WPS 2785

POLICY RESEARCH WORKING PAPER   2785

Improving Air Quality in Metropolitan Mexico City

An Economic Valuation

*The Mexico Air Quality Management Team**

The annual health benefits of a 10 percent reduction in ozone and PM10 in Mexico City, conservatively estimated, are approximately $760 million (in 1999 U.S. dollars) annually. Reducing PM10 has larger estimated health benefits than reducing ozone, with each microgram per cubic centimeter reduction in PM10 worth about $100 million per year.

The World Bank
Latin America and the Caribbean Region
Environmentally and Socially Sustainable Development Sector Unit
February 2002

Summary findings

Mexico City has for years experienced high levels of ozone and particulate air pollution. In 1995–99 the entire population of the Mexico City metropolitan area was exposed to annual average concentrations of fine particulate pollution (particulates with a diameter of less than 10 micrometers, or PM10) exceeding 50 micrograms per cubic meter, the annual average standard in both Mexico and the United States. Two million people were exposed to annual average PM10 levels of more than 75 micrograms per cubic meter. The daily maximum one-hour ozone standard was exceeded at least 300 days a year.

The Mexico Air Quality Management Team documents population-weighted exposures to ozone and PM10 between 1995 and 1999, project exposures in 2010, and computes the value of four scenarios for 2010:

- A 10 percent reduction in PM10 and ozone.
- A 20 percent reduction in PM10 and ozone.
- Achievement of ambient air quality standards across the metropolitan area.
- A 68 percent reduction in ozone and a 47 percent reduction in PM10 across the metropolitan area.

The authors calculate the health benefits of reducing ozone and PM10 for each scenario using dose-response functions from the peer-reviewed literature. They value cases of morbidity and premature mortality avoided using three approaches:

- Cost of illness and forgone earnings only (low estimate).
- Cost of illness, forgone earnings, and willingness to pay for avoided morbidity (central case estimate).
- Cost of illness, forgone earnings, willingness to pay for avoided morbidity, and willingness to pay for avoided mortality (high estimate).

The results suggest that the benefits of a 10 percent reduction in ozone and PM10 in 2010 are about $760 million (in 1999 U.S. dollars) annually in the central case. The benefits of a 20 percent reduction in ozone and PM10 are about $1.49 billion annually. In each case the benefits of reducing ozone amount to about 15 percent of the total benefits.

By estimating the magnitude of the benefits from air pollution control, the authors provide motivation for examining specific policies that could achieve the air pollution reductions that they value. They also provide unit values for the benefits from reductions in ambient air pollution (for example, per microgram of PM10) that could be used as inputs into a full cost-benefit analysis of air pollution control strategies.

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* The Mexico Air Quality Management Team

The Mexico Air Quality Management Team was led by Walter Vergara and includes the following authors:

Herman Cesar[1]

Victor H. Borja-Aburto[2]

Pablo Cicero-Fernandez[3]

Kees Dorland[1]

Roberto Muñoz Cruz[4]

Luke Brander[1]

Maureen Cropper[8]

Ana Citlalic Gonzalez Martinez[5]

Gustavo Olaiz-Fernandez[7]

Ana Patricia Martínez Bolivar[6]

Xander Olsthoorn[1]

Alberto Rosales-Castillo[2]

Gloria Soto Montes de Oca[4]

Victor Torres-Meza[2]

Ricardo Uribe Ceron[4]

Pieter Van Beukering[1]

Eduardo Vega López[4]

Max Magin Niño Zarazua[5]

Miguel Angel Niño Zarazua[5]

Walter Vergara[9]

[1] Institute for Environmental Studies (IVM), Vrije Universiteit, Amsterdam

[2] Secretaria de Salud, Mexico DF Centro Nacional de Salud Ambiental (CENSA)

[3] Environmental Health Sciences Department, School of Public Health, University of California Los Angeles (UCLA)

[4] Secretaría del Medio Ambiente del Gobierno del Distrito Federal, Mexico DF

[5] Unidad Asesora para Comunidades Empresariales, A.C. (UNACE)

[6] Pan American Health Organization (PAHO)

[7] Dirección General de Salud Ambiental, Mexico DF

[8] Development Research Group, World Bank, Washington,

[9] Latin America and Caribbean Environment Department, World Bank

Executive Summary

The Mexico City Metropolitan Area (Zona Metropolitana del Valle de Mexico (ZMVM)) has witnessed high levels of air pollution in the past few decades. Recent efforts to curb emissions have been reasonably successful, and 1999 had the lowest overall level of air pollution during the last decade. With the exception of lead, carbon monoxide and sulfur dioxide (SO2), however, pollution levels are still far above current air quality standards (See Table E.1).

Table E.1: Number of Days Per Year that Ozone and PM$_{10}$ Concentrations in Mexico City Satisfy Daily Air Quality Standards

	1995	1996	1997	1998	1999
Ozone	41	39	43	45	65
PM$_{10}$	273	186	212	176	345

Source: GDF (2000).

Further efforts to reduce polluting emissions are being developed by the Comision Ambiental Metropolitana (CAM) under the Third Air Quality Program 2001–2010. This study presents an economic valuation of benefits from reducing pollution in the ZMVM, as the main economic rationale for controlling emissions is the welfare gain from improvements in air quality. The current study focuses on the two most important economic impacts of air pollution, namely health impacts and restrictions imposed on economic activities through environmental contingencies (*contingencias ambientales*).

The health hazards associated with ozone and PM10 are studied because these substances are the most important in terms of violating pollution standards. Ozone pollution stems mainly from emissions of NO$_x$ and VOCs. Their concentration levels depend on the amount and location of emitted pollutants, geographical characteristics, meteorological conditions, and atmospheric chemistry and transport. The chemistry of ozone formation is complicated and nonlinear: under certain conditions, an increase in NO$_x$ emissions could reduce ozone concentrations. PM10 pollution stems mainly from direct emissions of particles, and from reactions of NO$_x$, and SO2 with other substances in the atmosphere. Likely emission sources are building and construction (road construction), diesel trucks and buses, forest fires, open-air refuse burning, some manufacturing industries, and resuspension of road dust.

The daily 1-hour maximum air quality standard for ozone is 0.110 ppm. During 1995–99, the highest concentration observed for ozone—0.349 ppm—was measured at the Pedegral station, in the southwest zone of the ZMVM. The Chapingo station in the northeastern zone was the least polluted, with a daily 1-hour maximum concentration of 0.210 ppm. The daily average air quality standard for PM10 is 150 μg/m3 and the annual average standard is 50 μg/m3. All stations violated both standards with the exception of the annual average standard at the Pedregal and Coacalco stations. The highest concentrations were in the east of the ZMVM with a daily maximum of 335 μg/m3 at the Netzahualcoyotl station. The highest annual average of 94 μg/m3 was observed at the Xalostoc station. In 1995, over 1.2 million

people were exposed to concentrations above the environmental contingency Stage I level of 300 μg/m3 at least once during the year.

The baseline scenario for 2010 assumes emissions of NO_x and VOCs, precursors of ozone and PM_{10}, to be the same as at the end of the 1990s. Likewise, we assume air quality in 2010 with respect to ozone and PM_{10} to be the same as the levels observed at the end of the 1990s. This assumption, however crude, seemed to be the most appropriate one in the absence of an integrated model of emission projections for 2010 for fixed and mobile sources in Mexico City.

Four alternative air pollution reduction scenarios for 2010 are evaluated. We do not appraise the policies needed to achieve the concentration reductions. The four scenarios are (population weighted exposure reductions are presented in table E.2):

- a 10-percent reduction in PM10 and ozone;

- a 20-percent reduction in PM10 and ozone;

- improved air quality compliance at an air quality standard of 50 μg/m3 for PM10 and 0.11 ppm 1-hour maximum for ozone in all ZMVM locations (AQS1);

- an air quality standard superimposing the required decrease in concentrations in the most polluted areas (Xalostoc for PM10 and Pedregal for ozone) across the ZMVM (68 and 47 percent reduction in ozone and PM10 concentrations, respectively) (AQS2).

Table E.2: Reduction in Population-Weighted Exposure for the Analyzed Scenarios

Scenario	Population weighted exposure to PM_{10} (μg/m^3/person)	Population weighted Exposure to ozone (ppm/person)
10 percent exposure reduction	6.41	0.0114
20 percent exposure reduction	12.81	0.0227
AQS compliance in each area – AQS1	14.06	0.0702
AQS compliance in worst area – AQS2	29.99	0.0778

The health risks due to air pollution (specifically ozone and PM10) are quantified by estimating the relationship between the incidence of adverse health effects and air quality. To this end, a number of quantitative estimates of exposure-response relations of known health effects from various cities have been pooled together (meta-analysis).

Health impacts include eye irritation, respiratory diseases, cardiovascular effects, and premature death. This paper, unlike studies such as Hernandez-Avila and others (1995), who focused only on hospital costs, assesses a wide range of health benefits of reducing air pollution: (i) reduced cost of illness (COI); (ii) reduced losses in productivity; (iii) willingness to pay (WTP) for reduced acute and chronic morbidity effects; and (iv) willingness to pay for mortality effects associated with acute and chronic exposure.

In each case the WTP concept captures aspects of the value of avoiding death and illness (for example, the pain and suffering avoided) above and beyond foregone earnings and COI (used here to refer to avoided medical costs). The largest single contributor to the benefit estimate is WTP for premature death. Because of the debate over using WTP for valuing health benefits, in particular when WTP is estimated using the Contingent Valuation Method (CVM), we compute the health benefits both including and excluding this benefit category.

Specifically, we present three sets of benefit estimates. The 'high estimate', the most comprehensive one, includes WTP to avoid illness, as well as avoided illness costs (COI) and reduced losses in productivity, to value reduced morbidity. Avoided premature mortality is valued using WTP. The 'central estimate' includes the same comprehensive measure of the value of reduced morbidity, but values avoided premature mortality using foregone earnings, a lower bound to WTP. The 'low estimate', the most conservative, values morbidity using COI and productivity measures alone and premature mortality using foregone earnings. The high and central estimates vary depending on the income elasticity used to transfer WTP estimates for morbidity and mortality from other countries to Mexico. Income elasticities of 1.0 and 0.4 are presented; however, we view the 1.0 elasticity as our central estimate.

Table E.3 summarizes the benefits of each control scenario, where results for ozone and PM_{10} are added together. Adding the benefits of these two pollutants is appropriate because the estimates for each pollutant controls for the level of the other pollutant. The central estimate of the annual benefits of a 10 percent reduction in ozone and PM10 is $759 million. High and low estimates of the value a 10 percent reduction are $1,607 million and $154 million, respectively. Obtaining air quality compliance (AQS1) offers benefits of approximately $2 billion per year, with high and low estimates of benefits of some $4 billion and $400 million, respectively.

Table E.3: Summary of Benefits From Each Scenario for Ozone and PM_{10} Combined
(in million US$ per year, 2010 values in 1999 prices, income elasticity 1.0)

Estimates	10%	20%	AQS1	AQS2
High	1607	3184	3952	7636
Central	759	1489	1928	3580
Low	154	275	368	618

The estimates presented in table E.3 clearly show that the calculated benefits associated with air pollution reduction provide an economic basis for expenditures to further reduce polluting emissions. Exactly how much is open to debate. Ideally, a study like this on economic benefits should be combined with estimates of emission abatement costs to determine an economically justifiable level of abatement. Hence, developing a cost-benefit model is the next logical step.

Table E-4 presents alternate estimates of health benefits, as well benefits from avoiding environmental contingencies, for ozone and PM10 separately. This is particularly useful as it

shows that the health benefits of PM10 reductions are roughly an order of magnitude higher than those of ozone.

Table E.4: Benefits from Reducing Air Pollution: Four Scenarios for Ozone and PM$_{10}$
(in million US$ per year, 2010 value in 1999 prices, 3 percent discount rate)

	Scenario							
	10%		20%		AQS1		AQS2	
Income elasticity	1.0	0.4	1.0	0.4	1.0	0.4	1.0	0.4
Ozone								
Health benefit estimate 1, including morbidity (Prod. Loss + COI +WTP) and WTP for mortality	116	183	232	365	717	1129	794	1250
Health benefit estimate 2, including morbidity (Prod. Loss + COI +WTP) and human capital losses for mortality	75	114	151	228	465	706	515	782
Health benefit estimate 3, including morbidity (Prod. Loss + COI) and human capital losses for mortality	18	18	35	35	109	109	121	121
Environmental contingencies benefits	36	36	45	45	45	45	45	45
PM$_{10}$								
Health estimate 1, including[1] morbidity (Prod. Loss + COI +WTP) and WTP for mortality	1451	2549	2903	5098	3186	5595	6793	11931
Health benefit estimate 2, including: morbidity (Prod. Loss + COI +WTP) and human capital losses for mortality	644	1184	1289	2367	1414	2598	3016	5540
Health benefit estimate 3, including: morbidity (Prod. loss + COI) and human capital losses for mortality	96	96	191	191	210	210	448	448
Environmental contingencies benefits	4	4	4	4	4	4	4	4

Prod. loss = Productivity losses; COI = Direct Cost of Illness; WTP = Willingness to Pay.

1. Introduction

The *Zona Metropolitana del Valle de Mexico* (ZMVM) (Mexico City Metropolitan Area) is one of the world's largest urban areas and one of the most notorious for its poor air quality. In the 1990s, however, efforts to control air pollution seem to have diverted the trend. Table 1.1 shows a decline in overall air pollution during the last decade (GDF 2000). However, with the exception of lead, carbon monoxide and sulfur dioxide, pollution levels are still far above air quality standards.

Table 1.1 Number of Days Per Year that Ozone and PM_{10} Concentrations in Mexico City Satisfied Air Quality Standards

	1995	1996	1997	1998	1999
Ozone	41	39	43	45	65
PM_{10}	273	186	212	176	345

Source: GDF (2000).

The Third Air Quality Program 2001–2010 ("the Program") being developed by the *Comision Ambiental Metropolitana* (CAM) includes further initiatives to improve air quality. These air quality efforts are expected to improve the health of the population and also reduce the number of environment-related alerts in the ZMVM. One element of the program, and the purpose of this study, is the economic evaluation of the benefits gained from improving air quality.

Air pollution has a range of negative effects on human health. It may also affect economic activity when excessive levels of pollution require *Contingencias Ambientales* (restrictions on environmentally polluting activities). Health-related impacts include eye irritation, respiratory diseases, cardiovascular effects, and premature death. When a *Contingencia Ambientale* is declared it limits activities of a range of manufacturing industries that generate emissions of air pollutants, and also restricts traffic.

In sum, current air pollution exceeds permitted standards, and prospects for the future, with some exceptions (such as lead), will not improve without more active air quality management and policies designed to improve air quality. Policy measures will be most useful if they can (1) be defined in terms of specific measures and the costs involved; (2) assess changes in air pollution using some form of air quality modelling; (3) assess and evaluate changes in air pollution impacts; and (4) rank the measures in terms of cost-effectiveness.

The rationale for making air quality policies is the welfare gain from improvements in air quality. This report attempts to assess in economic terms the reduced impacts on human health and economic activity associated with four prespecified air quality scenarios with a time horizon of 2010. It is limited to the impacts of ozone and PM_{10} because these substances are the most important in terms of exceeding their standards and because relevant health information is not fully available for other pollutants (such as NO_2).

Earlier efforts to assess the benefits of improvements in air quality for Mexico City by Hernandez-Avila and others (1995) estimated the direct medical costs and foregone income avoided if air quality standards were met. We use a different methodology for the economic valuation of reduced health risks (see section 6), and we use recent insights into the functional relationships between air quality and health impacts. We also deal with the economic benefits of avoiding *Contingencias Ambientales* (the use of environmental contingencies).

