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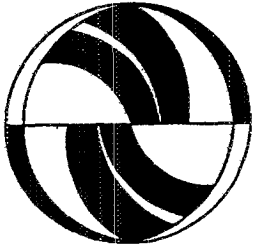
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Authors

McCubbin, Donald R.
Delucchi, Mark A.

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**The Social Cost of the Health Effects of
Motor-Vehicle Air Pollution**

Donald R. McCubbin
Mark A. Delucchi

Working Paper
UCTC No 321

**The University of California
Transportation Center**
University of California
Berkeley, CA 94720

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University of California
Transportation Center

108 Naval Architecture Building
Berkeley, California 94720
Tel. 510/643-7378
FAX 510/643-5456

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The Social Cost of the Health Effects of Motor-Vehicle Air Pollution

Donald R. McCubbin
Mark A. Delucchi

Institute of Transportation Studies
University of California
Davis, CA 95616

*Working Paper
August 1996*

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The University of California Transportation Center
University of California at Berkeley

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REPORTS IN THE SOCIAL-COST SERIES

There are 20 reports in this series. Each report has the publication number UCD-ITS-RR-96-3 (#), where the # in parentheses is the report number:

- Report 1:** The Annualized Social Cost of Motor-Vehicle Use in the U.S., 1990-1991: Summary of Theory, Methods, Data, and Results (M. Delucchi)
- Report 2:** Some Conceptual and Methodological Issues in the Analysis of the Social Cost of Motor-Vehicle Use (M. Delucchi)
- Report 3:** Review of Some of the Literature on the Social Cost of Motor-Vehicle Use (J. Murphy and M. Delucchi)
- Report 4:** Personal Nonmonetary Costs of Motor-Vehicle Use (M. Delucchi)
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- Report 11:** The Cost of the Health Effects of Air Pollution from Motor Vehicles (D. McCubbin and M. Delucchi)
- Report 12:** The Cost of Crop Losses Caused by Ozone Air Pollution from Motor Vehicles (M. Delucchi, J. Murphy, J. Kim, and D. McCubbin)
- Report 13:** The Cost of Reduced Visibility Due to Particulate Air Pollution from Motor Vehicles (M. Delucchi, J. Murphy, D. McCubbin, and J. Kim)
- Report 14:** The External Cost of Noise from Motor Vehicles (M. Delucchi and S. Hsu) (with separate 250-page data Appendix)
- Report 15:** U.S. Military Expenditures to Protect the Use of Persian-Gulf Oil for Motor Vehicles (M. Delucchi and J. Murphy)
- Report 16:** The Contribution of Motor Vehicles and Other Sources to Ambient Air Pollution (M. Delucchi and D. McCubbin)
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Send requests by fax (916 752-6572), e-mail (matillotson@ucdavis.edu), or mail (Institute of Transportation Studies, University of California, Davis, California 95616 attn: publications). For general information, call (916) 752-6548.

LIST OF ACRONYMS AND ABBREVIATIONS AND OTHER NAMES

The following are used throughout all 20 reports of the series, although not necessarily in this particular report

AER = *Annual Energy Review* (Energy Information Administration)
AHS = *American Housing Survey* (Bureau of the Census and others)
ARB = Air Resources Board
BLS = Bureau of Labor Statistics (U. S. Department of Labor)
BEA = Bureau of Economic Analysis (U. S. Department of Commerce)
BTS = Bureau of Transportation Statistics (U. S. Department of Transportation)
CARB = California Air Resources Board
CMB = chemical mass-balance [model]
CO = carbon monoxide
dB = decibel
DOE = Department of Energy
DOT = Department of Transportation
EIA = Energy Information Administration (U. S. Department of Energy)
EPA = United States Environmental Protection Agency
EMFAC = California's emission-factor model
FHWA = Federal Highway Administration (U. S. Department of Transportation)
FTA = Federal Transit Administration (U. S. Department of Transportation)
GNP = Gross National Product
GSA = General Services Administration
HC = hydrocarbon
HDDT = heavy-duty diesel truck
HDDV = heavy-duty diesel vehicle
HDGT = heavy-duty gasoline truck
HDGV = heavy-duty gasoline vehicle
HDT = heavy-duty truck
HDV = heavy-duty vehicle
HU = housing unit
IEA = International Energy Agency
IMPC = Institutional and Municipal Parking Congress
LDDT = light-duty diesel truck
LDDV = light-duty diesel vehicle
LDGT = light-duty gasoline truck
LDGV = light-duty gasoline vehicle
LDT = light-duty truck
LDV = light-duty vehicle
MC = marginal cost
MOBILE5 = EPA's mobile-source emission-factor model.
MSC = marginal social cost
MV = motor vehicle
NIPA = National Income Product Accounts
NO_x = nitrogen oxides

NPTS = Nationwide Personal Transportation Survey
OECD = Organization for Economic Cooperation and Development
O₃ = ozone
OTA = Office of Technology Assessment (U. S. Congress; now defunct)
PART5 = EPA's mobile-source particulate emission-factor model
PCE = Personal Consumption Expenditures (in the National Income Product Accounts)
PM = particulate matter
PM₁₀ = particulate matter of 10 micrometers or less aerodynamic diameter
PM_{2.5} = particulate matter of 2.5 micrometers or less aerodynamic diameter
PMT = person-miles of travel
RECS = Residential Energy Consumption Survey
SIC = standard industrial classification
SO_x = sulfur oxides
TIA = *Transportation in America*
TSP = total suspended particulate matter
TIUS = *Truck Inventory and Use Survey* (U. S. Bureau of the Census)
USDOE = U. S. Department of Energy
USDOL = U. S. Department of Labor
USDOT = U. S. Department of Transportation
VMT = vehicle-miles of travel
VOC = volatile organic compound
WTP = willingness-to-pay

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11. THE SOCIAL COST OF THE HEALTH EFFECTS OF MOTOR-VEHICLE AIR POLLUTION IN THE UNITED STATES

11.1 BACKGROUND

Motor vehicles and their related emission sources, such as petroleum refineries, emit many different kinds of air pollutants, which affect human health in a variety of ways. These health effects create a large economic cost to society. In this report, we estimate the social cost of many of the health effects of motor-vehicle air pollution.

The relationship between changes in emissions related to motor-vehicle use and changes in health welfare (measured in dollars) can be modeled in three steps: 1) relate changes in emissions to changes in air quality; 2) relate changes in air quality to changes in physical health effects; and 3) relate changes in physical health effects to changes in economic welfare. We have made a detailed model of this sort to estimate the cost of the health effects of motor-vehicle air pollution¹.

We estimate the human-health cost of motor-vehicle air pollution in the entire U.S., in urban areas of the U.S., in rural areas of the U.S., and in 11 major metropolitan statistical areas (MSAs): Boston, Denver, Houston, Los Angeles, Minneapolis, New York, Philadelphia, Phoenix, St. Louis, Spokane, and Washington D. C. We consider six types of motor vehicles: light-duty gasoline and diesel vehicles, light-duty gasoline and diesel trucks, and heavy-duty gasoline and diesel trucks. We estimate the number and type of health effects, and the monetized value of these effects, including total dollar costs, dollar costs per vehicle-mile of travel, and dollar costs per kg of pollutant emitted. Finally, we include an analysis of the three main sources of the costs: direct emissions from motor vehicles, emissions of road-dust particulate matter, and "upstream" emissions from gasoline stations, refineries, vehicle manufacturing, and so on.

11.1.1 Emissions and air quality

We estimate the *status quo* air quality in 1990, and then estimate the effect of reducing motor vehicle emissions by 10% and 100%, and reducing all anthropogenic emissions by 100%. We represent the *status quo* with measurements of actual ambient air quality at air-quality monitoring sites (EPA, 1993d). To estimate air quality without 10% or 100% of motor-vehicle related emissions, we use a simple model of emissions, dispersion, and atmospheric chemistry, developed in Report #16 in the social-cost series listed at the beginning of this report and summarized in Chapter 11.5.

¹Alternatively, we could have tried to use hedonic or household production studies to estimate the value of air pollution reduction but, as we discuss in Chapter 11.4, these sorts of studies have significant shortcomings. For a recent review of the literature on the air pollution damages of transportation, see Krupnick (1995). See also the recent methodological reviews by Krupnick (1993) and Cifuentes and Lave (1993). For a recent estimate of air-pollution damages in Los Angeles, see Small and Kazımi (1995).

We estimate the effects of a specific, “marginal” change in pollution: the difference between actual pollution and, what pollution would have been had there been either a 10% or a 100% reduction in motor vehicle-related emissions. We emphasize two points here. First, it is useful to consider a marginal change because the formation of ambient pollution from emissions of pollutants is a nonlinear process, and to some extent the effect of ambient pollution on people's health is nonlinear. Second, when we say “motor vehicle-related,” we include emissions from motor vehicles' tailpipes, road dust, the production of motor fuel at refineries, emissions from the servicing of motor vehicles, emissions from road construction, and so on.

11.1.2 Air pollution and health effects

We estimate the cost of four “criteria” pollutants (carbon monoxide, nitrogen dioxide, ozone, and particulate matter) and six “toxic” air pollutants (formaldehyde, acetaldehyde, benzene, 1,3-butadiene, gasoline particulates, and diesel particulates). The estimation methods for the criteria pollutants are different than the methods for the toxic air pollutants. Table 11.1-1 summarizes the pollutants and health effects covered in our analysis.

Criteria pollutants. We reviewed hundreds of clinical, animal, and epidemiological studies of the health effects of various pollutants, and constructed exposure-response functions for each criteria pollutant (ozone, carbon monoxide, etc.) and each of a variety of health effects (for example, asthma, or headaches). These functions relate the change in health effects to the change in exposure. We have developed mortality-risk estimates for those pollutants, such as fine particles, which according to some studies are associated with mortality. For most pollutants and health effects, we have established upper and lower-bound estimates of the effects of exposure (Table 11.1-1).

In general, a exposure-response function has the following form:

$$\Delta E = f(\Delta P, O) = f(PI, PP, O)$$

where:

ΔE = the change in the effect of interest (in this case, human health)

ΔP = the change in ambient air pollution

O = other variables (such as population in the county, or the incidence rate of a health problem or cause of death; see Chapter 11.3)

PI = the initial pollution level (estimated from data on actual ambient air quality in counties in the U.S.; see Chapter 11.2)

PP = the pollution level after the change in pollution -- in our analysis, the level had there been no motor-vehicle-related emissions (detailed in Report #16 in the social-cost series listed at the beginning of this report.).

Our estimate of the health effects of particulate matter, which is by far the most damaging pollutant, accounts for several important factors: 1) the likelihood

that smaller particles are more damaging than larger particles, 2) that geological material is less damaging than combustion material, and 3) that particulate-matter emission inventories are seriously mis-estimated.

Toxic air pollutants. Whereas the cost of the criteria pollutants is estimated on the basis of human epidemiological studies and ambient air-quality data, the cost of toxic air pollutants is estimated on the basis of unit-risk numbers and exposure to pollution in micro-environments. Unit-risk functions relate the probability of getting a particular type of cancer (e.g., leukemia) to the amount of exposure to a particular toxic air pollutant (e.g., benzene). Details are given in Chapter 11.6.

Note that our estimate of the cost of gasoline and diesel particulates as toxic air pollutants does not double-count any of the costs of particulate matter as a criteria pollutant, because the end points are different (lung cancer in the case of gasoline and diesel particulates, and acute respiratory deaths, acute cardiovascular deaths, and chronic cardiopulmonary deaths in the case of particulate/criteria pollutant).

11.1.3 Valuation of health effects

In the last step, we estimate the economic value of the estimated health effects. Our estimates of the dollar value of health effects are derived from studies of the value of lost work days, of restricted activity, of tolerating certain symptoms, and so on. When we estimate the value of life, which is the most important valuation parameter in the analysis, we distinguish future deaths from current deaths, and deaths that would have occurred soon anyway even if there were no pollution.

The total health cost then is equal to the change in the effect of interest (ΔE above; e.g., number of deaths due to motor-vehicle particulate air pollution) multiplied by the dollar value per effect (e.g., the value of life).

11.1.4 Summary of results

The most important result we found is the large cost of particulate matter pollution, and the potentially large contribution of motor vehicles to ambient particulate levels (Chapter 11.7). Generally, combustion emissions of particulate matter and precursors to particulate matter cause the greatest health costs, by far.

Particulates appear to cause a number of respiratory ailments, including chronic illness and mortality. Chronic illness and especially mortality are the most costly health effects because of the value that society places on avoiding them. Very small particulates formed from combustion processes and from atmospheric gases appear more harmful than larger, more soil-based particulates. Motor-vehicle use results in both types of emissions. Motor vehicles contribute the smaller, more dangerous particulates directly from tailpipe emissions and indirectly from the large amounts of "precursor" gases that they give off such as nitrogen dioxide. Motor vehicles also emit large amounts of fairly coarse soil-based particulates from road dust -- dust kicked up into the atmosphere from moving vehicles. Determining the relative harmfulness of these two classes of particulates (combustion vs. dust particles) has important policy implications. We model a variety of assumptions that try to capture the range of plausible values for these factors.

Carbon monoxide, nitrogen dioxide, ozone, sulfur dioxide and toxics appear to have much smaller effects than particulates. Aside from their contribution to particulate formation, emissions of nitrogen dioxide, sulfur dioxide and volatile organic carbon are relatively unimportant. Interestingly, in the cost ranking, ozone, which is formed from the interaction of nitrogen oxides and volatile organic carbon, is nearly in last place, well behind particulates, and less damaging even than carbon monoxide and nitrogen oxides. In part this might be due to our inability to capture all of the effects of ozone. Nevertheless, it appears that air pollution policy has focused too heavily on ozone control and not enough on particulate control.

11.1.5 Sources of uncertainty

There is considerable uncertainty at every stage of the modeling process. We review some of the uncertainties below, and discuss them in more detail in the relevant chapters of the report.

When estimating the *status quo* exposure (Chapters 11.2 and 11.6), we used a county average, which may poorly reflect the exposure levels of many people. Furthermore, we did not have ambient data in many areas, particularly rural areas, and had to rely on data in the same region of the country. When estimating ambient pollution levels after reducing motor vehicle emissions by 10% and 100% (Chapter 11.5 and Report 16), we used a simple model of pollution formation that took into account the average location of emission sources to main population concentrations, and for particulates that took into account the relative potency of particulates. We only looked at the emission sources in a particular air quality control region (AQCR), when determining how to allocate the cause of a particular county's ambient pollution level to different emission sources. We did not -- and could not without a much more sophisticated model -- account for the movement of pollution across AQCRs, as may occur with emissions from power plants with very high emission stacks.

In determining the health effects of ambient exposure (Chapters 11.3 and 11.6), there is uncertainty regarding the types and magnitudes of effects caused and the pollutants that are responsible. We could not estimate certain health effects that appear to occur and we did not treat certain populations as more or less susceptible to pollution. Particulates appear to have a particularly severe effect on the elderly population, however the difference has not been well quantified. Moreover, where we can quantify the health effects, we cannot rule out the possibility that other pollutants are involved. But perhaps most importantly, we could not reliably determine the effects of particulates based on their composition and size, and were forced to use a variety of assumptions that present a range of plausible values

Finally, there is considerable uncertainty in the valuation of health effects. We have used lower and upper bounds; in most cases, the upper bound is at least four times the lower bound. This difference by itself of course increases the uncertainty in the final damage estimates by a factor of four or more.

11.1.6 Report Organization

In Chapter 11.2, we present a number of the issues involved in estimating the *status quo* exposure for “criteria” pollutants that we consider -- carbon monoxide, nitrogen dioxide, ozone, and particulates. (We treat toxic pollutants differently; see the discussion in Chapter 11.6.)

Chapter 11.3, examines the health effects of carbon monoxide, nitrogen dioxide, ozone and particulates. Chapter 11.6 discusses how we quantify the link between exposure to toxics and the formation of cancer.

In Chapter 11.4, we discuss how we value health effects. Chapter 11.5 summarizes how we model the contribution of motor vehicles to ambient air quality. In Chapter 11.7, we discuss our results.

TABLE 11.1-1. POLLUTANTS AND QUANTIFIED HEALTH EFFECTS IN THIS STUDY

Criteria Pollutants	Health Effect^a	Population in Lower Bound	Population in Upper Bound
Carbon Monoxide (CO)	Headache	All Ages	All Ages
	Hospitalization	Adults (> 65)	Adults (> 65)
	Mortality	Adults (> 65)	Adults (> 65)
Nitrogen Dioxide (NO ₂)	Excess Phlegm Eye Irritation Sore Throat	All Ages	All Ages
Ozone (O ₃)	Asthma Attacks	All Asthmatics	All Asthmatics
	Eye Irritation	Adults (> 17)	All Ages
	Lower Respiratory Illness	Non-Asthmatics (> 17)	Non-Asthmatics of All Ages
	Upper Respiratory Illness	Adults (> 17)	All Ages
	Any other symptom (ARD ₂)	none	All Ages
Particulates (PM _{2.5} , PM ₁₀ , TSP)	Asthma Attacks	All Asthmatics	All Asthmatics
	Respiratory-Related Restricted Activity Days (RRAD)	Non-Asthmatics	Non-Asthmatics
	Air Obstructive Disease (AOD)	All Ages	All Ages
	Mortality	All Ages	All Ages
Toxic Pollutants	Type of Cancer Caused		
Acetaldehyde	Oral Cavity and Pharynx	All Ages	All Ages
Benzene	Leukemia	All Ages	All Ages
1,3-Butadiene	All Sites	All Ages	All Ages
Diesel Particulates	Lung	All Ages	All Ages
Formaldehyde	Oral Cavity and Pharynx	All Ages	All Ages
Gasoline Particulates	Lung	All Ages	All Ages

^aThe health effects are discussed and quantified in the sections below on the individual pollutants. Note that these are the quantified effects, there are some effects, such as the effect of CO on heart disease in persons under the age of 65, that we could not estimate. Some of the terms are defined in Table 11 3-1

11.2 POLLUTION EXPOSURE

The production, distribution and combustion of gasoline and diesel produces hundreds of compounds -- many of which are transformed into yet other compounds by atmospheric chemistry involving solar radiation, water vapor, and pollutants from other processes. Most of these pollutants are harmful. A comprehensive analysis to consider all of the pollutants and all of their effects is well beyond the scope of our effort here. Instead, we include in our analysis those pollutants for which we have air-quality data and dose-response functions: carbon monoxide (CO), nitrogen dioxide (NO₂), ozone (O₃), particulates (of two kinds), acetaldehyde, benzene, 1,3-butadiene, and formaldehyde. We do not include sulfur dioxide (SO₂) because there is no dose-response function (we discuss this more below), and we do not include lead because it has been phased out of gasoline.

Table 11.2-1 gives the National Ambient Air Quality Standards (NAAQS) for five so-called "criteria" pollutants.

11.2.1 Exposure To Criteria Pollutants

We estimate people's exposure to criteria pollutants using the county average of air quality monitors. This is broadly consistent with, but not necessarily identical to, the way in which exposure is estimated in the epidemiological studies that we use to estimate dose-response functions. For example, Schwartz and Zeger (1990: 63) used observations from an air-quality monitor two and a half miles from the study population. Dockery et al. (1992: 364) used a single monitor in St. Louis and in Knoxville to estimate people's exposure in each city. Ostro and Rothschild (1990: 240) used airport visibility data to estimate exposure to fine particulates in a metropolitan statistical area (a group of urban counties). Because we can not replicate *exactly* how each researcher chose to estimate exposure, we use the county average of all air quality monitors.¹

Of course, air-quality monitors obviously are not perfect estimates of people's exposure (Lipfert et al., 1988: 3; Schwab, 1990; Lippmann and Liroy, 1985: 245). Monitors may be miles from people and even if the monitors are in the center of a city, the monitor can not capture the variability in exposure levels that inevitably occurs. However, the proper objective is not necessarily to estimate exposure as accurately as possible, but rather to replicate as closely as possible the exposure estimates in the epidemiological studies that we use to estimate dose-response functions. (That is, the epidemiological studies that we use relate observed responses to a specific model of exposure, and so to use these studies to predict responses, we must use the same model of exposure as in the original studies.)

We note that variability in pollution levels is less of a problem for ozone and fine particulates, because they disperse relatively uniformly. Portney and Mullahy (1986) found that there was little difference between using ozone readings from the

¹We use data, supplied by the EPA (1993d), for all air monitoring stations in the US, and weight all observations equally. Future work should perhaps weight more heavily those monitors most closely associated with population exposure.

monitor nearest to their study population versus the average readings from all monitors within a five, ten and twenty mile radius.

11.2.2 Estimating Exposure and Treatment of Missing Data

We have no air pollution observations for the majority of the 3,000-plus counties in the U.S. and we have an incomplete set of observations for the rest (EPA, 1993d). To fill incomplete observation sets, we interpolate, and for counties with no observations we use as a proxy a county in one of the ten Federal regions in the U.S..

We aggregate our air pollution observations in the four ways dictated by the epidemiological studies that we use. We estimate the daily one-hour maximum pollution level, the daily average, the annual average and the cumulative hours of exposure above various cutoffs. We consider each in turn.

11.2.2.1 Estimating The One-Hour Daily Maximum Pollution Level

The one-hour daily maximum is the hour in the day with the highest average pollution level. Unfortunately, it is very common for one or more observations to be missing, because the data are not being collected, the air monitor is being serviced or for other reasons. The missing observations might have been the highest. Faced with missing observations, we must either pick the highest of the remaining hours or ignore the observations from that day. We use two algorithms to determine the one-hour maximum -- one used by the California Air Resources Board (CARB) and the other by the EPA:

i) CARB (1992: 42) uses the highest of the available observations in a day if there are at least six observations for each 1/3 day (hours 0 through 7, 8 through 15, and 16 through 23), and they are missing no more than two consecutive hourly observations.

ii) The EPA uses the highest of the available observations in a day if at least 75% of the observations (at least 9 hours) are available between 9 AM and 9 PM (see "Protection of Environment." Title 40 *Code of Federal Regulations*. Part 50, 1995 ed.).

We use CARB's method to estimate the one-hour maximum for CO and NO₂, because we estimate the health effects of CO and NO₂ using the work of Schwartz and Zeger (1990), who obtained their data from CARB.²

Our estimates of the health effects of ozone are derived from the work of Holguin et al. (1985), Kinney and Ozkaynak (1991), Ostro et al. (1993), and Whittemore and Korn (1980). We do not know which algorithm these studies used to estimate the one-hour maximum for ozone. We assume they used the EPA method, and so use the EPA method ourselves.³

²We also use Morris et al (1995), but it is unclear which algorithm they used

³ With both methods there is a possibility that the true one-hour maximum could be missing. If the authors of the health effects studies used these methods, however, then we should also our results will be biased

Filling in Missing Days of Data for the One-Hour Daily Maximum in Counties with Some Data

Some days either do not have enough observations to meet the CARB or EPA criterion, or else have no observations at all. To estimate these missing days observations, we considered two interpolation methods.

i) In their nationwide study of the health effects of ozone, Krupnick and Kopp (1988) placed the daily one-hour maximums recorded over the ozone season in "bins" of ten ppb width (there were 36 bins because the peak ozone value was 360 ppb) and scaled up the number of observations in each bin so that the total number of observations in the bins equaled the number of days in the ozone season. For example, if one bin had 50% of the observations then they assigned 50% of the missing observations to that bin, replacing the missing observations with the value of the midpoint of the bin.

ii) In their study of the effect of air pollution on mortality, Kinney and Ozkaynak (1991) used the average of eight air pollution monitors to estimate exposure in Los Angeles County. When one monitor did not have an observation for a day, they used the other monitors in the county to estimate the missing observation.

$$x_{ijk} = \bar{x}_{jk} \cdot \frac{\bar{x}_{1k}}{\bar{x}_{.k}}$$

where.

x_{ijk} = one-hour maximum reading missing from monitor i, on day j, in year k

\bar{x}_{jk} = mean of other monitors' reading for day j

\bar{x}_{1k} = annual mean of one-hour maximum readings for station 1 and year k

$\bar{x}_{.k}$ = annuals mean across stations for all stations with a one-hour maximum

There are drawbacks to Kinney and Ozkaynak's procedure. If a county does not have an observation from any monitor for a given day, we can not estimate that day's observation. We considered using monitors in nearby counties; however, such a procedure is unrealistic to consider for all 3110 counties in the U.S.

We chose to use Krupnick and Kopp's method, but with a few changes to make the estimated pollution levels account for seasonal differences.

For CO and NO₂, we use a two-stage binning method. In the first stage we consider the observations from a county in two-month blocks which allows us to control for differences in pollution levels that may occur over the course of the year. If there are six or more observations in a block we use the binning method on the available data in the block. If there are fewer than six observations in a block we go to the second stage which collects the remaining missing observations and assumes

that they have the same distribution as the observations found over the course of the year.

For ozone, we use observations from the ozone season separately from the non-ozone season. (Table 11.2-2 gives the ozone season for each state in the U.S.) In the first stage of binning for the ozone season we use two-month blocks; for ozone seasons with an odd number of months (e.g., Idaho's April through October ozone season), we use either July or August separately. After the first stage of binning we estimate any remaining missing observations using all observations from the ozone season.

Estimating the Pollution Level in Counties Without Pollution Data

Counties with no air quality monitors probably have relatively low pollution levels, because the air pollution monitors presumably are placed where there are pollution problems. We use the county with the lowest annual average pollution level in each of ten regions in the U.S. as a proxy for the counties with no observations. For CO and NO₂, we use the 10 regions established by the EPA for administrative purposes and choose the two counties (one urban and one rural) with the lowest annual average pollution level as the proxy for the other counties in the region.⁴ For ozone, we group counties by the nine ozone seasons and use the same procedure as we do for CO and NO₂.

11.2.2.2 Estimating The Daily Average Pollution Level

We use the daily average PM₁₀ level to estimate the effects of particulates on mortality and chronic illness, the daily average TSP level to estimate the effects of particulates on asthma, and the daily average PM_{2.5} level to estimate the effects of particulates on respiratory-related restricted activity days (RRADs) and mortality. Here, too, there are plenty of missing observations, in large part because the norm is to collect particulate measurements every six days. We describe first how we fill-in missing observations for counties with data, and then address counties with no particulate data at all.

Estimating Particulate Exposure In Counties With Some Particulate Data

For counties with particulate data, we determine the daily average PM₁₀ and TSP levels in two stages; the process for filling in TSP observations is exactly analogous to the PM₁₀ case.

i) We consider each county separately, and divide the year into six two-month blocks. If there are any observations for PM₁₀ in a two-month block, then we fill in the rest of the observations using Krupnick and Kopp's "binning" method, discussed above. We then use the "binning" method to fill in any remaining missing observations in the year.

⁴There are few rural CO monitors so we chose to use the lowest of both urban and rural counties as the proxy for rural areas. The states in each EPA region are given in Table 11.2-2

ii) If a county does not have PM₁₀ observations but does have TSP observations then we used the national ratio between PM₁₀ and TSP to adjust the TSP data to determine the PM₁₀.⁵ For example, if the PM₁₀/TSP ratio is 0.6 and the TSP level is 100 µg/m³ then the PM₁₀ level equals 60 µg/m³. After converting the TSP observations, we use Krupnick and Kopp's "binning" method to fill in the rest of the missing observations for the two-month block. We then use the "binning" method to fill in any remaining missing observations in the year.

Estimating Particulate Exposure In Counties Without Particulate Data

To estimate the pollution level in counties with no data, we use a two-stage procedure; the process for filling in TSP observations is exactly analogous to the PM₁₀ case.

i) If a county has no PM₁₀ data and has TSP data, then we adjusted the TSP observations by multiplying them by the national PM₁₀/ TSP ratio.

ii) For counties with no data, we use a proxy to estimate the pollution level. We use the county in the same EPA region with the lowest annual average PM₁₀ level, on the assumption that air-pollution monitors are placed where there are pollution problems.

