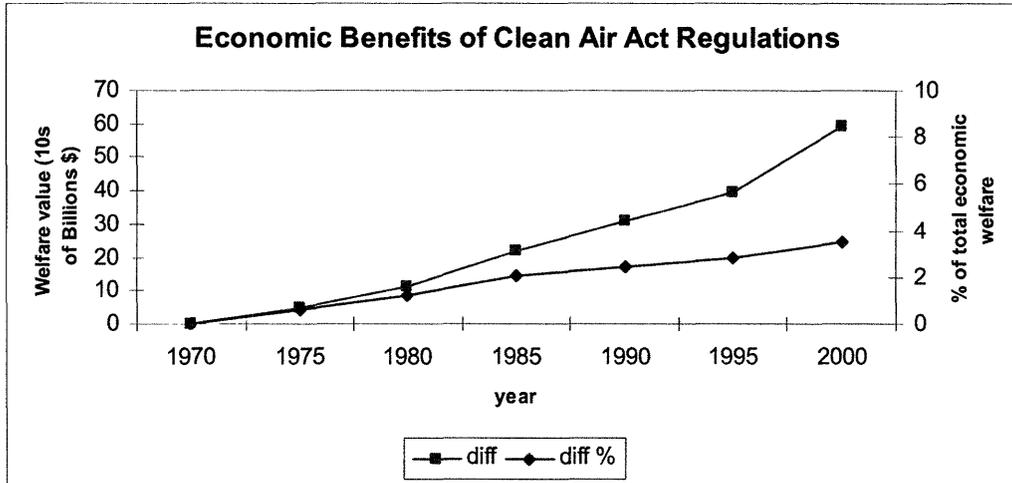


Fig. 17 Economic welfare loss for “no-control” scenario as compared to historical values

In absolute dollar terms, the economic benefits from tough air pollution control laws increases dramatically from less than 70 billion dollars in 1975 to almost 600 billion in year 2000 (an increase of 800%). As a percentage of the overall economic welfare of the nation, the results are slightly less dramatic – from less than 1% to almost 4% by 2000. The percentage growth is slower than the absolute value growth because the annual economic welfare (GDP) of the nation was rising also.

To determine which of the pollutants would have been the most egregious in causing health damages and in turn lowering the economic welfare of the US, we look at the individual contributions of each of the pollutants across the years:

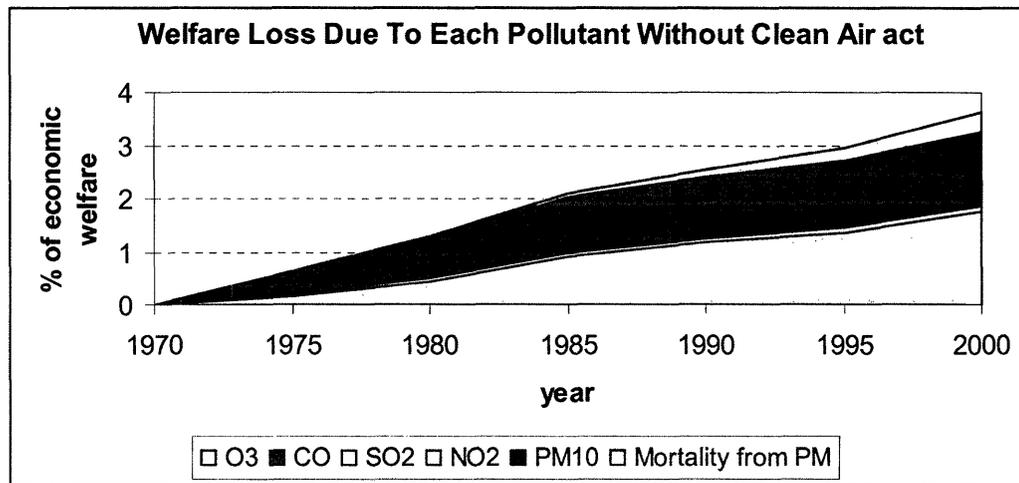


Fig. 18 Each pollutant contribution to welfare loss without air pollution control