Air pollution is the outcome of a range of physical processes. To understand its impacts one needs to know (1) the spatial and temporal patterns of pollutant emissions; (2) the chemical, physical and meteorological processes in the airshed; and (3) the effects of pollutants on people's health, how many people are exposed to them, what economic activities suffer from environmental contingencies, and, if the scope of interest extends beyond the urban area, how natural systems (for example, ecosystems and climate) are affected.

The structure of the report is as follows. Section 2 describes current emissions and air quality management in the ZMVM. Section 3 specifies the four air quality scenarios considered in a model of current and future air quality. Section 4 models the population exposure to air pollution and estimates the number contingency measures invoked. Section 5 discusses the functional relationship between exposure and health, and derives exposure-response functions specific to the ZMVM. Section 6 covers the economic valuation of the air quality scenarios set out in section 3 in terms of both the reduced health impacts and the reduced number of *Contingencias Ambientales*. Section 7 discusses the results.

2. Emissions and Current Air Quality in the ZMVM

For a quantitative understanding of air quality it is necessary to have an insight into the spatial and temporal pattern of emissions. The present study does not perform atmospheric transport modelling as this is outside the scope of the study. We will instead assume scenarios for current and future air quality and exposure (see sections 3 and 4). To provide a context for this study, however, we briefly characterize the pollution emissions that are the root of the air quality problems in the ZMVM. This information also indicates the available options for improving air quality. We shall also give a brief overview of current air quality in the ZMVM. Finally, we discuss the environmental contingency program that is currently applied to deal with high air pollution levels in the ZMVM.

Emissions

In recent years a number of different emission inventories have been taken. Table 2.1 summarizes the emission inventory by sector for 1996. Table 2.2 summarizes the emission inventory for 1998, but excludes emissions from heavy industry and open-air refuse burning.

Table 2.1 ZMVM Emissions Inventory, 1996

(tons/year)

	NO_x	VOC	PM_{10}
Industry	28,666	16,279	5,700
Services	7,832	234,991	337
Transport	84,961	193,100	7,745
"Natural sources" [a]		134,673	18,072
Total	121,459	579,043	31,854

a. Includes biogenic emissions, forest fires, and open-air refuse burning.
Source: INE (1997).

Table 2.2 ZMVM Emissions Inventory, 1998

(tons/year)

	NO_x	VOC	PM_{10}
Industry[a]	22,094	17,595	3,173
Area sources[b]	8,489	270,190	1,058
Transport[c]	142,603	198,253	8,545
Natural sources[d]	11,802	72,670	5,800
Total	184,988	558,708	18,576

a. Excludes heavy industry.
b. Includes lubricant industry, solvent emissions, forest fires, and services sector, and others.
c. Includes private vehicles, public transport, taxis, and trucks.
d. Includes biogenic emissions and soil erosion.
Source: Comision Ambiental Metropolitana (http://sma.df.gob.mx/inventario/emisiones_1998.htm on 25 July 2000).

Ozone air pollution is formed from the emissions of NO_x and VOCs. The amount produced depends on the amount and location of emitted pollutants; background pollution levels; atmospheric chemistry; geographical, climatological and meteorological characteristics; and atmospheric transport characteristics. Moreover, the chemistry of ozone formation is quite complicated and nonlinear: under certain conditions an increase in NO_x emissions can *reduce* ozone concentrations.

The origins of particulate pollution (PM_{10}) are less clear. PM_{10} may be emitted directly or formed from SO_2 and NO_x reacting with other substances in the atmosphere (secondary particle formation). Likely sources of directly emitted particles include building and construction (road construction), diesel trucks and buses, forest fires, open-air refuse burning, some manufacturing industries, and resuspension of road dust. The relationship between emissions and concentrations, however, is not straightforward due to secondary particle formation. The ambient concentration of air pollutants depends on the amount and location of emissions; the source dependent physical characteristics of the emitted PM_{10} and PM_{10} pre-

cursors such as SO_2 and NO_x[1]; background pollution levels (especially of ammonia); atmospheric chemistry; geographical, climatological and meteorological characteristics; and atmospheric transport characteristics.

Air Quality and the *Programa de Contingencias Ambientales* (PCA)

Most air quality information comes from measurement stations across the area—the *Red Automatica de Monitereo Atmosferica* (RAMA)—that compile time-averaged concentrations (see figure 2.1). The annual reports usually present this information in frequency tables giving the percentage of a year that a certain concentration occurred, or as a single annual average.

Day-to-day air quality data available on the Internet include daily maximum concentrations for five pollutants—PM_{10}, ozone, SO_2, NO_x, CO—and an ultraviolet (UV) index) for five zones[2]: downtown, Northwest, Northeast, Southwest and Southeast. These concentrations are expressed in IMECA points (*Indice Metropolitana del Calidad del Aire*). Table 2.3 shows how concentrations relate to the indicator points (100 = standard).[3]

Figure 2.1 Measuring PM_{10} and Ozone Concentrations across the ZMVM

> ▲ PM10 Stations
> ○ O3 stations
> ▢ Municipalities and Delegations

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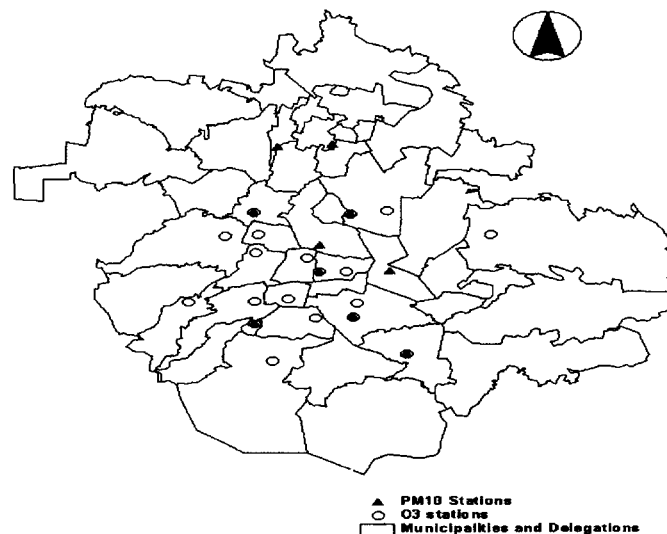
[1] If one would consider $PM_{2.5}$—a smaller mass than PM_{10}—emissions of SO_2 and NO_x become more important since these substances can be converted into particulate matter ($PM_{1.0}$) in the atmosphere. Furthermore, NO_x and VOCs can be attached to existing particulate matter in the atmosphere. The indications are that these small particles have disproportionately large health effects. However, given the lack of air quality information on $PM_{2.5}$ (and appropriate epidemiological data) it impossible to take account of this.

[2] http://sima.com.mx/sima/df (April 2000).

[3] For ozone, the IMECA indicator is proportional to ozone concentrations. For PM_{10} an IMECA number follows from linear interpolation between the values indicated in the table.

4

Table 2.3 The IMECA Indicator System for PM_{10} and Ozone

IMECA points	100	200	300	400	500
PM_{10} μg/m³ (daily average)	150	350	420	500	600
Ozone ppm (daily 1-hr. maximum)	0.110	0.232	0.355	0.477	0.600

Source: INE (April 2000) at http:///www.ine.gob.mx/dggia/indicatores.

Figure 2.2 shows the trend in the IMECA points between 1990 and 1999 for ozone and between 1995 and 1999 for PM_{10}. Table 2.4 shows the number of days per year satisfying air quality standards.

Figure 2.2 Daily Average Trends in the ZMVM for Ozone and PM_{10}

Ozone, daily average 1-hour maximum
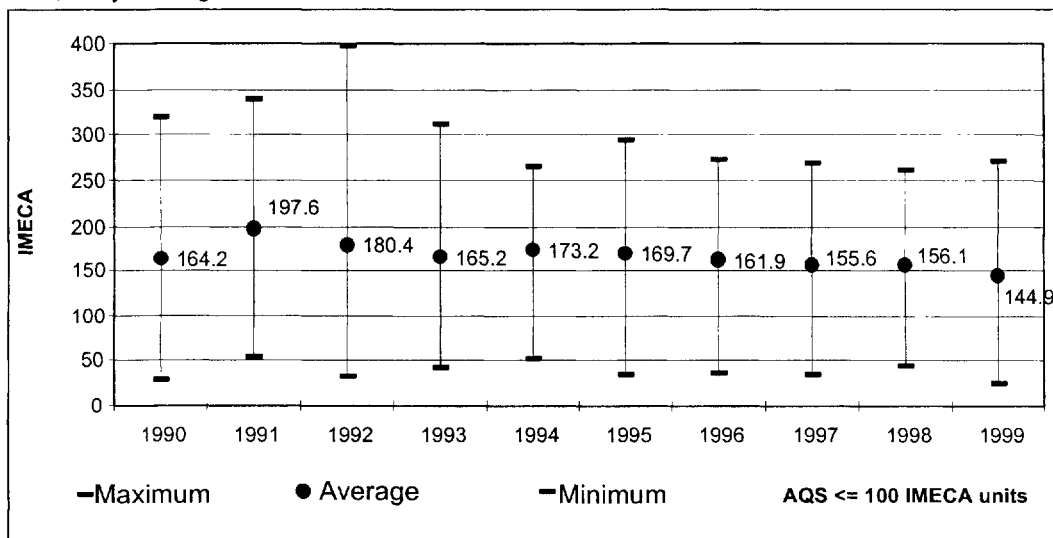
PM_{10}, daily average
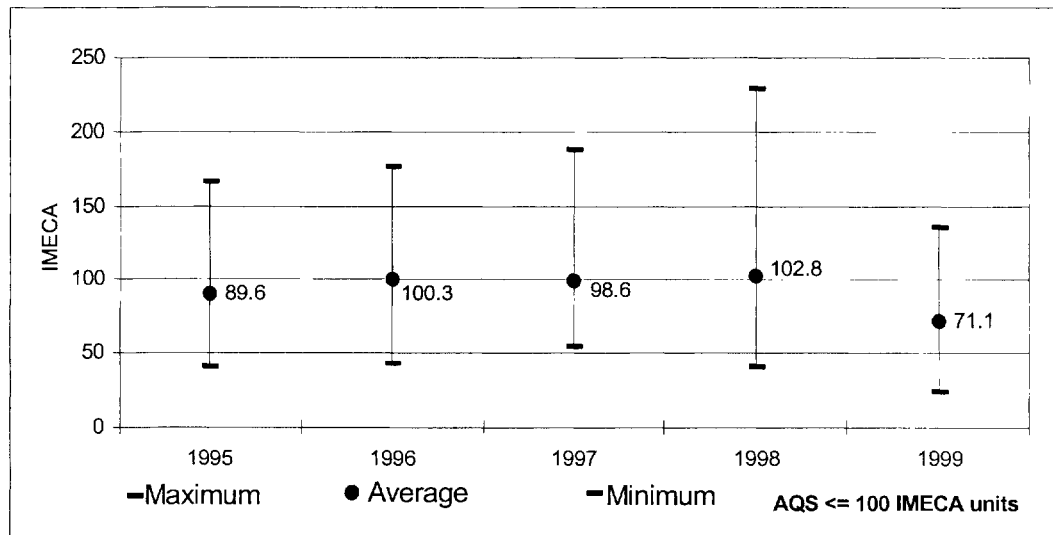
Source: SMA (1999).

Table 2.4 Number of Days Ozone and PM₁₀ Levels Satisfied Air Quality Standards

	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999
Ozone	37	12	34	41	21	41	39	43	45	65
PM₁₀	--	--	--	--	--	273	186	212	176	345

Source: SMA (1999).

For the period 1995–99, the highest value for ozone—0.349 ppm—was measured at the Pedegral station, which is located in the Southwest zone. Ozone air pollution at this station exceeded the standard for 276 days. The least polluted station was Chapingo in the Northeast, with a daily 1-hour maximum concentration of 0.210 ppm .

Air quality standards for PM_{10} include a daily average (150 $\mu g/m^3$) and an annual average (50 $\mu g/m^3$). All stations violated both standards apart from the annual average standard at the Pedregal and Coacalco stations (formerly Villa de las Flores). The highest concentration—a daily maximum of 335 $\mu g/m^3$ (190 IMECA)—was observed in the eastern zone at the Nezahualcoyotl station. The maximum annual average of 94 $\mu g/m^3$ was observed at Xalostoc station. This station has traditionally recorded the highest particulate matter concentrations in the metropolitan area, exceeding particulate standards 58 days per year (16 percent of the year). As a result, in 1995 about 1.2 million people were exposed at least once a year to PM_{10} concentrations above 300 $\mu g/m^3$, the trigger for a Phase I contingency (see table 2.6).

If air pollution goes above certain levels in one of the five zones a PCA is invoked (a contingency program). Table 2.5 describes the three levels of action: Precontingency, Phase I contingency, and Phase II contingency.

Table 2.5 The Environmental Contingencies Program

| Target	Precontingency	Phase I contingency	Phase II contingency
Public health	Suspend outdoor sport activities in schools and parks.	Epidemiological surveillance and communication of recommendations to address health risks.	Suspend activities in public offices, recreational activities and public services.
Transport sector		Restrict traffic (no circulation of hologram II vehicles every other day). Suspend use of publicly owned vehicles by 50%. Improve traffic circulation.	Suspend use of all hologram II vehicles and 80% of public service vehicles.
Industry and services		Reduce certain industrial activities 30-40%. Suspend fuel distribution activities, red brick fabrication, and the thermoelectric power plant Jorge Luque.	Reduce industrial activity by 50%.
Public services / infrastructure maintenance	Suspend maintenance of urban infrastructure.		
Additional actions	Surveillance and control of fires in natural and agricultural areas / landfills.		

Source: SMA (http://sma.df.gob.mx).

Precontingencias (precontingencies) apply to the zones where the corresponding threshold is exceeded. For ozone and the ozone-PM_{10} combination, Phase I applies to the entire ZMVM, while for PM_{10} alone Phase I applies only to the zone where the threshold is exceeded. If the situation persists, however, the *contingencia* is extended to the entire ZMVM. Phase II applies to the entire ZMVM irrespective of which zone exceeds the threshold.[4] Table 2.6 gives the air quality threshold levels applied since May 1998.

Table 2.6 PCA Threshold Air Quality Levels[a]

Levels	Ozone (IMECA)		PM_{10} (IMECA)	
Precontingencia	200–240	(0.233–0.281 ppm)	160–175	(270–300 $\mu g/m^3$)
Phase I	240–300	(0.281–0.355 ppm)	175–300	(300–420 $\mu g/m^3$)
Phase II	> 300	(>0.355 ppm)	> 300	(> 420 $\mu g/m^3$)

a. Since May 1998.

3. Air Quality Modelling

Three strands of science are combined to address the research question of this report: air quality and exposure modelling, epidemiology, and economics. This section focuses on air quality modelling of current and future scenarios. The next section deals with exposure modelling. Figure 3.1 shows the basic elements of the two sections.

Figure 3.1 Overall Approach for Exposure Modelling

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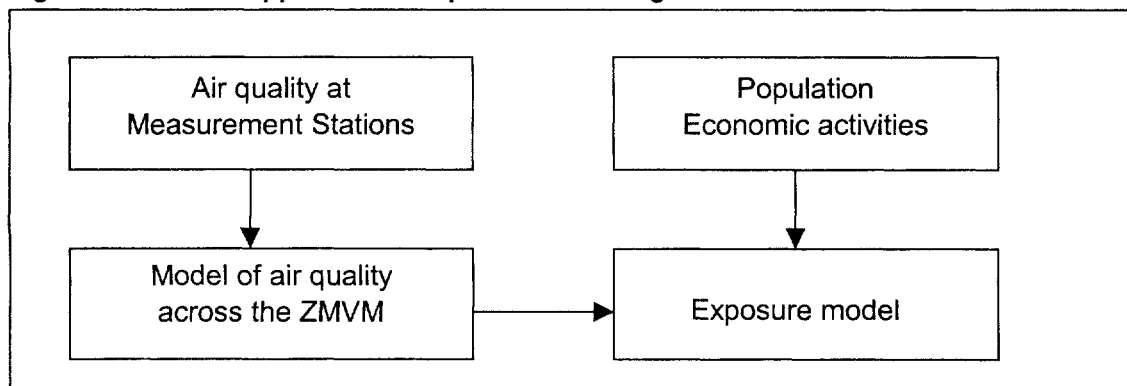
[4] For more information go to http://sma.df.gob.mx/contingencias2000.