There are not enough PM_{2.5} observations for us to use them directly to estimate RRADs. Instead, to estimate the daily average PM_{2.5} concentration, we simply multiply the daily PM₁₀ level by the national PM_{2.5}/PM₁₀ ratio.

11.2.2.3 Estimating The Annual Average County Pollution Level

We use the annual average CO level to estimate the effects of toxics, and we use the annual average PM_{2.5} level to estimate the effects of particulates on long-term mortality.

For CO, the county must have observations for 10% of the days over the course of a given year.⁶ For counties with insufficient observations, we use as a

⁵It is a fairly common procedure to assume that there is a constant relationship between PM₁₀ and TSP, and Simpson (1992) found that the ratio of PM₁₀ to TSP observations from Brisbane, Australia stayed constant over the course the year

To calculate the PM₁₀/TSP ratio, we take the annual average ambient level of PM₁₀ and TSP for each county in the US that has measurements of both pollutants, and then take an average of these ratios

⁶Previous work set a relatively high criterion (50% or 75%) for the inclusion of any given monitor. Because we are interested in estimating the effects of pollution in all counties in the U.S., we use a lower standard of completeness, under the assumption that a few observations is a better estimate of the county pollution level than the use of a proxy, such as the county with the lowest pollution level in that region

Johnson et al (1992b: 4) assumed that a monitor must have 75% of its maximum possible observations in order for it to be included in their data set, when estimating the carboxyhemoglobin levels caused by ambient CO. The EPA (1992a: 2-2), in its estimates of national ambient levels of criteria pollutants, used monitoring data if 50% of the observations were available. Portney and Mullahy (1990), when estimating the effect of ozone and TSP on chronic disease, used a monitor if it had at least half the maximum number of observations

proxy the county with the lowest annual average pollution level in the region, taking into account whether the county is urban or rural. For PM_{2.5}, we use the annual average of the daily PM_{2.5} readings, based on the daily PM₁₀ level adjusted by the national PM_{2.5}/PM₁₀ ratio.

11.2.2.4 *Estimating the Cumulative Hours of Exposure*

Following Abbey et al. (1991b; 1993; 1995), we use the cumulative hours of PM₁₀ exposure to estimate the effect of particulates on chronic disease. Each day above a given threshold (e.g., 100 µg/m³) contributes 24 hours to the year's hours of exposure.

11.2.3 Emissions from natural sources, and natural background pollution levels

To estimate the effects of motor-vehicle pollution, we estimate ambient pollution with (PI) and without (PP) motor-vehicle related emissions, taking into account all sources of pollution including natural sources. The ambient pollution with motor vehicles (PI) is the actual ambient air pollution measured at air-quality monitoring networks throughout the U. S. As explained in Report #16 in the social-cost series listed at the beginning of this report, we estimate the ambient air pollution without motor vehicles (PP) as follows:

$$\text{Assume } \frac{PP}{PI} = \frac{PP^*}{PI^*}$$

$$PP = PI \times \frac{PP^*}{PI^*}$$

where:

PP = the estimated actual pollution level, without motor-vehicle-related emissions

PI = the actual ambient pollution level (data from air-quality monitors; discussed in Reports 11, 12, and 13)

PP* = the modeled level of pollution, without motor-vehicle related emissions

PI* = the modeled level of total ambient pollution

Ambient pollution PP* and PI* is modeled as a function of emissions, dispersion, and atmospheric chemistry. For the purposes of this discussion, we may assume that PP*/PI* is proportional to the ratio of total emissions excluding motor-vehicle emissions to total emissions, where total emissions (T) consists of known emissions from natural sources (N), motor vehicles (M), and other sources (O):

assume

$$\frac{PP^*}{PI^*} = \frac{N+O}{N+M+O}$$

$$PP = PI \times \frac{N+O}{N+M+O}$$

(1)

Equation (1) is the simplest form for our model. However, the difficulty with equation (1) is that the data for N, emissions from natural sources, often are missing or incomplete (EPA, 1994a, 1995a, 1995b).

There are three ways to deal with missing or incomplete data for N: 1) estimate the missing data; 2) use instead an estimate of the natural background pollution concentration (PB) due to natural sources; or 3) combine the known N with estimate of the natural background pollution that remains on account of the unknown N. The first method is self-explanatory. With the second method, we in essence assume that the ratio of total anthropogenic pollution without motor vehicles to total anthropogenic pollution is equal to the ratio of total anthropogenic emissions without motor-vehicle emissions to total anthropogenic emissions:

$$\frac{PP - PB}{PI - PB} = \frac{O}{M+O}$$

$$PP = PB + (PI - PB) \times \frac{O}{M+O}$$

(2)

where:

PB = natural background pollution

PP-PB = total anthropogenic pollution without motor vehicles

PI-PB = total anthropogenic pollution

That the background concentration due to natural sources (PB) is proportional to emissions (N), as shown in equation (2), can be derived from equation (1):

$$PP = PI \times \frac{N + O}{N + M + O}$$

$$PB = PI \times \frac{N}{N + M + O}$$

$$PP = PB + \frac{PI \times O}{N + M + O}$$

$$PB \times N + PB \times M + PB \times O = PI \times N$$

$$N = \frac{PB \times M + PB \times O}{PI - PB}$$

$$PP = PB + \frac{PI \times O}{\frac{PB \times M + PB \times O}{PI - PB} + M + O}$$

$$PP = PB + \frac{PI \times O \times (PI - PB)}{PB \times M + PB \times O + M \times PI - M \times PB + O \times PI - O \times PB}$$

$$PP = PB + \frac{PI \times O \times (PI - PB)}{M \times PI + O \times PI}$$

$$PP = PB + (PI - PB) \times \frac{O}{M + O}$$

Equation (2) is the general form of our model: if PB is taken to be zero, and O includes N, then equation (2) becomes equation (1). Thus, where we have good, complete data on N, we assume that PB = 0, and include N in O. Alternatively, if we have no data on N, we use an estimate of PB. We also can use PB and N both, so long as the total contribution of natural sources is in effect exactly and completely apportioned between N and PB. (That is, we can estimate a fraction of the natural emissions, and then estimate PB such that it accounts for the remaining, unestimated natural emissions.)

The question, then, is whether we work solely with emissions N, solely with the background concentration PB, or with some combination of both. In the case of PM, we use emissions (N). In the case of CO, NO_x and ozone, we use a combination of N and PB.

Whenever we use PB, we can proceed in two ways: a) assume that natural sources contribute a constant fraction of ambient pollution, or b) assume that natural sources contribute a constant concentration to ambient pollution levels. Obviously, neither method is perfect: the true natural background pollution concentration is neither fixed nor a fixed percentage of total ambient pollution. Both the natural pollution level, and the fraction of total emissions that are from natural

sources, vary over time and space. The advantage of the fixed-percentage approach is that it can represent the variation in the natural pollution level that occurs, say, between seasons, and that it always results in a background pollution level that is less than observed ambient pollution levels. The disadvantage is that it ignores the fact that anthropogenic pollution levels are typically more variable than natural pollution levels⁷ On this last score the fixed-concentration assumption is preferred. We will use it here, whenever we use an estimate of PB.

11.2.3.1 Carbon Monoxide -- Natural Level

Natural sources of CO include plant emissions, oxidation of natural hydrocarbons, forest wildfires, oceans and methane oxidation. Our inventory only includes wildfire emissions, which are less than 5% of global emissions (Wuebbles and Edmonds, 1988: 50). Hence, we use an estimate of the natural background concentration (PB) and the available emission inventory, N.

Seiler and Junge (1970: Table 1) took CO measurements in many areas across the globe and found that CO in "clean air conditions" ranges between 0.1 and 0.2 ppm. Similarly, Graedel and Crutzen (1989: 62) reported that "clean atmospheres" in the Northern Hemisphere have 0.1 to 0.2 ppm CO. The EPA (1991: 4-15) reported that the average concentration of CO is 0.9 ppm and natural sources contribute 40% of emissions, which very roughly suggests a background rate of 0.3 to 0.4 ppm. In our lower bound case, we assume a background rate of 0.4 ppm, and in our upper bound case we assume 0.1 ppm.

11.2.3.1 Nitrogen dioxide-- Natural Level

Natural sources of NO_x include lightning, microbial processes in the soil, forest wildfires, stratospheric oxidation of N₂O and oceans. The most important sources appear to be microbial processes in the soil and lightning, with both sources contributing about the same amount of emissions (EPA, 1993c: 4-12). Emissions from oceans are negligible. Our emissions inventory includes emissions from soils and wildfires, but not production from lightning or stratospheric oxidation of N₂O. It appears that lightning and stratospheric oxidation of N₂O account for about half of all natural emissions of NO_x (EPA, 1993c: 4-12). To account for the missing emissions, we considered scaling up the emissions inventory by a factor of two, but it is unlikely that NO_x emissions from the soil are well correlated with NO_x generated from lightning.

Instead, we chose to assume a natural background level of NO₂, although we recognize this has difficulties for several reasons. First, most of the available data on natural emissions or natural background levels pertain to NO_x, which comprises

⁷The variation of anthropogenic sources occurs between seasons and geographic areas (types of fuels used, industrialization and population levels are important factors causing differences between geographic areas). Of course, the natural pollution level also varies between geographic areas and over time due to temperature, proximity to oceans, plant life, wildfires, volcanoes, and other factors. However, as a rule variation in anthropogenic pollution is the main determinant of variation in air quality, natural sources (such as volcanoes and wildfires) rarely dominate anthropogenic sources.

NO as well as NO₂. We assume that NO_x is equal parts NO and NO₂; i.e., that the ratio of NO and NO₂ in NO_x is 1:1. Second, the natural background concentration of NO_x varies greatly from over space and time, owing to variability in natural emissions of NO_x, and to the relatively short atmospheric residence time of NO_x (a few days, according to Logan, 1983: 10786). Third, it can be difficult to measure precisely the trace quantities of NO_x present in the atmosphere.

Due to these difficulties, the estimates of the background concentration of NO_x that we found vary by at least two orders of magnitude.⁸

- Logan et al. (1981: 7210) reviewed a number of estimates, including ground level estimates of NO in clean air in Wyoming and Colorado, and reported that the "few measurements available for NO and NO₂ in clean air," range from roughly 0.01 to 0.1 ppb.
- The EPA (1993c: Table 7-2) reported that "for isolated rural sites and coastal inflow areas in the United States, the NO_x concentrations generally range from a few tenths to one ppb." It added that remote maritime areas had NO_x concentrations ranging from 0.02 to 0.04 ppb, and measurements in remote tropical areas ranged between 0.02 and 0.08 ppb.

We attach more weight to terrestrial measurements, particularly when there is little chance that they will be contaminated by the anthropogenic pollution (it is common for urban pollution to be transported long distances). The remote tropical measurements may be the best indicator of natural pollution levels in the U.S., although we recognize that few areas in the U.S. resemble tropical areas.

In sum, it appears that the natural background level is between 0.01 and 0.20 ppb. We already have emissions from wildfires and microbial processes in the soil, which account for roughly half of natural emissions. So we use half of the estimated natural background rate: 0.005 ppb in our upper bound case and 0.1 ppb in our lower bound scenario.

11.2.3.3 Ozone -- Natural Level

As mentioned above, our treatment of ozone uses data on emissions of ozone precursors from natural sources (N), and data on background ozone levels (PB). The ozone formation equation discussed, in Report #16 in the social-cost series listed at the beginning of this report, takes into account the emissions of NO_x and VOC from soil and plants, but does not account for ozone that is injected from the stratosphere. To account for this, we have added a constant ozone contribution from the stratosphere.

A number of studies have estimated the ozone level in "clean" areas, which ostensibly are reasonably well isolated from anthropogenic sources⁹. The available

⁸NO_x observations over the middle of the ocean often are an order of magnitude lower than observations in clean air on land

⁹It is not clear, however, if these areas are unaffected by long-range transport

estimates of the background ozone level agree reasonably well. We review these studies below, with an eye towards identifying the contribution of stratospheric ozone (which is the only "natural" emissions source not accounted for in our emissions inventory).

- Hoffer et al. (1982) measured ozone levels in the Mojave Desert between 1976-1979 and found the daily one-hour maximum readings stayed near 0.04 ppm throughout the year. However, these readings may have been influenced by the transport of pollutants from Los Angeles (Pryor and Hoffer, 1991).
- Evans et al. (1983) reviewed observations collected in 1979 from six remote sites in the United States in the National Air Pollution Background Network. The mean daily one-hour maximum ranged from 0.033 ppm in the winter to 0.049 in the summer.
- The EPA (1986: 5-105) found that the arithmetic mean of one-hour ozone observations in rural areas varied between roughly 0.02 and 0.04 ppm. However they could not be certain that these readings were free from urban pollution
- Altshuller (1987: 1416) reviewed a number of methods to estimate the background level and concluded that in the spring and summer the background level ranges between 0.01 ppm and 0.02 ppm.
- Lefohn (1990: 200-201) collected observations from clean sites in American Samoa; Barrow, Alaska; Mauna Loa, Hawaii; the South Pole; and various remote sites in the United States and Canada. The average one-hour maximum for most of these sites ranged between roughly 0.03 and 0.045 ppm; the observations at the South Pole were closer to 0.015 ppm, probably representing the contribution of ozone from the stratosphere.

Our best estimate is that the *total* natural background level of ozone is around 0.025 to 0.035 ppm. We need to break this total into the fraction due to emissions from local biogenic emissions of VOC and NO_x (which we account for separately), and the fraction due to stratospheric ozone. Based on the above evidence, a reasonable range for the constant stratospheric contribution is 0.01 to 0.015 ppm.

11.2.3.4 Particulate Matter -- Natural Level

Windblown soil, forest fires, salt spray and secondary particulates formed from ammonia, nitrogen oxide, sulfur oxide and reactive organic gases contribute to the natural ambient particulate concentration. We have emissions estimates for windblown soil, forest fires and natural emissions of nitrogen oxides and reactive organic gases, the latter two of which we use to estimate secondary particulate formation. However, the inventory is not complete. Among other things, we do not have estimates of salt spray, natural sources of sulfur oxides, and lightning-

produced and stratospherically injected nitrogen oxides. Furthermore, the inventory for windblown soil does not include "dust devils", which are a potentially large source of emissions in the western states.

It is difficult to correct for these deficiencies. To account for missing natural emissions, we increase the estimates of wind erosion by 20% in our lower bound case and 10% in the upper bound. Furthermore, we account (to some extent) for missing emissions by restricting the maximum county PM₁₀ reading to 200 $\mu\text{g}/\text{m}^3$ and the maximum TSP reading to 325 $\mu\text{g}/\text{m}^3$, because readings above this are most likely due to dust storms caused by wind erosion of agricultural and non-agricultural land.

TABLE 11.2-1. CALIFORNIA AND NATIONAL AMBIENT AIR QUALITY STANDARDS (NAAQS)

Pollutant	Averaging Time	California Standard	National Primary Standard
Carbon Monoxide (CO)	8 hour	9 ppm	9 ppm
	1 hour	20 ppm	35 ppm
Nitrogen Dioxide (NO ₂)	Annual Average	n.a	0.053 ppm
	1 hour	0.25 ppm	n.a
Ozone (O ₃)	1 hour	0.09 ppm	0.12 ppm
Particulate Matter (PM ₁₀)	Annual Geometric Mean	30 µg/m ³	n.a
	Annual Arithmetic Mean	n.a	50 µg/m ³
	24 hour	50 µg/m ³	150 µg/m ³
Sulfur Dioxide (SO ₂)	Annual Arithmetic Mean	n.a	0.03 ppm
	24 hour	0.04 ppm	0.14 ppm
	1 hour	0.25 ppm	n.a

Source California Air Resources Board (1991c)
n.a =not applicable

TABLE 11.2-2. OZONE MONITORING SEASON BY STATE

EPA Region	State	Starting Month ^a	Ending Month ^a
4	Alabama	March	November
10	Alaska	April	October
9	Arizona	January	December
6	Arkansas	March	November
9	California	January	December
8	Colorado	March	September
1	Connecticut	April	October
3	Delaware	April	October
3	District of Columbia	April	October
4	Florida	January	December
4	Georgia	March	November
9	Hawaii	January	December
10	Idaho	April	October
5	Illinois	April	October
5	Indiana	April	September
7	Iowa	April	October
7	Kansas	April	October
4	Kentucky	April	October
6	Louisiana	January	December
1	Maine	April	October
3	Maryland	April	October
1	Massachusetts	April	October
5	Michigan	April	October
5	Minnesota	April	October
4	Mississippi	March	November
7	Missouri	April	October
8	Montana	June	September
7	Nebraska	April	October
9	Nevada	January	December
1	New Hampshire	April	October
2	New Jersey	April	October
6	New Mexico	January	December
2	New York	April	October
4	North Carolina	April	October
8	North Dakota	May	September
5	Ohio	April	October
6	Oklahoma	March	November
10	Oregon	April	October
3	Pennsylvania	April	October

(continued next page)

EPA Region	State	Starting Month ^a	Ending Month ^a
1	Rhode Island	April	October
4	South Carolina	April	October
8	South Dakota	June	September
4	Tennessee	April	October
6	Texas (AQCR 4, 5, 7, 10, 11) ^b	January	December
6	Texas (AQCR 1, 2, 3, 6, 8, 9, 12)	March	October
8	Utah	May	September
1	Vermont	April	October
3	Virginia	April	October
10	Washington	April	October
3	West Virginia	April	October
5	Wisconsin	April	October
8	Wyoming	April	October

Ozone season data is from "Protection of Environment " Title 40 *Code of Federal Regulations* Part 58, 1995 ed

AQCR = Air Quality Control Region (see "Protection of Environment" Title 40 *Code of Federal Regulations* Part 81, 1995 ed)

^aThe Code of Federal Regulations did not state the day that the ozone season starts or ends We assume that the entire month is included in the ozone season

^bTexas has two ozone seasons

11.3 HEALTH EFFECTS CAUSED BY CRITERIA POLLUTANTS

We estimate the health effects caused by criteria pollutants using dose-response functions derived from epidemiological studies that link ambient air pollution levels people face in their everyday life and the adverse health effects that people also experience.¹ A dose-response function is simply an equation that estimates the change in the incidence of adverse health effects caused by air pollution. In this section, we discuss why we chose particular dose response functions, what these dose-response functions look like, and how they are derived.

There are a variety of health effects probably associated with motor vehicle air pollution. These effects are often broken down between acute and chronic effects. Acute effects include eye irritation, headaches, respiratory illness, hospitalization and relatively quick death, and chronic effects include asthma, chronic bronchitis, emphysema and cancer. We reviewed many studies that examine the link between air pollution and adverse health, but unfortunately found that most of them cannot be used to develop dose-response functions. This is especially true of studies of chronic morbidity. To be included in our analysis, a study must meet three criteria:

- i). It must have a good statistical design, for example, we prefer that it estimate the effect of several pollutants simultaneously.
- ii). It must produce coefficients that we can use to predict the effect of changing the level of pollution (many studies fail on this score).
- iii). The health effect measured in the study must be economically evaluable. Thus, we cannot use studies that measure, say, lung functioning in terms of forced expiratory volume in one second (defined in Table 11.3-1), because we cannot estimate an economic value of this effect.

When quantifying adverse health effects, we chose not to use any of the numerous clinical or laboratory studies that have examined the link between air pollution and adverse health effects. Laboratory studies typically expose human subjects to low levels of a pollutant for short periods of time and estimate technical measurements of lung function such as forced expiratory volume in one second (FEV₁) (Table 11.3-1).² We chose not to use laboratory studies for a number of reasons. In order to use clinical studies to estimate health effects, one must replicate the conditions in the study, which requires that one model people's activities and personal exposure to pollution during the course of a day. This requires strong assumptions about people's activities or very large amounts of data. The measures of lung functioning that clinicians generally use are difficult to evaluate

¹When we use the term epidemiological study, we refer to studies that look at the effects of air pollution on people in their daily lives, as opposed to looking at the effects of air pollution in, say, a laboratory

²Laboratory studies have also looked at the effects of air pollution (sometimes very high levels) on animals. Obviously extrapolating from effects on animals to humans is difficult, and we chose not to do so given the choice of using epidemiological studies using humans. When quantifying the effects of toxics, we could not rely on epidemiological or laboratory studies so we used the results from animal studies, we discuss this further in Chapter 11.6

economically. What, for example, is the dollar cost of reduced FEV₁?³ Laboratory studies do not account for the actual mix of pollutants that people face, and they allow for only short exposure periods (Spektor et al., 1991). Finally, clinical studies may be less sensitive than epidemiological studies in determining the effect of low levels of pollution (Ostro et al., 1991: 700; Spektor et al., 1988b). For these reasons, we chose not to use clinical studies and to rely instead on epidemiological studies.⁴

11.3.1 Some issues to consider when using epidemiological studies to quantify the health effects of air pollution

A number of issues arise when using epidemiological studies to quantify the health effects of air pollution. The literature on the health effects of air pollution is large and disjointed -- studies use different measures of pollution, different measures of health effects and different statistical procedures, and the results vary widely. The uncertainty makes our estimates only rough approximations of the "true" cost. In recognition of this uncertainty, we estimate plausible upper and lower bounds, rather than point estimates.

11.3.1.1 Health effects thresholds

The threshold is the point where air pollution ceases to have an economic cost; below the threshold we assume there is no cost. The evidence is limited regarding thresholds, but the more researchers look the more harm -- and less evidence of threshold -- they find. Oak Ridge National Laboratory and Resources for the Future (1992: 2-13) reviewed the evidence regarding thresholds and concluded that "as has already been experienced with respect to regulating cancer risks, we may find that there are no 'safe' exposure levels to ozone and other air pollutants." Dockery et al. (1993) found that in six U.S. cities, fine particulates were associated linearly with mortality, with no apparent "no effects" threshold. Pope et al. (1995a) observe that acute health effects have been associated with particulate levels well below the current National Ambient Air Quality Standards. (This is important, because particulates appear to be by far the most damaging pollutant.)

We assume there is no threshold for NO₂, ozone and particulates and estimate effects down to the natural (or background) pollution level (see Section 11.2.3). Similarly, when estimating cancer incidence, we assume that there is no threshold. However, we derive our dose-response function for CO from the work of

³One perhaps can link clinical measures to understandable symptoms that are more easily valued. For example, Ostro et al. (1989) analyzed the results from ozone chamber studies and found that a 10% reduction in FEV₁ is linked to a 15% increase in the probability of a mild, moderate or severe lower respiratory symptom (which in turn can be valued economically) and a 6% increase in the probability of a moderate or severe symptom (which also can be valued economically). In our view, however, this tenuous linkage, stretches the already difficult task of valuation to the breaking point.

⁴Not everyone shuns clinical studies. Krupnick and Kopp (1988) reworked the results from a clinical study by McDonnell et al. (1983) to estimate the health effects of ozone, and Hall et al. (1992) used clinical studies to estimate the health effects of air pollution in Los Angeles. Criticism of the exposure model (REHEX) that they used is discussed in Harrison and Nichol's (1990) work.

Schwartz and Zeger (1990), who found a threshold for effects at 7 ppm. We assume that threshold here. We further discuss threshold levels below in the sections on each pollutant.

11.3.1.2 *Interactions among pollutants*

Pollutants may interact in ways that magnify or negate their individual effects. A mixture of pollutants – which is what people are exposed to – may be more or less harmful than is indicated by the simple sum of the effects of the pollutants measured in isolation.

The evidence for interactions is mixed. Krumm and Graves (1982) analyzed the causes of emergency cardiac and respiratory admissions to Cook County Hospital in Chicago and found a strong effect for SO₂ with little effect for coefficient of haze (COH) but significant interactions between SO₂ and COH. However, most studies have found little effect for SO₂ on hospital admissions and no interaction between SO₂ and particulates (Table 11.3-26). On the other hand, Krzyzanowski et al. (1992) found that in the presence of higher PM₁₀ levels, ozone has a more detrimental effect on lung functioning. And researchers studying rats and guinea pigs have found interactions between various pollutants (Last et al., 1983; Warren and Last, 1987; Last, 1991).

In any event, we do not have dose-response functions that explicitly model interactions with separate coefficients that indicate the strength of any interaction. If interactions do occur, then we bias our results by excluding them, but we can not say the direction of the bias.

11.3.1.3 *Proxy Pollutants*

The pollutants that we do consider may be proxies for the truly pernicious (but overlooked) pollutants in the atmosphere. If the proxy is not perfectly correlated with the true pollutant, then we generally underestimate the effects of air pollution, and we may misallocate the estimated effects if the pollution sources (e.g., power plants versus cars) emit different ratios of the proxy (measured) and true (unmeasured) pollutant.⁵

⁵Measurement error in the covariates can bias the estimated pollution coefficient downward or upward. The direction of the bias depends on the amount of measurement error and the correlation between the pollution variable and the other covariates. Generally, we feel that the exposure measurement error will dominate as found by Schwartz (1994c 653, 1994d 372, 1994e 597)

Schwartz (1994a 653, citing Judge et al., 1980) notes "Error in exposure measurements can produce bias in either direction in the estimated regression coefficient in a multivariate model. The total bias involves a sum of the term proportional to the ratio of the measurement error variance to the total variance in exposure, which always biases downward, and terms involving the correlation between exposure and covariates. These can have different signs, and therefore can bias in either direction."

A similar point was noted by Klepper et al. (1993 203) ". as long as measurement error in the pollution variables is classical, it will only cause the estimate of the pollution exposure coefficient to be biased asymptotically toward zero. On the other hand, measurement error in the other variables can have far more pernicious effects. It could cause the pollution variables to appear to have an effect on health when in fact they have no true effect or it could even cause them to appear to have an effect in the wrong direction."

11.3.1.4 *Collinearity Among Pollutants*

There is often a high degree of collinearity between pollutants, which makes it difficult to determine which pollutant is the culprit (Lipfert and Wyzga, 1995: 11). In studies of Los Angeles, Kinney and Ozkaynak (1991) and Shumway et al. (1988) reported that three pollutants predict mortality equally well. In studies of Steubenville, Ohio and Philadelphia, Moolgavkar et al. (1995) linked O₃, SO₂ and TSP to mortality and concluded that there is not a single or dominant culprit such as particulates. Abbey et al. (1995) estimated associations between a variety of health effects and the pollutants TSP, PM₁₀, PM_{2.5}, SO₄, and ozone, but could not separate the effects of the different pollutants in part because of high correlations between them. Kinney et al. (1995) found PM₁₀ correlated with ozone and CO in a study of the association between PM₁₀ and mortality in Los Angeles.

We note, however, that researchers have found a link between particulates and mortality in places with very low SO₂ levels (Pope et al., 1992; Fairley, 1990), but have not found a link between SO₂ and mortality in places with very low particulate levels. This suggests at least that particulates in general and not SO₂ specifically are associated with mortality.

11.3.1.5 *Not All Effects Considered*

We do not include the personal cost of avoiding pollution because we know little about how much people try to avoid pollution or mitigate its effects. Evans et al. (1988) interviewed residents of Los Angeles and found that even though people are anxious about pollution, they do little to avoid air pollution. Other researchers, however, believe that avoidance behavior is important. In any case, we do not know the size of the cost and so, reluctantly, ignore it. This biases our estimates of the cost of air pollution downward by an unknown amount.

11.3.1.6 *Unrepresentative Health Effects Studies*

Some of our dose-response functions are derived from epidemiological studies of relatively small populations. Nevertheless, we apply these functions to every county population group in the U.S.. To the extent that the pollution exposure and response of the measured population is not the same as the exposure and response of the [rest of the] populations in the U.S., our results might be inaccurate.