Without Clean Air regulations the two pollutants that would have had the largest economic effect in the last 30 years are particulate matters and ozone. Individually they would have caused \$3.7 and \$3.2 trillion of economic damage respectively. Most of the economic damages were from health effects such as respiratory hospital visits, restricted activity days, chronic bronchitis and cough, congestive heart failures, ischemic heart disease and increased mortality rates. Out of those, increased mortality definitely had the largest welfare impact. Decreased mortalities from acute exposures to ozone accounted for over 30% of the welfare gain from reduce ozone levels. With reduced particulate matters, mortality due to its chronic exposure also decreased over the 30 years to contribute about \$500 billion in economic welfare gain.

#### 4.3.2.1 Comparison to EPA cost benefit analysis

The values of benefits as calculated using this methodology was significantly different than the EPA's values in its 1990 and 2000 reports on the cost and benefit of the Clean Air Acts [ref 2 and 3]. In EPA's calculations, the total economic benefits from the clean air act over the last 30 years (not including

lead) are \$27.6 trillion in 2000 dollar value. Although this number far surpasses our estimate of \$7 trillion, the main difference arises from our new calculation of mortality from chronic exposure to particulate matter. Where the EPA immediately include the benefits of reduced mortality, we used a more conservative and accurate stock and flow model which slowly phases in the mortality differences over a population's lifetime (see section 3.4.3 for more details of the model differences). With the new model, most of the gains from reduced particulate matter concentration from 1970 to 2000 is not realized for until after 2000 and lasting till mid-century:

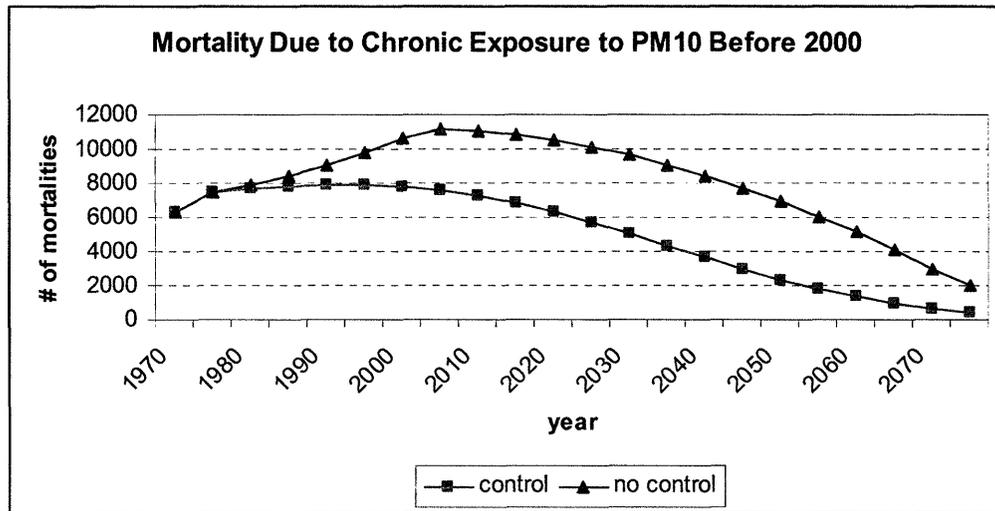


Fig. 19 Mortality difference between “control” and “no-control” scenarios

As a comparison, figure 20 shows EPA’s estimation of the number of mortalities saved due to the lowered PM concentration levels versus our model. In their calculations, they assumed immediate effect in terms of mortality from reduced ambient PM concentration level (see section 2.2 for more in-depth discussion of the different methodologies and the underlying epidemiological study results).