Modelling of Current Air Quality

The starting points for modelling air quality are the air quality measurements at specific locations in the ZMVM. Figure 2.1 shows the RAMA air quality measurement network.[5] The empirical data from the measurement stations is used to derive an air quality data field for the entire ZMVM. Since the use of an emission database and an atmospheric transport model are beyond the scope of this study[6], we apply a simple approach to generate the air quality fields. We use measurements at the stations between 1995 and 1999, spatial interpolation in a geographical information system (GIS), and take the average over the institutional units (16 delegations in FD) and 28 municipalities in the State of Mexico.

Because the measurement stations tend to be located in areas with high levels of pollution, information from relatively good air quality areas is patchy. Therefore, interpolating only on the basis of measured data could give unrepresentative results. We avoid such interpolation results by assuming pseudo air quality data at locations where low pollution is expected.[7]

Another difficulty was selecting a reference air quality year for comparison with future air quality. Given the variability of meteorological conditions the reference air pollution year—represented as an air quality frequency distribution—was derived from the distributions for 1995–99. This reference distribution preserves the following three baseline statistics: the average over the five-year interval; the standard deviation over the five-year interval; and the maximum concentration in the five-year interval (see Cesar and others 2000 for more details).

We developed the following distribution metrics for air quality:

- for ozone, a daily 1-hour maximum, a daily maximum 8-hour running average, and a daily average;
- for PM_{10}, a daily average.

Figure 3.2 presents estimates based on the regionally differentiated air quality models for annual average ozone (daily 1-hour maximum) and PM_{10} (daily average) over the ZMVM during 1995–99.

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[5] The network has 19 ozone stations and 10 PM_{10} stations.

[6] The German Fraunhofer Institute developed such a model for CAM during the period of the present study. Unfortunately, results of this development were not (yet) usable for the present project.

[7] We consulted experts to make the best estimate of air quality across the ZMVM.

Figure 3.2 Regionally Differentiated Model for Air Quality by Region, 1995–99
(for 16 delegations in FD and 28 municipalities in the State of Mexico)

Ozone
Annual average daily 1-hour maximum

PM_{10}
Annual average

Mun. & Deleg.
O3 AA 1 h max in PPM
0.08 - 0.094
0.094 - 0.109
0.109 - 0.123
0.123 - 0.138
0.138 - 0.152

Mun. & Deleg.
PM10 AA in ug/m3
40 - 50
50 - 60
60 - 70
70 - 80
80 - 95
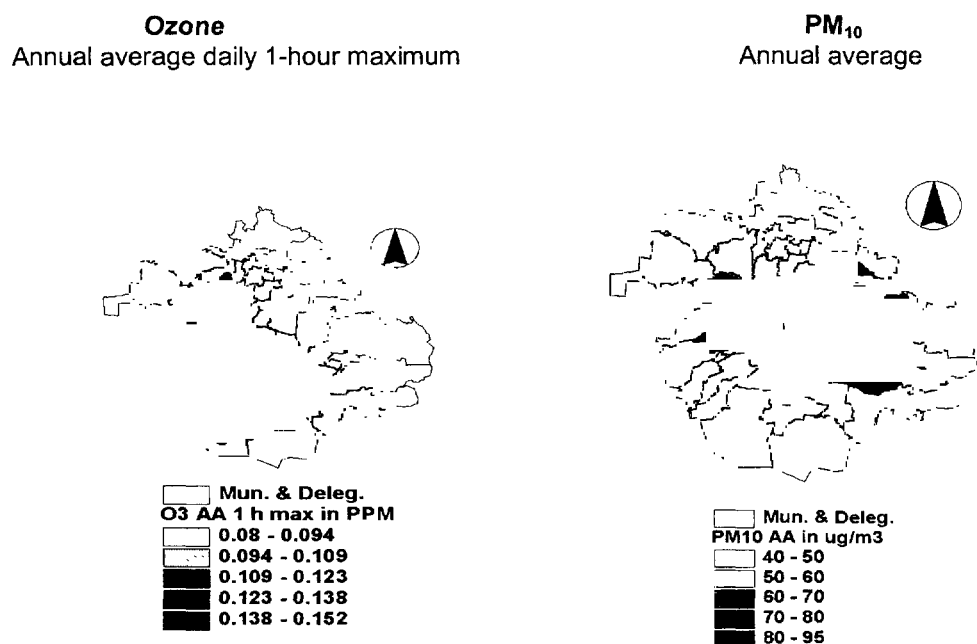
Modelling Future Air Quality

The previous section developed a baseline for air quality for the ZMVM thought to be representative of the end of the 1990s. Since a program to improve air quality would typically take some years to show results, we chose the year 2010 as the reference year for the future. So two research questions arise:

• What reference or business-as-usual scenario for the air quality in 2010 do we use, assuming no air quality policy beyond current measures?

• What future air quality scenario do we want to evaluate, assuming some air quality policy beyond current measures?

The Future Reference Scenario

For an assessment of future air quality one needs insight into factors that determine air quality. The main determinants are emissions and climate, although the latter is not likely to change significantly over the next 10 years. So, the key question is "How will emissions develop over the next decade?" In our speculations about future emissions we deal separately with ozone and PM_{10}, since the origins of these two problems are for the most part unrelated.

Ozone pollution is generated in the presence of NO_x (nitrogen oxides) and VOCs (volatile organic compounds or hydrocarbons) in the atmosphere, and depends on geographical, climatological and meteorological conditions. Most emissions of NO_x and VOCs—50 to 75

percent—come from the use of gasoline vehicles and associated gasoline distribution systems. As table 3.1 shows, emissions standards for new gasoline vehicles have helped to reduce emissions. The share of modern cars with three-way catalysts (electronic systems) has risen to over 10 percent of the total Mexican fleet of gasoline cars (about 70 percent of all cars). In the ZMVM all new gasoline cars are equipped with catalytic systems. According to SMA (1999), however, 32 percent of the cars in the ZMVM are pre-1980 models. And we know that emissions from pre-1986 cars are ten times greater than 1999 cars. Hence, total future emissions depend on the rate at which old cars are replaced with newer models.

Table 3.1 Environmental Characteristics of Cars Sold in Mexico around 1998

Year/model	Features	Percent of vehicle fleet
Pre–1986	With carburator	37.2
1986–1992	Fuel injection	23.8
1992–1993	Catalytic converter	28.6
1994 and after	Full electronic systems	10.4
Total		100.0

Source: Mexican Association of Vehicle Distributors (April 2000) as cited at
www.tradeport.org/ts/ countries/mexico/isa/isar0013.html.

As the composition of the future vehicle fleet in Mexico City is unknown, it is assumed here—in the absence of an integrated model on emission projections for 2010 for mobile sources—that emissions in 2010 will equal those of 1998. This could be the case, for instance, if all improvements in vehicle emissions were exactly offset by the growing importance of road transport in the ZMVM. This would also hold for VOC emissions. Hence, in the absence of more detailed information on emission patterns, we assume that NO_x and VOC emissions will be the same as at the end of the 1990s. Once again, we note the enormous uncertainty of baseline predictions.[8]

Table 3.2 Main Assumptions on Reference Scenario

Pollutant	Main observations	Result
NO_x and VOCs	There are two opposing trends: (i) increase in cars, buses and other pollution sources; (ii) the emissions per unit is decreasing over time. The resultant of these two trends is inconclusive.	Due to lack of information it is assumed that the baseline situation in 2010 is equal to current conditions.
PM_{10}	Origins of PM10 are uncertain. Trends in measured air quality (since 1995) are inconclusive.	Due to lack of information it is assumed that the baseline situation in 2010 is equal to current conditions.

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[8] As mentioned in the previous section, meteorology is an important explanatory variable for actual air quality, but is unlikely to change during the time horizon of this analysis. We, therefore, assume meteorological conditions in 2010 to be similar to the meteorological conditions of the 1995-99 reference air pollution year. Economic variables are equally uncertain and different growth patterns will greatly influence the actual future emissions.

Changes in PM_{10} emission sources and their contribution to air quality is even more unclear. Relevant sources of directly emitted particles include building and construction (for example road surface works), diesel engine vehicles (about 30 percent of all vehicles (SMA 1999)), forest fires, industry, and open-air refuse burning. Measurements of PM_{10} since 1995 (the year in which continuous PM_{10} air quality measuring started) do not indicate a trend, although in 1999 the number of days that standards were violated were the lowest in the five-year period. In the absence of any concrete trend data or integrated model on emission projections for 2010 for fixed and mobile sources, we assume the reference case in 2010 to be equal to the 1998 baseline air quality. We note the arbitrariness of 2010 baseline.

The Future Scenarios

To examine the implications of different levels of pollution control we developed four alternative 2010 scenarios in addition to the 2010 reference scenario:

- a 10 percent air pollution reduction scenario;
- a 20 percent air pollution reduction scenario;
- an air quality standard compliance scenario assuming air quality would improve to the standard (50 $\mu g/m^3$ for PM_{10} and 0.11 ppm 1-hour maximum for ozone) in all locations in the ZMVM—the AQS1 scenario;
- an air quality standard compliance scenario superimposing the needed percentage decrease in concentrations in the most polluted areas (Xalostoc for PM_{10} and Pedregal for ozone) across the ZMVM (68 percent and 47 percent reduction in ozone and PM_{10} concentrations, respectively)—the AQS2 scenario.

To enhance the potential plausibility of the scenarios, we compared them with air quality trends for the South Coast Air Basin of California (see box 3.1). The trends for this area show a decline in maximum ozone concentration of 24 percent in 10 years due to strict controls. Similarly, the PM_{10} annual average decreased by 40 percent, but maximum PM_{10} concentrations remained the same. Hence, the first two scenarios proposed seem plausible but the third and especially fourth scenario would be very difficult, but not impossible, to achieve. We do not consider the policies needed to achieve the concentration reductions in this study.

Box 3.1 Air Quality Trends for the South Coast Air Basin of California

Year	Ref Year	Ozone 1 hour max ppm	Stndrzd to Y0	8 hour max ppm	Stndrzd to Y0	Ref Year	PM10 24 hour max ug/m3	Stndrzd to Y0	AA ug/m3	Stndrzd to Y0
1980	0	0.49	1.00	0.34	1.00					
1981	1	0.39	0.80	0.28	0.84					
1982	2	0.40	0.82	0.27	0.79					
1983	3	0.39	0.80	0.26	0.77					
1984	4	0.34	0.69	0.25	0.74					
1985	5	0.39	0.80	0.29	0.86					
1986	6	0.35	0.71	0.25	0.75					
1987	7	0.33	0.67	0.21	0.62	0	219	1.00	73	1.00
1988	8	0.35	0.71	0.26	0.77	1	289	1.32	82	1.11
1989	9	0.34	0.69	0.25	0.75	2	271	1.24	81	1.11
1990	10	0.33	0.67	0.19	0.58	3	475	2.17	67	0.91
1991	11	0.32	0.65	0.20	0.60	4	179	0.82	65	0.89
1992	12	0.30	0.61	0.22	0.65	5	649	2.96	62	0.85
1993	13	0.28	0.57	0.20	0.58	6	231	1.05	58	0.79
1994	14	0.30	0.61	0.21	0.62	7	161	0.74	56	0.76
1995	15	0.26	0.52	0.20	0.60	8	219	1.00	52	0.71
1996	16	0.24	0.49	0.17	0.52	9	162	0.74	52	0.71
1997	17	0.21	0.42	0.17	0.51	10	227	1.04	56	0.77

Parameter	Ozone 1 hour max	8 hour max	PM10 24 hour max	AA
R Square	0.87	0.78	0.06	0.81
Obs	18	18	11	11
Intercept	0.88	0.88	1.53	1.07
P-value	>0.001	>0.001	0.004	>0.001
m	-0.0238	-0.0216	-0.0498	-0.0400
P-value	>0.001	>0.001	0.475	>0.001
Annual red.	-2.4%	-2.2%	-5.0%	-4.0%
Decade red.	-24%	-22%	-50%	-40%

Source: California Air Resources Board (p. 90, 1999).

Although the definition of baseline air quality in 2010 is highly uncertain, the benefit analyses that follow are valid for the reductions in ambient air pollution associated with each scenario. This is because the dose-response functions used to quantify health benefits and the economic values applied to these benefits are independent of baseline levels of air pollution—they depend only on *changes* in ambient pollutant concentrations.

4. Exposure Modelling and Contingency Estimates

This section describes both the modelling of exposure and the estimation of environmental contingencies (that is, alerts). First, exposure of the population to pollution is modelled by combining the air quality maps (section 3) with information on population distribution. This model will be used to estimate the health impacts of air pollution in the next sections. Second, we estimate the number of environmental contingencies declared to value the economic cost of these alerts (in section 6).

Population

The Mexico City Metropolitan Area (with a population of 17 million in 1995 (*Instituto Nacional de Estadística, Geografía e Informática*, INEGI, 1997) is composed of the Federal District (containing Mexico City and its 8.5 million inhabitants) and part of the State of Mexico. Demographic information used for the population distribution was obtained from Mexican National Institute of Statistics, Geography and Information (INEGI). Figure 4.1 presents a population distribution map of the ZMVM. The GIS working group at the Federal District Government provided political boundaries and geographic definitions. Each locality (represented by a point) is assigned to a municipality (in the State of Mexico) or to a delegation (in the Federal District).

Figure 4.1 Population Map for ZMVM by Municipality, 1995
(in the State of Mexico) or Delegation (in the Federal District)

Population 1995
☐ 14975 - 90543
☐ 90544 - 255838
■ 255839 - 552183
■ 552184 - 839692
■ 839693 - 1696609
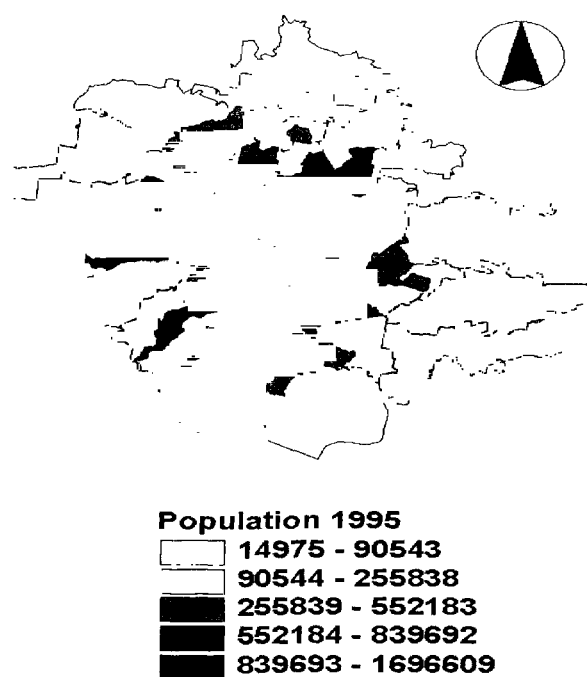
Exposure of the Population

By combining the map of population distribution and the maps of air quality it was possible to assess exposure of the population to air pollutants. This assessment of pollution exposure should correspond with the format of exposure defined and used in the epidemiological studies that our exposure-response models are based on (section 5). Exposure-response relations are constructed from epidemiological information and air quality measurements made at monitoring stations. The statistical exposure-response relationships reflect several factors in the cause-effect chain between air quality and health effects.