Moreover, some people are (exquisitely) sensitive to air pollution, and suffer effects of a different kind or to a different degree than does the rest of the population. Asthmatics, children, the elderly, individuals with pre-existing respiratory infections (who suffer more from ozone pollution [see Ostro et al., 1993]), and individuals with heart disease (who are more susceptible to angina induced by carbon monoxide) are examples of sensitive populations. Because the effects of air pollution on these small but sensitive populations might not show up in studies of

large populations composed of mainly not-sensitive people, it is preferable in principle to identify these sensitive groups and estimate effects for them separately.

In practice however, it is very difficult to identify and determine the effect of air pollution on all possible groups. To keep our analysis manageable, we consider separately (to at least some extent) three reasonably well-studied groups: asthmatics, children and the elderly.

Asthmatics and Air Pollution

A number of studies have focused on the effects of air pollution on asthmatics particularly in regard to the incidence of asthma attacks. We use these studies (e.g., Whittemore and Korn, 1980) to estimate the increased incidence of asthma attacks. It is possible that asthmatics are more susceptible to other types of effects caused by air pollution, such as the effect of NO₂ on coughing which we discuss below, however we have no way at the moment to quantify some of the possible differences between asthmatics and the general public.

Children and Air Pollution

Although there is no doubt that air pollution affects children's health, the type of the effect is uncertain. Children reportedly do not suffer from symptoms such as coughing as much as adults do. Avol et al. (1985) found air pollution has roughly the same effect on technical measurements of lung functioning in children and adults, but children seem to suffer fewer symptoms from a given pollution level. Portney and Mullahy (1986) and Krupnick et al. (1990: Table 4) reported that children did not develop the ozone-related respiratory symptoms suffered by adults, and they found that particulates caused significantly fewer respiratory symptoms in children than in adults.

On the other hand Avol et al. (1985) reported that children might experience more lung damage from pollutants than adults because they are less likely to notice (i.e., have a higher tolerance to irritation). Furthermore because children are more likely than adults to be outside (where pollutant levels are higher) and exercising, they probably receive a larger dose of pollution (Hall et al., 1989). We further discuss the evidence for the effects on children below in the sections on each pollutant.

The Elderly and Air Pollution

People over 65 have 16% more restricted activity days due to acute respiratory symptoms than do those aged 45-64 (Table 11.3-2), suggesting that the elderly may also suffer greater morbidity due to air pollution.⁶ In fact, many of the victims of air pollution do appear to be elderly, but until recently the epidemiological literature has not focused on elderly people. Schwartz and Dockery (1992a) reported a 10% increase in the mortality rate for people over the age of 65 and just 3% in people younger, due to a 100 µg/m³ increase in ambient total suspended particulates.

⁶ Morris et al (1995) reported that carbon monoxide causes an increase in hospital admissions due to congestive heart failure. Other recent studies examining the effect of air pollution on the elderly include Schwartz (1994a, 1994b) and Saldiva et al (1995)

Morris et al. (1995) reported that individuals over the age of 65 are more likely to enter the hospital for congestive heart failure because of CO.

We probably underestimate the effects of air pollution on the elderly because they usually are generally not well represented in epidemiological studies. Ostro (1989) confined their study of air pollution in Los Angeles to individuals under the age of 65, and Schwartz and Zeger (1990) examined the effects of air pollution on students nurses with a mean age of nineteen. The results of these studies probably should not be applied to the elderly. However for lack of more information we must treat the elderly similarly to other adults in most cases (an exception is our estimate of congestive heart failure caused by CO), and as a result we may underestimate the true effect of air pollution.

11.3.2 Carbon Monoxide

Carbon monoxide is dangerous because it binds with hemoglobin in the blood to form carboxyhemoglobin (COHb), thereby reducing the oxygen carrying capacity of the blood and limiting the release of oxygen from circulating hemoglobin (Allred et al., 1991: 90). CO adversely affects individuals with heart trouble (about seven million people in the U.S have heart disease) [EPA, 1991: 2-17]), and is linked to headaches (Schwartz and Zeger, 1990) (Tables 11.3-3 and 11.3-4). CO most strongly affects fetuses, infants, pregnant women, elderly people, people with anemia, and people with a history of cardiac, respiratory, or vascular disease (EPA 1991: 1-21).

11.3.2.1 Morbidity

CO affects the heart through the increase in the level of COHb in the blood. Unfortunately, we are unable to directly estimate the effect of COHb. There are two steps that we would have to perform: 1) use ambient CO data to estimate COHb blood levels, and 2) quantify the link between COHb and an "economically valuable" effect (i.e., an effect that has been valued by, say, estimated medical costs, a contingent valuation survey, etc.). Data and models are available for the first step, but, unfortunately, not for the second.

Johnson et al. (1992b) used a CO exposure model to track the CO exposure of individuals in Denver over the course of a day, and estimate the number of person-hours in which a Denver adult with ischemic heart trouble experienced various COHb levels.⁷ We considered extrapolating these results so that Denver would have been the model for the rest of the urban areas of the country. We would have estimated person-hours of exposure in each city by scaling the Denver results by the ratio of the annual average CO exposure in each city to that in Denver, with an adjustment for differences in altitude.

At this point, we would have estimated person-hours of exposure. However, we could not have proceeded to estimate the effect of the exposure on people's health, because we are not aware of a dose-response function that links person-

⁷This model was adapted for use as the mobile-source hazardous-air-pollutant-exposure model, HAPEM-MS, we describe in detail how we use HAPEM-MS to estimate toxics exposure in Chapter 11 6 of this report

hours of COHb exposure to an economically valuable effect. The EPA (1991: 1-20) reported that COHb levels of 3% to 6% increase angina symptoms, but they did not specify the relationship between hours of elevated COHb, the level of COHb and the likelihood and strength of the effect on angina. Other studies (e.g., Allred et al., 1989) have linked COHb levels to the onset of angina in individuals that are exercising, but we do not know how to value this effect. However, we concluded that there is not enough information available to develop a useful dose-response function.

Instead, we skipped the intermediate step of estimating COHb and focused on epidemiological studies that estimate the linkage between CO and adverse health effects. We begin with a review of studies of CO levels and hospital admissions.

A number of studies (Table 11.3-4) have examined the possible link between CO and hospital admissions, especially for cardiovascular problems. Kurt et al. (1978) found more cardiorespiratory admissions in an emergency room in Denver on days of high CO levels compared to days of low CO levels. The levels of O_x, NO_x and sulfur compounds (a subset of PM₁₀) were low and uncorrelated with CO and thus unlikely to confound the results. Ponka (1991) and Sunyer et al. (1991) also found a correlation between CO and hospital admissions, but the authors did not control for other pollutants, and calculated only the correlation between pollutants and admissions. Morris et al. (1995) and Schwartz and Morris (1995) both found CO significantly linked to congestive heart failure (ICD-9 code 428); the Schwartz and Morris study is particularly important because it controlled for PM₁₀, an important possible confounder. Not all researchers have found an effect of CO: in a study of residents of Steubenville, Ohio, Samet et al. (1981) did not find CO linked to respiratory-related emergency room admissions. Nevertheless, the weight of the evidence in these studies suggests that CO does have an effect on hospital admissions for congestive heart failure. We use the results of Morris et al. to quantify the link.

We reviewed four epidemiological studies (aside from hospital admission studies) that tested the link between CO and morbidity (Table 11.3-3). Robertson and Lebowitz (1984) and Lebowitz et al. (1987) found CO correlated with cough and rhinitis but they did not report the coefficients that we need to develop dose response functions. In a diary study of student nurses in Los Angeles, Schwartz and Zeger (1990) found a significant association between CO and headaches. Such a link is not surprising given that CO is known to affect humans' neurological functioning and blood vessels in the eyes (EPA, 1991: 10-177 and Table 1-2). In another diary study of 24 Denver asthmatics, Perry et al. (1983) did not find CO linked to asthma attacks, although they did find a significant response for (particulate) nitrates. The lack of an observed effect might have been due in part to the small sample size and short data collection period (three months).

Morbidity and Carbon Monoxide: Conclusion

Carbon monoxide causes heart problems, headaches, and perhaps other illnesses. We are able to estimate the effects of CO on hospitalization for congestive heart failure in persons over 65 and on headaches for the general population, on the basis of the work by Morris et al. (1995) and Schwartz and Zeger (1990). We do not

estimate any effects of CO on heart problems in individuals below the age of 65 and thus undoubtedly underestimate the effects of CO.

The results of Morris et al. (1995: Figure 2) suggest that CO has effects down to very low levels; Schwartz and Zeger (1990) did not report a threshold for the effect of CO. On the basis of this, we assume that there is no threshold.

There is little evidence concerning whether children, or other age groups, are more or less susceptible to an increase in headaches caused by CO. When estimating headaches caused by CO, we treat children identically to adults in both the lower and upper bound scenarios.

Estimating Headaches Caused by Carbon Monoxide

Schwartz and Zeger (1990) used logistic regression to analyze the health symptoms reported in the diaries of 110 student nurses living in Los Angeles from 1961 to 1964. The highest one-hour daily measurements of CO, NO₂, SO₂ and ozone -- but not particulates -- were taken from a monitors located within 2.5 miles of the hospital where they lived and worked.

There are a number of weaknesses in the study. The omission of particulates, which are associated with adverse health effects in many epidemiological studies, will bias the results if particulates affect the incidence of headaches and particulates and CO are correlated. The young and healthy student nurses are *not* representative of the national population. The estimated coefficient probably is smaller than it would have been with a more representative national sample. On the other hand, student nurses are more likely to be exposed to debilitating agents which might make them more susceptible to getting headaches. Nevertheless, in spite of the weaknesses, we use the study.

Using logistic regression, Schwartz and Zeger (1990: 63) estimated the effect of CO on the incidence of headaches, where incidence was defined as the presence of a symptom when the previous day was symptom free. The median bout of headache lasted one day (Schwartz and Zeger, 1990: Table 2).

To use the results of Schwartz and Zeger, we start with the basic assumption of the logistic model that:

$$\ln\left(\frac{p}{1-p}\right) = X \cdot \beta$$

where:

p = the probability of a day with a headache

X = the matrix of covariates

β = the vector of coefficients in the model.

We solve for the probability of a day with headache:

$$p = \frac{1}{1 + e^{-X\beta}}$$

We then use this equation twice: to calculate the probability of a day with a headache before and after a change in the ambient CO concentration. We calculate this change with the following equation:

$$\Delta \text{Headache} = \left[\frac{1}{1 + e^{-\Omega - \beta_c PP}} - \frac{1}{1 + e^{-\Omega - \beta_c PI}} \right] \text{Population}$$

where:

$\Delta \text{Headache}$ = change in the number of days with a headache

Ω = -2.107 (= the mean values of the other covariates multiplied by their coefficients)⁸

β_c = coefficient on CO, 0.0125 (Schwartz and Zeger, 1990: 63, Table 5)

PP = maximum one-hour CO level (ppm), after control

PI = maximum one-hour CO level (ppm), before control

Population = county population of all individuals, of all ages (U.S. Bureau of the Census, 1994).

Estimating Hospitalization for Congestive Heart Failure Caused by Carbon Monoxide

The work by Morris et al. (1995) quantifies the link between CO and hospitalization for congestive failure (ICD-9 code 428) in seven cities in the U.S. They report essentially a linear relationship between CO and hospitalization, although at very low CO levels (less than 2 ppm) the relationship is nonlinear and the effect of CO is stronger (Morris et al., 1995: Figure 2). Nevertheless, we assume that a linear approximation is reasonable (perhaps underestimating the true effect) and then estimate the average number of daily hospitalizations per 100,000 people over the age of 65 per ppm of CO in each city. We estimate a range of 0.05 to 0.12. Finally, we note that a certain fraction of the people that are admitted for congestive heart failure die, and their death should be valued much more highly than someone who is admitted for congestive heart failure and is then released. Based on the estimated number of cases of congestive heart failure reported in Morris et al. (1995) and the reported number of deaths from congestive heart failure (U.S. Department of Health and Human Services, 1991), we find 6% of the admittances for congestive heart failure result in death.

⁸Note that we assume that the other covariates do not change with a change in the CO level, and we set the covariates at their mean values. The coefficients and the mean values of the covariates in the headache estimation are

<i>Covariate</i>	<i>Coefficient</i>	<i>Mean Value of Covariate</i>
Intercept	-2.218	1
CO	0.0125	16.71 ppm
Monday	0.386	0.143
Pollen Allergy	0.328	0.17 prevalence in sample

We estimate the change in congestive heart failure with the following equation:

$$\Delta \text{Congestive Heart Failure Admits} = \beta (PP - PI) (1 - \text{probability of death}) \text{Population Over 65}$$

where:

Δ Congestive Heart Failure Admits = change in the number of hospital admittances for congestive heart failure
probability of death = 6% of hospital admittances for congestive heart failure result in reported mortality for congestive heart failure

β = 0.05 (lower bound), 0.12 (upper bound)

PP = maximum one-hour CO level (ppm), after control

PI = maximum one-hour CO level (ppm), before control

Population Over 65 = county population of all individuals over the age of 65 (U.S. Bureau of the Census, 1994).

11.3.2.2 Mortality

Because of the high value of mortality and the high level of CO emissions from motor vehicles, it is important to establish if there is any relationship between CO emissions and mortality. Lipfert (1985: 765-766) charged that several studies have linked CO with increased mortality, but that "these findings have been largely overlooked." The EPA (1991: 1-12) concluded in the latest CO criteria document that the evidence is "suggestive but not conclusive" that CO may lead to sudden death in people with coronary artery disease. After examining a number of studies (Table 11.3-4), we also conclude that it is likely that CO is responsible for increased mortality, however most of the evidence does not allow us to distinguish between the effect of CO and the effect of particulates; the work of Morris et al. (1995) which we use to estimate the increased incidence of hospital admittances for congestive heart failure. As we noted, roughly 6% of these cases result in death; we use this to estimate the effect of CO on mortality, recognizing that it is likely an underestimate of the true effect of CO on mortality. We present our review of the evidence regarding the link between CO and mortality:

- Hexter and Goldsmith (1971) reported that CO in Los Angeles caused increased mortality, however they measured only CO and O₃. CO may be capturing the mortality effect of other pollutants not included in the analysis, especially particulate matter. We believe that this study is not reliable.
- In an epidemiological study carried out in Baltimore, Kuller et al. (1975) found no association between ambient CO and myocardial infarction-caused mortality, although they did not rule out such an association.
- Mendelsohn and Orcutt (1979: 98-101) found that CO may cause between 7,000 and 35,000 deaths annually in the U.S., however the high standard

errors of the coefficients and the fact that NO₂ reportedly saved 63,936 lives per year makes us skeptical of the relationship between CO and mortality. The authors themselves state that sulfate is the dominant effect and that CO may have little, if any effect, on mortality.

- In another study of Los Angeles -- this one measuring several pollutants, including particulates (KM), hydrocarbons (HCs), and CO -- Shumway et al. (1988: 231) found a positive relation between mortality and three measures of pollution: KM, HC and CO.⁹ However, the pollutant measures were so highly correlated that the authors could not attribute the mortality effects to any one pollutant. They concluded (p. 233) that future modeling efforts need use only one of these pollution measures. The evidence that we have seen indicates that particulates are the main cause of mortality.
- Kinney and Ozkaynak (1991), using the same data set as Shumway (1988), found a significant relationship between mortality and ozone and a group of pollutants: CO, NO_x and KM. Due to collinearity, they could not distinguish whether CO, NO_x or KM was the responsible agent, so they concluded for predictive purposes that any one of the three is suitable.
- Finally, we note epidemiological studies by Stern et al. (1981), Edling and Axelson (1984) and Stern et al. (1988) that looked at the mortality rates of people in occupations exposed to high levels of automobile pollution -- motor vehicle examiners, garage workers and tunnel officers. All three studies found an abnormally high number of cardiovascular deaths, which is consistent with the hypothesis that CO causes cardiovascular problems. Moreover, Stern et al. (1981) found a higher incidence of cancer. However these studies did not control for other pollutants such as toxic gases and particulates.
- Kirney et al. (1995) estimated the risk of daily mortality as a function of pollution in Los Angeles from 1985 through 1990, and found that levels of PM₁₀, CO, and O₃ were, by themselves (in separate single-pollutant models) significantly associated with increased mortality risk. However, CO and O₃ levels were somewhat correlated with PM₁₀ levels. When CO and PM₁₀ were included simultaneously, the relative risk of both dropped and was no longer significant (at p = 0.05). When O₃ and PM₁₀ were included simultaneously, PM₁₀ was significantly associated with increased mortality risk, but O₃ was not. Kinney et al. (1995) do not draw any definitive conclusion about the relationship between CO or O₃ and mortality.

⁹Describing KM, Shumway et al (1988 227) reported that "KM monitors [draw] ambient air through a segment of porous tape during two-hour intervals and then measured the amount of light transmitted through the tape "

Mortality and Carbon Monoxide: Conclusion

The available evidence *suggests* to us that an increase in the concentration of CO does increase the risk of death for some people. However, nobody has quantified this risk convincingly and in a way that we can use to establish a dose-response function that directly links CO with mortality. However, we estimate an effect of CO on mortality indirectly by using the work of Morris et al. (1995) and assuming that 6% of the estimated cases of hospital admittances for congestive heart failure result in mortality. We estimate this effect with the following equation:

$$\Delta \text{Congestive Heart Failure Deaths} = \beta \cdot (PP - PI) \text{ (probability of death) Population Over 65}$$

where:

Δ Congestive Heart Failure Deaths = change in the number of hospital admittances for congestive heart failure resulting in death
probability of death = 6% of hospital admittances for congestive heart failure result in reported mortality for congestive heart failure
 β = 0.05 (lower bound), 0.12 (upper bound)
PP= maximum one-hour CO level (ppm), after control
PI= maximum one-hour CO level (ppm), before control
Population Over 65 = county population of all individuals over the age of 65 (U.S. Bureau of the Census, 1994).

11.3.3 Nitrogen Dioxide

A number of laboratory and epidemiological studies suggest that NO₂ increases minor respiratory symptoms and eye irritation (Table 11.3-5). We did not find evidence linking NO₂ to more serious illnesses such as emphysema, chronic bronchitis and cancer. Of course it is possible that NO₂'s contribution to more serious illness has simply escaped detection up until now. Populations most at risk include those people with preexisting respiratory disease, children and the elderly (EPA, 1993c: 1-23).

11.3.3.1 Morbidity

Minor Respiratory Symptoms

Schwartz and Zeger (1990) analyzed diary data collected from 110 student nurses living in Los Angeles, and found an association between NO₂ and the incidence of sore throat and phlegm attacks. A number of other studies have found similar results. Samet and Utell (1990) reported that NO₂ increases the susceptibility of people to respiratory infection and decreases lung functioning, and Goings et al. (1989: 1075) found NO₂ increases the susceptibility of adults to respiratory infections

Several studies have found NO₂ linked to cough, rhinitis and more general respiratory symptoms (Table 11.3-5), although they do not give the information necessary to determine a dose-response function. Robertson and Lebowitz (1984) and Lebowitz et al. (1987) examined symptoms reported by "normal" groups and an

asthmatic groups living in Tucson. Using spectral analysis, both studies linked NO₂ to coughing in both the normal and asthmatic groups, however spectral analysis is not useful for quantifying the effects of pollutants. Koo et al (1990), in a study of mothers and their primary school children in Hong Kong, found NO₂ linked to cough and chronic rhinitis in non-smoking mothers, but did not provide the information necessary to develop a dose-response function. Hasselblad et al. (1992) performed a meta-analysis (using the results from twelve studies) and found a significant relationship between respiratory effects in children under the age of twelve and a 30 µg/m³ increase in NO₂ -- roughly the increase that would be expected with the use of a gas stove in one's home. The meta-analysis, however, only measured the effect of 30 µg/m³ increase in NO₂ and did not allow for the interpretation of different changes in NO₂ levels, and thus we can not use it for this analysis.

Against these positive findings, three well-run epidemiological studies did not find that NO₂ increases respiratory symptoms (Table 11.3-5). Harrington and Krupnick (1985) reanalyzed the data base collected by Shy and Love (1980) in Chattanooga, Tennessee, and found that NO₂ did not increase respiratory disease in children. Instead, they found a "U" shaped dose-response curve, with the estimated illness rate at the highest observed two-week maximum concentration (384 µg/m³) lower than that at the lowest observed concentration (27 µg/m³). In a study of 5000 individuals living in four communities in southern California, Krupnick et al. (1990) found no relationship between NO₂ and acute respiratory disease. And Ostro et al. (1993), analyzing a subset of the data used by Krupnick et al. (1990), also did not find NO₂ linked to either upper or lower respiratory symptoms.

We did not find any evidence that NO₂ is associated with asthma attacks. Holguin et al. (1985), Dockery et al. (1989) and Roemer et al. (1993) all reported no link between NO₂ and asthma (Table 11.3-5).

Eye Irritation

Schwartz and Zeger (1990: 66) reported that NO₂ increases the incidence of eye irritation, probably because it is a precursor in the formation of peroxyacetyl nitrate. Lebowitz et al. (1987) also found NO₂ linked to eye irritation. Ostro et al. (1993), however, found ozone linked to eye irritation but not NO₂.

Severe Respiratory Symptoms

We found little evidence that NO₂ causes chronic respiratory problems or respiratory symptoms severe enough to require hospitalization (Table 11.3-5). Lafuma et al. (1987) did find evidence that rats exposed to NO₂ may develop more severe emphysema, but the rats in the study were exposed to levels of NO₂ higher than people commonly receive, which makes the study of doubtful usefulness in determining the effects of NO₂ on humans. In a study of Seventh-Day Adventists living in California, Euler et al. (1988) did not find any evidence of NO₂ increasing the incidence of COPD, and Dockery et al. (1989) did not see any link between NO₂ and bronchitis, chronic cough or persistent wheeze.

In Table 11.3-6 we review the effects of NO₂ on admissions to hospitals. Samet et al (1981), Bates and Sizto (1983), Sunyer et al. (1991) and Lipfert and Hammerstrom (1992) did not find NO₂ linked to hospital admissions. Richards et al. (1981), Bates et al. (1990) and Ponka (1991) did find NO₂ correlated with hospital admissions, but they did not control for the effects of other pollutants.

Morbidity Conclusion

It appears that NO₂ increases respiratory symptoms and eye irritation, but not chronic respiratory disease or hospital admissions. We use Schwartz and Zeger's (1990) work to estimate the increase in the number of days with sore throat, excess phlegm and eye irritation. We develop a dose-response function and apply it to all age groups -- including children, as explained next.

NO₂ adversely affects children. Although Roemer et al. (1993), Hoek et al. (1993), Dockery et al. (1989) and Harrington and Krupnick (1985) did not find an effect for children, the meta-analysis of 12 studies of children performed by Hasselblad et al. (1992) found that NO₂ causes respiratory problems in children.¹⁰ The EPA (1993c: 1-17) also concluded that NO₂ adversely affects children 5-12 years old after it reviewed a number of indoor air epidemiology studies. The EPA found an insignificant (but directionally correct) effect for infants two years and younger.¹¹

We estimate the effects of NO₂ down to its background level. Schwartz (1989) reported reductions in pulmonary functioning below the NAAQS standard of 0.05 ppm, and Schwartz and Zeger (1990: 65, Figures 3 and 4) reported that even very small amounts of NO₂ increase sore throat and eye irritation. Both of these findings suggest that there is no safe level of NO₂.

Estimating Sore-Throat Days Caused by Nitrogen Dioxide

Using logistic regression, Schwartz and Zeger (1990) estimated the effect of NO₂ on the incidence of sore throat, where incidence was defined as the presence of a symptom when the previous day was symptom free. The median bout of sore throat lasted two days (Schwartz and Zeger, 1990: Table 2), so we multiply the estimated effect by two to account for the total number of days with a sore throat.

We calculate the change in the number of days with sore throat with the following equation:

$$\Delta \text{ Sore Throat} = 2 \cdot \left[\frac{1}{1 + e^{-\Omega - \beta_n PP}} - \frac{1}{1 + e^{-\Omega - \beta_n PI}} \right] \text{ Population}$$

where:

Δ Sore Throat = change in the number of days with sore throat

¹⁰Meta-analysis is a more powerful test than individual studies and thus more likely to reject the null hypothesis (i.e., NO₂ has no effect on children) when it is false

¹¹In the infant analysis, the odds ratio was 1.09 with a 95% confidence interval of 0.95 to 1.26 (EPA, 1993c 1-18)

$\Omega = -3.782$ (= the mean values of the other covariates multiplied by their coefficients)¹²

β_n = coefficient on NO₂, 2.571 (Schwartz and Zeger, 1990: 63, Table 5)

PP= maximum one-hour NO₂ level (ppm), after control

PI = maximum one-hour NO₂ level (ppm), before control

Population = county population of all individuals, of all ages (U.S. Bureau of the Census, 1994).

Estimating Excess-Phlegm Days Caused by Nitrogen Dioxide

Schwartz and Zeger (1990) estimated the effect of NO₂ on the incidence of excess phlegm, where incidence was defined as the presence of a symptom when the previous day was symptom free. The median bout of excess phlegm lasted two days (Schwartz and Zeger, 1990: Table 2); hence we multiplied the estimated effect by two, to account for the total number of days with a excess phlegm.

We calculate the change in the number of days with excess phlegm with the following equation:

$$\Delta \text{ Excess Phlegm} = 2 \left[\frac{1}{1 + e^{-\Omega - \beta_n PP}} - \frac{1}{1 + e^{-\Omega - \beta_n PI}} \right] \text{ Population}$$

where:

Δ Excess Phlegm = change in the number of days with excess phlegm

$\Omega = -3.386$ (= the mean values of the other covariates multiplied by their coefficients)¹³

¹²The coefficients and the mean values of the covariates in the sore throat estimation are

Covariate	Coefficient	Mean Value of Covariate
Intercept	-2.311	1
NO ₂	2.571	0.13 ppm
Temperature	-0.0238	71.8 (degrees Fahrenheit)
Monday	0.4570	0.143
Sinusitis	0.7514	0.23 prevalence in sample

¹³The coefficients and the mean values of the covariates in the excess-phlegm day estimation are

Covariate	Coefficient	Mean Value of Covariate
Intercept	-2.379	1
NO ₂	0.843	0.13 ppm
Temperature	-0.0169	71.8 (degrees Fahrenheit)
Monday	0.626	0.143
Smoking	0.207	0.564 ^a

^a Smokers are categorized as 0=nonsmoker, 1=as much as 2 pack-years, and 2=more than 2 pack-years. We do not know the percentage of the sample in each category. We do know that 38.4% of the sample smoked and 18% of the sample smoked more than 15 cigarettes a day. We assume that 18% of the sample smoked more than 2 pack-years and the balance of the smokers (20.4% of the population) smoked less than 2 pack-years.

β_n = coefficient on NO₂, 0.843 (Schwartz and Zeger, 1990: 63, Table 5)
 PP = maximum one-hour NO₂ level (ppm), after control
 PI = maximum one-hour NO₂ level (ppm), before control
 Population = county population of all individuals, of all ages (U.S. Bureau of the Census, 1994).

Estimating Eye-Irritation Days Caused by Nitrogen Dioxide

Schwartz and Zeger (1990) estimated the effect of NO₂ on the incidence of eye irritation, where incidence was defined as the presence of a symptom when the previous day was symptom free. The median bout of eye irritation lasted one day (Schwartz and Zeger, 1990: Table 2).

The equation to estimate the change in the number of days with eye irritation is slightly more complicated than the other logistic equations because the NO₂ variable is the mean of the current and previous days' ambient levels. We use the daily one-hour maximum NO₂ level, however, rather than the two-day moving average because our procedure for filling in missing observations does not account for the exact day of the missing observation.