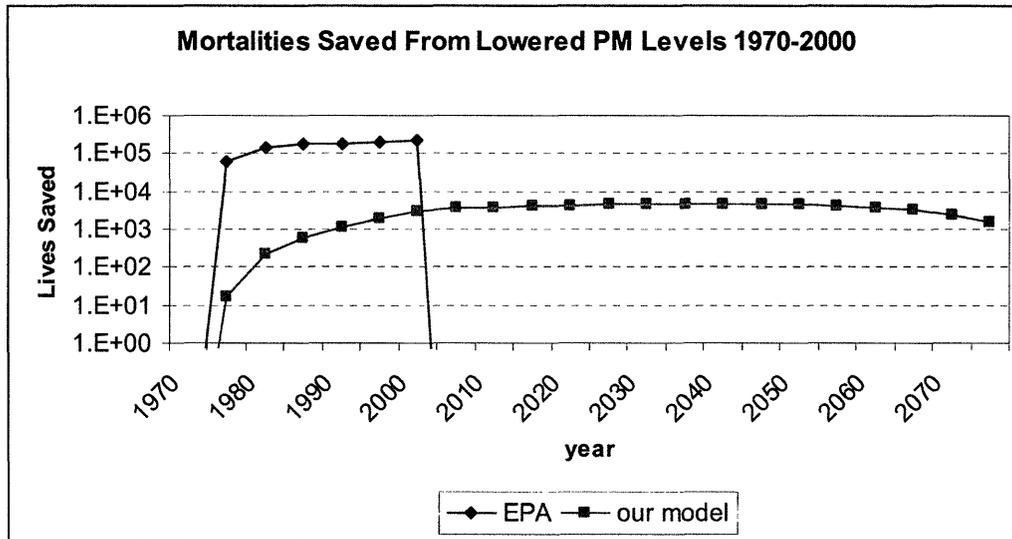


Fig. 20 EPA and our model comparison for mortalities saved from reduced PM<sub>10</sub> levels due to Clean Air Act regulations

In EPA's calculations, all of the benefits from reduced PM levels were felt during the 1970-2000 period. The fact that most of these effects did not take place until after 2000 greatly changes the welfare impact of these regulations. Using our mortality figures, the total economic benefit from the lowered PM levels from 1970-2000 are:

	Gross Total	Total Discounted at 3% annually back to 2000	Total Discounted at 5% annually	Total Discounted at 10% annually
<b>Economic Benefit of future mortality saved (trillions of 2000 dollars)</b>	18.9	6.9	4.1	1.6

Table 10 Economic value of extra mortality due to chronic exposure to PM<sub>10</sub> before 2000.

With the added benefits of future mortality savings, the total welfare benefit values are much closer to the values estimated by EPA:

	<b>EPA calculated benefits</b>	<b>Our estimate + future mortality not discounted</b>	<b>Our estimate + future mortality Discounted at 3% annually</b>	<b>Our estimate + future mortality Discounted at 10% annually</b>
<b>Total economic benefit from clean Air Acts (trillions of 2000 dollars)</b>	\$27.6	\$25.8	\$13.8	\$9.5

Table 11 Total economic benefits from having Clean Air Act regulation vs. no-control

Even with our lowest future mortality valuation (discounted at 10% back to 2000), the benefits of the Clean Air Acts still far outweighs its cost. According to the EPA, the total cost of all the regulation from 1970 to 2000 was approximately \$500 billion. The calculated benefits from the improved air quality conditions are at least 20 times the cost of implementing all the regulations.

#### 4.3.3 “Green” Scenario

The “green” scenario refers to the hypothetical case where the ambient background pollution levels of urban cities are consistent with the levels seen in natural non-polluted areas of the world. By modeling such a scenario, we can estimate the remaining economic burden of degraded air quality.

The commonly accepted “natural” background concentration levels for our criteria pollutants are listed in Table 9 of section 4.2. At those levels, there would have been very few health impacts on the society and the resulting economic growth from 1970 to 2000 would have been much higher than the known historical records. Figure 21 compares the economic differences of the “green” and the “control” scenarios.