One of these factors is actual exposure and inhalation. This element is important since indoor air quality differs greatly from outdoor air quality, and an individual's behavior determines what he/she actually inhales. In epidemiological studies (see meta-analysis in section 5), however, it is assumed that exposure is proportional to measured air quality at a specific outdoor air quality measurement station. Actual exposure will, however, differ from measured concentrations, depending on human activity patterns. Exposure-response functions incorporate the behavior of people in the particular study area and we need to assume that the behavior of people in Mexico City is similar to that of the people in the study areas where the epidemiological studies are performed. Also other factors, such as state of health, age, diet, and so forth may lead to differences in the exposure-response functions in different locations. Due to the lack of information and knowledge as to how these characteristics influence the estimated functions we have to assume there are no differences between the characteristics of people represented in the epidemiological studies and those living in Mexico City.

For exposure to PM_{10}, the metric we use is the annual average of the 24-hour average concentration in an area. For exposure to ozone, the metric we use is the annual average of the daily 1-hour maximum concentration in an area (see Cesar and others 2000 for more details.)

Figure 4.2 summarizes population exposure in the 1995–99 reference air quality scenario we developed in the previous section. The baseline scenario for exposure can be summarized by computing population-weighted exposure for each pollutant. For PM_{10} and ozone this is 64.06 $\mu g/m^3$ /person and 0.114 ppm/person, respectively. The 10 percent reduction scenario would lead to a reduction of 6.41 $\mu g/m^3$ PM_{10}/person and 0.0114 ppm ozone/person, respectively. A 20 percent reduction would double this figure. The AQS1 scenario would result in reduced exposures of 14.06 $\mu g/m^3$/person and 0.0702 ppm/person for PM_{10} and ozone, respectively. The AQS2 scenario would result in the reduced exposures of 29.99 $\mu g/m^3$ /person and 0.0778 ppm/person for PM_{10} and ozone, respectively. These results are summarized in table 4.1.

This table indicates that for PM_{10}, a compliance strategy aimed at achieving the air quality standard in each area and no further air quality improvement would have only a slightly higher benefit than a uniform 20 percent reduction in the annual average. Hence, an emission abatement strategy should target sources in highly polluted and populated areas of the metropolitan area.

Figure 4.2 Reference Scenario for Population Exposure to Ozone and PM₁₀ for the ZMVM, 1995–99

Ozone, maximum and annual average daily 1-hour maximum

Population exposed to the stated concentration
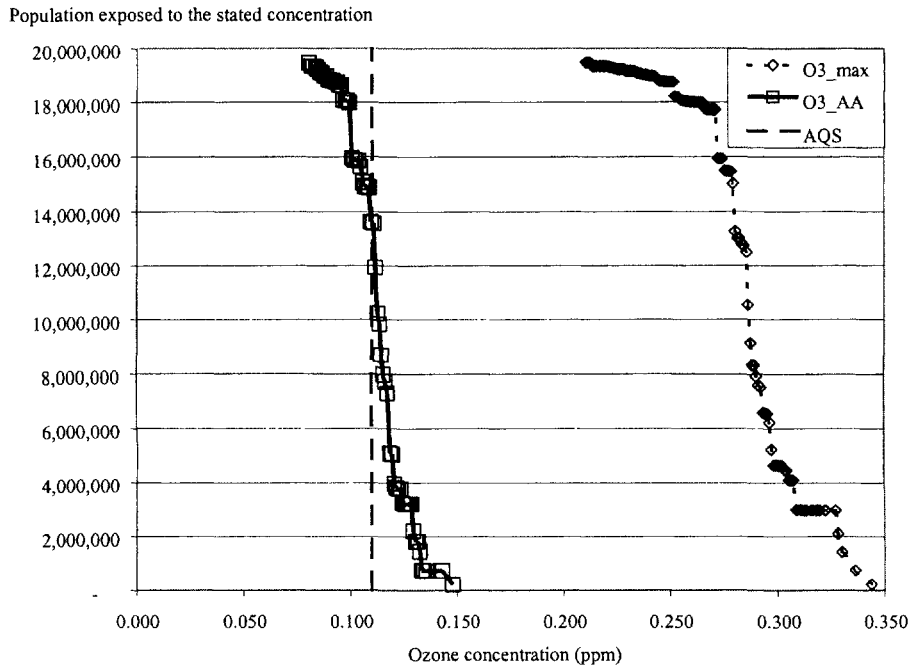
PM₁₀, maximum daily and annual average population exposure

Population exposed to the stated concentration

Table 4.1 Reduction in Population-Weighted Exposure for the Analyzed Scenarios

Scenario	Population weighted exposure to PM_{10} $(\mu g/m^3 / person)$	Population weighted Exposure to ozone $(ppm / person)$
10 percent exposure reduction	6.41	0.0114
20 percent exposure reduction	12.81	0.0227
AQS compliance in each area—AQS1	14.06	0.0702
AQS compliance in worst area—AQS2	29.99	0.0778

Environmental Contingencies

This section estimates the number of times a contingency is declared; that is, the number of days that ozone or PM_{10} concentration measured at each station exceeds the relevant standard for the reference scenario and for each of the future scenarios. Implicitly it is assumed that a contingency is declared if the measured concentration is above the concentration levels stated for the contingency (see section 2 for details on the *Contingencias Ambientales* program). In practice this is not always the case, for instance when air quality on a specific day is expected to improve considerably because of changing meteorological conditions. Therefore, our predictions give an upper estimate of the number of contingencies.

The highest ozone concentrations in the period 1995–99 have been observed at the Pedegral station. The five-year composite frequency distribution of the highest daily 1-hour maximum ozone concentrations for the whole ZMVM is shown in figure 4.3 together with the future scenarios (see section 3). *Contingencia I* is triggered at IMECA=240, which corresponds to 0.281 ppm. According to the frequency distribution a *contingencia I* would be invoked for 10 days, and a *contingencia II* would be invoked for 0 days. (In the 10 percent reduction scenario: 2 days for *contingencia I*. In the *20* percent reduction scenario: 0 days). Noted that the threshold levels from May 1998 are used to make this calculation. Table 4.2 gives the results based on pre-May 1998 threshold levels.

Figure 4.3 Cumulative Frequency Distribution of Pedegral Station and Ozone Air Quality Scenarios, 1995–99
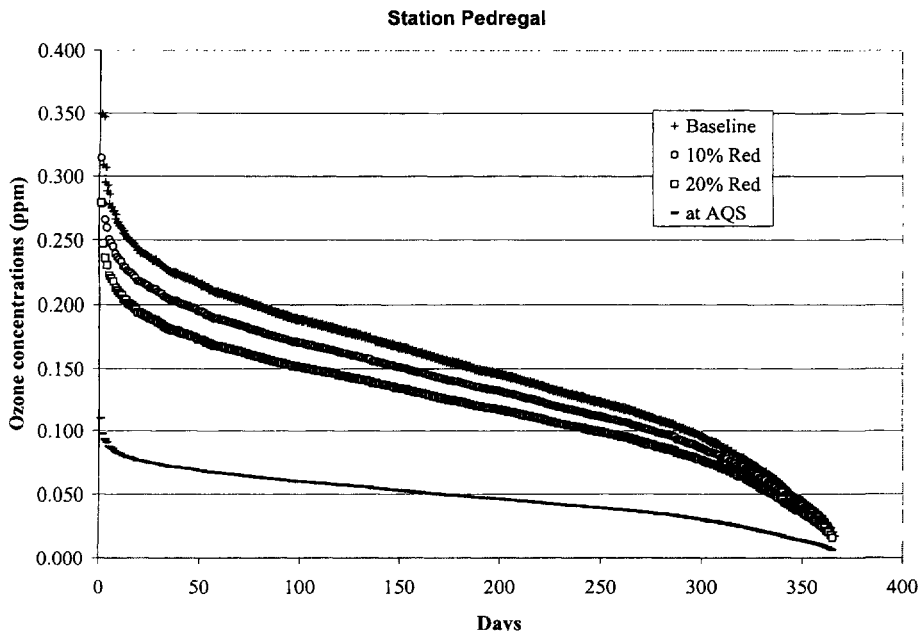
Table 4.2 Days above Ozone Daily 1-hour Maximum Standards and Contingency Stages

Ozone (ppm)	Baseline		10 % Red		20% Red		At standard	
>0.355	0	0%	0	0%	0	0%	0	0%
>0.281	8	3%	2	1%	0	0%	0	0%
>0.233	60	18%	25	8%	5	1%	0	0%
>0.110 AQS	319	87%	306	84%	285	79%	0	0%

The highest frequency distribution for the PM_{10} daily maximum concentration in the ZMVM shows that air quality at that specific place would trigger one *contingencia* (see table 4.3 and figure 4.4). The highest values are measured at the stations Nezahualcoyotl or Xalostoc.

Table 4.3 Days above the PM_{10} Daily Maximum Standards and Contingency Stages

PM_{10} (µg/m3)	Baseline		10% Red		20% Red		At standard	
>420	0	0%	0	0%	0	0%	0	0%
>300_Neza	1	1%	1	0%	0	0%	0	0%
>270_Xal	2	1%	1	0%	0	0%	0	0%
>150 AQS	87	16%	59	12%	34	7%	0	0%

Figure 4.4 Cumulative Frequency Distribution of Xalostoc Station PM_{10} Air Quality Scenarios, 1995-99
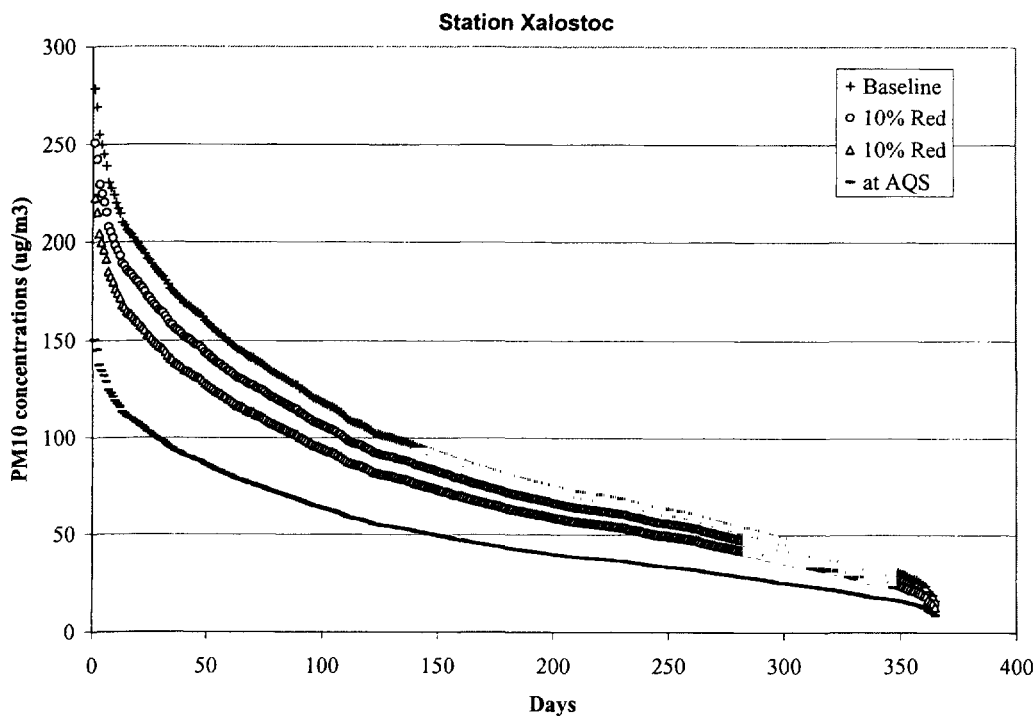
5. The Physical Effect of Air Pollution

This section deals with assessing the effect of improvements in air quality in the ZMVM on human health and environmental contingencies. We begin with an introduction to the health effects of ozone and PM_{10}. Then we discuss the relationships between public health and air quality (exposure-response relations) and quantify the health benefits of the air quality improvement scenarios in physical terms. The economic benefits of these health benefits and of contingencies will be quantified in section 6.

Effects of Ozone and PM_{10} on Health

Not all air pollutants have the same capacity to damage human health. The differences in toxicity are due to the physical and chemical properties of the components of pollution. First, we briefly discuss the types of health effects caused by exposure to air pollution. Then we describe the properties of PM_{10} and ozone as they relate to toxicity. More details can be found in Cesar and others (2000). In the second section we discuss the development of the exposure response functions that are used in this study in more detail.

Health Effects Due to Short- and Long-Term Exposure to Air Pollutants

Susceptibility to air pollution exposure varies greatly among individuals. Individual risk is determined by genetics, age, nutritional state, presence and severity of respiratory and cardiac conditions, and the use of medications. The variability in the estimates found in epidemiological studies may reflect these differences in the populations studied. A good example of variation in individual risk occurs in the evaluation of maximum expiratory flow in healthy children, children with minor respiratory disease and those with asthma, with and without pharmacological treatment, and all exposed to various environmental pollutants. The results show an association between exposure and disease only in children with asthma under pharmacological treatment, in other words, those children who are most seriously ill (Roemer and others 1999). Genetic susceptibility is another factor that could be associated with respiratory diseases (Möller, Schuetzle, and Autrup 1994). Age is an important factor as well, with preadolescents (<13 years) and the elderly (>65) at greatest risk (Wilson and Spengler 1996, Ghio and others 1999).

Toxic effects attributable to short-term exposure to high levels of air pollution (hereafter "acute effects and acute exposure") vary widely. Episodes of high pollution and the associated increases in diverse respiratory and heart diseases and death have been reported since the beginning of the industrial revolution. The most serious acute effect is mortality. Many reports describe an increase in total mortality (not including accidental death) associated mainly with exposure to particulate matter (PM), ozone, and sulfates. (Schwartz 1994a, Wilson and Spengler 1996).

Many studies report increases in mortality due to respiratory complications, and this relationship can obviously be related to exposure to air pollution. Many reports also claim an increase in death due to cardiovascular disease, which would also imply an indirect effect

from air pollution. Both causes of death are associated with exposure to PM, ozone, and sulfates. Mortality attributable to exposure to air pollution occurs mainly in individuals who already suffer from cardiac and/or respiratory diseases. Increased mortality in these groups occurs within one to five days following the hazardous exposure (Schwartz 1994a, Wilson and Spengler 1996).

Short-term exposure to high levels of air pollutants is also associated with diseases of the respiratory tract, both upper and lower: bronchitis, pneumonia, chronic obstructive pulmonary disease, and cough with phlegm. Symptoms aggravated by exposure to certain pollutants such as ozone and PM include asthmatic attacks, cough without phlegm, and wheezing (Wilson and Spengler 1996, Ghio and others 1999).

Episodes of extremely high pollution documented in cities around the world have demonstrated the consequence of human exposure to high concentrations of air pollution. These episodes, however, occur sporadically, whereas exposure to low concentrations of pollutants over long periods of time is a daily phenomenon. Recent studies have focussed on establishing the effects of long-term exposure to low levels of air pollutants.

Health effects due to long-term, low-level exposure to air pollution (hereafter "chronic effects and chronic exposure") are similar to those reported for short-term exposure to high levels of air pollution. A synthesis of the available information concerning chronic exposure is an extremely complex task because many different factors can cause the same symptoms. There are several reports of increased mortality related to chronic exposure, however, most cases involve mainly elderly individuals for whom respiratory and cardiovascular problems are already the principal cause of death (Pope and Dockery 1999). Increased respiratory diseases (such as bronchitis) have also been reported as associated with chronic exposure.

In both acute and chronic exposure to air pollutants, populations are exposed to a complex mixture of compounds whose combined toxic effects could differ from that of each compound alone. A study performed on volunteers exposed to ozone with and without preexposure to H_2SO_4 showed that the preexposed group suffered more severe toxic effects than the group that was not preexposed (Wilson and Spengler 1996).