We calculate the change in the number of days with eye irritation with the following equation:

$$\Delta \text{ Eye Irritation} = \left[\frac{1}{1 + e^{-\Omega - \beta_n PP}} - \frac{1}{1 + e^{-\Omega - \beta_n PI}} \right] \text{Population}$$

where:

Δ Eye Irritation = change in the number of days with eye irritation

Ω = -3.463 (= the mean values of the other covariates multiplied by their coefficients)¹⁴

β_n = coefficient on NO₂, 1.604 (Schwartz and Zeger, 1990: 63, Table 5)

PP = maximum one-hour NO₂ level (ppm), after control

PI = maximum one-hour NO₂ level (ppm), before control

Population = county population of all individuals, of all ages (U.S. Bureau of the Census, 1994).

3.3 2 Mortality

The evidence in the literature linking NO₂ and mortality is weak (Table 11.3-6). In a time-series study of mortality records in St. Louis, Dockery et al. (1992) found

¹⁴The coefficients and the mean values of the covariates in the eye irritation estimation are

Covariate	Coefficient	Mean Value of Covariate
Intercept	-3.705	1
NO ₂	1.604	0.13 ppm
Oxidant*Oxidant	0.000767	10.24 pphm
Monday	0.527	0.143
Pollen Allergy	0.5066	0.17 prevalence in sample

PM₁₀ linked to mortality but not NO₂. Shumway et al. (1988), in a study of mortality in Los Angeles, found no link between NO₂ and mortality. Kinney and Ozkaynak (1991) used the same data as Shumway et al. (1988) and found an association between NO₂ and mortality, but high collinearity between the pollution variables prevented them from distinguishing between the effect of CO, NO₂ and KM (an indirect measure of particulates). Because the best evidence links particulates and mortality, we assume that KM, and not NO₂, is the best predictor of mortality in the Kinney and Ozkaynak (1991) work. And because we estimate mortality using particulate data, to also use NO₂ would invite double counting. Therefore, we conclude that there is as yet no satisfactory evidence of a link between NO₂ and mortality.¹⁵

11.3.4 Ozone

Ozone is a strong oxidant linked with a number of adverse health effects. It alters the mechanical functions of the lung, injures cells in the respiratory tract (Mehlman and Borek, 1987: 38), and reduces the lungs' ability to expel foreign (possibly carcinogenic) material (Pinkerton et al., 1989). It causes respiratory infection (Gilmour et al., 1993), inflammation in the respiratory tract (Koren et al., 1989) and acute respiratory symptoms, including asthma attacks (Holguin et al., 1985; Ostro et al., 1993). Moreover, Kinney and Ozkaynak (1991) and Moolgavkar (1995) reported that ozone is linked with mortality. All people are susceptible to the effects of ozone, but people with asthma, COPD, bronchitis and allergies are especially sensitive (Bresnitz and Rest, 1988: 395).

11.3.4.1 Acute Morbidity

Eye Irritation

In a review of the health effects of ozone Lippmann (1989c) noted that oxidants other than ozone such as peroxyacetyl nitrate and aldehydes are responsible for increased eye irritation. This is consistent with the laboratory results of Kulle et al. (1985), who did not find that ozone increases eye irritation. Since peroxyacetyl nitrate and aldehydes are generally found together with ozone, ozone may estimate their effects. If motor vehicles cause the same percentage of peroxyacetyl nitrate, aldehydes and ozone then we may use ozone as a proxy to estimate the effect of motor vehicles on eye irritation.¹⁶

Zagraniski et al. (1979), Schwartz and Zeger (1990) and Ostro et al. (1993) presented evidence linking ozone (and oxidants) to eye irritation, but only Schwartz and Zeger (1990) published coefficients for a dose-response function.

¹⁵In analyses similar to ours, Schwartz et al (1985), Rowe et al (1986) and Hall et al (1989) also concluded that there is not enough evidence to link NO₂ and mortality

¹⁶Motor vehicles actually may contribute equally to ambient ozone and aldehyde concentrations. Gasoline and diesel vehicles are responsible for roughly 40% of national emissions of VOCs (EPA, 1995a, 1995b), and motor vehicles are also responsible for roughly 40% of the ambient concentrations of formaldehyde and acetaldehyde (see Table 11 6-8)

Asthma Attacks

A number of studies have linked ozone and other oxidants to asthma attacks (Tables 11.3-7 to 11.3-9). Whittemore and Korn (1980) studied 443 asthmatics living in California and found both oxidants and TSP linked to asthma attacks. Dockery et al. (1989) found ozone significantly related to the asthma rates of grade school children. Holguin et al. (1985), in a careful study of Houston asthmatics, found ozone linked to asthma. Lebowitz et al. (1987), using spectral analysis, found ozone linked to wheeze, coughing and reduced pulmonary functioning in asthmatics. And Kulle et al. (1985) and Schwartz and Zeger (1990) found ozone related to chest discomfort, which is consistent with the hypothesis that ozone causes asthma attacks.

However, the evidence has not shown that ozone increases the number of *new* asthmatics. Portney and Mullahy (1990) and Abbey et al. (1991a) examined the long-term consequences of air pollution, and did not find that ozone increases the number of new asthmatics. Bates et al. (1990), Ponka (1991) and Cody et al. (1992) found that ozone causes asthma-related hospital admissions, but Richards et al. (1981) and Schwartz et al. (1993) have not reported this effect.

To estimate the effect of ozone on the incidence of asthma we use the results of Whittemore and Korn (1980) in our lower-bound case, and the results of Holguin et al. (1985) in our upper-bound case.

Acute Respiratory Symptoms Other than Asthma Attacks

Several studies have linked ozone to broad groups of respiratory symptoms. Portney and Mullahy (1986) used the 1979 Health Interview Survey and found ozone significantly related to minor respiratory-related restricted days (mRRADs) in adults. Ostro and Rothschild (1989) used six years of the Health Interview Survey (1976-1981) and found that ozone causes mRRADs, although the results were decidedly mixed – some years of data yielded significant negative coefficients and other years yielded significant positive coefficients.¹⁷ Finally, Ostro et al. (1993), in a re-analysis of data from a study of 290 families in Southern California (by Krupnick et al., 1990), found ozone linked to lower and upper respiratory symptoms.

In both the lower-bound and the upper-bound cases, we use the coefficients from Ostro et al. (1993) to estimate the effect of ozone on respiratory ailments. The work of Ostro et al. (1993) is preferable to that of Portney and Mullahy (1986) and Ostro and Rothschild (1989) because the latter studies relied on the number of incidents occurring over a two-week period rather than daily incidence and used a broadly defined dependent variable.

Because the lower respiratory illnesses counted by Ostro et al. (1993) include asthma attacks, which we estimate separately, we avoid double-counting respiratory ailments by subtracting the number of asthma attacks from the number of days with lower respiratory illness. This subtraction assumes that RRADs that include asthma

¹⁷Ostro and Rothschild's (1989) results for 1979 are close to Portney and Mullahy's (1986)

attacks include no other symptoms. To the extent that this is not true, we undercount symptoms and underestimate the effects of ozone.

Other Symptoms

In a study of 290 families in southern California, Krupnick et al. (1990) found ozone significantly related to a combined measure, termed ARD2, of any respiratory complaint, headache and eye irritation. This measure thus includes all of the effects already enumerated above -- eye irritation, asthma attacks, and upper and lower respiratory illnesses -- but also other effects, such as headaches.

In our upper-bound case, we use the coefficients from Krupnick et al. (1990) to estimate the effect of ozone on any symptom (ARD2) other than eye irritation, asthma attack, and upper and lower respiratory illness. Because ARD2 does include eye irritation, asthma, and lower and lower and upper respiratory illness, we must subtract the estimated number of cases of these from the estimated number of cases of ARD2¹⁸.

We do not include any of these other [residual] symptoms in our lower-bound case.

Ozone and Morbidity: Conclusion

We found reasonably persuasive evidence linking ozone with eye irritation, asthma attacks, other acute lower and upper respiratory symptoms, and other symptoms (Table 11.3-10). We use Schwartz and Zeger's (1990) work to estimate the change in eye irritation associated with ozone, using ozone as an acceptable proxy for the oxidants that often appear together with ozone and directly cause eye irritation. We estimate the change in asthma attacks using the results of Whittemore and Korn (1980) in our lower-bound case, and the results of Holguin et al. (1985) in our upper-bound case¹⁹. We use the work of Ostro et al. (1993) to estimate the change in lower and upper respiratory symptoms other than asthma. Finally, we use the work of Krupnick et al. (1990) to estimate the change in all other symptoms (such as headaches), in our upper bound case only. Table 11.3-11 presents the health effects studies used by previous economic analyses.

In the upper-bound case, we treat children the same as adults when applying the dose-response functions for eye irritation, asthma, other lower and upper respiratory symptoms, and any other symptom, in the upper bound scenario. In the lower bound case, we do not estimate an effect for children for eye irritation, or for

¹⁸We could use the ARD2 measure alone to estimate all symptoms associated with ozone, and not bother to estimate eye irritation, asthma, and respiratory illnesses separately. However, we feel that it is more precise to add separate estimates of the individual effects than to use a broad, catch-all measure to count everything.

¹⁹Because the number of cases of respiratory illness other than asthma equals the total number of cases of respiratory illness (Ostro et al., 1993) minus the number of asthma cases, the difference between the lower-bound (Whittemore and Korn, 1980) and the upper-bound (Holguin et al., 1985) number of asthma cases changes the number of asthma versus non-asthma cases of respiratory illness, but not the *total* number of cases of respiratory illnesses. However, the change in the distribution of asthma versus non-asthma cases matters because the former are much more costly than are the latter (Table 11 4-4).

lower and upper respiratory symptoms. Although children suffer a decline in lung function due to ozone (Detels et al., 1991: 357), they are not as likely as adults to display symptoms such as coughing following ozone exposure.²⁰ The reason for this difference is not clear. However, even if children do not display symptoms it is possible that they still suffer damage from ozone exposure that will manifest itself when they are older.

We assume no threshold and estimate the effect of ozone down to its natural background level (0.015 ppm in the upper bound and 0.010 in the lower bound) because even very low levels of ozone cause adverse health effects.

Our assumptions regarding ozone are summarized below:

Symptom	Lower-bound case		Upper-bound case	
		Children?		Children?
Eye irritation	Schwartz and Zeger (1990)	No	Schwartz and Zeger (1990)	Yes
Asthma attacks	Whittemore and Korn (1980)	Yes	Holguin et al. (1985)	Yes
Respiratory illnesses except asthma attacks	Respiratory cases (Ostro et al. 1993) minus asthma cases	No	Respiratory cases (Ostro et al. 1993) minus asthma cases	Yes
All other symptoms	none	No	ARD2 cases (Krupnick et al., 1990) minus all of the above cases	Yes

Estimating Eye Irritation Caused By Ozone

Schwartz and Zeger (1990) analyzed data collected from 110 student nurses living in Los Angeles in the early 1960's and estimated the effects of CO, SO₂, NO₂, and oxidants on eye irritation, headache, chest discomfort, and other symptoms. Using logistic regression they found oxidants linked to the incidence of eye irritation. The median bout of eye irritation lasted one day.²¹

We calculate the change in the number of days with eye irritation with the following equation:

²⁰Avol et al (1985) and McDonnell et al (1985a) found that children suffer a decline in the amount of air that they can expel in one second (FEV1) but do not suffer from the coughing that afflicts adults following ozone exposure (Folinsbee et al, 1988, McDonnell et al, 1983) Krupnick et al (1990) and Portney and Mullahy (1986) found ozone linked to acute respiratory illness in adults but not in children

On the other hand Whittemore and Korn (1980) and Holguin et al (1985) found ozone provokes asthma attacks in both children and adults Berry et al (1991) found children suffering cough and runny nose due to ozone exposure, although the results of this study are weakened because other pollutants were not included in the analysis

²¹They define incidence as the presence of a symptom when the previous day was symptom free

$$\Delta \text{ Eye Irritation} = \left[\frac{1}{1 + e^{-\Omega - \beta_o (PP^2)}} - \frac{1}{1 + e^{-\Omega - \beta_o (PI^2)}} \right] \text{Population}$$

where:

Δ Eye Irritation = change in the number of incidences of days with eye irritation

Ω = -3.335 (= the mean values of the other covariates multiplied by their coefficients)²²

β_o = coefficient on ozone, 7.67 (Schwartz and Zeger, 1990: 64, Table 5)²³

PP = maximum one-hour oxidant level (ppm), after control

PI = maximum one-hour oxidant level (ppm), before control

Oxidant = Ozone/0.9 (Krupnick and Kopp, 1988: 2-22)

Population = county population of all individuals, of all ages in the upper bound case. In the lower bound case, the population includes only individuals above the age of 17 (U.S. Bureau of the Census, 1994).

Estimating Asthma Attacks Caused by Ozone -- Whittemore and Korn (1980)

Whittemore and Korn (1980) studied 443 physician-confirmed asthmatics from six communities in the Los Angeles. The participants lived within two miles of an air quality monitor and kept a daily diary regarding their health and any asthma medication used.

The dependent variable was the rate of asthma attacks per day among the respondents. Pollution variables were 24-hour average concentration ($\mu\text{g}/\text{m}^3$) of TSP and the daily maximum hourly average (ppm) of oxidants (including, but not limited to, ozone).

Following Krupnick and Kopp (1988: 2-37), we calculate the change in the number of days with asthma attacks with the following equation:

²²The coefficients and the mean values of the covariates in the eye irritation estimation are

Covariate	Coefficient	Mean Value of Covariate
Intercept	-3.705	1
NO ₂	1.604	0.13 ppm
[Oxidant] ²	0.000767	10.24 pphm
Monday	0.4570	0.143
Pollen Allergy	0.5066	0.17 prevalence in sample

²³Schwartz and Zeger (1990) used oxidants in parts per hundred million, so the published coefficient is 10⁴ times smaller than shown here

$$\Delta \text{ Asthma Attacks} = \left(\frac{m}{1+m} - p \right) \text{ Asthmatic Population,}$$

$$m = \frac{p}{1-p} e^{[\beta_o (PP-PI)]}$$

where:

Δ Asthma Attacks = change in the daily number of asthma attacks

p = daily incidence rate for asthma = 0.0271 (Krupnick, 1988: 4-6).

β_o = coefficient on ozone, 1.66 (Whittemore and Korn, 1980: 691, Table 5)

PP = maximum one-hour O_x level (in ppm), after control

PI = maximum one-hour O_x level (in ppm), before control

Oxidant = Ozone/0.9 (Krupnick and Kopp, 1988: 2-22)

Asthmatic Population = 4.45% of the county population, based on the average of the asthma rate between 1988 and 1991 (Table 11.3-12); population data from the U.S. Bureau of the Census (1994).

As explained above, we use this equation in our lower bound.

Estimating Asthma Attacks Caused by Ozone -- Holguin et al. (1985)

Holguin et al. (1985) studied 51 non-smoking physician-diagnosed asthmatics from two Houston communities living within 2.5 miles of an ozone monitor. Using diaries kept by respondents, Holguin et al. (1985) determined the 12-hour asthma attack rates over a six month period.

Because it is impractical have air quality monitors at each person's home, Holguin et al. (1985) measured ozone inside and outside of the home over a relatively short period of time and regressed each set of observations on the ozone readings at a nearby outside monitor. Using the resulting regression coefficients, they estimated hourly exposure levels inside and outside of the home during the length of the study.²⁴

To avoid overestimating exposure and following Holguin et al. (1985) as closely as possible, we adjust for the estimated time that people spend indoors and outdoors and for the difference in the ozone levels between the two. We use 57.5% of the ambient reading at fixed-site monitors to estimate asthma attacks between 7 am-7 pm and 52.5% of the ambient reading between 7 pm-7 am.²⁵

²⁴Using entries from an hourly diary kept by the participants, Holguin et al (1985) estimated the exposure of each respondent for each hour in every 12-hour period (e g , 7 am-7 pm) A weighted average was used when respondents split an hour between indoors and outdoors Holguin et al (1985) used the highest hourly reading for a given 12-hour period to help predict asthma attacks

²⁵Except for some ozone forming from the use of photocopiers and laser printers, ozone originates from outdoor sources, and because it is readily absorbed and destroyed on surfaces, indoor levels tend to be lower than outdoors (Graedel, 1988 148) Weschler et al (1989) reviewed the literature and found that the indoor/outdoor ratio most likely ranges roughly between 0.2 and 0.8 with the precise value depending on the air exchange rate between the two environments and whether air filters are used Since we do not know the indoor/outdoor ratio for the study's participants, we assume it equaled 0.5,

Following Krupnick and Kopp (1988: 2-37), we calculate the change in the number of days with asthma attacks with the following equation, applied twice (once for daytime, once for nighttime):

$$\Delta \text{ Asthma Attacks} = \left(\frac{m}{1+m} - p \right) \text{ Asthmatic Population,}$$

$$m = \frac{p}{1-p} \cdot e^{[\beta_o (w \text{ PP} - \text{PI})]}$$

where:

Δ Asthma Attacks = change in the number of asthma attacks for the 7 am-7 pm or 7 pm-7 am period

p = daily incidence rate for asthma = 0.0271 (Krupnick, 1988: 4-6).

β_o = coefficient on ozone, 6.20 (Holguin et al., 1985: 276, Table 4)

PP = maximum one-hour ozone level (in ppm) for 7 am-7 pm, adjusted for indoor exposure, after control

PI = maximum one-hour ozone level (in ppm) for 7 am-7 pm, adjusted for indoor exposure, before control

w = weight used to estimate the maximum ozone concentration at night relative to the known maximum during the day; thus, when the equation is run for the daytime (7 am to 7 pm), the weight w on the daytime maximum ozone levels (X_1 and X_0) is 1.0, and when the equation is run for the nighttime (7 pm to 7 am), the weight w on the [same] daytime maximum ozone levels is 0.37, on the assumption that the nighttime maximum is 37% of the observed daytime maximum (Krupnick and Kopp, 1988: 2-37)

Asthmatic Population = 4.45% of the county population, based on the average of the asthma rate between 1988 and 1991 (Table 11.3-12); population data from the U.S. Bureau of the Census (1994).

We use this equation in our upper bound.

Estimating Lower and Upper Respiratory Symptoms Caused By Ozone

Ostro et al. (1993) examined the effect of COH, NO₂, ozone, SO₂ and SO₄ on lower and upper respiratory symptoms in 321 nonsmoking adults living in Azusa, California. Controlling for temperature, sex, day of study, gas stove use and the presence of chronic respiratory disease, they found ozone fairly closely linked to lower respiratory symptoms and upper respiratory symptoms. Although they considered the pollutants separately, the correlation between ozone and SO₄ is fairly low ($r=0.24$). No effect was found for the other pollutants.

and using the results from time-activity studies, we assume that people spend roughly 85% of their time indoors between 7 am-7 pm and 95% between 7 pm-7 am (EPA, 1993a. 5-29) We use 57.5% (= 85%*0.5 + 15%*1.0) of the ambient reading at fixed-site monitors to estimate asthma attacks between 7 am-7 pm and 52.5% (=95%*0.5 + 5%*1.0) between 7 pm-7 am

Because Ostro et al. (1993) reported the odds ratio for a 0.1 ppm change in the ozone level, we estimate the change caused by ozone in lower and upper respiratory symptoms using a different type of approximation (derived below) than that used in the logistic models of Schwartz and Zeger (1990).

Lower Respiratory Illness

To calculate the change in the incidence of lower respiratory illness, we use the following equation (which we derive later):

$$\Delta \text{Lower Respiratory Illness} = [p (1-p) \beta_o (PP - PI)] \text{Population}$$

where:

Δ Lower Respiratory Illness = change in the number of days with lower respiratory illness

$p = 0.039$ = mean daily incidence rate (Ostro et al., 1993: 693)

$\beta_o = 1.0436$ = ozone coefficient calculated from the odds ratio (=1.11; Ostro et al., 1993: Table 5) estimated from a 0.10 ppm change in the ozone level

PP = maximum one-hour ozone level (ppm), after control

PI = maximum one-hour ozone level (ppm), before control

Population = county population of non-asthmatic individuals, of all ages in the upper bound case. In the lower bound case, the population includes only non-asthmatics above the age of 17 (U.S. Bureau of the Census, 1994).

Upper Respiratory Illness

To calculate the change in the incidence of upper respiratory illness, we use the following equation (which we derive later):

$$\Delta \text{Upper Respiratory Illness} = [p (1-p) \beta_o (PP - PI)] \text{Population}$$

where:

Δ Upper Respiratory Illness = change in the number of days with upper respiratory illness

$p = 0.015$ = mean daily incidence rate (Ostro et al., 1993: 693)

$\beta_o = 0.7696$ = ozone coefficient calculated from the odds ratio (=1.08; Ostro et al., 1993: Table 5) estimated from a 0.10 ppm change in the ozone level

PP = maximum one-hour ozone level (ppm), after control

PI = maximum one-hour ozone level (ppm), before control

Population = county population of all individuals, of all ages in the upper bound case. In the lower bound case, the population includes only individuals above the age of 17 (U.S. Bureau of the Census, 1994).

Deriving the Equation for Estimating Respiratory Illness

We start with the basic assumption of the logistic model, namely that the log of the odds is a linear function of the covariates in the model:

$$\ln\left(\frac{p}{1-p}\right) = X \beta \quad (1)$$

where:

p = the probability of respiratory illness

X = the matrix of covariates

β = the logistic regression coefficients of the model.

We solve for the probability of respiratory illness

$$p = \frac{1}{1 + e^{-X\beta}} \quad (2).$$

When estimating the effect of ozone on eye irritation, we use equation (2); however Ostro et al. reported the odds ratio but not the estimated logistic regression coefficients (β), which forces us to use a different approach.

Taking the first derivative of equation (2) with respect to the concentration of ozone, holding the other covariates constant, we find:

$$\frac{\partial p}{\partial X_i} = (1 + e^{-X\beta})^{-2} e^{-X\beta} \beta_i \quad (3)$$

where:

X_i = the ozone concentration

β_i = the ozone coefficient.

Using equation (2), we solve for $e^{-X\beta}$ in terms of p :

$$p = \frac{1}{1 + e^{-X\beta}} \Rightarrow 1 + e^{-X\beta} = \frac{1}{p} \Rightarrow e^{-X\beta} = \frac{1-p}{p} \quad (4).$$

We enter the solution from equation (4) into equation (3) and derive:

$$\frac{\partial p}{\partial X_i} = \left(1 + \frac{1-p}{p}\right)^{-2} \cdot \left(\frac{1-p}{p}\right) \beta_i = p(1-p) \beta_i \quad (5).$$

To make equation (5) useful for estimating health effects, we use a first order Taylor series approximation, equation (6), and then solve for Δp :

$$\frac{\partial p}{\partial X_i} = \frac{\Delta p}{\Delta X_i} \quad (6)$$

$$\Delta p = p(1-p) \beta_1 \Delta X_1 \quad (7)$$

We use equation (7) to estimate the effect of ozone on lower and upper respiratory illness. We discuss below the calculation of the ozone coefficient, β_1 , using the odds ratio.

Calculating the Logistic Regression Coefficient from the Odds Ratio

Ostro et al. (1993) published the odds ratio for respiratory illness from a 0.1 ppm increase in the ozone level. We use this odds ratio and backtrack to estimate the logistic regression coefficient originally used to determine the odds ratio! With the coefficient we can then estimate the effect of any change in the ozone level, consistent with the ozone concentrations experienced by the sample in Ostro et al.'s (1993) study. We present the calculations used to estimate the logistic regression coefficient for lower respiratory illness; we did analogous calculations to determine the coefficient for upper respiratory illness.

We define the odds ratio with the following table, which shows the percentage of people that have lower respiratory illness given a high level and a baseline level of ozone exposure:

Percentage Of People With And Without Respiratory Illness High And Baseline Ozone

Ozone Level	Lower Respiratory Illness	No Illness
High Ozone (Baseline + 0.1 ppm)	A %	B %
Baseline Ozone	C %	D %

We calculate the odds of lower respiratory illness for each ozone level and the ratio of the two to get the "odds ratio:"

$$\text{Odds of Lower Respiratory Illness High } O_3 = \frac{A}{B}$$

$$\text{Odds of Lower Respiratory Illness Baseline } O_3 = \frac{C}{D}$$

$$\text{Odds Ratio} = \frac{\frac{A}{B}}{\frac{C}{D}}$$

The logistic model used by Ostro et al. (1993) assumed that the log of the odds is a linear function of the covariates in the model, so we can solve for the odds of lower respiratory illness at the baseline ozone level and when it is 0.1 ppm higher:

$$\ln\left(\frac{A}{B}\right) = X_h \cdot \beta \Rightarrow \frac{A}{B} = e^{X_h \beta}$$

$$\ln\left(\frac{C}{D}\right) = X_b \beta \Rightarrow \frac{C}{D} = e^{X_b \beta}$$

where:

X_h = model covariates with high ozone level (baseline plus 0.1 ppm)

X_b = model covariates with baseline ozone level

β = model coefficients.

We determine an expression for the odds ratio (= 1.11) for lower respiratory illness in terms of the change in the ozone level and the ozone coefficient:

$$\text{Odds Ratio} = \frac{\frac{A}{B}}{\frac{C}{D}} = \frac{e^{X_h \beta}}{e^{X_b \beta}} = e^{X_h \beta - X_b \beta} = e^{\beta_i (X_{i,h} - X_{i,b})} = 1.11$$

where:

β_i = ozone coefficient

$X_{i,h}$ = high ozone level

$X_{i,b}$ = baseline ozone level.

Finally we solve for the ozone coefficient, β_i , for lower respiratory illness:

$$\beta_i = \frac{\ln(1.11)}{X_{i,h} - X_{i,b}} = \frac{\ln(1.11)}{0.1} = 1.0436$$

Estimating ARD2 (any symptom) Caused By Ozone

As mentioned above, Krupnick et al. (1990) studied 290 families in Southern California and found ozone significantly related to a combined measure of any respiratory complaint, headache and eye irritation (termed ARD2). In the upper bound case, we estimate the change in the number of ARD2 symptoms with the following equation:

$$\Delta \text{ARD2} = \beta_o (PP - PI) \text{ Population}$$

where:

$\beta_o = 0.20$ = ozone coefficient from Krupnick and Kopp (1988: 2-34)

PP = maximum one-hour ozone level (ppm), after control

PI = maximum one-hour ozone level (ppm), before control

population = people of all ages (U.S. Bureau of the Census, 1994)

The change in ARD2 includes cases of eye irritation, asthma attacks, and upper and lower respiratory illness, which we have counted already (above). Hence, we subtract these from the estimated change in ARD2, to end up with the number of cases of other symptoms (such as headaches). We include these other symptoms in our upper bound estimate only.

11.3 4 2 *Chronic Morbidity*

While scientists have linked ozone to less severe forms of chronic disease such as asthma, hay fever and sinusitis (Table 11.3-8), they have not yet found strong evidence that ozone increases the number of people suffering from chronic illness. Nevertheless it is likely that such a link exists. A recent series of papers by Balmes (1993), Bates (1993), Devlin (1993), Lippman (1993) and Ostro (1993a) have discussed the evidence for the link and suggested future avenues of research.

In a national study, Portney and Mullahy (1990) examined the effect of both ozone and TSP on hay fever, sinusitis, and a combined measure of chronic respiratory problems (asthma, chronic bronchitis and emphysema). Controlling for TSP, they found ozone significantly related to the sinusitis and hay fever. However, because they used only one year of health data, it is not clear if ozone increased the number of people who suffered from hay fever and sinusitis, increased the number of symptoms among existing sufferers, or both. In any event, given that we already use the work of Ostro et al. (1993) to estimate the effect of ozone on the incidence of upper respiratory symptoms (which includes both sinusitis and hay fever), it would be double counting to use Portney and Mullahy's (1990) work to estimate the increase in new cases.