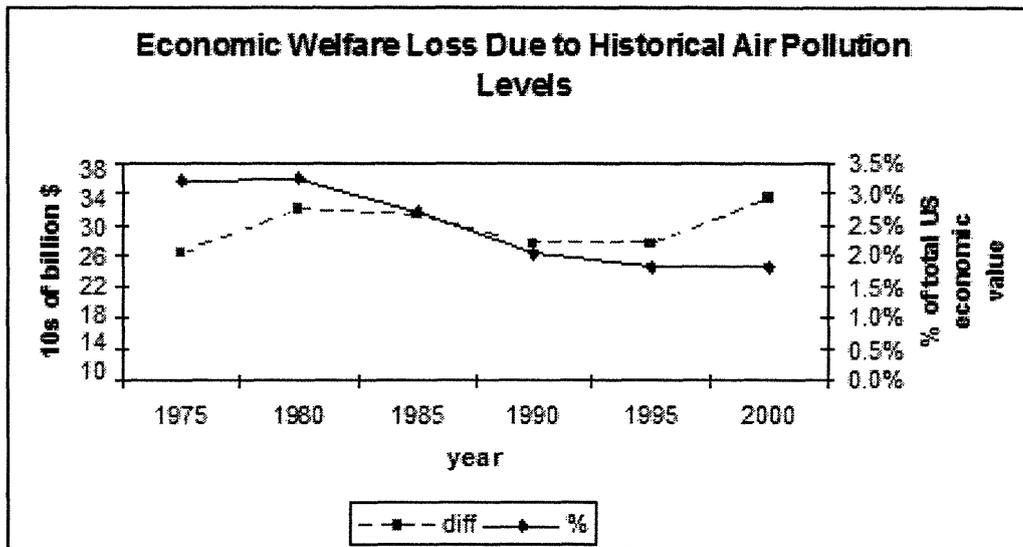


Fig. 21 Economic welfare loss due to historical pollutant levels 1975-2000

Even though the pollution levels steadily decreased from 1975-2000 (see figure 13), the actual economic real \$ impact of the pollutions were fairly consistent at approximately \$300 billion per year. This can be mainly attributed to the overall population increases during that time (~2% per year) and the increase in the percentage of population that lived in high pollution urban areas (see above section). The economic burden as a percentage of the overall welfare of the US economy has decreased steadily from over 3% of the economy to less than 2%.

In absolute terms, the total economic burden accrued during those 30 years is \$8.2 trillion. Out of that, the largest effect by far was from particulate matters which accounted for approximately 45% of the total welfare loss or (~\$3.8 trillion). And mortality from chronic exposure to the particulate matters accounted for 50% of that at \$1.9 trillion. This value is lower than other studies of similar design because of our new stock and flow model (see chapter 3 for a full description). The new model means that the mortalities from chronic exposure in the 1970 to 2000 years are mostly due to exposure from past years;