Particulate matter and ozone are often correlated spatially and over time, making it difficult to separate the effects of the individual pollutants. The mixture of PM_{10} and ozone, however, has proven more toxic than the individual compounds alone (Katsouyanni 1995). Unfortunately models and protocols to analyze the different interactions among environmental pollutants are not yet available (Samet and Speizer 1993). Thus, it is not clear how much each pollutant individually influences elevated mortality and morbidity rates. As a result some cost-benefit studies have chosen to use one index air pollutant rather than estimating effects for multiple air pollutants individually and then adding their effects to get a total air pollution effect. Focusing on a single pollutant provides a conservative approach to estimating air pollution effects. In fact, recent analyses (for example Thurston and Ito 1999) suggest that ozone and PM air pollution effects are relatively independent, since controlling for one pollutant has only modest effects on the concentration-response of the other. Thus, use of a single index pollutant underestimates the overall public health effects and monetary valuations

of air pollution changes. Given that the effect of ozone on mortality independent of particulates is still being debated, we re-evaluated the effect of ozone restricting the analysis to those studies that controlled for particles in the statistical analysis.

Properties of PM₁₀ and Ozone

Aerosol air pollutants (molecular aggregations) have been shown to be more toxic than gases. This is because gaseous compounds are eliminated by the respiratory system much more easily than aerosols, which are rapidly deposited or absorbed. (Wilson and Spengler 1996).

PM₁₀. In the field of air pollution epidemiology, there is now much more interest in the study of PM_{10} and $PM_{2.5}$ particles, and the organic and inorganic compounds in them (Wilson and Spengler 1996, Pooley and Mille 1999). The particles produce toxic effects according to their chemical and physical properties. Their effects on susceptible individuals are much more severe than those produced in normal individuals (Schlesinger 1995, Wilson and Spengler 1996).

The extent of particle penetration into the respiratory system is determined by particle size. Only particles less than 10 μm in diameter enter the respiratory system. This is the reason for focusing on PM_{10} (particles less than about 10 μm).[9] Once particles have entered the respiratory tract, depending on their size, they can accumulate in different sites. Evidence suggests that many of the health effects associated with PM_{10} can be attributed to even smaller particles (Pope and Dockery 1999, Ghio and Samet 1999). Since, however, most epidemiological information refers to PM_{10}, and for the ZMVM there is little air quality information on smaller particles, we restricted our analysis to PM_{10}.

The chemistry of suspended particles complicates empirical epidemiology enormously and has not been analyzed in detail yet, so little epidemiological evidence is available on the influence of the chemical composition of particles. Since little is known about the chemistry of PM_{10} found in Mexico City, this aspect is not accounted for. This contributes to the uncertainties associated with exposure-response modelling.

Ozone. Ozone is a poorly soluble but highly reactive gas. "Bad" ozone (as opposed to "good" ozone in the stratosphere) is mainly produced in the troposphere (ground level) by a series of sunlight-driven reactions involving oxides of nitrogen and volatile organic compounds. Inhaled ozone is partially depleted in the upper airways but a major fraction reaches the lower airways. In the body ozone can react with uric acid, which is secreted by human submucosal airway glands and is present in near millimolar/liter (mmol/l) concentrations of nasal surface liquid. Pryor and his colleagues have proposed that some of the toxic products of the latter reaction (hydroxyhydroperoxides, hydroxyaldehides) are important mediators of ozone effects on underlying epithelium. Bromberg (1999) has calculated that ozone per se does not even reach the epithelial cell apical membrane in conducting airways.

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[9] Actually, the metric is not the size, but the aeolic behavior of particles, as measured in equipment that mimics the human respiratory system. This metric comes close to size.

The proportion of ozone uptake attributed to surface liquid dynamics decreases progressively as a surface liquid thins and its reactivity with ozone diminishes, so that the highest epithelial tissue dose is predicted for the terminal bronchiole-respiratory bronchiole region, which is, indeed, a site of damage in ozone-exposed animals. Bronchoscopic sampling along airways also indicates that a substantial fraction (35 percent) of orally inspired ozone is taken up in the upper airway and trachea and that ozone in exhaled air is limited to the initially expired volume representing airways dead space (Bromberg 1999).

The toxicity of ozone inhalation in large airways is supported by evidence of ciliated cell loss and increased epithelial mitotic index in small animals, netrophilic inflammation in humans, increased bronchial artery blood flow in sheep, and by the symptoms of cough and substernal pain exacerbated by deep inspiration in humans (Bromberg 1999).

Development of Exposure-Response Models for Mexico City

Meta-analysis

Although the number of published studies on the health effects of air pollution has grown during the past decade, specific studies of the ZMVM are still limited. We, therefore, decided to summarize relevant international and national published reports through a meta-analysis, which combines the results from various studies to identify consistent patterns. Due to the rapid growth of the field of epidemiology since the 1960s, the number of publications is overwhelming and a classical narrative review is no longer appropriate for summarizing findings. Despite limitations, statistical analysis of compiled published results has become more common when dealing with an extensive offering of differing and inconclusive results.

Identification, Selection and Classification of Bibliographical Information

The meta-analysis involved an exhaustive search of published studies on human health effects due to exposure to ozone and PM_{10} using Medline, Pubmed, Biomed-net and Aries databases. Manual library searches examined mainly Mexican publications. Not all the bibliographic material collected was appropriate for the statistical analysis.

Criteria for inclusion were

- peer-reviewed published papers evaluating the association between exposure to ozone or particles and clinically identifiable human health effects (biochemical and molecular effects were not included); and

- papers that quantified any type of particles: Total Suspended Particles (TSP), black smoke (BS), coefficient of haze (CoH), or any PM.[10]

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[10] We used the approach of Dockery and others (1993) to convert air quality expressed in these metrics into PM_{10} concentrations.

Criteria for exclusion were

- papers that did not present information for the variance, standard error or confidence intervals of the association estimate;

- reports based on small populations or excessively large confidence intervals or standard errors;

- papers that did not control for temperature and seasonal variation over the study time period; and

- papers that did not correct for ozone effects when addressing PM_{10} and vice versa.

According to these criteria, 126 publications were selected for the statistical analysis of ozone and PM_{10} health effects (the list appears in Cesar and others 2000).

Exposure-Response Functions

Most studies express the health effect (y) as a function of the degree of change in health and the measured change in air pollutant levels (ΔC). The calculation of the corresponding change in health impact (Δy) depends on the exposure-response (ER) functions from epidemiological studies. The ER-function estimations may differ from each other in several ways, for example, in the use of standard definitions of health endpoints, baseline populations and the functional form of the estimated relationship. Some studies assume linear relationships, while others use log-linear functions.

The linear relationship is of the form

$$y = \alpha + \beta \cdot C \tag{5.1}$$

The log linear relationship is of the form:

$$y = \alpha \cdot e^{\beta \cdot C} \text{ or, equivalently } \ln(y) = \alpha + \beta \cdot C \tag{5.2}$$

Despite some statistical limitations, results from different studies were transformed to percent changes in the health effect for each 10 units of variation in the pollutant concentration.

Pooled Estimates

We obtained a single pooled estimate of the health effects reported from the selected studies by using a weighted average. Deciding on a method to obtain an average estimate is not an easy task. Estimates from different studies could be different because of random variation and also because of a true difference coming from differences in exposure and susceptibility factors. To take into account heterogeneity of effects of reported studies we applied a random-effects model to pool the studies (DerSimionian and Laird 1986.) Random-effect models assume that the true effect is decomposed into the mean population effect and between-study variability. With a random-effects model the estimate of the average value is the

weighted average of the study estimates taking into consideration the sampling error and the between-study variability. Note that the within-study variability is not taken into account and only the average estimate is used in the quantification of the health benefits.

Since the analysis applies to Mexico City, articles based on Mexico City population were given double the weight of international cases because they are thought to better reflect the Mexican reality in terms of susceptibility and sociodemographic characteristics. The potential influence of long-term exposures on health, and especially in the reduction of life expectancy, could be one of the most influential end points. This is discussed in more detail in box 5.1. An example of mortality due to acute exposure to PM_{10} is presented in box 5.2.

Table 5.1 summarizes the ER functions and the background rates for health effects as they are used in the present study. The exposure response coefficients in the second and third columns of the table come from the meta-analyses described above. (For some health endpoints, a meta-analysis was not possible and the source of the estimate is a single study.) The studies used to derive each coefficient reported in table 5.1 may be found in Cesar and others (2000). In the next two sections we will include only nonoverlapping health endpoints to prevent double counting of the benefits from air pollution reduction.

Box 5.1 Premature Mortality Due to Long-Term Exposure to PM_{10}

Cross-sectional studies and cohort studies have been conducted to study the effect of long-term exposure to particles and premature mortality. Cohort studies are preferable to cross-sectional studies because cohort studies can control for other factors related to mortality such as smoking status or occupation. To date three cohort studies in the United States have followed a significant number of individuals for at least 8 and up to 17 years (Dockery and others 1993, Pope and others 1995, Abbey and others 1993). During the study period, air pollution data were gathered from local monitoring stations to estimate average pollution exposures for individuals within the study. The 1999 U.S. EPA Report to Congress on the Benefits and Costs of the Clean Air Act 1990 to 2010 and other authors prefer to use the Pope and others (1995) study, which is based on extensive evaluation of confounders, as well as a larger sample size and greater geographic coverage. This cohort study found a concentration-response coefficient of 17 percent for a 24.5 $\mu g/m^3$ increase of $PM_{2.5}$, or a 6.6 percent increase for a 10 $\mu g/m^3$ increase of $PM_{2.5}$. For comparison with PM_{10} studies, this is equivalent to an increase in mortality rates of 3.84 percent for a 10 $\mu g/m^3$ increase of PM_{10}.

Table 5.1 Best Estimates of Exposure-Response Functions for the ZMVM for the General Population (unless stated otherwise)

Endpoints	Percent change per 10 ppb daily 1-h max ozone	Percent change per 10 μg/m3 daily average PM_{10}	Background rates (per 100,000 persons)	Notes
Hospital admission				
Respiratory	3.76	1.39	411	[1]
Cardiocerebrovascular	0.98	0.60	403	[1]
Congestive heart failure	–	1.22	5.1	
Emergency room visits (ERV)				
Respiratory	3.17	3.11	3,168	[1]
Restricted activity days (RAD)				
Total (adults)	–	7.74	646,050	
Work loss days (adults)[g]	–	7.74	236,520	
Total (children)[g]	–	7.74	646,050	Assumed same as total RAD background rate in adults
Work loss days women due to RAD in children[h]	–	7.74	332,000	
Minor restricted activity days (MRAD)				
Total (adults)	2.20	4.92	780,000	
Effects in Asthmatic [c]				
Asthma attacks[a]	2.45	7.74	12,740	
Cough without phlegm (children)	–	4.54	21,200	
Cough with phlegm (children)	–	3.32	2,120	0.1* chronic cough without phlegm
Cough with phlegm & bronchodilator usage[d]	–	10.22	56,174	
Some respiratory symptoms (children)	0.66	–	21,200	Same as cough without phlegm
Lower respiratory symptoms[d]	0.23	–	8,810	
Respiratory symptoms				
Upper respiratory symptoms[d]	1.50	4.39	22,400	[1]
Lower respiratory symptoms[d]	2.20	6.85	9,000	[1]
Wheeze[d]	1.32	–	10,600	
Acute bronchitis[d]	–	11.0	4,400	
Morbidity-Chronic Exposure				
Chronic bronchitis, additional cases	–	3.60	707	
Chronic cough, prevalence (children)	–	0.30	5,770	
Mortality-Chronic Exposure				
Total[e]	–	3.84	b	[1]
Mortality-Acute Exposure				
Total[f]	0.59	1.01	577.9	[1]
Infant[f]	–	3.52	3,133	[1]

Source: Cesar and others (2000), Summary Tables, II.4.

a. Included in MRAD (U.S. EPA 1999).

b. Estimated with life expectancy and survival probability tables by 1-year age interval, see section III.2.2.

c. ER-functions to be applied to asthmatics in population only (5 percent of population).

d. Included in RAD for PM_{10} (U.S. EPA 1999).

e. Originally identified for people age 30+ but applied to all population.

f. Not included in aggregated benefit estimates because of methodological problems of separating morality associated with acute exposure from mortality associated with chronic exposure.

g. Assumed the same ER-function as RAD total adults.

h. WLD in adult women due to RAD of their children.

1. Boletín de Información Estadística. Daños a la Salud. Secretaría de Salud, Septiembre, Mexico (1996).

Box 5.2 Percent Change in Mortality Due to Acute Exposure to PM$_{10}$

Of all the toxic effects attributed to PM$_{10}$, death has been the most thoroughly documented. Death due to the acute effects of air pollution occurs generally between one and five days after the hazardous exposure. Since the 1950s studies have recorded increased mortality associated with high levels of pollution. This analysis includes the major studies carried out in the Americas, Europe, Australia and Asia since 1970.

The figure below shows the percent change in general mortality associated with an increase in air pollution. The percent change, considering all the cases, establishes an increase in mortality of between 0.06 and 2.82 percent. These data are for total, nonaccidental deaths.
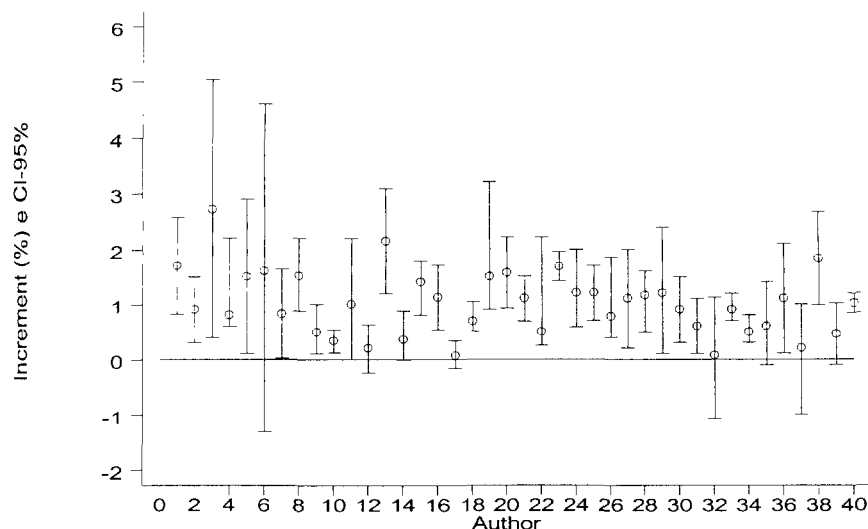
Note: Percent change in general, nonaccidental mortality for each 10 μg/m^3 increase in PM$_{10}$. The numbers represent the following studies: 1. Anderson and others 1996 (London), 2. Ballester and others 1996 (Valencia), 3. Borja-Aburto and others 1997 (Mexico), 4. Bremner and others 1999 (London), 5. Dockery and others 1992 (St. Louis), 6. Dockery and others 1992 (Tennessee), 7. Gamble and Lewis 1996 (Chicago), 8. Gamble and Lewis 1996 (Utah), 9. Ito and Thurston 1996 (Chicago), 10. Kelsall and others 1997 (Philadelphia), 11. Kinney, Ito, and Thurston 1995 (Los Angeles), 12. Lee and Schwartz 1999 (Seoul), 13. Mazumdar and Sussman 1983 (Pittsburgh), 14. Moolgavkar and Luebeck 1996 (Ohio), 15. Moolgavkar and others 1996 (Philadelphia), 16. Neas, Schwartz, and Dockery 1999 (Philadelphia), 17. Ostro 1995 (California), 18. Ostro and others 1996 (Santiago), 19. Pope and Kalkstein 1996 (Utah), 20. Pope III 1999 (Ogdem), 21. Pope, Hill, and Villegas 1999 (Provo), 22. Pope, Hill, and Villegas 1999 (Utah), 23. Samet and others 1998 (Philadelphia), 24. Schwartz 1994c (Cincinnati), 25. Schwartz and Dockery 1992a (Philadelphia), 26. Schwartz and Dockery 1992b (Steubenvile), 27. Schwartz 1993 (Birmingham), 28. Schwartz 1994b (Detroit), 29. Schwartz 1994c (Ohio), 30. Simpson and others 1997 (Brisbane), 31. Spix and others 1993 (Erfurt), 32. Sunyer and others 1996 (Barcelona), 33. Touloumi and others 1996 (Athens), 34. Touloumi, Samoli, and Katsouyanni 1996 (Athens), 35 Verhoeff and others 1996 (Amsterdam), 36. Wordley, Walters, and Ayres 1997 (Birmingham), 37. Zmirou and others 1996 (Lyon), 38. Castillejos and others 2000 (México), 39. Cropper and others 1997 (Delhi), 40. Pooled estimate.