In a study of Seventh-Day Adventists living within five miles of their current California residence for at least ten years, Abbey et al. (1991a; 1993) found an increase in the number of physician-confirmed asthmatics in areas with higher levels of ozone. However Abbey et al. included only one pollutant at a time and the significant effect for ozone could be due to other pollutants. TSP was more strongly linked to asthma than ozone, and in the same study TSP was linked to chronic bronchitis and all chronic respiratory problems. Since we already use PM₁₀ (based on Abbey et al., 1995) to estimate the effect of chronic respiratory illness we choose not to use Abbey et al.'s (1991a; 1993) work to estimate a possible link between ozone and chronic respiratory illness. The foregoing suggests to us that ozone causes new cases of asthma, but we are not able to measure them due to the close correlation of ozone with TSP.

There is little evidence that ozone causes more severe symptoms such as emphysema and chronic bronchitis. Neither Euler et al. (1988) or Abbey et al (1991a; 1993) reported ozone causing chronic bronchitis or emphysema. Although Fujinaka et al. (1985) found that ozone caused significant long-term damage in the lungs of monkeys exposed to 0.64 ppm ozone for eight hours per day for a year, the high ozone levels and the usual problems with using the results of animal studies to estimate the effects in humans, persuade us not to use this study.

11.3 4.3 Mortality

Hexter and Goldsmith (1971), Shumway et al. (1988), Schwartz (1991) and Dockery et al. (1992) reported no significant correlation between ozone and mortality. However, Kinney and Ozkaynak (1991) found that ozone increased acute mortality in Los Angeles, Mills (1991) found that it may increase the incidence of cancer of Seventh-Day Adventists living in California, Moolgavkar (1995) found that ozone increased acute mortality in Philadelphia during the summer, and Ozkaynak et al. (1995) found that ozone and TSP or PM₁₀ were both significantly associated with daily mortality in Toronto, Canada.

Recently, Kinney et al. (1995) estimated the risk of daily mortality as a function of pollution in Los Angeles from 1985 through 1990, and found that levels of PM₁₀, CO, and O₃ were, by themselves (in separate single-pollutant models) significantly associated with increased mortality risk. However, CO and O₃ levels were somewhat correlated with PM₁₀ levels. When O₃ and PM₁₀ were included simultaneously, PM₁₀ was significantly associated with increased mortality risk, but O₃ was not. Kinney et al. (1995) believe that “definitive conclusions regarding a possible role of O₃ in daily mortality cannot be drawn from this small data set, nor from the existing literature” (p. 68).

To explain why ozone may be linked to mortality, Kinney and Ozkaynak (1991: 115) suggested that ozone is most deadly for people who have already serious health problems, and is acting as the proverbial last straw. Along similar lines, Cody et al. (1992: 193) found that ozone causes an increase in hospital admissions from asthma attacks and suggested that ozone-induced asthma attacks cause increased mortality.

Nevertheless, the evidence regarding an independent link between ozone and mortality is mixed, and one reasonably might doubt that ozone has any nontrivial effect apart from the effect of other pollutants. We already include the effects of particulates and CO on mortality. It is likely that we would be double counting the effects of air pollution by including a separate estimate for ozone. While we recognize that there could be a link between ozone and mortality, we do not include to avoid double counting the effects of air pollution.

11.3.5 Particulate Matter

Particulates have been linked to a variety of adverse health effects – reduced pulmonary functioning, asthma attacks, respiratory symptoms, hospital admissions for cardiac and respiratory problems, chronic respiratory disease and mortality. All people are affected by particulates, but those with previous respiratory symptoms and the elderly are particularly prone to its adverse effects. Tables 11.3-13 to 11.3-17 summarize the epidemiological evidence that we discuss further below.

Table 11.3-18 presents a variety of the particulate measures used in epidemiological studies and in setting air pollution standards, both in the U.S. and in Britain. Ideally whatever measure(s) we choose would capture all of the health effects associated with particulates. We use three particulate measures – PM_{2.5}, PM₁₀ and TSP -- to estimate health effects, because these measures and the

associated health effects studies cover the broadest range of effects. Health effects researchers have used a variety of particulate measures (e.g., H⁺, sulfates, PM_{2.5}, PM₁₀, COH, TSP, and British Smoke), depending on the availability of air pollution and health data in a particular geographic area. A number of studies have used TSP to measure particulates, although the number of these studies has declined as the research focus has shifted to inhalable particulates. The current criteria pollutant is PM₁₀, and most recent studies link PM₁₀ to morbidity (e.g., Pope, 1989) and mortality (e.g., Dockery et al., 1992). However, as discussed in Chapter 11.1, many researchers now believe that PM_{2.5} or even finer particulates, or acidic particles specifically, will prove to be the most dangerous.

11.3.5.1 Are the effects associated with particulate matter a function of the size of the particles, the composition of the particles, or the number of particles?

Particulate matter is a heterogeneous mix of solid or liquid compounds, including organic aerosols, sulfates, nitrates, and metals, suspended in the atmosphere. It comprises a very wide range of chemical constituents, structures, and sizes, which in turn can have a wide range of physiological and biological effects.

The epidemiological studies that we use to quantify health effects relate the effects to an undifferentiated mass of particles (usually all particles less than 10 μm in diameter); they do not relate health effects to individual PM compounds, or to the absolute number of particles inhaled. However, because the size and chemical characteristics of PM mixes vary from one emissions source to the next (for example, PM from diesel engines is quite a bit different than PM from road dust), it is likely that some emissions sources contribute more to the estimated overall harm (from undifferentiated PM) than do others. Some sources of PM even might be harmless. Thus, in order for us to correctly attribute the estimated health effects to specific emissions sources, we must know what specific properties of PM are causing the health effects, and where the specifically harmful PM comes from.

Current research, reviewed below, indicates that smaller particles are more dangerous than larger particles, that acidic particles are more dangerous than dust particles, and that more particles (of any size or composition) are more dangerous than fewer particles.

The effect of particulate size.

It matters a lot whether health effects are related to the size of the PM, because PM from combustion generally is much smaller than PM from dust. If size does not matter, then most of the health effects might be attributed to fugitive dust, which accounts for the bulk of the airborne particulate matter to which people are exposed.

However, there is a near-consensus among researchers that fine particulates (less than 2.5 μm in diameter), which can penetrate deeply into the lungs, are more dangerous than coarser particulates, although exactly which are the more dangerous

sizes is not known.²⁶ Here is a sampling of some recent findings and reviews regarding the relationship between particle size and health effects:

- Pope et al. (1995: 669) single out particulates less 2.5 microns as being a larger public health concern than the coarser fraction of PM₁₀, because the smaller particles can be inhaled more deeply into the lungs.
- Dockery et al. (1995) aver that “an overall evaluation of the epidemiologic evidence suggests that fine particulates (PM_{2.5}), which are primarily from combustion sources, are more closely associated with human health effects than coarser particles” (p. 120).
- Marrack (1995a) offers a similar perspective, concluding that “particles less than 3.0 μm diameter have biological properties that make them much more significant than the larger components” (p. 8).
- The cross-sectional mortality study of Ozkaynak and Thurston (1987), and the hospitalization study of Thurston et al. (1994), found a stronger effect for fine than coarse particulates.

It is possible that the most harmful component of particulate matter is smaller still -- less than 1.0 μm :

- Bates (1995) reviews a recent colloquium on particulate air pollution and suggests that the harmful particles might be less than 1.0 μm in size, because in general very small particles can penetrate more easily into the lungs, and because some very small particles appear particularly dangerous.
- In laboratory studies of rats Oberdorster et al. (1992) found particulates less than 0.02 microns as having a much stronger effect than particulates less than 0.2 microns. In later work, Oberdorster et al. (1995) found that ultrafine particles of polytetrafluoroethylene were extremely toxic and caused acute mortality in rats at very low inhaled mass concentrations.
- Ostro (1990) found that sulfate particulates, which range from about 0.05 to 5.0 μm (Allen, 1995), have the strongest effect on respiratory effects, possibly through a link to acid aerosols.
- Miller et al. (1995) argue that a “reasonable argument can be made that, for some individuals, the inhalation of high concentrations of particles

²⁶In recent years, ambient particulate data collection efforts have gradually focused on finer particulates, moving from TSP, to PM₁₀ and PM_{2.5} and even PM_{1.0}. Undoubtedly, more resources will be devoted to collecting data on fine particulates -- PM_{2.5}, PM_{1.0} and even finer particulates

between about 0.1 and 0.3 μm may result in a localized overloading of the lung's clearance mechanisms that triggers a cascade of events leading to acute morbidity and/or mortality" (p. 630).

We believe, then, that it is likely that $\text{PM}_{2.5}$ or $\text{PM}_{1.0}$ is more dangerous than the coarse fraction of PM_{10} . Still, it is doubtful that the coarse fraction is altogether harmless. In the first place, many of the studies cited above state that the coarse particles are *less* harmful, not harmless. Furthermore, some studies have found that the coarse fraction of PM_{10} can have the same effect as the fine fraction. For example, Douglas Dockery et al. (1992) in a time-series study of mortality in St. Louis and Eastern Tennessee found roughly the same effect for $\text{PM}_{2.5}$ and the coarser fraction of PM_{10} . And Ozkaynak et al. (1995) report that in a study of daily mortality and pollution in Toronto, they "could not distinguish the estimated mortality effects of TSP from those associated with exposures to PM_{10} " (p. 812)²⁷. Consequently, we feel that it is still premature to ignore the coarser fraction of PM_{10} altogether²⁸.

In sum, most epidemiological and toxicological research suggests that $\text{PM}_{2.5}$ is more dangerous than the coarser fraction of PM_{10} , but does not demonstrate that the coarser fraction of PM_{10} is completely harmless. Our own view is that qualitatively, fine PM is at least twice as dangerous as coarse PM, and perhaps an order of magnitude more dangerous. Therefore, when we apportion total particulate damages to individual sources (such as motor vehicles), we assume that $\text{PM}_{2.5}$ is from 2.0 (lower bound) to 10.0 (upper bound) times as potent (in terms of deaths per gram) as is the coarse fraction of PM_{10} . In defense of this range, we can say only that, on the basis of the evidence we reviewed, it seems reasonable to us. Obviously, one could make different assumptions.

The effect of particulate composition.

Are all particle types of a given size equally dangerous, or are, say, combustion particles of a given size generally more dangerous than dust particles of the same size? The answer is important because road dust is the largest single source of particulate matter in the U.S. emissions inventory, and one legitimately might wonder whether road-dust particles of a given size really are as harmful as diesel-engine PM of the same size.

²⁷Of course, this does not prove that all particle sizes were equally harmful. It is possible that TSP and PM_{10} were so closely correlated that their effects simply were not statistically separable.

²⁸It is likely that the relationship between particle size and health effect is different for different health effects. For example, it might be that coarse particles affect asthma as much as do fine particles, but affect mortality much less than do fine particles. Nevertheless, we assume one $\text{PM}_{2.5}/\text{PM}_{10}$ potency ratio for all health effects, we do not establish different $\text{PM}_{2.5}/\text{PM}_{10}$ potency ratios for different health effects.

It appears that composition does matter. Two recent studies suggest that mineral-based and related PM from dust are not as dangerous as other types of particulates, of the same size:

- Kleinman et al. (1995) tested the biological effects of road dust, nitrates, and sulfates. Road dust had some significant adverse effects on cells, but generally had fewer adverse effects than did nitrates and sulfates. All of the particles (of all three types) in their experiments were relatively large, 4 μm . The concentration of road dust administered was about 12 times greater than the concentration of sulfates, and 3 times greater than the concentration of nitrates. The experimenters conclude that "in terms of potency relative to the concentration of each component administered, the effects could be ordered as $\text{SO}_4^{2-} > \text{NO}_3^- > \text{road dust}$ " (p. 600).
- Koenig et al. (1995) studied the relationship between visits to the emergency room and levels of two different kinds of fine particulate matter: those from combustion sources, such as automobiles, and those from dust (mainly dust storms). They note that "at first glance, it appears that fine particles from the earth's crust are less likely to be associated with adverse health outcomes than fine particles from combustion sources," but warn that "the individual characteristics of each air shed make comparisons risky" (p. 806). (See also Heflin, 1994).
- In a study of the relationship between PM_{10} , ozone, and respiratory-related hospital emissions, Schwartz (1996) tested to see if agricultural dust had less of an effect than did the "average" PM_{10} .

Windblown dust from agricultural land is an important source of PM_{10} in the fall, after crops are harvested. If this particulate is less toxic than average, a lower PM_{10} slope should be seen during the fall. To test this possibility, I repeated the basic regression with an interaction term between PM_{10} and the fall season. The interaction term was negative, indicating a lower slope, but it had a t-statistic less than one, and there was no improvement in model fit (p. 23-24).

This perhaps does hint, barely, that agricultural dust is less harmful than average PM_{10} , but the lack of significance, and lack of improvement in model fit, make the finding weak at best.

Many other studies did not specifically control for size, but nevertheless suggest that all else equal, dust particles are not as dangerous as combustion particles:

- Xu et al. (1994: 222) suggested that natural soil dust may be less dangerous than anthropogenic pollution, because soil is coarse and not readily inhalable and is more inert than, say, particulates from coal combustion.

- Cormier et al. (1990) found that organic dust increased the incidence of cough and sputum production but had little lasting effect on the respiratory health of peat moss factory workers.
- Yano (1990) found only a small increase in respiratory symptoms in an area with high levels of volcanic ash (which was relatively biologically inert).
- Ozkaynak and Thurston (1987) reported that coal particulates increased mortality rates, while soil and auto particulates did not.²⁹
- Lewtas (1995) suggests that particulate organic matter might be the "causative agent" for human mortality and morbidity.
- Marrack (1995a) concludes that the principal health effects of particulate matter are due to acidic PM_{2.5}, to hazardous air pollutants absorbed onto carbonaceous PM, and to mutagenic organic matter, most of which apparently are derived from combustion sources. However, he does also identify adverse effects of mineral and vegetable dusts (see below).
- In a review of a recent colloquium on particulate air pollution and human health, Bates (1995) suggests that studies have shown that ash from residual fuel oil is more toxic than generic dust in Dusseldorf Germany, which in turn is more toxic than volcanic ash from Mount St. Helens.
- Many researchers believe that acid particles, which come from combustion sources but not fugitive dust, are the most dangerous component of particulate air pollution (Marrack, 1995a, 1995b; Samet et al., 1995; Schlesinger, 1995; Utell and Frampton, 1995; Waldman et al., 1995). These particles reportedly cause increased morbidity and mortality (Amdur, 1989; Lippmann, 1989a, 1989b; Ostro et al., 1991). For example, Samet et al. (1995) remark that "current concepts of particle toxicity emphasize the role of particle acidity and the induction of inflammation at sites of injury" (p. 5). And Thurston et al. (1994) report that in their research, "the acidity of an aerosol has been confirmed to be one important particulate matter attribute to be considered.." (p. 287)³⁰.

²⁹We do not attach much significance to the surprising result regarding auto particulates Ozkaynak and Thurston used observations from only 25 MSAs in the mid-west -- and their negative result contradicts a substantial body of work that shows that diesel and gas particulates are carcinogenic (EPA, 1993a, Mauderly, 1994, Schlesinger, 1995, Cohen and Higgins, 1995), and contradicts recent work by Saldiva et al (1995) that found a significant link between PM₁₀ and daily mortality of elderly persons in Sao Paulo, Brazil, where the primary source of pollution comes from motor vehicles

³⁰However, a number of studies have not found a significant effect for acidity Pope et al (1992) linked daily mortality with PM₁₀ that turned out to have very low particle acidity Similarly, Dockery et al (1993) found that "mortality was more strongly associated with the levels of inhalable, fine, and

Still, not all researchers are convinced that one can distinguish particle types according to harm. Lipfert and Wyzga (1995: 7) reported "no evidence for a differential effect of particle chemistry on mortality". Dockery and Pope (1994) note that researchers have found that particulates affect people regardless of location, and that the magnitude of the effect is reasonably similar, which suggests that the composition of particulates is not very important. For example, the estimated logistic regression coefficients from Utah (Pope et al., 1992) and St. Louis and eastern Tennessee (Dockery et al., 1992) are remarkably close -0.00147 versus 0.00150 and 0.00160 . Likewise, mortality studies in London have produced results similar to those in the U.S. (Schwartz and Dockery, 1992a: 602). In light of this, Dockery and Pope (1994), in their recent review of the effects of particulate air pollution, argue against distinguishing particles by potency:

The mass concentration of PM₁₀ includes a wide array of potentially toxic chemical species. It is, therefore, presumptuous to assign these observed health effects solely to the mass concentration of particulates. On the other hand, the consistency of these observed effects across so many communities suggests that, lacking an explicit hypothesis, these associations should be assigned to a nonspecific definition of inhalable or fine particulate concentrations common to urban areas. Until controlled animal- and human -exposure studies identify the active component(s) of these complex mixtures and can characterize their underlying mechanisms of toxicity, it is prudent to ascribe health effects observed by epidemiologists to the undifferentiated particle mass rather than to any specific component (1994: 127)

Moreover, regardless of whether it is reasonable to distinguish particle potency by composition, it certainly is the case that fugitive dust, although perhaps less harmful than, say, sulfates, is not harmless. Marrack (1995a) points out that:

A number of "inert" mineral PM-10 when inhaled cause adverse tissue changes which may be recognized as disease. These exposures may be at levels below the NAAQS. The exposures do not have to be long term to result in tissue damage effects but the latent period prior to recognizable changes is usually years. Oxides and salts of Beryllium, Manganese, Iron, Cadmium and Nickel on their own or as part of mixed chemical composition PM-10 have been implicated in such tissue damage. A variety of crystalline silicon oxide (silica) containing minerals when inhaled as small particles can cause lung tissue damage which is usually progressive without additional silica exposure (p. 4)

In the same vein, Miller et al. (1995) argue that a "tracheobronchial deposition of coarse mode particles should not be dismissed as potentially contributing to the acute morbidity seen in asthmatics" (p. 630). As mentioned above, Kleinman et al. (1995) and Koenig et al. (1995) found that dust does have adverse effects, albeit apparently not as serious as the effects of combustion particles and sulfates.

Finally, Fairley (1990) notes that re-entrained road dust contains "... in addition to soil particles, engine oil which contains various metals; tire particles; and sulfates" (p. 161) -- that is, some of the compounds that people suspect of being

sulfate particles than with the levels of total suspended particles, the sulfur dioxide levels, the nitrogen oxide levels, or the acidity of the aerosol" (p. 1755). Dockery et al. (1992) have a similar finding, although they emphasize that they cannot rule out aerosols as a risk factor.

particularly dangerous. Similarly, road dust is involved in the atmospheric chemistry that produces some of the putatively more harmful particulates, such as nitrates (Yang et al., 1994). And to the extent that, say, acidic sulfate particles would not exist in the absence of road dust, road dust in effect is as dangerous as is sulfate.

This evidence suggests that particulates from paved and unpaved roads, road construction and agricultural tillage are less harmful than other types of particulates, but not entirely harmless. Qualitatively, it appears to us that dust of a given size class could be anywhere from about half as potent as other (mainly combustion) particles, to an order of magnitude less potent. Thus, in our lower bound, we assume particulates from paved roads, unpaved roads, construction, agricultural tillage, and livestock are 10% as potent as other particles; in our upper bound, we assume that they are 50% as potent. We do not pretend that this range has any real quantitative basis; we suggest only that it is not unreasonable given the evidence. One certainly could make different assumptions.

The effect of the number of particles.

Some researchers have suggested that health effects are related directly to the number-concentration of particles inhaled (number of particles per ml), regardless of size and composition. The greater the number concentration, the greater the irritation, and the more the immune system is overwhelmed locally (Chen et al., 1995). As noted above, Miller et al. (1995) argue that a high concentration of particles might overwhelm the lung's defenses and eventually cause morbidity and mortality.

Generally, however, the number of particles is a function of the size of the particles, because small particles can be inhaled more deeply into the lung. Thus, sources that emit smaller particles are more likely to cause a localized PM overload in the lungs and eventually adverse health effects. Consequently, if the number concentration of particles really is the determinant of health effects, we may as a reasonable approximation apportion health effects of PM on the basis of the size distribution of particles from emissions sources.

11.3.5. 2 Acute Morbidity

Table 11.3-13 summarizes the epidemiological work examining the link between particulates and pulmonary functioning. The bulk of the work suggests that particulates cause a decline in lung functioning (which, unfortunately, we cannot value in dollars). Tables 11.3-14 and 11.3-15 consider the effects of particulates on effects that we can value. We consider, in turn, the effects of particulates on asthma, general respiratory ailments and hospital admissions.

Asthma

Five studies found a significant effect between particulates and asthma:

- Cohen et al. (1972) studied 20 asthmatics living in West Virginia and found various particulate measures and SO₂ linked to asthma, although no single pollutant was found to be the cause of asthma attacks.

- In a study of Los Angeles asthmatics, Whittemore and Korn (1980) controlled for the effect of O_x and found TSP linked to asthma attacks.
- Perry et al. (1983) found particulate nitrates linked to asthma in a study of 24 Denver asthmatics. However, a later study by Ostro et al. (1991) (discussed below) did not support the results of this paper.
- In a study of 207 Denver asthmatics, Ostro et al. (1991) did not find an effect for nitrates but did find that PM_{2.5} increased the incidence of asthma attacks. Because Ostro et al. did not publish the coefficient for PM_{2.5}, we can not use the study to develop a dose-response function.
- Finally, in a study of industrial towns in the Netherlands, Roemer et al. (1993) found SO₂ and particulates associated with asthma attacks. (The pollutants were considered separately.)

The available evidence, then, indicates that particulate air pollution causes asthma attacks. We use the Whittemore and Korn (1980) to develop a dose-response function, because theirs is sophisticated statistical analysis, is amenable to developing a dose-response function, and considers ozone and particulates simultaneously.

Respiratory Symptoms

In Table 11.3-14 we review a number of studies that examined a possible link between particulates and acute respiratory ailments other than asthma attacks.

- Portney and Mullahy (1986) used the 1979 national Health Interview Survey to study the effects of ozone and SO₄ on over 3000 adults 17 and older. They found that ozone increases significant minor respiratory symptoms but they found no effect for SO₄.
- Ostro and Rothschild (1989) used six years (1976-1981) of data from the Health Interview Survey and found PM_{2.5} associated with both minor and more serious respiratory illness, which give rise to respiratory-related restricted activity days (RRADs). They found significant effects in most years, including 1979, the year that Portney and Mullahy (1986) analyzed
- In a study of Los Angeles residents, Krupnick et al. (1990) found that the coefficient of haze (COH) increases all types of respiratory symptoms, especially in adults.
- Using observations from 321 nonsmoking adults --a subset of Krupnick et al.'s (1990) data -- Ostro et al. (1993) did not find COH to be a significant predictor of lower respiratory illness ($p = 0.14$), but did find SO₄ significantly linked to lower respiratory illness.

Although the evidence is mixed, in part because a variety of particulate measures have been used, it does indicate that particulates are linked to respiratory problems. We use the study by Ostro and Rothschild (1989) to estimate the link because the study is well done, and because PM_{2.5} can be reasonably approximated using PM₁₀ data and probably is a better measure of particulate air pollution than are either SO₄ or COH.

Hospital Admissions

Eleven of the fourteen studies that we review, and all of the studies since 1983, found particulates significantly related to hospital admissions (Table 11.3-15). Hospital admissions, however, overlap with our other measures of morbidity (asthma attacks and RRADs) and acute mortality. To avoid double-counting we do not estimate the incidence of hospital admissions due to particulates.

Our Conclusion

We estimate the effect of particulates on asthma and RRADs. There is some overlap between RRAD and asthma attacks -- to avoid double-counting we estimate RRAD only for the nonasthmatic population. We do not estimate the incidence of hospital admissions because these are reasonably well captured by our estimates for asthma, RRADs and mortality. The health effects studies that we use are summarized in Table 11.3-19; the studies used by previous economic analyses are in Table 11.3-20.

Particulates definitely have a negative effect on pulmonary functioning and symptoms in children (Ostro, 1989; Pope, 1989; Krupnick et al., 1990; Roemer et al., 1993). Krupnick et al. (1990) compared the effects of particulates on children and adults and reported that the coefficient for COH was 59% smaller for children than it was for adults. In the lower bound, we use a coefficient that is 59% smaller for children. We do not adjust the number of asthma attacks, because the majority of participants in Whittemore and Korn's (1980) study were children, and asthma is prevalent among children (Table 11.3-12).

Although the evidence is mixed, we assume that there is no threshold level below which particulates are harmless. Chestnut et al. (1991) did not find reduced pulmonary functioning when the quarterly average TSP level fell below 60 µg/m³. Similarly, Vedal et al. (1991) found no effect on respiratory symptoms when the annual average TSP and PM₁₀ levels fell below 45-50 µg/m³ and 35-40 µg/m³. On the other hand, studies that link particulates with mortality have reported no threshold. Schwartz (1991), Dockery et al. (1992), Pope et al. (1992) and Schwartz and Dockery (1992b) reported an effect for particulates well below the NAAQS standard.

Estimating Asthma Attack-Days Caused by Particulates

Whittemore and Korn (1980) studied 443 physician-confirmed asthmatics from six communities in the Los Angeles, who kept daily diaries in the period 1972 to 1975. Each asthmatic lived within two miles of an air monitor.

The dependent variable is the rate of asthma attacks per day among the respondents. The pollution variables included the 24-hour average concentration

($\mu\text{g}/\text{m}^3$) of TSP and the daily maximum hourly average (ppm) of oxidants (which include ozone).

Following Krupnick and Kopp (1988), we calculate the change in the number of days with asthma attacks with the following equation:

$$\Delta \text{Asthma Attacks} = \left(\frac{m}{1+m} - p \right) \text{Asthmatic Population},$$

$$m = \frac{p}{1-p} \cdot e^{\beta_{pm} (PP-PI)}$$

where:

Δ Asthma Attacks = change in the daily number of asthma attacks

p = daily incidence rate for asthma = 0.0271 (Krupnick, 1988: 4-6).

β_{pm} = coefficient on TSP, 0.00079 (Whittemore and Korn, 1980: 691, Table 5)

PP = daily average TSP level ($\mu\text{g}/\text{m}^3$), after control

PI = TSP level ($\mu\text{g}/\text{m}^3$), before control

Asthmatic Population = 4.45% of the county population, based on the average of the asthma rate between 1988 and 1991; these values are reported in Table 11.3-12; population data from the U.S. Bureau of the Census (1994).

Estimating Respiratory-Related Restricted Activity Days (RRADs) Caused by Particulates

Using data from 68 MSAs collected by the Health Interview Study (HIS) for the years 1976-1981, Ostro and Rothschild (1989) examined the effect of ozone and PM_{2.5} on working adults 18-65 years old. The PM_{2.5} data was estimated from airport visibility data. The pollutants were considered simultaneously in a Poisson regression, and the correlation between the two pollutants was low over the six years of data ($r = -0.03$ to 0.25).

The dependent variable is the incidence of respiratory-related restricted activity days (RRADs) and minor RRADs over a two-week period. Ostro and Rothschild found the two-week daily-average PM_{2.5}, lagged one period, a significant predictor of both RRADs and mRRADs, although the best results were found for RRADs. The results for ozone were mixed and indicated that ozone is responsible for minor respiratory ailments.