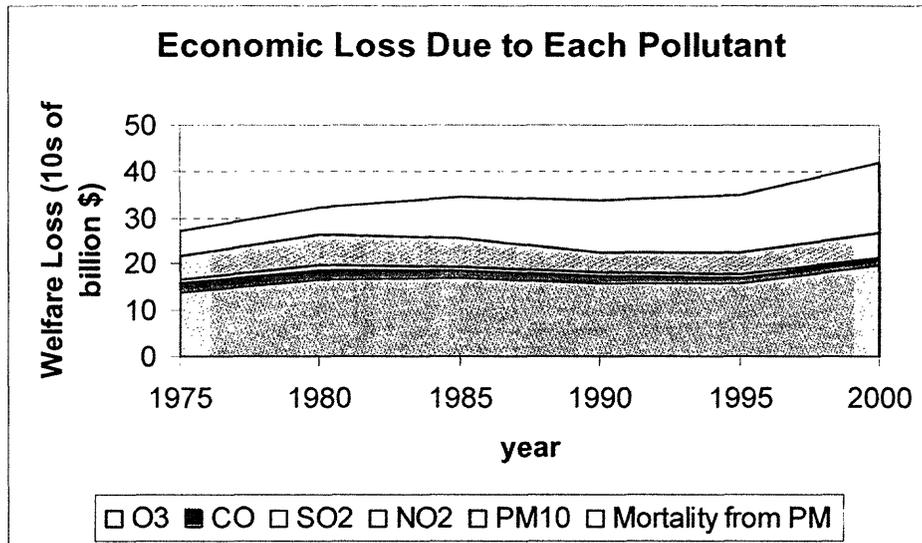


Fig. 22 Economic loss due to each pollutant "control" vs "green" scenario

Because we wanted to measure the economic burden during the years of 1975-2000, we have assumed for the "green scenario" that the particulate matter concentration previous to 1975 were also at natural levels. With that assumption, we can calculate the actual mortality burdens during 1970 to 200 due to chronic exposures during and before that time period.

Other pollutants that caused large economic burdens on the society included ozone, and carbon monoxide. Those two pollutants combined contributed to 55% of the economic welfare burden and caused increased mortalities, congestive heart failures, asthma attacks and other diseases such as asthma that restricted the activities of the population.

#### 4.3.4 Sensitivity Analysis

In Chapter 2, we had noted that many of the health effects from air pollution are not fully agreed upon by the scientific community due to uncertain epidemiological studies. In the previous analyses described in this chapter, we have used the set of health effects compiled by ExternE that are most accepted

by the scientific community. Along with that list, ExternE has also listed a set of health effects that are more controversial. These health effects are not included in the original set but identified as less certain because of the relatively fewer or conflicting epidemiological evidence. Table 12 shows the list of these health effects along with the pollutants in question.

<b>Pollutant</b>	<b>Uncertain Health Effect</b>
Carbon Monoxide (CO)	<ul style="list-style-type: none"> <li>• Mortality from acute exposure</li> </ul>
Ozone (O <sub>3</sub> )	<ul style="list-style-type: none"> <li>• Asthma induced emergency room visits</li> </ul>
Nitrates	<ul style="list-style-type: none"> <li>• Emergency Room Visit for Chronic Obstructive Pulmonary Disease</li> <li>• Asthma induced emergency room visits</li> </ul>
Particulate Matter (PM)	<ul style="list-style-type: none"> <li>• Emergency Room Visit for Chronic Obstructive Pulmonary Disease</li> <li>• Asthma induced emergency room visits</li> </ul>
Nitrogen Dioxide (NO <sub>2</sub> )	<ul style="list-style-type: none"> <li>• Respiratory hospital visit</li> <li>• Mortality from acute exposure</li> </ul>

Table 12 Uncertain health effects of pollutants

For these health effects, we have decided to use them as sensitivity analysis on our economic burden analysis for 1970-2000. By not including them in the original dataset, we are acknowledging their uncertainty but at the same time we felt it was worthwhile to explore the results of our study with those health end points included as well. While some of these, such as asthma induced hospital visits, have relatively low economic costs, others such as higher acute mortality rates will have very large effects on the economy. And the result of this analysis should be thought of as a maximum ceiling of the economic burden on the United State's economy due to our historical levels of ambient air pollution.

For example, during 1975 if we had counted all of the uncertain health effects as costs because of air pollution, the total economic burden to society would have almost doubled from \$291 billion to \$533.5 billion. The majority of the increase, \$200 billion, is from the suspected connection between a high level of mortality and CO concentration:

	<b>Economic Burden of uncertain health effects</b> <b>(\$ billions)</b>	<b>Burden as % of total economic welfare</b>
<b>Ozone (O<sub>3</sub>)</b>	0.58	0.004%
<b>Carbon Monoxide (CO)</b>	200.1	2.6%
<b>Nitrates</b>	4.7	0.06%
<b>Particulate Matter (PM<sub>10</sub>)</b>	3.7	0.05%
<b>Nitrogen Dioxide (NO<sub>2</sub>)</b>	33.4	0.4%

Table 13. Sensitivity analysis for 1975 air pollution economic burden from uncertain relationships of health endpoint vs. increased pollutant concentration.