Pooled Estimate of the Effect of PM$_{10}$ in Total Mortality

Mortality	Mean	CI95%
Total	1.01	0.83,1.19

Health Effects

The morbidity avoided due to a reduction in exposure to PM_{10} and ozone are now calculated as follows:

$$I = \Delta Y \cdot Y_b \cdot \Delta C_{PopW} \cdot Pop \qquad (5.3)$$

With:

I	= Impact
ΔY	= ER-function coefficient (percent change in impact per unit of pollutant)
Y_b	= Background health impact rate (impacts/100,000 persons)
ΔC_{PopW}	= Population weighted change in exposure (concentration/person)
Pop	= Population exposed (persons)

Box 5.3 presents estimates of avoided hospital admissions for respiratory problems with a 10-percent reduction in annual average daily 1-hour maximum ozone concentrations. The avoided morbidity impacts are expressed in number of cases.

When quantifying the avoided mortality impacts it is important to take into account that the exposure-response functions provide estimates of premature mortality rather than additional deaths. The economic valuation of an additional death is quite different from the valuation of only a limited number of years of life lost (YOLL). Following ExternE (1999) we have assumed that acute and chronic premature mortality leads, on average, to 0.75 and 5 years of life lost per case respectively. The quantification of the number of YOLL related to mortality associated with acute exposure is thus equal to the number of premature deaths times the average YOLL (0.75 years). The quantification of the avoided YOLL related to mortality associated with chronic exposure is more complex as death occurs later. Therefore, the age-specific life expectancy and death rates are taken into account. A more detailed discussion of the method followed is presented in Cesar and others (2000).

Tables 5.2 and 5.3 present the morbidity health benefits for the air pollution reduction scenarios discussed in sections 3 and 4. Table 5.4 gives the mortality health benefits for the air pollution reduction scenarios.

Box 5.3 Estimation of Avoided Hospital Admissions for Respiratory Problems Due to Ozone Pollution Improvements in 2010

- A 10 percent improvement of air quality results in a reduction of population weighted exposure of 0.011357 ppm/person (ppm relates to annual average daily 1-hour max ozone concentration), see section 4.

- The background rate for this type of hospital admissions is 411 per 100,000 persons per year.

- The exposure-response relation is 3.76 percent per 10 ppb ozone concentration change.

So the number of avoided admissions is: 0.0376/10 change/ppb × 1,000 ppb/ppm × 0.00411 admissions/person × 0.011357 ppm/person × 18,787,934 persons = 3,300 admissions.

Table 5.2 Reduction in Morbidity Health Impacts Due to Ozone Pollution Reduction Scenarios for the ZMVM in 2010

Endpoints	Scenario			
	10%	20%	AQS1	AQS2
Hospital admission				
Respiratory	3,300	6,600	20,404	22,597
Cardiocerebrovascular	842	1,684	5,207	5,767
Emergency room visits				
Respiratory	21,429	42,857	132,501	146,746
Minor restricted activity days				
Total (adults)	2,495,805	4,991,610	15,432,494	17,091,616
Effects in asthmatics				
Asthma attacks[b]	3,330	6,660	20,591	22,805
Some respiratory symptoms (children)	404	809	2,501	2,770

Table 5.3 Reduction in Morbidity Health Effects Due to PM$_{10}$ Pollution Reduction Scenarios for the ZMVM In 2010

Endpoints	Scenario			
	10%	20%	AQS1	AQS2
Hospital admission				
Respiratory	688	1,376	1,510	3,221
Cardiocerebrovascular	291	582	638	1,361
Congestive heart failure (elderly)	0.36	0.71	0.78	1.66
Emergency room visits				
Respiratory	11,858	23,717	26,029	55,507
Restricted activity days				
Total (adults)	4,102,282	8,204,565	9,004,464	19,202,173
Work-loss days (adults)	998,116	1,996,233	2,190,854	4,672,035
Total (children)	1,630,710	3,261,421	3,579,391	7,633,112
Work-loss days for women due to RAD in children	428,269	856,537	940,045	2,004,662
Minor restricted activity days				
Total (adults)	3,148,315	6,296,630	6,910,516	14,736,794
Effects in asthmatics				
Cough without phlegm (children)	1,569	3,139	3,445	7,346
Cough with phlegm (children)	115	230	252	537
Chronic morbidity				
Chronic bronchitis, new cases	3,063	6,126	6,723	14,337
Chronic cough, prevalence (children)	574	1,148	1,260	2,686

Table 5.4 Reduced Deaths or YOLL Related to Ozone and PM_{10} Pollution Reduction Scenarios for the ZMVM in 2010[a]

Endpoints	Scenario			
	10%	20%	AQS1	AQS2
Mortality-acute exposure				
Total population—YOLL Ozone	546	1,091	3,374	3,737
Mortality-chronic exposure				
Total population—YOLL PM_{10}	14,131	28,261	31,016	66,143

a. 3% discount rate, average YOLL per death are 0.75 and five years for morality associated with acute and chronic exposures, respectively.

6. Economic Valuation of Scenarios

Earlier studies suggested that improving air quality in Mexico City would bring limited benefits (Hernandez-Avila and others 1995). These studies, however, used a narrow definition of health benefits. Estimates of the effects of air pollution on human health were quantified for fewer endpoints than in section 5. In addition, health benefits were valued using a very narrow definition of benefits. Reductions in premature mortality were valued by the associated increase in earnings (the human capital approach). Reductions in illness were valued using the savings in medical costs and reductions in lost work time that result from reducing illness (the Cost of Illness approach). In the present study we use a broader definition of the value of health benefits: In addition to valuing avoided illness costs and productivity losses, we estimate the amount that people are willing to pay to avoid the discomfort associated with illness and the disutility associated with premature death. This section first discusses the methods used to value health benefits in this study. Next we present the main results of the health benefit analysis and the economic benefits of a reduction in the number of contingencies experienced.[11]

Economic Valuation of Premature Mortality and Morbidity

Economists value avoided premature mortality by the amount that people are willing to pay to reduce their risk of dying (Hernandez-Avila and others 1995). Ideally, "willingness to pay" (WTP) should capture the loss in satisfaction—from consumption, leisure time, interaction with friends and family—that occurs when life is shortened.[12] It should, in particular, exceed the monetary value of the consumption (or income) lost when a person dies prematurely. In studies conducted in the United States (Viscusi 1993) estimates of WTP to reduce risk of death suggest that WTP is between 8 and 20 times as large as the corresponding gain in earnings from living longer. (The methods used to estimate WTP for reduced risk of death are discussed below.) Ideally, changes in premature mortality should be valued using WTP. The value of earnings lost when a person dies prematurely (the Human Capital measure of

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[11] A more detailed discussion of the methodology, the assumptions and the results is presented in Cesar and others (2000).

[12] Typically, WTP to reduce risk of death is expressed in terms of the Value of Statistical Life (VSL). If each of 10,000 people are willing to pay $100 to reduce their risk of dying by 1 in 10,000, they are together WTP $1,000,000 for risk reductions that sum to one statistical life. The $1,000,000 is termed the Value of a Statistical Life.

the value of reduced risk of death) will, in general, understate the economic value of reduced risk of death (Freeman 1993).

Avoided morbidity is also valued by the amount a person will pay to avoid a particular illness. For minor illnesses (such as respiratory infections) the correct valuation concept is what an individual would pay to avoid the illness with certainty.[13] This should capture the value of the pain and suffering avoided, as well as the value of time lost due to illness (both leisure and work time) and the costs of medical treatment. In cases where some of these costs are not borne by the individual, and are therefore not reflected in his WTP, the value of the avoided costs must be added to WTP to measure the social benefits of reduced morbidity. It is often the case that the costs of medical treatment (hereafter referred to as COI) and time lost from work (Productivity Loss) are not borne by the sick person. We therefore measure the value of avoided mortality by WTP to avoid lost leisure time and the discomfort associated with illness, but add to this the value of lost productivity and the costs of medical treatment. As in the case of mortality, it can be argued that the avoided value of lost productivity and medical costs alone will understate the economic value of reduced morbidity (Freeman 1993).

Estimates of WTP to reduce risk of death and estimates of WTP to avoid illness unfortunately do not exist for Mexico. It is therefore necessary to transfer to Mexico estimates from countries where WTP studies have been conducted. When extrapolating estimates of WTP from one country to another, adjustments must be made for the effect of income on WTP. In general, WTP (both for mortality and for morbidity) should increase with income. In transferring estimates from country A to Mexico the formula used is

$$WTP_{MEXICO} = WTP_A \, [Income_{MEXICO}/Income_A]^{\varepsilon}$$

where ε represents the income elasticity of WTP—the percentage change in WTP corresponding to a one percent change in income.

It should be acknowledged that there is considerable uncertainty regarding estimates of the income elasticity of WTP, especially for mortality, as well as uncertainty regarding the estimates of WTP themselves. We handle this uncertainty in two ways. First, we use two estimates of the income elasticity of WTP—1.0, and 0.4. Holding WTP_A constant, the 0.4 elasticity results in a larger WTP estimate for Mexico than the 1.0 elasticity. Indeed, when WTP estimates from the United States are transferred to Mexico using Purchasing-Power-Parity-adjusted income, an income elasticity of 0.4 implies a WTP for Mexico that is about the size of WTP in the US. We therefore view WTP estimates based on an income elasticity of 0.4 as upper bound estimates, and estimates based on an income elasticity of 1.0 as central case estimates.

Second, to handle uncertainty about the size of WTP, especially WTP for reduced mortality, we also present conservative, lower bound estimates of the value of mortality and morbidity. Specifically, we measure the value of avoided premature mortality using the Human Capital/foregone earnings approach, as well as by transferring estimates of WTP to reduce

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[13] In the case of rarer events, such as heart attack or stroke, the correct valuation concept is what a person would pay to reduce his risk of the illness occurring.

risk of death from other OECD countries to Mexico. In the case of morbidity we present estimates of avoided illness costs and productivity losses alone (i.e., without WTP) as conservative, lower bound estimates of the benefits of reduced morbidity.

To summarize, 3 sets of benefit estimates are provided for each of the four air quality scenarios analyzed. (See Table 6.1.) Health Benefit Estimate 1, the most comprehensive, includes WTP to avoid illness, as well as avoided illness costs (COI) and reduced losses in productivity, in valuing reduced morbidity. Avoided premature mortality is valued using WTP. Health Benefit Estimate 2 includes the same comprehensive measure of the value of reduced morbidity, but values avoided premature mortality using foregone earnings, a lower bound to WTP. Health Benefit Estimate 3, the most conservative, values morbidity using COI and productivity measures alone and premature mortality using foregone earnings. Health Benefit Estimates 1 and 2 vary depending on the income elasticity used to transfer WTP estimates for morbidity and mortality from other countries to Mexico.

For reasons described more fully below, we view Health Benefit Estimate 1, with an income elasticity of 1.0 used for benefits transfer, as a Central Estimate of the value of health benefits. Health Benefit Estimate 1, using an income elasticity of 1.0, is viewed as a High Estimate and Health Benefit Estimate 3 as a Low Estimate. We interpret Health Benefit Estimate 1, using an income elasticity of 1.0 for benefits transfer, as a "Central Estimate" of the health benefits of pollution reduction. This is motivated by the belief that the estimates of WTP for reduced morbidity used in the analysis are more reliable (and certainly less controversial) than the estimates of WTP for reduced risk of death. It is also the case that WTP for reduction in risk of death is based on small risk changes. Applying a marginal WTP estimate to the large risk changes in AQS1 and AQS2 may yield implausibly large estimates of WTP. Health Benefit Estimate 3, which uses a lower bound estimate for morbidity (= Productivity Loss + COI) and mortality (Human Capital approach), is a conservative, lower bound estimate to benefits.

Table 6.1 Overview of Health Benefit Estimates Presented in the Study

	Income elasticity of WTP
Components of Health Benefits	1.0
1. Health benefit estimate 1 including morbidity (Prod. Loss + COI +WTP) and WTP for mortality	High estimate
2. Health benefit estimate 2 including morbidity (Prod. Loss + COI +WTP) and human capital losses for mortality	Central estimate
3. Health benefit estimate 3 including morbidity (Prod. Loss + COI) and human capital losses for mortality	Low estimate

The following sections explain in more detail how productivity losses, COI, human capital losses and WTP are measured. For a more detailed discussion of these methods see Cesar and others (2000).

Loss of Productivity

Loss of productivity (also referred to as the "change in productivity" method or "effect on production") is a valuation method that computes the loss in output due to illness or some other event. The loss of productivity method is applied in two situations.

First, environment-induced health effects reduce production. Foregone income as a result of illness, which is assumed to be evenly distributed over time, is valued by using the average wages in the formal and informal sectors (see Cesar and others 2000 for a further discussion). Assuming an annual increase of 2.45 percent the formal and informal daily wage level in 2010 are US$ 24.8 and US$ 10.3, respectively (2010 values in 1999 prices).[14] For those air pollution–related health effects where we are not able to identify the age of the people affected we use the population-weighted wages for the whole ZMVM population. This leads to an average daily wage of US$ 6.49 (2010 values in 1999 prices). Using total population-weighted wages to estimate morbidity damage for specific age groups in the ZMVM would lead to an underestimation of the damages if only adults or children are affected and an overestimation of the damages if only the elderly are affected.[15] For effects in the elderly we assume no economic losses occur. For effects in adults and children we use an adult population-weighted wage in of US$ 9.52 (2010 values in 1999 prices). The assumed number of "days lost" due to air pollution is presented in table 6.2

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[14] In the absence of data on the expected wage growth in Mexico we have used the growth in GNP per capita as a proxy by deducting the population growth rate (1 percent) from the expected growth in GNP (3.7 percent).

[15] Effects resulting in loss of time for children leads to productivity losses in adults resulting from care for the children.

Table 6.2 Days Lost Per Case in Mexico City for the General Population
(unless stated otherwise)

Endpoints	Days lost[a]	Source
Hospital admission		
Respiratory	8	ExternE (1999, 2000)
Cardiocerebrovascular	45	ExternE (1999, 2000)
Congestive heart failure (elderly)	7	ExternE (1999, 2000)
Emergency room visits		
Respiratory	5	ExternE (1999, 2000)
Restricted activity days		
Total (adults and children)	0[b]	
Work-loss day	1	
Minor restricted activity days		
Total (adults)	0	Assumed
Effects in asthmatics		
Asthma attacks	1	ExternE (1999, 2000)
Cough without phlegm (children)	1	Assumed
Cough with phlegm (children)	1	Assumed
Some respiratory symptoms (children)	1	Assumed
Chronic morbidity		
Chronic bronchitis, additional cases	7	Extrapolated from ERISCA[c]
Chronic cough, prevalence (children)	7	Extrapolated from ERISCA[c]

a. Including recovery days at home.
b. The loss-of-productivity part is accounted for by the work-loss-day part of RAD.
c. See Cesar and others (2000).

Second, loss of productivity occurs during environmental contingencies (ECs) or alerts. As discussed in section 2.2 these alerts lead to temporary closures in production infrastructure to avoid further air pollution. Here the loss of productivity is measured by estimating the difference in gross national product (GNP) with and without an environmental alerts. A distinction has been made between production losses in the industry and the transport sectors.