We calculate the change in the daily (rather than bi-weekly) incidence of RRADs with the following equation (derived below):

$$\Delta \text{RRAD} = \text{RRAD}_0 \cdot \left[e^{(PP-PI) \beta_{pm}} - 1 \right] \text{Population}$$

where:

Δ RRAD = change in the number of RRADs in a day

$RRAD_0 = 0.118/14$, the average number of RRADs in a day (Ostro and Rothschild, 1989: 242) (Ostro and Rothschild estimated 0.118 RRADs over two weeks; we assume one-fourteenth this amount per day)³¹
 PP = daily average PM_{2.5} level ($\mu\text{g}/\text{m}^3$), after control
 PI = daily average PM_{2.5} level ($\mu\text{g}/\text{m}^3$), before control
 $B_{\text{pm}} = 0.0158$, the weighted average of the PM_{2.5} coefficients, where each coefficient is weighted by the inverse of its variance (Ostro and Rothschild, 1989: 242)³²
 Population = county population of non-asthmatic individuals of all ages (U.S. Bureau of the Census, 1994).

Deriving the Equation for Estimating RRAD

To calculate the change in RRADs, we start with the basic assumption of the Poisson regression model used by Ostro and Rothschild (1989):

$$E(RRAD_0) = e^{X_0 \beta} \quad (1)$$

where:

$E(RRAD_0)$ = the expected number of RRAD per day, given the baseline values for the covariates (where the baseline value is denoted by a subscript '0')

X_0 = the matrix of covariates (including PM_{2.5}), at baseline values

B = vector of coefficients.

Changing only the PM_{2.5} level, we calculate the expected number of RRAD per day:

$$E(RRAD_1) = e^{X_1 \beta} \quad (2)$$

³¹Ostro and Rothschild (1989) estimated the incidence of RRADs over a two-week period, using the average PM_{2.5} pollution level over two weeks. Ideally, we would replicate their procedure exactly: we would estimate the average PM_{2.5} pollution level over two weeks, calculate the change in RRADs over a two-week period, with respect to the two-week average incidence of RRADs, and do this calculation 26 times for each year. However, because we estimate PM_{2.5} levels with respect to daily PM₁₀ levels, it is easier for us to calculate the change in RRADs daily, with respect to a daily average incidence of RRADs which we assume to be one-fourteenth of the average incidence over two weeks, and run the equation 365 times per year. This method of using the daily average pollution level and the daily average incidence of RRADs will not necessarily yield the same result as will the preferred approach of using the two-week average incidence of RRADs and the two-week daily average pollution level, because as can be seen from the equation the change in RRADs is linear with respect to the average incidence but nonlinear with respect to pollution.

³²Using the published standard deviations, we determine that the weighted average equals 0.0150. We assume the difference between this and Ostro and Rothschild's value of 0.0158 is attributable to rounding error, and that their calculations carry more significant figures and thus are more desirable.

Dividing equation (1) by equation (2), we find that the other covariates drop out, and the expression on the right-hand side of equation (3) is a function only of the change in the ambient PM_{2.5} concentration and the PM_{2.5} coefficient:

$$\frac{E(RRAD_1)}{E(RRAD_0)} = \frac{e^{X_1 \beta}}{e^{X_0 \beta}} = e^{(PP-PI) \beta_{pm}} \quad (3)$$

where:

PP = the new PM_{2.5} ambient concentration

PI = the baseline PM_{2.5} ambient concentration

B_{pm} = the PM_{2.5} coefficient.

Multiplying both sides of equation (3) by equation (1), we get:

$$E(RRAD_1) = E(RRAD_0) \cdot e^{(PP-PI) \beta_{pm}} \quad (4).$$

Finally, subtracting equation (1) from both sides of equation (4), we get:

$$\Delta E(RRAD) = E(RRAD_0) \left[e^{(PP-PI) \beta_{pm}} - 1 \right] \quad (5)$$

We use equation (5) to estimate the effect of particulates on RRAD

11.3.5.3 Chronic Morbidity

A number of studies have found that particulates cause chronic respiratory problems in people of all ages (Table 11.3-16). Chapman et al. (1985), in a study of young adults, and Dockery et al. (1989) and Vedal et al. (1991), in studies of grade school children, found particulates linked to chronic cough, chronic phlegm, wheezing, chest illness, and bronchitis. In a study of two closely matched counties in the Utah Valley, Archer (1990) found more deaths from nonmalignant respiratory disease in the county with higher pollution levels. Finally, in a series of studies of adult Seventh Day Adventists living in California, Euler et al. (1987; 1988) and Abbey et al. (1991a; 1993; 1995) found TSP and PM₁₀ significantly linked to airway obstructive disease (AOD), which includes asthma, chronic bronchitis and emphysema.

The evidence clearly points to a link between particulates and chronic disease. For several reasons, Abbey et al.'s (1995) study is the most useful: they used a panel design which allows us to determine the incidence of new cases of chronic disease, they reported statistics that allow us to develop dose response functions; and they measure ambient particulate levels using PM₁₀ rather than TSP.

Estimating Chronic Respiratory Disease Caused by Particulates

Abbey et al. (1995) used multiple logistic regression in a panel study to link PM₁₀ with asthma, chronic bronchitis, and emphysema. They based the analysis on health surveys completed by 3,914 Seventh Day Adventists over the age of 25 living within five miles of their current residence in California for at least ten years (as of 1977). Health surveys were conducted in 1977 and again in 1987.

Abbey et al. (1995: 140) classified individuals as definitely having asthma, chronic bronchitis or emphysema if the following conditions were met:

- *asthma*: having been told by their physician that they had asthma as well as having a history of wheezing, or
- *chronic bronchitis*: symptoms of cough and /or sputum production on most days, for at least three months per year, for two years or more, or
- *emphysema*: having been told by their physician they had emphysema as well as having shortness of breath when walking either normal paced or hurried.

Their method of classification appears to allow new cases of chronic disease to be classified in more than one group: 7.38% (N=3,237, cases=239) got chronic bronchitis, 2.17% (N=3,634, cases=79) got asthma, yet only 8.50% (N=3236, cases=275) got AOD. Although Abbey et al. did not specify why the sample sizes differ, the smaller percentage for AOD suggests overlapping definitions and a small percentage of people getting emphysema (roughly 0.7%). (Abbey et al. reported an insufficient number of cases to do a separate analysis on emphysema; they did not report the number of emphysema cases.)

The percentage of new cases of chronic bronchitis (7.38%) that occurred between 1977 and 1987 in Abbey et al.'s sample seems high compared to the national figures of chronic bronchitis prevalence (5.01%) we report in Table 11.3-12. Four possible explanations are:

- *A reversal rate of roughly 50%*. The reversal rate equals the number of cases that had definite symptoms in 1977 who did not have definite symptoms in 1987 divided by the number who had definite symptoms in 1977.
- *Different definitions of chronic bronchitis*. The National Center for Health Statistics (1992) -- the source of the national rate -- asked the respondent whether he or she has had "bronchitis," while Abbey et al. asked if the respondent had "cough and/or sputum production on most days, for at least three months per year." It seems likely that Abbey et al.'s would produce more positive responses.
- *Misreporting by respondents*. Regarding asthma, Abbey et al. (1993: 42), who used the same data set as Abbey et al. (1995), reported confirming 30

out of 49 cases of asthma by checking the respondents' medical records. A 61.2% confirmation rate is low for a serious disease like asthma. Obviously people either did not confide in their doctors or they misunderstood the health survey; the latter seems more likely.

- *California specific factors.* A brief review of the counties with non-attainment areas (EPA, 1992a) suggests that California has a disproportionate share. Higher pollution rates (or other factors) in California, relative to the rest of the U.S., may have caused a higher proportion of cases.

We assume that it is better to use our own best estimate of the annual incidence rate of new cases, rather than the original estimate of Abbey et al. (1995), even though we take our coefficient (beta) from Abbey et al. To account for the misreporting that apparently occurred in the Abbey et al study, we multiply their reported incidence rates by 61%, which as noted above, appears to be the ratio of the true (doctor-confirmed) incidence rate to the reported rate. Hence we assume the incidence of new cases over ten years is 5.185% (rather than 8.5%) for AOD, and to determine the annual rate we divide these percentages by ten.

We do not make any adjustment for the high reversal rate. An adjustment would be called for if the reversal rate in Abbey et al. (1995) differed from the reversal rate in Krupnick and Cropper (1992) and Viscusi et al. (1991) – studies that we use to value chronic illness. We have no information regarding any difference between these studies.

Abbey et al. (1995) estimated the cumulative number of hours of PM₁₀ exposure above thresholds of 40, 50, 60, 80 and 100 µg/m³, from 1973-1977. They deemed these exposure levels representative of the study population's long-term exposure to PM₁₀; they picked the exposure variable that gave the best statistical fit. Little is known about the best way to estimate long-term exposure, so we accept their procedure .

Particulates emitted in any of the exposure periods may have been only partially responsible for the estimated increase in AOD. Particulates may quickly affect susceptible people or the effect may be more gradual, with many years of exposure necessary to contract chronic disease – in which case no single year or episode of exposure would be responsible. We assume that the estimated new cases of AOD will occur over a 20-year period, with the effects distributed in a bell-shape over this period (Table 11.3-21). Clearly a different lag structure between exposure and disease could have been chosen, but until more is known about the etiology of AOD and its link to particulates, we consider a 20-year lag a reasonable approximation.

We use the following equation (derived in Section 11.3.4.1) to determine the number of new cases of AOD caused by PM₁₀:

$$\Delta AOD = p (1 - p) \beta_{pm} \cdot (PP - PI) \text{ Population}$$

where:

Δ AOD = change in the number of cases of airway obstructive disease (AOD)
 $p = 0.005185$ = estimated annual incidence of new cases of AOD

β_{pm} = coefficient on PM_{10} , $5.401 \cdot 10^{-5}$ ($40 \mu\text{g}/\text{m}^3$ threshold); = $5.791 \cdot 10^{-5}$ ($50 \mu\text{g}/\text{m}^3$ threshold); = $7.402 \cdot 10^{-5}$ ($60 \mu\text{g}/\text{m}^3$ threshold); = $1.001 \cdot 10^{-4}$ ($80 \mu\text{g}/\text{m}^3$ threshold); = $1.699 \cdot 10^{-4}$ ($100 \mu\text{g}/\text{m}^3$ threshold) (Source: Table 11.3-22)

PP = one individual's annual hours of PM_{10} exposure above the threshold, after control

PI = one individual's annual hours of PM_{10} exposure above the threshold, before control

Population = county population of all individuals (U.S. Bureau of the Census, 1994).

It is not obvious which of the five coefficients we should use. To allow for the uncertainty in our choice, we use the mean estimated number of cases in our lower bound, and in our upper bound we use the maximum.

Calculating The Regression Coefficient From The Relative Risk Estimate

Abbey et al. (1995) published the relative risk but not the estimated coefficients for all of the exposure levels; they presented coefficients only for exposure exceeding $100 \mu\text{g}/\text{m}^3$. The fact that particulates adversely affect people at much lower exposure levels and that $100 \mu\text{g}/\text{m}^3$ is rarely exceeded in many communities in the United States suggests that we should estimate the coefficients for other exposure thresholds.

We use the relative risk estimates published by Abbey et al., summarized in Table 11.3-22, to find the "odds ratio," and then estimate the logistic regression coefficient. We present the calculations for the estimated coefficient for AOD and exposure in excess of $100 \mu\text{g}/\text{m}^3$; we do exactly analogous calculations for other exposure thresholds.

The relative risk is the ratio of the risk with and without PM_{10} exposure, and can be formally defined with a table showing the percentage of people that have AOD, depending on their PM_{10} exposure.

Percentage Of People With AOD In High PM_{10} And Baseline PM_{10} Exposure Cases

PM_{10} Level	AOD	No AOD
High PM_{10} Exposure (Baseline + 1000 hours/year at $> 100 \mu\text{g}/\text{m}^3$)	A	B
Baseline PM_{10} Exposure	C	D

We can calculate the "risk" of AOD at the two exposure levels and then the "relative risk" (which is the ratio of the two risks):

$$\text{Risk of AOD With High PM}_{10} \text{ Exposure} = \frac{A}{A+B}$$

$$\text{Risk of AOD With Baseline PM}_{10} \text{ Exposure} = \frac{C}{C+D}$$

$$\text{Relative Risk} = \frac{\frac{A}{A+B}}{\frac{C}{C+D}}$$

Abbey et al. (1995: Table 3) reported that the incidence rate new cases of AOD in their sample was 8.498%, which we assume equals the baseline incidence rate (C in the table above). Given the relative risk (= 1.17) of AOD from 1000 hours of PM₁₀ above 100 µg/m³, and the fact that the sums of A and B, and C and D, both equal 100%, we can then calculate A, B, C and D:

$$\text{Relative Risk} = \frac{\frac{A}{A+B}}{\frac{C}{C+D}} = \frac{A}{C} = 1.17$$

$$B = 100\% - A = 90.057\%$$

$$D = 100\% - C = 91.502\%$$

It is then easy to calculate the odds ratio:

$$\text{Odds Ratio} = \frac{\frac{A}{B}}{\frac{C}{D}} = 1.1888$$

Finally, using the formula developed in Section 11.3.4.1, we estimate the logistic coefficient for PM₁₀:

$$\beta_{PM_{10}} = \frac{\ln(\text{Odds Ratio})}{X_h - X_b} = \frac{\ln(1.1888)}{1000} = 1.729 \cdot 10^{-4}$$

where:

X_h = 1000 hours above 100 µg/m³, over the course of a year

X_b = 0 hours above 100 µg/m³, over the course of a year.

Our estimated coefficient ($\beta_{PM_{10}} = 1.729 \cdot 10^{-4}$) is close to Abbey et al.'s (1995: Table 3) published coefficient ($\beta_{PM_{10}} = 1.699 \cdot 10^{-4}$). The difference probably is due to

rounding error in the relative risk: a coefficient of $1.699 \cdot 10^{-4}$ implies a relative risk of 1.1668, which rounds to the published figure of 1.17³³. In any event, our estimated coefficient of $1.729 \cdot 10^{-4}$ is close enough to the published estimate of $1.699 \cdot 10^{-4}$ that we may use the relative risk to estimate the (unpublished) coefficients for the exposures at other threshold levels (Table 11.3-22).

11.3.5.4 *Particulates and mortality: "acute" versus "chronic" deaths, "harvest" versus "non harvest" deaths, and the relationship to times-series versus prospective cohort and cross-sectional studies*

For years researchers have debated what (if any) effect particulates have on mortality. In 1985, Morgan et al (1985: 665) claimed "almost any answer is possible...depending on which expert is consulted."

Since their statement, a number of results have been published providing convincing evidence that particulates are positively associated with acute mortality, and that the size of the estimated effect is consistent across the U.S (see Dockery and Pope, 1994; Schwartz, 1994a, 1994b). A variety of particulate measures have been used, and yet all measures have found a significant effect: Ozkaynak and Thurston (1987) studied sulfates; Fairley (1990), coefficient of haze; Dockery et al. (1992) and Pope et al. (1992), PM₁₀; Schwartz, (1991), Schwartz and Dockery (1992a and 1992b), TSP; and Schwartz and Marcus (1990), British smoke. Furthermore, two prospective cohort studies (Dockery et al., 1993; Pope et al., 1995) and a number of "ecologic" studies (e.g., Ozkaynak and Thurston, 1987) using annual pollution levels have found a significant link between mortality and a measure of chronic particulate exposure. Thus, there are two strands of evidence linking particulates to both "acute" and "chronic" deaths. As we discuss below, it appears that we should divide acute deaths into two groups: "harvest" deaths that would have occurred anyway, and "non-harvest" deaths that would have occurred significantly later in life.

Defining Acute and Chronic Deaths

The epidemiological evidence suggests that there are at least two and most likely three types of deaths that we need to distinguish when we value mortality: "acute harvest" deaths, "acute non-harvest" deaths, and "chronic" deaths.

Acute deaths are those that occur only a relatively short time -- say, less than a week -- after exposure to particulate air pollution. There are two kinds of acute deaths due to pollution: *harvest deaths* and *non-harvest deaths*. Acute harvest deaths are those that would have occurred in a few days anyway even if there had been no particulate pollution. For example, if particulate air pollution hastens by a few days or weeks the death of an already moribund person, the death is called "acute harvest". An acute non-harvest death is one that would not have occurred

³³It also is possible, but in our view unlikely, that the difference is due to the baseline incidence rate (C in the calculation above) being different from 8.498%. If the relative risk was exactly 1.17, then Abbey et al.'s (1995) published coefficient of $1.699 \cdot 10^{-4}$ implies a baseline incidence rate of 7.0%. However, the 8.498% that we assume is equal to the number of new cases divided by the sample size, which is the definition of the incidence rate in the Abbey et al. study

soon had there been no particulate air pollution. If particulate air pollution kills a seriously ill person who otherwise would have recovered and lived a long time afterwards, the death is called "acute non-harvest". We distinguish between harvest and non-harvest deaths because the former cost only a few days or weeks of life, whereas the latter cost years, and we believe that the value of the death is related at least crudely to the number of days or years lost (Chapter 11.4).

Chronic deaths are those that occur many years after the precipitating exposure to particulate air pollution. If exposure to air pollution initiates a cancer or cardiopulmonary disease that years later is fatal, the resulting death is called a "chronic" death. We distinguish between acute and chronic because the latter occur much later than do the former and hence have a lower present value, all else equal (Chapter 11.4).

It would be nice if the available epidemiological studies separately estimated acute harvest deaths, acute non-harvest deaths, and chronic deaths, but they do not -- quite. Instead, we have in general three different kinds of epidemiological studies: time-series studies, and prospective cohort and cross-sectional studies.³⁴ Time-series studies (e.g., Pope et al., 1992) link daily mortality with ambient particulate concentration. They estimate total acute deaths, both harvest and non-harvest, but do not necessarily distinguish between acute harvest and acute non-harvest deaths. They cannot capture chronic deaths³⁵. Prospective cohort studies (e.g., Pope et al., 1995) keep track of individuals over many years and link observed mortality with long-term average pollution levels. They are designed to control for important confounding factors such as smoking and are able to estimate acute non-harvest deaths and chronic deaths, but do not distinguish between the two³⁶. Cross-sectional studies (e.g., Ozkaynak and Thurston, 1987), also called "ecologic" regressions, link (retrospectively) annual average pollution level with annual mortality rates in metropolitan regions. Although these regressions are common, so too is the criticism that they do not adequately control for important confounding variables, such as smoking, except by using city-wide averages.³⁷ Like prospective cohort studies, the cross-sectional studies estimate acute non-harvest deaths and chronic deaths.

To estimate acute harvest deaths, we must deduct from total time-series deaths an estimate of the number of acute non-harvest deaths therein. To estimate chronic deaths, we must deduct from total prospective cohort deaths (or cross-

³⁴Lipfert and Wyzga (1995) give a useful summary of different types of mortality studies

³⁵Actually, time-series studies would capture chronic deaths if the day-by-day pollution pattern were the same every year, and if the time from exposure to long-term death was absolutely invariant. Of course, these conditions never obtain.

³⁶And as discussed below, it also is possible, albeit not necessarily likely, that some chronic deaths are not captured by either type of study.

³⁷Criticisms of cross-sectional studies are considered in Evans et al (1984b), Lipfert et al (1988) and Lipfert and Wyzga (1995), among others.

sectional deaths³⁸) an estimate of the number of acute non-harvest deaths therein. We must estimate acute non-harvest deaths as some fraction of total time-series or total prospective cohort deaths. Given this, our approach is:

- i) estimate the number of all acute deaths (time-series study);
- ii) estimate (on the basis of the review below) the fraction of total time-series acute deaths that are non-harvest deaths;
- iii) estimate the number of acute non-harvest deaths, and the number of acute harvest deaths (multiply time-series total of step i by fraction from step ii)
- iv) assume that the number of acute non-harvest deaths estimated in iii), from the time-series study, also is the number of acute non-harvest deaths captured by the prospective cohort study (or cross-sectional study);
- v) estimate chronic deaths, by subtracting from total estimated prospective cohort and cross-sectional deaths the estimated number of acute non-harvest deaths.

We emphasize two points here. First, to estimate acute non-harvest deaths we separate harvest from non-harvest deaths in the time-series study, rather than separate chronic from non-harvest in the prospective cohort (or cross-sectional) study, because more time-series studies provide evidence bearing on the separation. Second, we *assume* (step iv) that the number of acute non-harvest deaths in the cross-sectional studies is the same as the number in the time-series studies. *Statistically*, this need not be so, simply because the time-series studies estimate differences due to daily pollution levels, and the cross-sectional studies estimate differences due to annual pollution levels. (At the extreme, time-series studies will not capture any acute non-harvest deaths (or any deaths at all) in places with the same pollution level everyday throughout the year, and cross-sectional studies will not capture any acute non-harvest deaths (or any deaths at all) in places with the same annual mean pollution level.) We make this assumption only to keep our effort manageable.

Separating acute non-harvest deaths from acute harvest deaths and chronic deaths.

How can we separate acute non-harvest deaths from acute harvest deaths (in time-series studies) or from chronic deaths (in prospective cohort or cross-sectional studies)? As discussed above, the issue is central to our estimation of the cost of mortality. If all acute deaths are harvest deaths -- i.e, if in the time-series studies the daily pollution simply had hastened (or "harvested") deaths by a few days -- then the

³⁸To estimate non-harvest acute deaths and chronic deaths, we use a cross-sectional study (Ozkaynak and Thurston, 1987) in the lower bound and a prospective cohort study (Pope et al.) in the upper bound

cross-sectional studies estimate a completely different population of individuals, ones who had died from (chronic) long-term effects associated with air pollution.³⁹ In this case, we would multiply estimated time-series (acute-harvest) deaths by the cost of an acute death, multiply estimated cross-sectional (chronic) deaths by the present-value cost of a chronic death, and add the two together. On the other hand, if all acute deaths are non-harvest deaths, we would multiply estimated time-series deaths by the cost of an acute non-harvest death, subtract all time-series deaths from the total number of cross-sectional deaths in order to arrive at the number of chronic deaths, and multiply the resulting number of chronic deaths by the cost of a chronic death.

We have found three studies that bear (weakly) on the question of acute non-harvest versus acute harvest, and one that bears on the question of acute non-harvest versus chronic.

1). Spix et al. (1993) provide weak evidence that in the daily mortality studies, pollution is harvesting deaths. They estimated a pollution*prior-mortality interaction term with two different pollution variables to determine the amount of harvesting (i.e., hastening of death). After some search, they used the mortality level the previous two weeks in this interaction term and found a negative (but insignificant, $p=.5$ and $p=.1$ for SO_2 and suspended particulates) coefficient, which indicates that as mortality two weeks previously increases air pollution will have less of an effect. They found that the relative risk of air pollution rises 50% ($RR=1.08$ to 1.12 for SO_2 and $RR=1.14$ to 1.27) when going from high to low periods of previous mortality. If one interprets these results to mean that air pollution kills fewer people when there are fewer people near death, then one can infer that air pollution is killing those already near death. In their own assessment, Spix et al. (1993) wrote that "...suspended particulates do not cause deaths in the sense of acute toxicity, but they may in some cases lead to the extra stress that causes the death of a moribund person on a certain day, instead of slightly later [a harvest death], and in some cases lead to the death of a seriously ill person who would otherwise have recovered [an acute non-harvest death]" (p. 523; brackets added).

We note, though, that Spix et al.'s (1993) own assessment is merely a statement of the logical possibilities (time-series deaths either are acute harvest deaths or else acute non-harvest deaths), not a specific conclusion supported by their findings. If, as we said above, one believes their findings and interprets them to mean that air pollution kills fewer people when there are fewer people near death, then air pollution causes mainly or exclusively harvest deaths. To the extent that air pollution causes acute non-harvest rather than harvest deaths, one would not expect to find a negative interaction term. If one does not believe their findings at all, one cannot make any specific conclusion at all. The point is that this study either is weak evidence that in the daily mortality studies, pollution is harvesting deaths, otherwise it is silent on this issue.

³⁹If particulates hastened death *only* a few days, then virtually no effect would be found by the prospective cohort and cross-sectional studies

2). On the other hand, Utell and Frampton (1995) argue that the "harvesting effect...has not been evident in the epidemiology of particulate exposure" (p. 647).

3). Pope et al.'s (1992) study of particulate levels and mortality in the Utah Valley, where a steel mill is the dominant contributor to ambient particulate levels, also provides weak evidence that in the time-series studies, the deaths are non-harvest. On the basis of their estimated Poisson regression coefficient, they predicted a 2.3% drop in the mortality level when the steel mill shut down for a year. If time series studies are capturing a large proportion of deaths that are premature by only, say, a few days or weeks, then the actual drop in the annual mortality level should have been close to zero. In fact the actual drop was 3.2%⁴⁰. This suggests that in the daily-mortality (time-series) studies, the deaths are acute non-harvest deaths.

4). In a study of six U.S. cities, Dockery et al. (1993) reported a significant association between long-term pollution exposure and lung cancer, as did Pope et al. (1995) in a study of 151 metropolitan statistical areas. This finding indicates that the prospective cohort studies (and presumably cross-sectional studies) are capturing chronic deaths, not acute non-harvest deaths.

What can we make of this evidence? It appears to us that there is little ground for assuming that in the time-series studies, pollution *mainly* is harvesting deaths. Rather, it appears to us that harvested deaths are in the minority. Thus, we assume that acute harvest deaths are 25% (upper-bound cost case) to 50% (lower-bound cost case) of total time-series-estimated acute deaths. Acute non-harvest deaths therefore are 75% to 50%. We subtract these estimated acute non-harvest deaths from total cross-sectional deaths, to obtain chronic deaths.

It turns out that nationally, and in almost every county in the U.S., the estimated number of cross-sectional deaths exceeds the number of acute non-harvest deaths estimated with respect to the time-series results. However, in a few counties, the reverse is true, and the number of acute-non harvest deaths estimated with respect to the time-series results exceeds the total number of cross-sectional deaths and hence the number of acute non-harvest deaths included in the cross-sectional studies.⁴¹ In those few counties, we have two options as regards the number of acute non-harvest deaths: 1) assume that it is as estimated originally, with respect to the time-series results, or 2) assume that it is equal to the (lower) number of cross-sectional deaths. We have chosen the former: we always estimate the number of acute non-harvest deaths with respect to the time-series results, even if the result is more than the total number of cross-sectional deaths.

⁴⁰It is not clear why the actual was greater than the predicted drop. Perhaps unusual weather or infectious disease contributed to some of the difference.

⁴¹It is possible for acute non-harvest deaths estimated from the time-series studies to exceed cross-sectional deaths in one county, but for the reverse to be true in another county because the time-series equations -- used to estimate acute non-harvest deaths -- do not have the same structure as the lower-bound cross-sectional equation. The time-series equations estimate deaths as a nonlinear function of PM₁₀ and the background death rate, whereas the lower-bound cross-sectional equation estimates death as a linear function of PM_{2.5} and the total population.

Nonlinear Effect of Particulates on Mortality

It is possible that sudden jumps in the particulate level cause deaths directly, independent of the average annual pollution level. Cross-sectional studies, which relate differences in death rates to differences in average annual particulate levels, do not capture this possible effect of jumps, because by hypothesis the direct deaths due to jumps are independent of the average annual particulate level (i.e., are in addition to any deaths associated with the rise in the average annual particulate level caused by the jumps). Time-series studies capture any immediate deaths due to jumps, but of course do not capture any long-term deaths due to jumps. (A death due to a cancer initiated by a sudden rise in the particulate level might be an example of a long-term death due to a jump.) Thus, it is at least theoretically possible that not only might there be little overlap between, say, prospective cohort and time-series studies, there might even be a type of PM-related death not captured by either: long-term deaths due to jumps. However, we have not found any evidence in support of this hypothesis, and so have not accommodated it formally.

Beyond this, it is not clear if in general the relationship between particulate air pollution and mortality is essentially linear or significantly nonlinear. Samet et al. (1995), in a re-analysis of Schwartz and Dockery's (1992a) study of daily mortality in Philadelphia, find statistical indications that "strongly support the hypothesis that the effects of SO₂ and TSP are nonlinear" (p. 31). Lipfert and Wyzga (1995) also suggest that the best functional forms might be nonlinear. However, Pope et al. (1995b) conclude from their review of the epidemiological literature that "health effects increase monotonically with pollution levels, often with a near linear dose-response relationship" (p. 14).