## Chapter 5 Conclusions

Exposure to harmful air pollution causes many harmful health effects such as respiratory illness, bronchitis, reduced lung function, pulmonary disease and early mortality. In this thesis, we described a new methodology for calculating the economic burden of urban air pollution in the United States from 1970 to 2000 and the benefits of reductions achieved through the Clean Air Act regulations. Past pollution health cost and benefit studies have mostly used a direct multiplication of the number of illnesses and the value of each to come up with an economic welfare cost of air pollution. Here, we have developed a methodology for fully integrating the health effects from exposure to air pollution into a computable general equilibrium economic model. This model represents the first attempt at fully incorporating the economic valuation of air pollution in an integrated economic model that has endogenously built-in consumer demand and preference curves to accurately represent the demand for air pollution health. Furthermore, we also described a new stock and flow model to track the extra mortalities from chronic exposure to particulate matters. This new framework allows the tracking of total pollution in-take and its effect on mortality levels in current and future years as pollution levels change.

Using these new models, we valued the economic benefit of reduced air pollution due to the Clean Air Act regulations to be over \$7 trillion from 1970 to 2000 or 2.1% of the aggregate economic welfare of the United States in this period. This does not include the hundreds of thousands of lives saved from early mortality in years after 2000 due chronic exposure during 1970 to 2000. The economic benefit of those saved mortalities is another \$7 trillion using a 3% discount rate. Furthermore, we estimated the economic burden from the rest of the unmitigated pollution levels (actual historical pollution) to be approximately \$9 trillion in the last 30 years.

There were several important assumptions and simplifications that were made by the author in this model that may need further inspection in future studies:

1. The assumptions of no threshold and linearity in the relationship of health effects and pollution level. The linearity assumption states that while individuals may respond differently to various pollution levels, the population as a whole will have health effects that rise linearly with pollution levels. The threshold assumption says that there is not a minimum level of pollution below which there are no health effects.

Both of these assumptions are widely debated in the environmental health community. The linearity and no threshold assumptions we used are commonly used in benefit studies.

2. The assumption of no trade-off between medical services and time away from work to recover from illnesses due to exposure to health effects. This assumption simply states that for different illnesses, different combinations of professional medical service and rest are needed to recover. This combination, on average, does not change as more or less cases of these illnesses develop, or in response to changing prices or income levels. This assumption is extreme perhaps as there may well be willingness to forgo purchased healthcare under certain economic conditions. While this assumption should be examined especially if we are to use this model for other regions of the world, it is consistent with the valuation literature that would simply multiply per case costs times the number of cases.
3. The assumption that an increase in pollution exposure of  $x$  amount in one year is equal to an increase of  $\frac{x}{n}$  amount exposure every year in the lifetime where  $n$  is the age of the person. This assumption is a crucial part of the stock and flow model developed in this thesis (see chapter 3) for the calculation of mortalities due to

chronic exposure to particulate matters, and is another implication of the linearity assumption.

4. The simplification of modeling the 1970 US economy as a smaller version of the 1997 economy in the CGE model. While it would have been ideal to construct the US 1970 economy to match the exact production and technology levels of the historical record, it would have been an extremely time-consuming event. In this thesis, we were able to scale the 1997 US economy values as found in the original EPPA CGE model to 1970 based on GDP changes, and approximately match the change in the change in the real wage rate. We believe that this simplification does not have a large effect on our results, as the valuation of damages in the model is most directly affected by the total size of the economy and the wage rate.