Cost of Illness

The cost of illness for the different morbidity endpoints is quantified in terms of direct costs for treatment of an illness. These costs are dependent on the social security system. In Mexico the most common health systems are the public health insurance system for uninsured people (*Poblacion Abierta*), the public health system for low-income employed people (IMSS), and the private health insurance system (*Privado*). Hernandez-Avila and others (1995) conducted a COI study for Mexico by including the costs of consultations, laboratory tests, and medication. The inflation-corrected numbers they obtained are presented in table 6.3.

Table 6.3 Cost of Illness Per Case in Mexico for the General Population (unless stated otherwise)
(costs in US$, 2010 values in 1999 prices)

Endpoints	Cost of Illness[a]				
	Public services	IMSS	Private	Others	Average[b]
Hospital admission					
Respiratory	939	1,252	3,131	1,565	1,870
Cardiocerebrovascular[c]	2,818	3,757	9,392	4,696	5,611
Congestive heart failure (elderly)[d]	939	1,252	3,131	1,565	1,870
Emergency room visits					
Respiratory	211	50	83	50	91
Restricted activity days[e]	10	10	10	10	10
Minor restricted activity days					
Total (adults)	ng	ng	ng	ng	ng
Effects in asthmatics					
Asthma attacks	271	199	572	199	337
Cough without phlegm (children)	ng	ng	ng	ng	ng
Cough with phlegm (children)	ng	ng	ng	ng	ng
Some respiratory symptoms (children)	ng	ng	ng	ng	ng
Respiratory symptoms	10	10	10	10	10
Chronic morbidity					
Chronic bronchitis	153	168	326	168	218
Chronic cough (children)	169	136	279	136	190

ng = Assumed negligible.
a. From Hernandez-Avila and others (1995).
b. Based on National Health Survey, ENSA II 1994, 18.6% public insurance, 31.9% IMSS, 33.3% private insurance, and 16.2% other.
c. Assumed three times respiratory hospital admissions
d. Assumed same as respiratory hospital admissions.
e. Assumed same as respiratory symptoms. Only 46% of the work-loss-day portion of restricted activity days are valued with a COI component (Krupnick 2000).

Human Capital Loss

The human capital approach is used for valuing the lost productivity associated with mortality. This approach assumes that the value of a person is equal to what he or she would have produced, that is, the discounted present value of a person's expected future earnings. The value of lost productivity may also include nonmarket productivity, for example, the value of household production. Other dimensions of illness and death, such as pain, suffering, and loss of leisure are excluded. The difference between the productivity loss and human capital is that the former accounts for the short-term production losses caused by morbidity, while the latter focuses on the production losses in the long term caused by increased mortal-

ity. Therefore discounting is applied only to human capital loss. Following Pearce and Ulph (1995) a social discount rate of 3 percent has been used.[16]

Willingness to Pay

As noted above, economists consider the appropriate value of avoided premature mortality to be what an individual would pay to reduce his risk of death. This should reflect the value of foregone consumption and leisure time and the loss of contact with loved ones. WTP can be estimated using the contingent valuation method (CVM) and hedonic pricing.

CVM estimates the WTP or willingness to accept (WTA) a change in the quantity and/or quality of a good by using survey techniques (Mitchell and Carson 1989 and Hoevenagel 1994). In the questionnaire a hypothetical change is described and the respondent is asked directly for his WTP or WTA this change. The main values derived through the CVM in this study are for health impacts such as asthma attacks and premature death.

Hedonic pricing estimates the WTP/WTA through (i) the difference in the value of the same property in different areas with different environmental risks (property value differential); or (ii) the wage differential people are willing to pay (or accept) for a decrease (or increase) in risk of death related to a job.

In this study we focus on the WTP estimated through CVM and wage differential studies. Because CVM is a costly and complex method, studies have been conducted in only a limited number of countries for a limited number of environmental goods and services. In the United States and Europe numerous CVM studies have been conducted on the WTP to reduce the risk of mortality and morbidity impacts. Wage differential studies are also numerous in these countries. WTP/WTA estimates, based on both CVM and wage differential studies, are not available for Mexico. Therefore, we estimate the WTP for risk reduction through "benefit transfer" of WTP studies performed outside Mexico.

Benefit transfer is an application of monetary values from a particular valuation study in one area to a policy decision setting in another geographic area (Navrud 1999). When transferring values it is important to know when data from other studies can be used and under what conditions. The value that people attach to avoided health risks depends on the type and magnitude of risk (low probability, high impact), the extent to which the risk is experienced voluntarily, on cultural settings, income, and the futurity of the risk. The most important factors for applying benefit transfer in this study are the level of real per capita income, represented by purchasing power parity (PPP) per capita income, and the income elasticity of WTP (ε).[17] For reasons explained above we assume a best estimate for the income elasticity of 1.0.[18]

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[16] A more extensive discussion on the discounting can be found in Cesar and others (2000).

[17] It seems plausible that risk preferences might also change with the status of development. However, we have not included this difference in risk aversion between countries in our benefit transfer due to lack of data.

[18] A more elaborate discussion on WTP and benefit transfer is presented in Cesar and others (2000).

The original values of WTP for the morbidity endpoints are based on report from the Centre for Social and Economic Research on the Global Environment (Pearce and others 1999), U.S. EPA (1999), and ExternE (1999).[19] Table 6.4 shows the values derived for Mexico using different income elasticities.

Table 6.4 WTP Estimates for Morbidity Impacts Obtained with CVM
(in US$, 2010 values in 1999 prices)

Health endpoint	Income elasticity		
	0	0.4	1
Hospital admission			
Respiratory	550	330	153
Cardiocerebrovascular	550	330	153
Congestive heart failure (elderly)	550	330	153
Emergency room visits			
Respiratory	284	170	79
Restricted activity days			
Total[a,b]	49	35	21
Minor restricted activity days[c]			
Total (adults)	49	35	21
Effects in asthmatics			
Asthma attacks	52	31	15
Cough without phlegm (children)[c]	49	35	21
Cough with phlegm (children)[c]	49	35	21
Some respiratory symptoms (children)[c]	49	35	21
Chronic morbidity			
Chronic bronchitis, new cases	422,991	253,899	118,074
Chronic cough, prevalence (children)	287	199	116

a. All RADs are valued using WTP. The work-loss days, a subset of RADs, are not valued separately to prevent double counting.

b. We value restricted activity days (RADs) as a cough episode and thus equal to a minor restricted activity day. This underestimates the WTP for RADs. However, Pearce and others (1999) found the value of a bed day, which might be seen as an overestimate of the RAD as not all RADs are bed days, to be only 30% higher than WTP to avoid cough.

c. Following ExternE (1999) we value most cases of effects in asthmatics, cases of respiratory symptoms, and minor restricted activity days (MRADs) as a cough case (episode). For MRADs this give the same value as used by U.S. EPA (1999).

In estimating the WTP for premature mortality it is important to realize that the number of life-years lost due to acute and chronic exposure to air pollution is limited. Because we intend to value only the reduction in the number of life-years lost, the "years of life lost" (YOLL) approach has been applied.[20] The YOLL approach is particularly recommended for deaths arising from exposure to air pollution. The value will depend on a number of factors, such as how long it takes for the exposure to result in an illness and eventually death. In this

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[19] See Cesar and others (2000) for a more detailed discussion

[20] An alternative for the YOLL approach is the "value of a statistical life" (VSL) approach. A comparison of the two approaches is provided in the Cesar and others (2000).

study, the YOLL approach is used both in cases where the hazard has a significant latency period before impact (mortality associated with chronic exposure), and cases where the impact takes place within a short period of time (mortality associated with acute exposure). In estimating the values of mortality arising from chronic exposure to particulate matter we assume that latency and mortality risks are spread out evenly over a period of 15 years and the life time reduction is 5 years on average (ExternE 1999). For mortality associated with acute exposure in the general population we assume no latency and the average life time reduction to be 0.75 years (ExternE 1999). The resulting "value of life-year" lost (VOLY) based on benefit transfer using the PPP approach is reproduced in table 6.5.

Table 6.5 Value of Life Year (VOLY)
(in US$, (2010 values in 1999 prices, 3 percent discount rate) [a]

	VOLY mortality-acute exposure		VOLY mortality-chronic exposure	
	Male	Female	Male	Female
Income elasticity = 0	184,750	179,776	140,611	138,308
Income elasticity = 0.4	131,961	128,409	100,434	98,789
Income elasticity = 1.0	79,660	77,515	60,628	59,635

a. Using a VSL of 4.28, 3.06, and 1.85 million US$ (2010 values in 1999 prices) after benefit transfer of the European estimate of VSL of 3.36 million US$ (1999 values in 1999 prices) with income elasticity 0, 0.4 and 1, respectively.
b. Differences in values for males and females arise from unequal distributions of survival probabilities and life expectancy.

Results

This section presents the main results of the economic valuation of the benefits of improving air quality in Mexico City. A distinction is made between economic health benefits for the air pollution reduction scenarios presented in section 4 and the benefits arising from the reduction in environmental contingencies. Both categories of effects are then aggregated and summarized.

Economic Health Benefits

As explained in earlier sections health-related benefits consist of effects resulting from reducing acute morbidity, mortality associated with acute exposure, chronic morbidity and mortality associated with chronic exposure. For the first three categories a straightforward procedure is followed by multiplying the physical health impacts (see section 5) by the monetary values for each unit of health impact (see previous section). The procedure to assess the damages from mortality associated with chronic exposure—combining information on life expectancy, age dependent mortality rates, and VOLYs—is more complicated.

Figure 6.1 presents the configuration of the health-related benefits of a reduction in air pollution. The productivity losses, the cost of illness, and the willing to pay are included in the estimated morbidity benefits. In the mortality benefit estimates either the human capital benefits or the WTP are included.[21]

Tables 6.6 to 6.9 present the results for WTP estimates derived by benefit-transfer with income elasticities of 0.4 and 1.0. The results show that the main health damages are caused by WTP for a reduction of health impacts. For PM_{10}, the economic value of preventing premature death dominates the overall outcome. A summary of the damages including and excluding WTP benefits are presented in table 6.10.

Figure 6.1 Health-Related Benefits of Reduction of Air Pollution

Loss of Productivity
+
Cost of Illness
+
Willingness to Pay
} Acute & chronic morbidity

Human Capital Loss
or
Willingness to Pay
} Acute & chronic mortality

} Health-related Benefits

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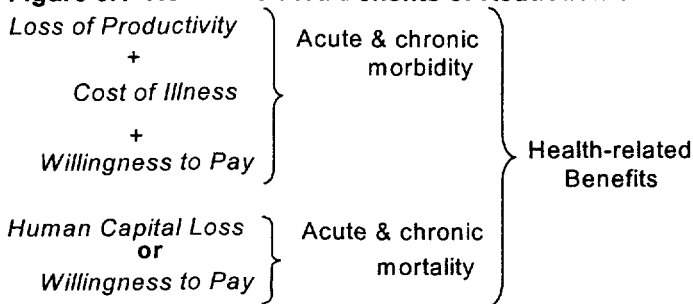
[21] In the literature used here, only studies that specifically differentiated between the costs categories (COI, productivity loss, and WTP) were considered.

Table 6.6 Health Benefits of Ozone Air Pollution Reduction in the ZMVM

(in million US$ per year [a] for income elasticity 0.4, 2010 values in 1999 prices)

Endpoints	Scenario			
	10%	20%	AQS1	AQS2
Morbidity impacts (Prod. Loss + COI + WTP)				
Hospital admission				
Respiratory	7.43	14.86	45.95	50.90
Cardiocerebrovascular	5.25	10.50	32.45	35.94
Emergency room visits				
Respiratory	6.30	12.60	38.95	43.14
Minor Restricted activity days				
Total (adults)	86.49	172.98	534.79	592.28
Effects in asthmatics				
Some respiratory symptoms (children)	0.02	0.04	0.11	0.12
Lower respiratory symptoms	0.01	0.02	0.06	0.06
Respiratory symptoms				
Upper respiratory symptoms	3.67	7.34	22.69	25.13
Lower respiratory symptoms	2.14	4.28	13.22	14.64
Wheeze	1.23	2.46	7.60	8.41
Morbidity impacts (Prod. Loss + COI)				
Hospital admission				
Respiratory	6.34	12.69	39.22	43.44
Cardiocerebrovascular	4.97	9.94	30.74	34.04
Emergency room visits				
Respiratory	2.65	5.29	16.37	18.13
Effects in asthmatics				
Some respiratory symptoms (children)	0.00	0.01	0.02	0.03
Lower respiratory symptoms	0.00	0.00	0.01	0.01
Respiratory symptoms				
Upper respiratory symptoms	1.19	2.37	7.33	8.12
Lower respiratory symptoms	0.69	1.38	4.27	4.73
Wheeze	0.19	0.39	1.20	1.33
Mortality impacts—WTP				
Mortality-acute exposure (total)	70.07	140.13	433.25	479.83
Mortality impacts—Human capital losses				
Mortality-acute exposure (total)	1.67	3.34	10.33	11.44
Total – Morbidity (Prod. Loss+ COI +WTP) and WTP for mortality	183	365	1129	1250
Total – Morbidity (Prod. Loss+ COI +WTP) and human capital losses mortality	114	228	706	782
Total – Morbidity (Prod. Loss + COI) and human capital losses mortality	18	35	109	121

a. Discount rate 3%.

Table 6.7 Health Benefits of Ozone Air Pollution Reduction in the ZMVM

(in million US$ per year [a] for income elasticity 1.0, 2010 values in 1999 prices)

Endpoints	10%	20%	AQS1	AQS2
Morbidity impacts (Prod. Loss + COI + WTP)				
Hospital admission				
Respiratory	6.85	13.70	42.35	46.91
Cardiocerebrovascular	5.10	10.20	31.53	34.93
Emergency room visits				
Respiratory	4.35	8.69	26.87	29.76
Minor Restricted activity days				
Total (adults)	55.21	104.42	322.83	357.54
Effects in asthmatics				
Some respiratory symptoms (children)	0.01	0.02	0.08	0.08
Lower respiratory symptoms	0.01	0.01	0.04	0.04
Respiratory symptoms				
Upper respiratory symptoms	2.68	5.37	16.60	18.39
Lower respiratory symptoms	1.56	3.13	9.67	10.71
Wheeze	0.82	1.64	5.06	5.60
Morbidity impacts (Prod. Loss + COI)				
Hospital admission				
Respiratory	6.34	12.69	39.22	43.44
Cardiocerebrovascular	4.97	9.94	30.74	34.04
Emergency room visits				
Respiratory	2.65	5.29	16.37	18.13
Effects in asthmatics				
Some respiratory symptoms (children)	0.00	0.01	0.02	0.03
Lower respiratory symptoms	0.00	0.00	0.01	0.01
Respiratory symptoms				
Upper respiratory symptoms	1.19	2.37	7.33	8.12
Lower respiratory symptoms	0.69	1.38	4.27	4.73
Wheeze	0.19	0.39	1.20	1.33
Mortality impacts—WTP				
Mortality-acute exposure (total)	42.30	84.59	261.53	289.65
Mortality impacts—Human capital losses				
Mortality-acute exposure (total)	1.67	3.34	10.33	11.44
Total – Morbidity (Prod. Loss+ COI +WTP) and WTP for mortality	116	232	717	794
Total – Morbidity (Prod. Loss+ COI +WTP) and human capital losses mortality	75	151	465	515
Total – Morbidity (Prod. Loss + COI) and human capital losses mortality	18	35	109	121

a. Discount rate 3%.