We use non-linear functions to estimate short-term or "acute" deaths from particulate air pollution, and a linear function to estimate long-term or "chronic" deaths from air pollution.

11.3.5.5 Particulates and acute mortality

A number of time-series studies have been performed which, when taken together, present a coherent picture that particulates increase the daily mortality rate. Bates (1992) reported on a series of particularly persuasive studies in Utah:

The recent analyses of the respiratory consequences of air pollution in Utah are unique. Air monitoring has shown that in this location, oscillations occur in PM₁₀ levels without any significant SO₂, NO₂, or acid aerosol H₂SO₄ accompaniments. About 40% of the particles originate from a single source. An initial report related hospital admissions for respiratory disease to the PM₁₀ levels (Pope, 1989), this was confirmed in a later analysis (Pope, 1991). A third report indicated an excess of respiratory mortality in the impacted region (Archer, 1990), and a fourth (Pope, 1991) has shown that PM₁₀ levels are associated with reduction in PEF_R [i.e., lung functioning] in normal children and increased symptoms and medication use in a panel of adult asthmatics in the area of concern. Thus in one location, without exposure to multiple pollutants, four different adverse health indices have been found to be associated with variations in the PM₁₀ levels below the present US standard: respiratory mortality, increased respiratory hospital admissions, reduced lung function, and increased symptoms and medication use. This is a unique example therefore of remarkable coherence and is particularly valuable as multiple pollutants were not present (p. 340).

The weight of the evidence, then, clearly indicates that particulate pollution causes mortality. Schwartz (1994a) sums up the case:

The associations of particulates with daily mortality have now been reported at lower concentrations in locations with humid climates and air pollution peaking in cold weather. in locations with semiarid climates and air pollution peaking in cold weather. and in locations with humid climates and air pollution peaking in warm weather, suggesting that the possibility of confounding by weather and seasonal factors is unlikely. Furthermore, the relative risks reported in the studies were quite similar, which also makes confounding less likely (p 34)

The epidemiologic studies of particulate air pollution and daily mortality show consistency of finding, evidence of dose-dependent increases in risk, similarity in slopes, and exposure preceding effect. These findings also are coherent with studies relating airborne particles to symptoms and pulmonary function and hospital attendance. Overall, this pattern meets the generally accepted criteria for drawing causal inference in epidemiology. In light of such a pattern, it would seem that the burden of proof has now shifted, and it is incumbent upon those who disbelieve the causality of the reported associations to provide evidence showing that they are confounded. In the absence of such evidence, it is only prudent to treat these associations as causal (pp 34-35)

Although some investigators have questioned the statistical association between daily mortality and particulate air pollution (Li and Roth, 1995; Moolgavkar et al., 1995), a recent independent and sophisticated re-analysis of six of the more prominent studies, including those questioned by Li and Roth (1995) and Moolgavkar et al. (1995), validated the original results. The independent re-analysis concluded that "the original findings cannot be explained by an arbitrary choice of statistical model, but appear to be robust to a variety of modeling procedures" (*Particulate Air Pollution and Daily Mortality*, 1995).

Two well-run studies allow us to quantify mortality. We use the results from Pope et al.'s (1992) study, however, rather than Dockery et al.'s (1992) because it allows us to separately estimate total, cardiovascular, respiratory, and all other deaths. For simplicity in our results we sum the estimated cardiovascular and respiratory deaths.

Using Poisson regression, Pope et al. (1992) found a significant association between the five-day moving average of PM₁₀ and cardiovascular and respiratory deaths and no effect for PM₁₀ on the incidence of "all other deaths." In both the lower and upper bound scenarios, we use the coefficients for cardiovascular and respiratory deaths. We assume that there is no effects threshold, and estimate the effects down to particulates' estimated natural background level.

Estimating Acute cardiovascular Deaths Caused By Particulates

As discussed earlier, we distinguish between "acute" mortality and "chronic" mortality. Acute mortality refers to deaths linked to current pollution levels. Chronic mortality refers to deaths caused by air pollution but not linked to current air pollution levels. In the case of particulate air pollution, we will distinguish between acute cardiovascular deaths and acute respiratory deaths because we have separate equations for them.

We estimate the number of acute cardiovascular deaths caused by PM₁₀ with the following equation (derived in Section 11.2.4.1):

$$\Delta \text{Cardiovascular Deaths} = \text{Cardiovascular Deaths}_0 \cdot \left[e^{(PP-PI) \beta_{pm}} - 1 \right]$$

where:

Δ Cardiovascular Deaths = change in the daily number of cardiovascular deaths

Cardiovascular Deaths₀ = county daily average number of cardiovascular deaths (U.S. Department of Health and Human Services, 1991)

PI = daily average PM₁₀ level (µg/m³), after control⁴²

PP = daily average PM₁₀ level (µg/m³), before control

B_{pm} = 0.00179, PM₁₀ Poisson regression coefficient (Pope et al., 1992: 214).

Estimating Acute respiratory Deaths Caused By Particulates

We estimate the number of acute respiratory deaths caused by PM₁₀ with the following equation:

$$\Delta \text{Respiratory Deaths} = \text{Respiratory Deaths}_0 \left[e^{(PP-PI) \beta_{pm}} - 1 \right]$$

where:

Δ Respiratory Deaths = change in the daily number of respiratory deaths

Respiratory Deaths₀ = county daily average number of respiratory deaths (U.S. Department of Health and Human Services, 1991)

PP = daily average PM₁₀ level (µg/m³), after control

PI = daily average PM₁₀ level (µg/m³), before control

B_{pm} = 0.00361, PM₁₀ Poisson regression coefficient (Pope et al., 1992: 214).

11.3.5.6 *Chronic Mortality*

Air pollution might have a gradual or cumulative mortal effect, as well as an immediate mortal effect. We term the gradual, cumulative effect “chronic mortality.”

The evidence indicates that air pollution causes chronic mortality. Detels et al. (1991), Jedrychowski and Krzyzanowski (1989), and van der Lende et al. (1980) found that long-term exposure to air pollution causes a significant decline in lung functioning, which is a significant predictor of chronic disease and early death (Anderson et al., 1988; Krzyzanowski and Wysocki, 1986; Cullen, 1983; Beaty et al., 1982; Higgins and Keller, 1970). Archer (1990) found higher mortality from non-

⁴²When estimating cardiovascular deaths and respiratory deaths, we use the daily average PM₁₀ level (rather than the five-day moving average used by Pope et al (1992)), because our procedure for filling in missing observations (Section 11.2.2) does not account for the exact day of the missing observation

malignant respiratory disease in the more polluted of two closely paired counties in the Utah Valley.

To estimate the upper bound of chronic mortality, we use the prospective cohort study of Pope et al. (1995a), which is perhaps the most comprehensive study to date. Using a proportional hazards model, Pope et al. (1995a) find that the number of annual cardiopulmonary deaths rises by 31% (i.e., the hazard ratio, or relative risk, = 1.31) when the annual average PM_{2.5} pollution level increases from 9.0 to 33.5 $\mu\text{g}/\text{m}^3$. We can use Pope et al.'s relative risk to estimate the change in cardiopulmonary deaths as a function of a change in pollution.

The proportional hazards model assumes that the ratio of the pollution hazard, or risk, at pollution level PI to the risk at pollution level PP, is a nonlinear function of the difference between PI and PP.

$$RR \equiv \frac{h(t, PI, x_i)}{h(t, PP, x_i)} = e^{B' (PI-PP)}$$

where:

RR = relative risk (the hazard ratio)

$h(t, P, x_i)$ = the pollution risk as a function of time, the pollution level, and the other covariates x_i

t = time

x_i = the other covariates that affect the probability of dying

PI = the initial pollution level; in our analysis, the initial annual average PM_{2.5} level ($\mu\text{g}/\text{m}^3$) (data from ambient air-quality monitors; see discussion in Chapter 11.2)

PP = the final pollution level (after the reduction in emissions); in our analysis, the annual average PM_{2.5} level ($\mu\text{g}/\text{m}^3$), after the reduction in emissions (see discussion in Report #16 in the social-cost series listed at the beginning of this report)

B' = the vector of coefficients of from the proportional hazards model (on the assumption that the covariates other than pollution remain the same, this vector becomes the coefficient on pollution, B_{pm})

The pollution risk at a given pollution level, $h(t, P, x_i)$, is defined as the ratio of the probability of dying at time t divided by the cumulative probability of having died by time t:

$$h(t, P, x_i) \equiv \frac{f(t, P, x_i)}{F(t, P, x_i)}$$

$$F(t, P, x_i) = \int_0^t f(t, P, x_i) dt$$

where:

$f(t, P, x_i)$ = the probability density (the probability of dying, as a function of the time t , the pollution level P , and the other covariates x_i)

$F(t, P, x_i)$ = the cumulative density function (the probability of *having* died, as a function of the time t , the pollution level P , and the other covariates x_i)

The function $h(t, P, x_i)$ thus expresses the death rate. However, to estimate this death rate, one need not actually specify the probability functions; instead, one can estimate the death rate as the ratio of the number of deaths in year to the total number of people dead or alive in the year. Thus, one can calculate the relative risk -- the ratio of the death rate at pollution level PI to the death rate at pollution level PP -- as:

$$\text{Relative Risk (RR)} = \frac{\frac{A}{A+B}}{\frac{C}{C+D}}$$

$$\text{Risk of death at PP} = \frac{A}{A+B}$$

$$\text{Risk of death at PI} = \frac{C}{C+D}$$

where A, B, C, D, are the percentage of people who at the end of the year are:

	Dead	Alive
at PI	A	B
at PP	C	D

Pope et al. (1995) use this formula for the relative risk to estimate that the risk of death at $33.5 \mu\text{g}/\text{m}^3$ (PP) is 1.31 times the risk at $9 \mu\text{g}/\text{m}^3$ (PI). With this, we can estimate the coefficient B_{pm} in the proportional hazards model:

$$RR \equiv \frac{h(t, 33.5, x_i)}{h(t, 9, x_i)} = e^{B_{pm} (33.5-9)} = 1.31$$

$$B_{pm} = \frac{\ln(1.31)}{24.5} = 0.011022$$

Finally, we can derive the equation to estimate the change in cardiopulmonary deaths (Δ CPD), which is our ultimate objective:

$$\Delta CPD = CPD_{PI} - CPD_{PP}$$

$$CPD_{PI} = RR \cdot CPD_{PP}$$

$$\Rightarrow \Delta CPD = CPD_{PI} - \frac{CPD_{PI}}{RR}$$

$$\Rightarrow \Delta CPD = CPD_{PI} \left(1 - e^{-B_{pm}(PI-PP)} \right)$$

where:

Δ CPD = the change in cardiopulmonary deaths

CPD_{PP} = total cardiopulmonary deaths at pollution level PP

CPD_{PI} = total cardiopulmonary deaths at pollution level PI = county's annual number of cardiovascular and respiratory deaths (ICD-9 401-440 and 460-519) (U.S. Department of Health and Human Services, 1991).

B_{pm}, PI, and PP are as defined or derived above

We will use this equation to estimate deaths in our upper-bound case⁴³.

Example calculation To estimate the change in deaths due to, say, a decrease from 20 to 9 $\mu\text{g}/\text{m}^3$ of PM_{2.5}:

$$\Delta CPD = CPD_{20} \left(1 - e^{-0.011022(20-9)} \right) = 0.114 \times CPD_{20}$$

where:

CPD₂₀ = cardio-pulmonary deaths at 20 $\mu\text{g}/\text{m}^3$.

Pope et al. (1995a) estimated the relative risk of 1.31 from a Cox proportional hazard model. They also tried a simple regression model, and estimated an increase of 8.0 deaths per 100,000 people per year per $\mu\text{g}/\text{m}^3$ increase of PM_{2.5}. We have used their Cox hazard model rather than their regression model because they controlled for more potentially confounding variables in the former.

In our lower bound, we estimate the change in deaths with an equation from Ozkaynak and Thurston (1987):

⁴³Pope et al (1995a) also tried a simple regression model, and estimated an increase of 8.0 deaths per 100,000 people per year per $\mu\text{g}/\text{m}^3$ increase of PM_{2.5}. We have used their Cox hazard model rather than their regression model because they controlled for more potentially confounding variables in the former

$$\Delta \text{Deaths} = B_{pm} (\text{Annual Avg PM}_{2.5} - \text{Background}) \text{Population} / 100,000$$

where.

B_{pm} = 2.2 deaths per 100,000 people per year per $\mu\text{g}/\text{m}^3$

Annual Avg $\text{PM}_{2.5}$ = annual average $\text{PM}_{2.5}$ level ($\mu\text{g}/\text{m}^3$), before control

Background = annual average $\text{PM}_{2.5}$ level ($\mu\text{g}/\text{m}^3$), after control

Population = county population of all individuals, of all ages (U.S. Bureau of the Census, 1994).

It turns out that the upper bound equation, from Pope et al. (1995a), estimates two times as many deaths as does the lower-bound equation at any given pollution level (see Chapter 11.7), and more deaths than does any other equation that we found. Consequently, one reasonably might question whether we should use it, even as an upper bound. We are uneasy with the both the magnitude of the damages that result from the use of the Pope et al. equation, and the sensitivity of the results to this magnitude. However, for three reasons, we are not so uneasy as to ignore the Pope et al study altogether. First, the Pope et al. study is comprehensive and well done. Second, previous work by Pope et al. recently has been validated (Samet et al., 1995). Third, Pope et al's regression model, which we do not use, actually estimates still more deaths than does their Cox proportional-hazard model, which we do use. As mentioned above, the Pope et al. regression model estimates 8.0 deaths per 100,000 people per year per $\mu\text{g}/\text{m}^3$ increase of $\text{PM}_{2.5}$, which is 3.6 times higher than the lower-bound coefficient of 2.2 from Ozkaynak and Thurston whereas the Cox proportional-hazard model estimates two times more deaths than does the Ozkaynak and Thurston equation.

11.3.6 Sulfur Dioxide

SO_2 by itself appears to increase the breathing difficulty of asthmatics and may be linked to mortality (Tables 11.3-24 to 11.3-27; California Air Resources Board, 1991b). SO_2 has an indirect effect on morbidity and mortality through the formation of sulfate particulates. In this section, we concern ourselves with the direct effect of SO_2 .

11.3.6.1 Morbidity

Two laboratory studies found that SO_2 increases bronchoconstriction in asthmatics down to 0.10 ppm (Sheppard et al., 1980; Sheppard et al., 1981). An epidemiological study by Schwartz (1992) found SO_2 linked to chest discomfort primarily in asthmatics.

It is not clear if SO_2 affects lung functioning in the general population. The epidemiological studies in Table 11.3-24 suggest that SO_2 does not have an effect on lung functioning in the general population; however, Xu et al. (1991) found SO_2 (considered separately from TSP) to be linked to reduced pulmonary functioning in Beijing, China residents. This suggests that high levels of SO_2 (found in Beijing) have a measurable impact on lung functioning.

Two epidemiological studies (Charpin, 1988; Schwartz et al., 1991) found that SO₂ increases various respiratory symptoms in children, but did not provide enough information for us to make a dose-response function. Most of the studies that we reviewed (Table 11.3-25) did not find a link between SO₂ and respiratory health. Also, there is no link to chronic illness.

A number of studies have examined the link between SO₂ and hospital admissions, with most finding no effect (Table 11.3-26). Three studies found a link:

- Fishelson and Graves (1978: 788-89) measured both SO₂ and coefficient of haze (a measure of particulates) in a study of admissions to a hospital emergency room in Chicago due to SO₂, finding a significant effect for SO₂ and none for particulates. Other researchers disagree. Mazumdar and Sussman (1983) performed an identical study in Pittsburgh and found the opposite result: a positive link between COH and hospital admissions and none between SO₂ and hospital admissions.
- Bates et al. (1990) studied the relationship between temperature and pollution and admissions to the emergency room in hospitals in Vancouver over a three period (1984-1986), finding a significant correlation between SO₂ and respiratory-related hospital admissions -- in some cases independent of other pollutants (coefficient of haze, NO₂, ozone and SO₄). Bates et al. (1990) presented only the correlation coefficients between hospital admissions and SO₂ which by themselves are not useful for a dose-response function because they give the direction of the effect but not the magnitude.
- Lipfert and Hammerstrom (1992) reported SO₂ linked to hospital admissions in southern Ontario, controlling for other pollutants. Because they did not publish the regression coefficients that they estimated, we cannot develop a dose-response function.

Most studies that control for the effects of other pollutants reject the linkage between SO₂ and hospital admissions. (Lipfert and Hammerstrom [1992] is the exception.) Richards et al. (1981), Goldstein and Weinstein (1986), Cody et al. (1992), Tseng et al. (1992), and Schwartz et al. (1993) all failed to find a link. Schwartz et al. (1991) found a significant effect for SO₂, but after controlling for TSP and NO₂, the effect disappeared. (And we discard the group of studies that found a significant effect for SO₂ but did not control for other pollutants [Samet et al., 1981; Bates and Sizto, 1983; Díaz-Caneja et al., 1991; Ponka 1991; and Sunyer et al., 1991].)

Morbidity and SO₂: Conclusion

SO₂ negatively affects asthmatics and may affect the general population. However, the evidence for an effect on the general population is mixed at best (most studies that controlled for other pollutants did not find an effect), and what positive evidence there is cannot be used to develop dose-response functions. We conclude that the current evidence does not support a quantifiable link between SO₂ and morbidity.

11.3.6.2 Mortality

Most of the studies that we reviewed (Table 11.3-27) found no link between SO₂ and mortality. However, two studies by Xu et al. (1994) and Moolgavkar et al. (1995) reported that SO₂ has a strong effect, independent of TSP levels. Examining mortality rates in Philadelphia, Moolgavkar et al. found a significant relationship between SO₂ levels and mortality in the fall, winter and spring, controlling for TSP and ozone. Xu et al. reported on mortality rates in Beijing, China, where pollution rates are quite high because of the extensive burning of coal. The results are particularly significant since SO₂ had a significant effect, both modeled separately and with TSP. TSP had a significant effect in the summer but not in the winter. Xu et al. (1994: 222) suggested that the dry north winds in the winter carrying natural soil dust may be less dangerous than anthropogenic pollution; soil is coarse and not readily inhalable and is more inert than, say, particulates from coal combustion. Since the winds contribute 40-60% of ambient TSP it would not be surprising that the TSP coefficient is insignificant. Future work should use PM₁₀ measurements rather than TSP, since PM₁₀ is more widely dispersed (and thus a better indicator of exposure) and is inhalable.

The work of Xu et al. (1994) and Moolgavkar suggests a link between SO₂ and mortality; however their conclusion is weakened because they used TSP rather than an inhalable particulate measure. Furthermore the bulk of the evidence we reviewed suggests that SO₂ has only an indirect effect through its effect on particulate formation. Researchers have found a link between particulates and mortality in places with very low SO₂ levels (Pope et al., 1992; Fairley, 1990), but have not found a link between SO₂ and mortality in places with very low particulate levels. We conclude that the current evidence does not allow us to quantify an direct link between SO₂ and mortality independent of the link between particulates and mortality.

ABBREVIATIONS USED IN CHAPTER 11.3 TABLES

Pollutants

British Smoke = particulate measure used in the United Kingdom
CO = carbon monoxide
COH = coefficient of haze (general particulate measure)
H⁺ = hydrogen ion (measure of acidity)
HC = hydrocarbons
KM = measure of particulates (similar to the coefficient of haze)
NO = nitrogen oxide
NO₂ = nitrogen dioxide
NO₃ = particulate nitrate
NO_x = nitrogen oxides (including but not limited to NO₂)
NH₃ = ammonia
NH₄ = particulate ammonium
O₃ = ozone
O_x = oxidants,
PM = particulate matter
PM₁₀ = particulate matter with a diameter of 10 microns or less
PM₁₅ = particulate matter < 15 micrometers in diameter
PM_{2.5} = particulate matter with a diameter of 2.5 microns or less
Coarse PM₁₀ = particulate matter with a diameter between 2.5 and 10 microns
SO₂ = sulfur dioxide
SO₄ = particulate sulfate
SO_x = sulfur oxides
SOA = secondary organic aerosols
SPM = suspended particulate matter
TSP = total suspended particulates
VOCs = volatile organic compounds

Measures of pulmonary functioning

FEV₁ = forced expiratory volume in one second
FVC = forced vital capacity
MMEF = maximal midexpiratory flow
PEF = peak expiratory flow
RRAD = respiratory-related restricted activity day
Some measures of pulmonary functioning are defined in Table 3-1

TABLE 11.3-1. DEFINITIONS OF MORBIDITY AND PULMONARY FUNCTIONING MEASURES

Health Effect ^a	Definition
AOD	Airway obstructive disease This includes chronic bronchitis, emphysema and asthma Other names for this are COPD (chronic obstructive pulmonary disease) and CORD (chronic obstructive respiratory disease)
ARD2	Any respiratory symptom, headache, or eye irritation This symptom group was used by Krupnick et al (1990) to determine the effects of air pollution in Los Angeles
RAD	Restricted activity day This is a day during which a respondent is forced to alter his or her normal activity The RAD includes days of work loss or bed disability as well as more minor restrictions, due to both respiratory and nonrespiratory conditions
mRAD	Minor restricted activity day An mRAD is a restricted activity day that does not result in either work loss or bed disability and therefore involves more minor conditions and reductions in activity Both respiratory and nonrespiratory conditions are included in mRAD "
RRAD	Respiratory-related restricted activity day A respiratory-related restricted activity day is a combined measure of respiratory ailments that includes the common cold, upper respiratory infections, influenza, acute bronchitis, pneumonia, and other respiratory conditions, including asthma attacks It includes three types of conditions 1) bed disability days, 2) work or school loss days and 3) minor restricted activity days
mRRAD	Minor respiratory-related restricted activity day Involves more minor conditions and reductions in activity than does an RRAD.
Pulmonary Functioning ^b	Definition
FVC	Forced vital capacity The volume of air that can be expelled from fully inflated lungs by unassisted maximal subject effort
FEV ₁	Forced expiratory volume in one second The volume of air expelled from the lungs during the first second of an FVC maneuver
FEF ₂₅₋₇₅	Flow rate between 75 and 25% of vital capacity during the FVC maneuver, also known as maximal mid-expiratory flow rate
PEFR	Peak expiratory flow rate during an FVC maneuver

^aSource National Center for Health Statistics (1992), Krupnick et al (1990), Ostro and Rothschild (1989), Ostro (1987)

^bSource. Lippmann (1989c)

TABLE 11.3-2. NUMBER OF RESPIRATORY-RELATED AND OTHER RESTRICTED ACTIVITY DAYS BY AGE IN 1991 (100 PERSONS/YEAR)

Respiratory Conditions^a	All Ages	<5	5-17	18-24	25-44	45-64	>65
All Restricted Activity Days	733.3	966.1	720.5	712.4	682.9	643.1	877.4
All Respiratory Conditions	341.6	526.3	407.2	297.7	285.8	309.4	358.7
Common Cold	70.8	150.7	77.5	82.0	54.5	51.9	73.4
Other Acute Upper Resp Infections	31.5	50.8	49.4	18.2	28.0	23.9	23.7
Influenza	184.5	236.9	235.2	169.1	166.2	181.6	139.3
Acute Bronchitis	22.5	51.4	24.0	15.7	17.0	25.1	18.1
Pneumonia	23.0	24.1	10.8	10.6	15.3	18.9	78.5
Other Respiratory Conditions	9.3	12.5	10.3	2.2	4.8	8.1	25.6
Eye Conditions	1.8	4.6	0.3	4.5	0.1	1.7	4.9
Headache, Excluding Migraine	5.2	--	7.6	4.9	5.2	3.3	8.0

Source: National Center for Health Statistics (1992 Table 16)

^aSome of these terms are defined in Table 11.3-1.

TABLE 11.3-3. SUMMARY OF EPIDEMIOLOGICAL STUDIES CONCERNING THE HEALTH EFFECTS OF CARBON MONOXIDE

Study	Population and Study Period	Pollutants	Results
Perry et al. (1983)	24 asthmatics living in Denver Data collected from January 9, 1979 through March 28, 1979.	CO, NO ₃ , O ₃ , PM _{2.5} , PM ₁₅ , SO ₂ , SO ₄ and TSP considered independently	CO not linked to asthma Only NO ₃ significantly linked to asthma
Robertson and Lebowitz (1984)	3800 whites living in Tucson Data collected from August 8, 1974 to May 4, 1977	CO, NO ₂ , O ₃ and TSP considered jointly with stepwise regression	One pollutant at most after stepwise regression for a variety of symptoms, weather and pollen variables generally more important CO linked to cough and rhinitis
Lebowitz et al (1987)	204 people living in Tucson (45 asthmatics, 68 with airway obstructive disease, 62 with allergies and 29 individuals without a history of respiratory problems)	CO, NO ₂ , O ₃ and TSP considered jointly	No effect of pollutants on individuals without a history of respiratory problems CO linked to daily peak flow and rhinitis in asthmatics, and cough in people with airway obstructive disease
Schwartz and Zeger (1990)	Approximately 100 student nurses in Los Angeles Data collected November 1961 to May 1964	CO, NO ₂ , O _x and SO ₂ considered jointly.	Air pollutants linked to a variety of health effects CO linked to the incidence of headache

TABLE 11.3-4. SUMMARY OF HEALTH EFFECTS STUDIES EXAMINING EMERGENCY ROOM ADMISSIONS AND MORTALITY AND CARBON MONOXIDE

Study	Population and Study Period	Pollutants	Results
Hexter and Goldsmith (1971)	Mortality records for deaths from all causes in Los Angeles County from January 1, 1962 to December 31, 1965	CO and O _x considered independently	In a linear regression model, CO was found to be significantly linked to mortality. No effect found for O _x .
Kuller et al (1975)	Heart attack-caused mortality in adults, from 25-64 years of age, living in Baltimore, between June 1970 and June 1972	CO	Ambient CO not linked to mortality. Heart attack victims had higher carboxyhemoglobin levels, however, the authors concluded the difference was probably due to cigarette smoking.
Kurt et al (1978)	Cardio-respiratory-related emergency room admissions in Denver between November 1, 1975 and February 1, 1976	CO, NO _x , O ₃ , and sulfur compounds. The analysis focused on CO alone, as the other pollutants had low levels and were not highly correlated with CO.	CO correlated with emergency room admissions for cardiorespiratory complaints (i.e., shortness of breath, difficulty in breathing, nontraumatic pain in the chest, asthmatic attack and increased cough).
Mendelsohn and Orcutt (1979)	Cross-sectional study of 408 counties across the U.S. Demographic data (including mortality rate) are from 1970. Pollution data is from 1974.	CO, nitrates, NO ₂ , O ₃ , sulfates, SO ₂ and TSP considered jointly.	In estimating the annual effects of air pollution, significant effects were found for CO (7,000 to 35,000 deaths), SO ₂ (7,000 to 40,000 deaths), and sulfates (164,000 to 212,000 deaths). Significant negative effects were found for NO ₂ . No effect found for O ₃ , nitrates and TSP.
Samet et al (1981)	All ages in Steubenville, Ohio. Population = 31,000. Data collected for the months of March, April, October and November in 1974-1977.	CO, NO ₂ , O ₃ , SO ₂ and TSP considered independently in a regression analysis.	Small, but significant link found between SO ₂ and TSP and emergency room admissions for respiratory problems. No effect for CO and other pollutants.
Stern et al (1981)	1,558 white male motor vehicle examiners in New Jersey employed for a minimum of six months between 1944 and 1973.	CO measured although it was not used in the statistical analysis.	Motor vehicle examiners had significantly more cardiovascular deaths within 10 years after employment, compared to national rates. They also had higher cancer mortality found 30 years after employment.