The economic accounting of the welfare costs of air pollution is complicated in that it involves an understanding of many disciplines including health epidemiological studies, the modeling of human health physiology for health effects from both acute and chronic exposure to air pollution, and the economic valuation of different health effects for both morbidity and mortality illnesses. This thesis presented one framework for integrating these effects together in a rigorous economic modeling framework. We focused on developing the method for valuing the effects within a dynamic economic model of the economy, and that is the main new contribution of the thesis. This required identifying when mortality cases occur, and tracking how the loss will affect the economy over the remaining expected lifetime of the individual. The underlying epidemiological relationships and the resulting direct use of hospital services, and lost time associated with morbidity and mortality were taken directly from the existing literature. The method developed here helps provide both a deeper understanding of the complicated relationships of all the variables in the problem

and a general framework for providing sound economic welfare analysis for policy makers.

## APPENDIX A

Historical urban air pollution levels in US urban areas from 1970-2000

	1970	1975	1980	1985	1990	1995	2000
CO	12.8	11.8	8.8	7.36	6.124	4.827	3.425
Nox	0.023111	0.026	0.0275	0.0246	0.0231	0.0215	0.01954
SO2	0.016141	0.015	0.015	0.009987	0.00881	0.00598	0.005284
Ozone	0.153	0.153	0.143	0.125	0.11676	0.1158	0.10259

	PM10 concentration
1923	94.07894737
1940	105.2631579
1945	108.5526316
1950	110.5263158
1951	111.8421053
1955	105.9210526
1960	101.9736842
1965	92.10526316
1968	85.52631579
1970	78.94736842
1975	51.31578947
1980	42.76315789
1985	28.94736842
1990	26.97368421
1995	26.64473684
2000	25

## APPENDIX B

Hypothetical urban air pollution levels in 1970-2000 for "no-control" scenario

All values in ppb except for PM10 which is in  $\mu\text{g}/\text{m}^3$

	1970	1975	1980	1985	1990	1995	2000
CO	12.8	12.92381	11.06286	11.21524	10.49829	9.194286	7.241429
Nox	0.023111	0.031056	0.038194	0.038267	0.039142	0.039417	0.03908
SO2	0.016141	0.017885	0.021923	0.014404	0.013384	0.00943	0.008434
Ozone	0.153	0.1683	0.1716	0.16875	0.17514	0.19107	0.184662
PM10	78.94737	54.33437	55.34056	40.86687	41.25387	44.66912	45.58824

## APPENDIX C

Historical US economic activity record 1970-2000

	GDP (in 2000 \$)	real wage
1970	3.55E+12	1
1975	4.05E+12	0.983159
1980	4.87E+12	0.921397
1985	5.69E+12	0.909689
1990	6.68E+12	0.870471
1995	7.54E+12	0.85571
2000	9.19E+12	0.913714

## References

- Ref. 1. Kunzli, N. Kaiser, R. Medina S. Studnicka M. Chanel O. Filliger P. Herry M. Horak F. Jr. Puybonnieux-Textier, V. Quenel P. Schneider, J. Seethaler R. Vergnaud, J-C. Sommer, H. Public health impact of outdoor and traffic-related air pollution: a European assessment. *The Lancet*, Vol 356, September 2, 2000.
- Ref. 2. Pope, C. Arden. Burnett, Richard T. Thun, Michael J. Calle, Eugenia E. Krewski, Daniel. Ito Kazuhiko. Thurston, George D. Lung Cancer, Cardiopulmonary mortality and long-term exposure to fine particulate air pollution. *JAMA*, march 6, 2002. vol 287, No. 9. pg 1132-1141
- Ref. 3. Pope, C. Arden. Thun, Michael J. Namboodiri, Mohan M. Dockery, Douglas W. Evans, John S. Speizer E. Heath, Clark W Jr. Particulate air pollution as a predictor of mortality in a prospective study of US adults. *Am J. Respir Crit Care Med*; Vol 151. pp669-674, 1995
- Ref. 4. Dockery DW, Pope CA III, Xu X, et al. An association between air pollution and mortality in six US cities. *N England J. Med.* 1993; 329; 1753-1759
- Ref. 5. European Commission, "ExternE, Externalities of Energy", Volume 7: Methodology, 1998 update, Directorate-General XII, Science Research and Development, 1998
- Ref. 6. Dixon. Estimating the Health Impact of air pollution: methodology and an application to Jakarta.
- Ref. 7 WHO database on mortality levels across different countries and regions - <http://www3.who.int/whosis/menu.cfm>
- Ref. 8 US census database – population level across different age groups - [http://www.censusscope.org/us/chart\\_age.html](http://www.censusscope.org/us/chart_age.html)
- Ref. 9 US Environmental Protection Agency, Office of Air and Radiation, Office of Policy, The Benefits and Costs of the Clean Air Act 1970 to 1990. November, 1989.
- Ref. 10 US Environmental Protection Agency, Office of Air and Radiation, Office of Policy, The Benefits and Costs of the Clean Air Act 1990 to 2010. November, 1999.
- Ref 11. Beattie J, Covey J, Dolan P, Hopkins L, Jones-Lee M, Loomes G, Pidgeon N, Robinson A, Spencer A. On the Contingent Valuation of Safety and the Safety of Contingent Valuation: Part 1 – caveat investigator.