Table 6.8 Health Benefits of PM_{10} Air Pollution Reduction in the ZMVM
(in million US$ per year[a] for income elasticity 0.4, 2010 values in 1999 prices)

Endpoints	Scenarios 10%	20%	AQS1	AQS2
Morbidity impacts (Prod. Loss + COI + WTP)				
Hospital admission				
Respiratory	1.55	3.10	3.40	7.25
Cardiocerebrovascular	1.81	3.63	3.98	8.48
Congestive heart failure (elderly)	0.00	0.00	0.00	0.00
Emergency room visits				
Respiratory	3.49	6.97	7.65	16.32
Restricted activity days				
Total (adults)	161.10	322.20	353.62	754.09
Work-loss days (adults)	14.32	28.63	31.42	67.01
Total (children)	64.04	128.08	140.57	299.76
Work-loss days (working women due to RAD in children)	6.14	12.28	13.48	28.75
Minor restricted activity days				
Total (adults)	109.10	218.20	239.47	510.68
Effects in asthmatics				
Cough without phlegm (children)	0.07	0.14	0.15	0.32
Cough with phlegm (children)	0.01	0.01	0.01	0.02
Chronic morbidity				
Chronic bronchitis, new cases	778.48	1,556.96	1,708.75	3,643.94
Chronic cough, prevalence (children)	0.26	0.52	0.57	1.22
Morbidity impacts (Prod. Loss + COI)				
Hospital admission				
Respiratory	1.32	2.65	2.90	6.19
Cardiocerebrovascular	1.72	3.43	3.77	8.04
Congestive heart failure (elderly)	0.00	0.00	0.00	0.00
Emergency room visits				
Respiratory	1.46	2.93	3.22	6.86
Restricted activity days				
Total (adults)	18.94	37.89	41.58	88.67
Work-loss days (adults)	14.32	28.63	31.42	67.01
Total (children)	7.53	15.06	16.53	35.25
Work-loss days (working women due to RAD in children)	6.14	12.28	13.48	28.75
Minor restricted activity days				
Total (adults)	0.00	0.00	0.00	0.00
Effects in asthmatics				
Cough without phlegm (children)	0.01	0.03	0.03	0.07
Cough with phlegm (children)	0.00	0.00	0.00	0.01
Chronic morbidity				
Chronic bronchitis, new cases	0.81	1.61	1.77	3.77
Chronic cough, prevalence (children)	0.15	0.29	0.32	0.69
Mortality impacts—WTP				
Mortality (Acute exposure) – Infant	-	-	-	-
Mortality (Chronic exposure) – Total	1,408.53	2,817.07	3,091.71	6,593.13
Mortality impacts – Human capital losses				
Mortality (Acute exposure) – Infant	-	-	-	-
Mortality (Chronic exposure) – Total	43.28	86.55	94.99	202.57
Total – Morbidity (Prod. Loss+ COI +WTP) and WTP for mortality	2,549	5,098	5,595	11,931
Total – Morbidity (Prod. Loss+ COI +WTP) and human capital losses mortality	1,184	2,367	2,598	5,540
Total – Morbidity (Prod. Loss + COI) and human capital losses mortality	96	191	210	448

a. Discount rate 3%.

Table 6.9 Health Benefits of PM$_{10}$ Air pollution Reduction in the ZMVM
(in million US$ per year[a] for income elasticity 1.0, 2010 values in 1999 prices)

	Scenarios			
Endpoints	10%	20%	AQS1	AQS2
Morbidity impacts(Prod. Loss + COI + WTP)				
Hospital admission				
Respiratory	1.43	2.86	3.13	6.69
Cardiocerebrovascular	1.76	3.52	3.87	8.24
Congestive heart failure (elderly)	0.00	0.00	0.00	0.00
Emergency room visits				
Respiratory	2.40	4.81	5.28	11.26
Restricted activity days				
Total (adults)	104.76	209.52	229.94	490.36
Work-loss days (adults)	14.32	28.63	31.42	67.01
Total (children)	41.64	83.29	91.41	194.92
Work-loss days (working women due to RAD in children)	6.14	12.28	13.48	28.75
Minor restricted activity days				
Total (adults)	65.86	131.72	144.56	308.28
Effects in asthmatics				
Cough without phlegm (children)	0.05	0.10	0.10	0.22
Cough with phlegm (children)	0.00	0.01	0.01	0.02
Chronic morbidity				
Chronic bronchitis, new cases	362.46	724.92	795.59	1,696.61
Chronic cough, prevalence (children)	0.21	0.43	0.47	1.00
Morbidity impacts (Prod. Loss + COI)				
Hospital admission				
Respiratory	1.32	2.65	2.90	6.19
Cardiocerebrovascular	1.72	3.43	3.77	8.04
Congestive heart failure (elderly)	0.00	0.00	0.00	0.00
Emergency room visits				
Respiratory	1.46	2.93	3.22	6.86
Restricted activity days				
Total (adults)	18.94	37.89	41.58	88.67
Work-loss days (adults)	14.32	28.63	31.42	67.01
Total (children)	7.53	15.06	16.53	35.25
Work-loss days (working women due to RAD in children)	6.14	12.28	13.48	28.75
Minor restricted activity days				
Total (adults)	0.00	0.00	0.00	0.00
Effects in asthmatics				
Cough without phlegm (children)	0.01	0.03	0.03	0.07
Cough with phlegm (children)	0.00	0.00	0.00	0.01
Chronic morbidity				
Chronic bronchitis, new cases	0.81	1.61	1.77	3.77
Chronic cough, prevalence (children)	0.15	0.29	0.32	0.69
Mortality impacts—WTP				
Mortality (Acute exposure) - Infant	-	-	-	-
Mortality (Chronic exposure) – Total	850.28	1,700.55	1,866.35	3,980.02
Mortality impacts – Human capital losses				
Mortality (Acute exposure) – Infant	-	-	-	-
Mortality (Chronic exposure) – Total	43.28	86.55	94.99	202.57
Total - Morbidity (Prod. Loss+ COI +WTP) and WTP for mortality	1,451	2,903	3,186	6,793
Total - Morbidity (Prod. Loss+ COI +WTP) and human capital losses mortality	644	1,289	1,414	3,016
Total - Morbidity (Prod. Loss + COI) and human capital losses mortality	96	191	210	448

a. Discount rate 3%.

Table 6.10 Summary Health Benefits Due to Ozone and PM$_{10}$ Air Pollution Reduction
(in million US$ per year[a] 2010 values in 1999 prices, 3 percent discount rate)

	Scenario							
	10%		20%		AQS1		AQS2	
Income elasticity	1.0	0.4	1.0	0.4	1.0	0.4	1.0	0.4
Ozone benefits								
Total – Morbidity (Prod. Loss+ COI +WTP) and WTP for mortality	116	183	232	365	717	1129	794	1,250
Total – Morbidity (Prod. Loss+ COI +WTP) and human capital losses mortality	75	114	151	228	465	706	515	782
Total – Morbidity (Prod. Loss + COI) and human capital losses mortality	18	18	35	35	109	109	121	121
PM$_{10}$ benefits								
Total – Morbidity (Prod. Loss+ COI +WTP) and WTP for mortality	1,451	2,549	2,903	5,098	3,186	5,595	6,793	11,931
Total – Morbidity (Prod. Loss+ COI +WTP) and human capital losses mortality	644	1,184	1,289	2,367	1,414	2,598	3,016	5,540
Total – Morbidity (Prod. Loss + COI) and human capital losses mortality	96	96	191	191	210	210	448	448

Environmental Contingencies

The economic effects of ECs in the ZMVM industry sector have been explored through an analysis of value added losses in affected industries. Using 1994 data, value added has been estimated here to decrease 39 percent during one day of PM$_{10}$ contingency and 42 percent in an ozone contingency.[22] The costs of a one-day contingency for PM$_{10}$ are lower than for ozone as fewer industries are involved in PM$_{10}$ (the service sector is left out in PM$_{10}$, but included in ozone).

As mentioned in the earlier sections environmental contingencies (ECs) have two main cost components: production losses in industry and transportation. Productivity losses in the transport sector are much less straightforward to estimate, and the lack of data proved to be more severe in the transport sector. Given these constraints we focussed solely on production losses in industry. Knowing the value of production, the value added, and labor costs per day (percent participation in the total costs) allowed us to calculate the costs of production in a normal situation (without environmental contingency). From the normal situation scenario, the costs of ECs can be derived considering a decline of 33 percent of production per day (see table 6.11).[23]

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[22] This is larger than the average decrease of production of 33 percent for all industries combined, as explained in detail in Cesar and others (2000). The discrepancy stems from the fact that most workers go to work and get paid even on days when the production stops partially. The result is a higher cost of production per unit of product.

[23] For a more detailed analysis, see Cesar and others (2000).

Table 6.11 Value Added Losses to ZMVM Industry during PM$_{10}$ and Ozone Contingencies (value added per day in thousands US$ 1995 values, 1 US$=9.28 Mex. Peso)

Industry subsectors	Percent growth in PM$_{10}$ contingency (percent)	Percent growth in ozone contingency (percent)	VA loss in PM$_{10}$ contingency	VA loss in Ozone contingency
Nonmetallic minerals extraction	−42	−42	17.45	17.92
Food, beverages, and tobacco	−41	−41	259.01	392.08
Textiles and leather industry	−43	−43	68.61	99.89
Wood and wood products	−45	−45	7.86	5.10
Paper industry and printing	−46	−47	40.24	123.16
Chemical industries	−43	−43	215.15	247.84
Nonmetallic minerals industries (no oil)	−38	−39	282.05	173.06
Basic Metallic industry	−57	−58	70.89	45.09
Metallic products	−48	−47	236.40	276.49
Other manufacturing	−41	−43	16.20	0.99
Services	—	−39	—	2.36
Electricity generation	−22	−22	−7.26	−7.00
TOTAL	−39	−42	1,306.57	1,376.94

Source: d= datgen. Emissions Inventory 2000; i= INEGI . Instituto Nacional de Estadística, Geografía e Informática, Censo Industrial. Mexico (1997).

To calculate the total losses resulting from environmental alerts, the costs per day has been multiplied by the number of days that the contingency is expected to be in place for each of the scenarios described in section 4. Here, the number of days for the first phase contingencies are counted. The precontingencies do not have explicit economic costs, while the second phase is never attained. The costs per year for phase I contingencies are given in table 6.12 in millions of US$ per year.[24]

Table 6.12 Industry Losses in Four Scenarios for PM$_{10}$ and Ozone Phase I Contingencies (value added per year in millions of US$, 2010 values in 1999 prices)

	Base case	Scenario I (10%)	Scenario II (20%)	Scenario III (AQS1)	Scenario IV (AQS2)
Days with PM$_{10}$ contingency	1.0	0.0	0.0	0.0	0.0
Production losses due to PM$_{10}$ Phase I contingency	4.8	0.0	0.0	0.0	0.0
Days with ozone contingency	10.0	2.0	0.0	0.0	0.0
Production losses due to ozone Phase I contingency	45.4	9.1	0.0	0.0	0.0

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[24] An exchange rate of 4 Pesos to the US$ was taken for 1995. These numbers were converted to 1999 US$.

7. Conclusions and Recommendations

The health benefits included in this study are:[25] (i) reduced cost of illness, (ii) reduced productivity losses, (iii) willingness to pay (WTP) for reduced acute and chronic morbidity effects, measured using the contingent valuation method (CVM); and (iv) WTP for reduced mortality effects associated with acute and chronic exposure. The WTP concept in each case captures aspects of the value of avoiding death and illness (for example the pain and suffering avoided) above and beyond foregone earnings and COI. The largest single contributor to the benefit estimate is WTP for premature death due to chronic exposure to air pollution. Given the continuing debate over the use of WTP for valuing health benefits, particularly CVM, we estimate the health benefits both including and excluding this benefit category. The human capital and COI can then be interpreted as lower bounds to WTP for reduced mortality and for reduced morbidity, respectively.

Table 7.1 presents the overall benefit estimates from this study at different income elasticities used in the benefit transfer of WTP estimates from Europe and the United States to Mexico. The central estimate of the annual benefits of a 10-percent reduction in ozone and PM10 is $759 million (1999 US$). High and low estimates of the value a 10-percent reduction are $1,607 million and $154 million, respectively.

Because estimates of the health benefits of reducing each pollutant control for the levels of other pollutants, it is appropriate to add the benefits of ozone and PM reduction together for each scenario. This is done in table 7.2, which summarizes the benefits of each control scenario, assuming an income elasticity of one in benefits transfer. The 'high' estimate given in Table 7.2 uses benefits of reduced morbidity in terms of Productivity loss, cost of illness and willingness to pay and of reduced mortality in terms of willingness to pay. The 'central' estimate is the same as the 'high' estimate except that mortality is measured in human capital losses rather than WTP. The 'low' estimate deviates from the 'central' case in that it excludes WTP estimates for morbidity.

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[25] In the literature on economic valuation of morbidity effects used here, only studies that specifically differentiated between the costs categories (COI, productivity loss, and WTP) were considered.

Table 7.1 Summary of Total Benefits of a Reduction in Air Pollution in Four Scenarios for Ozone and PM$_{10}$

(in million US$ per year, 2010 value in 1999 prices, 3 percent discount rate)

	Scenario							
	10%		20%		AQS1		AQS2	
Income elasticity	1.0	0.4	1.0	0.4	1.0	0.4	1.0	0.4
Ozone								
Health benefit estimate 1 including: Morbidity (Prod. loss+ COI +WTP) and WTP for mortality	116	183	232	365	717	1129	794	1250
Health benefit estimate 2 including: Morbidity (Prod. loss+ COI +WTP) and human capital losses for mortality	75	114	151	228	465	706	515	782
Health benefit estimate 3 including: Morbidity (Prod. loss + COI) and human capital losses for mortality	18	18	35	35	109	109	121	121
Environmental contingencies benefits	36	36	45	45	45	45	45	45
PM$_{10}$								
Health estimate 1 including: Morbidity (Prod. loss+ COI +WTP) and WTP for mortality	1451	2549	2903	5098	3186	5595	6793	11931
Health benefit estimate 2 including: Morbidity (Prod. loss+ COI +WTP) and human capital losses for mortality	644	1184	1289	2367	1414	2598	3016	5540
Health benefit estimate 3 including: Morbidity (Prod. loss + COI) and human capital losses for mortality	96	96	191	191	210	210	448	448
Environmental contingencies	4	4	4	4	4	4	4	4

Prod. loss = Productivity losses; COI = cost of illness; WTP = willingness to Pay.

Table 7.2 Summary of Benefits From Each Scenario Using Income Elasticity of 1.0

(in million US$ per year, 2010 values in 1999 prices)

Estimates	10%	20%	AQS1	AQS2
High	1607	3184	3952	7636
Central	759	1489	1928	3580
Low	154	275	368	618

The estimates in tables 7.1 and 7.2 clearly show that the calculated benefits associated with air pollution reduction give an economic basis for spending to further reduce polluting emissions. Exactly how much is open to debate. Ideally, this study on economic benefits should be combined with estimates of emission abatement costs to determine an economically justifiable level of abatement. Hence, conducting a cost-benefit analysis is the logical next step. This would also necessitate further advances in atmospheric chemistry modeling for Mexico City, which is needed to compare costs from emissions reductions with benefits of lower concentrations of pollutants.

The current valuation study uses meta-analyses and benefit transfers. Additional epidemological and health-economic studies in Mexico City would allow estimates of health benefits solidly based on local data. Also, the uncertainties regarding all the estimates presented above are considerable. Further research that allows a reduction of these uncertainties is highly recommended.

It should also be noted that the monetary estimates of health benefits give a lower boundary to actual benefits. For instance, the human misery associated with a person suffering from chronic pollution-related morbidity may be much larger than monetary estimates indicate. This is especially the case if this person is the main wage earner of a poor family who could slide further into poverty due to a lack of safety net.

As other studies have found (U.S. EPA 1997, 1999), the health benefits from reducing ozone and PM_{10} are dominated by the benefits of reducing particulate matter. In the case of Mexico City, the benefits associated with reductions of PM_{10} are roughly an order of magnitude higher than those of ozone. The results must, however, be interpreted with caution. Pollution control strategies that reduce the precursors of ozone, especially NO_x, may also reduce particles.

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