- Ref 12. Shoven J, Whalley J. Applied General-Equilibrium Models of Taxation and International Trade: an Introduction and Survey., *Journal of Economic Literature*, Vol. XXII 9.1984. pp 1007-1051
- Ref 13. Babiker M, Reilly J, Mayer M, Eckaus R, Wing I, Hyman R. The MIT Emissions Prediction and Policy Analysis (EPPA) Model: Revisions, Sensitivities and Comparisons of Results. Report #71, 2/2001
- Ref 14 Mayer M., Wang C., Webster M., Prinn R. Linking Local Air Pollution to Global Chemistry and Climate. *Journal of Geophysical Research*, Vol. 105, No. D18, pgs 22,869-22,896, 9/27/2000
- Ref 15. Smith, Kerry V., Pattanayak Subhrendu K, Houtven, George Van. "Valing Environmental Health Risks: from Preference Calibration to Estimation". Working paper 3/2003
- Ref 16. Smith, Kerry V., Pattanayak Subhrendu K, Houtven, George Van Kerry Smith. "Using Preference Calibration for VSL Estimation". Working paper 3/2003
- Ref 17. Nordhaus, William D., Kokkelenberg, Edward C., et al. Nature's Numbers – Expanding the National Economic Accounts to Include the Enviroment, National Research Council, 1999
- Ref 18. Prinn, R.G., H.D. Jacoby, A.P. Sokolov, C. Wang, X. Xiao, Z. Yang, R. Eckaus, P.H. Stone, A.D. Ellerman, J. Melillo, J. Fitzmaurice, D. Kicklighter, Y. Liu & G. Holian Integrated Global System Model for Climate Policy Assessment: Feedbacks and Sensitivity Studies, may 1998,
- Ref 19. Z. Yang, R. Eckaus, A.D. Ellerman & H.D. Jacoby The MIT Emissions Prediction and Policy Analysis (EPPA) Model, May 1996 (49 p.)  
<http://web.mit.edu/globalchange/www/rpt6.html>
- Ref 20. Seinfeld, John H. Pandis, Spyros N., *Atmospheric Chemistry and Physics*, John Wiley & Sons, Inc, 1998
- Ref 21. Curtill, Laurianne. Urban Air Pollution Climate Policy. MIT Masters Thesis, 6/2002
- Ref 22. US environmental protection agency. Air Pollution Trends for Major Pollutants.  
<http://www.epa.gov/airtrends>.
- Ref 23. Babiker, Metcalf, reilly, Tax Distortions and Global Climate Policy. Report #85 for MIT Joint Program on the Science and Policy of Global Change
- Ref 24. Council of Economic Advisors, Economic Report of the President. 2/2003

Ref 25. Smith, Kerry V., Pattanayak Subhrendu K, Houtven, George Van Kerry Smith. "Benefit Transfer Functions for Avoided Morbidity: A Preference Calibration Approach". Working paper 3/2003