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Study	Population and Study Period	Pollutants	Results
Edling and Axelson (1984)	Cohort of workers (clerks, bus drivers, and garage workers) in a bus company, from 1951-1978	No measured pollutants	Garage workers suffered more than the expected number of cardiovascular-related deaths. The authors suggest that the results are in accordance with the hypothesis that CO causes cardiovascular problems.
Shumway et al. (1988)	All nonaccidental mortality records from Los Angeles County, with subcategories for respiratory-related deaths and cardiovascular-related deaths. Data collected for 1970-1979.	CO, HC, KM, NO ₂ , O ₃ , SO ₂ considered independently, some pollutants (e.g., CO, KM and HC) were highly corrected ($r > 0.8$)	CO, KM and HC were found to be approximately equal predictors of mortality. No significant effect was found for NO ₂ , SO ₂ and O ₃ .
Stern et al. (1988)	5,529 New York City bridge and tunnel officers, employed at one of nine major water crossings, between January 1, 1952 and February 10, 1981.	CO	Tunnel officers suffered significantly more cardiovascular deaths than expected, based on New York City's population. The relatively more-exposed tunnel officers also had a higher risk than bridge officers. The risk for tunnel officers declined when switched to working at the less-exposed bridges.
Kinney and Ozkaynak (1991)	All nonaccidental mortality records from Los Angeles County, 1970-1979. The study also considered respiratory-related deaths and cardiovascular-related deaths.	CO, KM, NO ₂ , O ₃ and SO ₂ considered jointly	CO, KM and NO ₂ each significantly related to total mortality and cardiovascular-related mortality in models with O ₃ . It was not possible to determine which pollutant (besides O ₃) caused mortality.
Ponka (1991)	All ages living in Helsinki, Finland. Data collected between 1987-1989.	CO, NO, NO ₂ , O ₃ , SO ₂ and TSP considered independently and in a step-wise regression.	All of the pollutants were correlated with asthma-related hospital admissions. Only NO ₂ and O ₃ linked to admissions for children. Asthma-related emergency room admissions were more highly correlated with SO ₂ , TSP and O ₃ . All asthma-related hospital admissions (including emergency room) were more correlated with CO, NO and NO ₂ .

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Study	Population and Study Period	Pollutants	Results
Sunyer et al. (1991)	All ages in Barcelona, Spain Data collected from 1985-1986	British Smoke, CO, NO ₂ , O ₃ and SO ₂ considered independently	CO a significant predictor of emergency room admissions for COPD British Smoke and SO ₂ also linked to admissions Each pollutant was considered separately, so it is not possible to determine if CO would still be significant, controlling for other pollutants
Morris et al (1995)	Hospital admissions for congestive heart failure in persons over 65 in seven metropolitan areas of the US	CO, NO ₂ , O ₃ and SO ₂ considered jointly	CO significantly linked to hospital admissions for congestive heart failure in both single-pollutant and multi-pollutant models
Schwartz and Morris (1995)	Hospital admissions for ischemic heart disease, dysrhythmias, and congestive heart failure in persons over 65 in Detroit, Michigan	CO, O ₃ , PM ₁₀ and SO ₂ considered jointly	CO significantly linked to hospital admissions for congestive heart failure in both single pollutant and multi pollutant models No link to ischemic heart disease and dysrhythmias
Kinney et al (1995)	Daily counts of total deaths in Los Angeles County from January 1 1985 to December 31 1990, excluding accidents and suicides	PM ₁₀ , CO, and O ₃ considered independently, plus PM ₁₀ + O ₃ , and PM ₁₀ + CO	In the single-pollutant models, each of the three pollutants had relative risks just significantly different from 1.0 (p value about equal to 0.05) In PM ₁₀ + O ₃ model, PM ₁₀ risk was same as in PM ₁₀ -only model, but O ₃ risk was 1.0 (no risk) In PM ₁₀ + CO model, risk of both was less than in single-pollutant models

TABLE 11.3-5. SUMMARY OF EPIDEMIOLOGICAL STUDIES CONCERNING THE HEALTH EFFECTS OF NITROGEN DIOXIDE

Study	Population and Study Period	Pollutants	Results
Robertson and Lebowitz (1984)	3800 whites living in Tucson Data collected from August 8, 1974 to May 4, 1977	CO, NO ₂ , O ₃ and TSP considered jointly with stepwise regression	One pollutant at most included in the stepwise regression for a variety of symptoms, weather and pollen variables generally more important NO ₂ linked to cough
Harrington and Krupnick (1985)	2928 children living in Chattanooga, Tennessee Data collected between Spring 1972 to Spring 1973	NO ₂ , SO ₄ and TSP considered jointly in a pooled cross-sectional time series	A U-shaped relationship between acute respiratory disease and NO ₂ , no explanation was found for this result SO ₄ was positively linked to acute respiratory disease
Holguin et al (1985)	51 asthmatics (aged 7-55) living in Houston Data collected between May-October 1981	CO, NO ₂ , O ₃ , PM ₁₅ , SO ₂ and TSP CO and NO ₂ considered jointly	NO ₂ not significantly linked to asthma attacks, controlling for O ₃ The effects of the other pollutants were not considered in the published results
Lebowitz et al (1987)	204 people living in Tucson (45 asthmatics, 68 with airway obstructive disease, 62 with allergies and 29 individuals without a history of respiratory problems)	CO, NO ₂ , O ₃ and TSP considered jointly	No effect of pollutants on individuals without a history of respiratory problems NO ₂ linked to wheeze and cough in asthmatics, and wheeze and eye irritation in people with airway obstructive disease
Euler et al (1988)	7,445 Seventh-Day Adventists 25 years and older living in California. Data collected from 1966-1976	NO ₂ , O ₃ , SO ₂ , and TSP considered independently and in two- and three-pollutant models	NO ₂ not linked to chronic obstructive pulmonary disease (COPD) The authors concluded TSP is the best predictor of COPD
Dockery et al (1989)	Elementary school students, 10-12 years old, living in six cities in the US Data collected during the 1980-1981 school year	NO ₂ , O ₃ , PM _{2.5} , PM ₁₅ , SO ₂ , SO ₄ and TSP considered independently	NO ₂ not linked to bronchitis, chronic cough, chest illness, persistent wheeze and asthma O ₃ linked to asthma PM ₁₅ linked to bronchitis and chronic cough
Schwartz (1989)	National survey of people aged 6-24 years old, conducted between 1976-1980	Nitric acid, NO ₂ , O ₃ , SO ₂ , sulfuric acid and TSP considered independently	NO ₂ , O ₃ and TSP significantly linked to pulmonary function (measured by FEV ₁ , FVC and peak flow)

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Study	Population and Study Period	Pollutants	Results
Koo et al (1990)	362 primary school children and 319 mothers in Hong Kong Data collected in May 1985.	NO ₂	NO ₂ linked to chronic cough and rhinitis in non-smoking mothers No effect found for children The authors concluded, other (unmeasured) pollutants may be the cause of the reported effects
Krupnick et al (1990)	290 families living in Azusa, California Data collected from September 1978 to March 1979	COH, NO ₂ , O ₃ and SO ₂ considered jointly	NO ₂ had a significant, negative relationship with respiratory symptom in adults, and had an insignificant, negative effect on the incidence of respiratory symptoms in children COH was positively related to the incidence of respiratory symptoms in both adults and children O ₃ significantly related to effects only in adults
Schwartz and Zeger (1990)	Approximately 100 student nurses in Los Angeles Data collected November 1961 to May 1964	CO, NO ₂ , O _x and SO ₂ considered jointly	Air pollutants linked to a variety of health effects NO ₂ linked to the incidence of eye irritation, phlegm and sore throat
Schwartz et al (1991)	Young children in five German cities Data collected from January 1983 to August 1985 in Duisberg, from September 1984 to April 1987 in Koln, from January 1986 to December 1987 in Stuttgart, Tubingen and Freudenstadt	NO ₂ , SO ₂ and TSP considered jointly	NO ₂ , SO ₂ and TSP were significant predictors of croup-related visits to doctors and hospitals, in single pollutant models In two pollutant models, NO ₂ (t=1.06) and TSP (t=1.44) were both not significant NO ₂ (t=1.83) was almost significant with SO ₂ (t <1) TSP (t=2.80) was significant with SO ₂ (t <1)
Braun-Fahrlander et al (1992)	Preschool children living in four cities in Switzerland Volunteers recruited collected between November 1985 and December 1986 to fill out six-week symptom diaries	NO ₂ , O ₃ , SO ₂ and TSP considered jointly	NO ₂ associated with the duration of respiratory symptom episodes, but the authors concluded that this effect may be due to confounding with TSP TSP linked to both the incidence and duration of respiratory symptoms No effect found for SO ₂ or O ₃
Hasselblad et al (1992)	Meta-analysis (synthesis) of 12 health effects studies looking at the effects of NO ₂ on children under that age of 12	NO ₂	Individually, the studies often do not show NO ₂ having a significant effect Combined, the studies show that a 30 µg/m ³ increase in NO ₂ will increase respiratory illness by 20 percent

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Study	Population and Study Period	Pollutants	Results
Hoek et al (1993)	Children from four years of age to seven years of age living in three nonindustrial towns in the Netherlands. Data collected from March 1989 to July 1989	NO ₂ , O ₃ , PM ₁₀ and SO ₂ considered jointly	NO ₂ not significantly linked to pulmonary functioning after controlling for O ₃ . Daily O ₃ levels were negatively correlated with lung functioning (measured by FEV ₁ , FVC, MMEF and PEF). A small effect was found for PM ₁₀ , independent of O ₃ .
Ostro et al (1993)	321 adults (18 years and older) from Azusa, California. A subset of the data used by Krupnick et al (1990). Data collected from September 1978 to March 1979	COH, NO ₂ , O ₃ , SO ₂ and SO ₄ considered independently	NO ₂ not linked to lower respiratory symptoms, upper respiratory symptoms, or eye irritation. SO ₄ and O ₃ had significant effects.
Roemer et al (1993)	74 children with chronic respiratory problems from two, small industrial towns in the Netherlands. Data collected from December 17, 1990 to March 17, 1991	British Smoke, NO ₂ , PM ₁₀ and SO ₂ considered independently	NO ₂ not linked to any adverse health effects. SO ₂ , PM ₁₀ and British Smoke linked to asthma attacks, wheeze and runny nose.

TABLE 11.3-6. SUMMARY OF HEALTH EFFECTS STUDIES EXAMINING EMERGENCY ROOM ADMISSIONS AND MORTALITY AND NITROGEN DIOXIDE

Study	Population and Study Period	Pollutants	Results
Richards et al (1981)	Children in Los Angeles Data collected from August 1, 1979 to January 31, 1980	COH, HC, NO, NO ₂ , O ₃ , SO ₂ and SO ₄ considered independently	NO, NO ₂ , COH and HC positively linked to asthma-related emergency room admissions SO ₂ and O ₃ <u>negatively</u> linked to asthma-related emergency room visits
Samet et al (1981)	All ages in Steubenville, Ohio Population = 31,000 Data collected for the months of March, April, October and November in 1974-1977	CO, NO ₂ , O ₃ , SO ₂ and TSP considered independently in a regression analysis	NO ₂ not linked to emergency room admissions for respiratory problems Small, but significant effects were found for SO ₂ and TSP
Bates and Sizto (1983)	All ages in southern Ontario Data collected from the months of January, February, July and August 1974, 1976-1978	COH, NO ₂ , O ₃ and SO ₂ considered independently	NO ₂ <u>negatively</u> correlated with respiratory-related admissions to hospitals Significant positive correlations found for SO ₂ and O ₃
Shumway et al (1988)	All nonaccidental mortality records from Los Angeles County (1970-1979), with subcategories for respiratory-related deaths and cardiovascular-related deaths	CO, HC, KM, NO ₂ , O ₃ and SO ₂ considered independently, some pollutants (e.g., CO, KM and HC) were highly correlated ($r > 0.8$)	CO, KM and HC were found to be approximately equal predictors of mortality NO ₂ , SO ₂ and O ₃ not linked to mortality
Bates et al (1990)	All ages in Vancouver Data collected between July 1984 and October 1986	COH, NO ₂ , O ₃ , SO ₂ and SO ₄ considered independently	NO ₂ related to respiratory-related admissions for adults (aged 61 years and over) during November and April SO ₂ and SO ₄ also had a significant effect on this group of people
Kinney and Ozkaynak (1991)	All nonaccidental mortality records from Los Angeles County, 1970-1979 The study also considered respiratory-related deaths and cardiovascular-related deaths	CO, KM, NO ₂ , O ₃ and SO ₂ considered jointly	CO, KM and NO ₂ each significantly related to total mortality and cardiovascular-related mortality in models with O ₃ It was not possible to determine which pollutant (besides O ₃) caused mortality

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Study	Population and Study Period	Pollutants	Results
Ponka (1991)	All ages living in Helsinki, Finland Data collected between 1987-1989	CO, NO, NO ₂ , O ₃ , SO ₂ and TSP considered independently and in a step-wise regression	All of the pollutants were correlated with asthma-related hospital admissions Only NO ₂ and O ₃ linked to admissions for children Asthma-related emergency room admissions were more highly correlated with SO ₂ , TSP and O ₃ All asthma-related hospital admissions (including emergency room) were more correlated with CO, NO and NO ₂
Sunyer et al (1991)	All ages in Barcelona, Spain Data collected from 1985-1986	British Smoke, CO, NO ₂ , O ₃ and SO ₂ considered independently	NO ₂ not a significant predictor of emergency room admissions for COPD CO, British Smoke and SO ₂ linked to admissions
Dockery et al (1992)	All nonaccidental mortality records from the St Louis metropolitan statistical area and eastern Tennessee from September 1985 to August 1986	H ⁺ , NO ₂ , O ₃ , PM _{2.5} , PM ₁₀ , SO ₂ , SO ₄ jointly	NO ₂ not significantly linked to mortality A strong association was found between PM ₁₀ and mortality
Lipfert and Hammerstrom (1992)	Respiratory-related admissions to 79 acute-care hospitals in southern Ontario, between 1979 and 1985	COH, NO ₂ , O ₃ , SO ₂ , SO ₄ and TSP.	O ₃ , SO ₂ , SO ₄ and TSP were significantly associated with respiratory admissions No single pollutant was determined to be the cause There were no reported effects for COH and NO ₂
Dockery et al (1993)	Health data collected over 14-16 years (study ended in 1991) from 8,111 white persons in six cities in the U.S -- Portage, WI, Topeka, KS, Watertown, MA, Kingston, TN, St Louis, MO, and Steubenville, OH	NO ₂ , O ₃ , PM _{2.5} , PM ₁₀ , PM ₁₅ , SO ₂ , SO ₄ , TSP and aerosol acidity considered independently	Mortality more strongly linked to PM _{2.5} , PM ₁₀ , PM ₁₅ and SO ₄ than with NO ₂ , SO ₂ , TSP and aerosol acidity O ₃ levels varied little and was not significant Air pollution linked to lung cancer and cardiopulmonary disease-related deaths

TABLE 11.3-7. SUMMARY OF STUDIES CONCERNING ACUTE MORBIDITY AND OZONE

Study	Population and Study Period	Pollutants	Results
Whittemore and Korn (1980)	443 asthmatics living in southern California between 1972 and 1975	NO _x , O _x , RSP, SO _x and TSP All except O _x were highly collinear In the analysis, TSP was used as a "surrogate" for all of these pollutants	O _x and TSP were both significantly related to asthma attacks, in multiple logistic regressions
Perry et al (1983)	24 asthmatics living in Denver Data collected from January 9, 1979 through March 28, 1979	CO, NO ₃ , O ₃ , PM _{2.5} , PM ₁₅ , SO ₂ , SO ₄ and TSP considered independently	O ₃ not linked to asthma Only NO ₃ significantly linked to asthma
Robertson and Lebowitz (1984)	3800 whites living in Tucson Data collected from August 8, 1974 to May 4, 1977	CO, NO ₂ , O ₃ and TSP considered jointly with stepwise regression	One pollutant at most after stepwise regression for a variety of symptoms; weather and pollen variables generally more important No reported effect for O ₃
Holguin et al (1985)	51 asthmatics (aged 7-55) living in Houston Data collected between May-October 1981	CO, NO ₂ , O ₃ , PM ₁₅ , SO ₂ and TSP CO and NO ₂ considered jointly	O ₃ significantly linked to asthma attacks, controlling for NO ₂ The effects of the other pollutants were not shown in the published results
Portney and Mullahy (1986)	3,347 adults (aged 17 and older) from the 1979 national Health Interview Survey	O ₃ and SO ₄ considered independently and jointly.	The two-week average of the one-hour daily maximum of O ₃ linked to minor RRADs over this same two-week interval O ₃ not found linked to any acute illnesses in children No significant effects found for SO ₄
Lebowitz et al (1987)	204 people living in Tucson (45 asthmatics, 68 with airway obstructive disease, 62 with allergies and 29 individuals without a history of respiratory problems)	CO, NO ₂ , O ₃ and TSP considered jointly.	O ₃ linked to wheeze, cough and reduced pulmonary functioning in asthmatics No effect of pollutants on individuals without a history of respiratory problems
Dockery et al (1989)	Elementary school students, 10-12 years old, living in six cities in the U S Data collected during the 1980-1981 school year	NO ₂ , O ₃ , PM _{2.5} , PM ₁₅ , SO ₂ , SO ₄ and TSP considered independently	O ₃ significantly related to asthma rates Particulate measures (especially PM ₁₅ and TSP) were associated with increased bronchitis, chronic cough and chest illness TSP was consistently associated with reduced pulmonary functioning

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Study	Population and Study Period	Pollutants	Results
Ostro and Rothschild (1989)	Six groups of roughly 8,000 adults (aged 18-65) from the 1986-1981 national Health Interview Survey	O ₃ and PM _{2.5} considered jointly	O ₃ significantly linked to minor RRADs in three of the years (1976, 1979 and 1980), and negatively related in two of the years (1977 and 1981) PM _{2.5} significantly related to RRADs in most years
Schwartz (1989)	National survey of people aged 6-24 years old, conducted between 1976-1980	Nitric acid, NO ₂ , O ₃ , SO ₂ , sulfuric acid and TSP considered independently	O ₃ significantly linked to pulmonary function (measured by FEV ₁ , FVC and peak flow) Significant effects also found for TSP and NO ₂
Krupnick et al (1990)	290 families living in Azusa, California Data collected from September 1978 to March 1979	COH, NO ₂ , O ₃ and SO ₂ considered jointly	O ₃ significantly related to effects only in adults COH was positively related to the incidence of respiratory symptoms in both adults and children The pollutants were considered simultaneously Significant negative effects found for NO ₂ and SO ₂
Schwartz and Zeger (1990)	Approximately 100 student nurses in Los Angeles Data collected November 1961 to May 1964	CO, NO ₂ , O _x and SO ₂ considered jointly	Air pollutants linked to a variety of health effects O ₃ linked to the incidence of chest discomfort and eye irritation
Braun-Fahrlander et al (1992)	Preschool children living in four cities in Switzerland. Volunteers recruited collected between November 1985 and December 1986 to fill out six-week symptom diaries	NO ₂ , O ₃ , SO ₂ and TSP considered jointly	No effect found for O ₃ or SO ₂ on respiratory symptoms suffered by preschool children NO ₂ associated with the duration of respiratory symptom episodes, but the authors concluded that this effect may be due to confounding with TSP TSP linked to both the incidence and duration of respiratory symptoms
Castillejos et al (1992)	148 schoolchildren (aged 7-9 years old) in Mexico City Data collected between January 1 and June 30, 1988	O ₃	O ₃ linked to decreased lung functioning -- children with a history of chronic phlegm production are especially susceptible The results are somewhat weakened because no other pollutants were measured

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Study	Population and Study Period	Pollutants	Results
Schwartz (1992)	Approximately 100 student nurses in Los Angeles. Data collected November 1961 to May 1964	CO, NO ₂ , O _x and SO ₂ considered jointly	O _x increased the <u>duration</u> of cough, phlegm and sore throat SO ₂ increased the duration of chest discomfort, primarily in asthmatics.
Hoek et al (1993)	Children from four years of age to seven years of age living in three nonindustrial towns in the Netherlands Data collected from March 1989 to July 1989	NO ₂ , O ₃ , PM ₁₀ and SO ₂ considered jointly	Daily O ₃ levels were negatively correlated with lung functioning (measured by FEV ₁ , FVC, MMEF and PEF) A small effect was found for PM ₁₀ , independent of O ₃ NO ₂ not significantly linked to pulmonary functioning after controlling for O ₃
Ostro et al (1993)	321 adults (18 years and older) from Azusa, California A subset of the data used by Krupnick et al (1990) Data collected from September 1978 to March 1979	COH, NO ₂ , O ₃ , SO ₂ and SO ₄ considered independently	Controlling for temperature, O ₃ linked to lower respiratory symptoms (p=0.07), upper respiratory symptoms (p=0.06), and eye irritation (p<0.0001) SO ₄ also had a significant effect on lower respiratory illness The pollutants were considered separately

TABLE 11.3-8. SUMMARY OF STUDIES CONCERNING CHRONIC MORBIDITY AND OZONE

Study	Population and Study Period	Pollutants	Results
Euler et al (1988)	7,445 Seventh-Day Adventists 25 years and older living in California Data collected from 1966-1976	NO ₂ , O ₃ , SO ₂ , and TSP considered independently and in two- and three-pollutant models	O ₃ significantly linked to an increased risk of COPD In three pollutant models with TSP and SO ₂ , only TSP was significant
Portney and Mullahy (1990)	1,318 adults (over the age of 17) in a national survey in 1979	O ₃ and TSP Both the annual average pollution level and six-year average (1974-1979) were used in this analysis	Marginally significant relationship found between TSP and emphysema, chronic bronchitis and asthma The six-year O ₃ level was significantly related to the incidence of sinusitis and hay fever O ₃ not related to a combined measure of more severe chronic disease (emphysema, chronic bronchitis and asthma)
Abbey et al (1991b)	6,303 nonsmoking Seventh-Day Adventists living in California in 1977, and who had resided within 5 miles of their current residence for 10 years Health data collected between 1977 and 1987. (Mortality and cancer analysis used full observation set, 3,914 observations for the respiratory symptom analysis)	O ₃ and TSP considered independently and jointly The average pollution level from 1973-1977 was used to determine pollution exposure Estimated the number of hours of pollution exposure above four thresholds	O ₃ linked to asthma and respiratory cancer No link found between O ₃ and total mortality TSP significantly linked with all malignant neoplasms, airway obstructive disease and asthma The pollutants were considered separately
Abbey et al (1993)	3,914 nonsmoking Seventh-Day Adventists living in California in 1977, and who had resided within 5 miles of their current residence for 10 years Health data collected in 1977 and 1987	O ₃ , SO ₂ and TSP considered independently and jointly The average pollution level from 1973-1977 was used to determine pollution exposure in published results Used the average annual number of hours of pollution exposure above five thresholds	O ₃ linked to new cases of asthma and symptom severity of asthma When O ₃ modeled with TSP the results were unstable, Abbey et al concluded both O ₃ and TSP are significantly linked to new cases of asthma and symptom severity

TABLE 11.3-9. SUMMARY OF STUDIES CONCERNING HOSPITAL ADMISSIONS AND MORTALITY AND OZONE

Study	Population and Study Period	Pollutants	Results
Hexter and Goldsmith (1971)	Mortality records for deaths from all causes in Los Angeles County from January 1, 1962 to December 31, 1965	CO and O _x considered independently	In a linear regression model, CO was found to be significantly linked to mortality. No effect found for O _x .
Richards et al (1981)	Children in Los Angeles. Data collected from August 1, 1979 to January 31, 1980.	COH, HC, NO, NO ₂ , O ₃ , SO ₂ and SO ₄ considered independently	NO, NO ₂ , COH and HC positively linked to asthma-related emergency room admissions. SO ₂ and O ₃ <u>negatively</u> linked to asthma-related emergency room visits.
Samet et al (1981)	All ages in Steubenville, Ohio. Population = 31,000. Data collected for the months of March, April, October and November in 1974-1977.	CO, NO ₂ , O ₃ , SO ₂ and TSP considered independently in a regression analysis.	Small, but significant link found between SO ₂ and TSP and emergency room admissions for respiratory problems. No effect for CO and other pollutants.
Shumway et al (1988)	All nonaccidental mortality records from Los Angeles County, with subcategories for respiratory-related deaths and cardiovascular-related deaths. Data collected for 1970-1979.	CO, HC, KM, NO ₂ , O ₃ and SO ₂ considered independently, some pollutants (e.g., CO, KM and HC) were highly correlated ($r > 0.8$)	CO, KM and HC were found to be approximately equal predictors of mortality. NO ₂ , SO ₂ and O ₃ not linked to mortality.
Bates et al (1990)	All ages in Vancouver. Data collected between July 1984 and October 1986.	COH, NO ₂ , O ₃ , SO ₂ and SO ₄ considered independently.	O ₃ correlated with total emergency hospital admissions for all age groups (ages 1-4, 15-60, and 61+). No significant effect found for respiratory- and asthma-related emergency admissions. Significant effects found for SO ₂ and SO ₄ on respiratory- and asthma-related emergency admissions.

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Study	Population and Study Period	Pollutants	Results
Kinney and Ozkaynak (1991)	All nonaccidental mortality records from Los Angeles County, 1970-1979. The study also considered respiratory-related deaths and cardiovascular-related deaths.	CO, KM, NO ₂ , O ₃ and SO ₂ considered jointly.	CO, KM and NO ₂ each significantly related to total mortality and cardiovascular-related mortality in models with O ₃ . It was not possible to determine which pollutant (besides O ₃) caused mortality.
Ponka (1991)	All ages living in Helsinki, Finland. Data collected between 1987-1989.	CO, NO, NO ₂ , O ₃ , SO ₂ and TSP considered independently and in a step-wise regression.	All of the pollutants were correlated with asthma-related hospital admissions. Only NO ₂ and O ₃ linked to admissions for children. Asthma-related emergency room admissions were more highly correlated with SO ₂ , TSP and O ₃ . All asthma-related hospital admissions (including emergency room) were more correlated with CO, NO and NO ₂ .
Schwartz (1991)	All nonaccidental mortality records in Detroit, Michigan, from 1973 to 1982.	O ₃ , SO ₂ and TSP. TSP predicted from daily observations of the visibility extinction coefficient.	O ₃ not significantly linked to mortality, controlling for TSP. TSP strongly linked to mortality, the size of the coefficient was not significantly affected by the inclusion of SO ₂ . No link between SO ₂ and mortality.
Sunyer et al (1991)	All ages in Barcelona, Spain. Data collected from 1985-1986.	British Smoke, CO, NO ₂ , O ₃ and SO ₂ considered independently.	O ₃ negatively and significantly correlated with emergency room admissions for COPD. (This may have been caused by a high correlation between O ₃ and temperature.) CO, British Smoke and SO ₂ were linked to admissions.
Cody et al. (1992)	All ages living in central and northern New Jersey. Data collected from May through August in 1988 and 1989.	O ₃ , PM ₁₀ and SO ₂ and visibility (as a proxy for sulfate) considered independently.	O ₃ significantly associated with asthma-related emergency room admissions. PM ₁₀ and SO ₂ not significantly linked.

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Study	Population and Study Period	Pollutants	Results
Dockery et al. (1992)	All nonaccidental mortality records from the St. Louis metropolitan statistical area and eastern Tennessee from September 1985 to August 1986	H ⁺ , NO ₂ , O ₃ , PM _{2.5} , PM ₁₀ , SO ₂ , SO ₄ considered jointly	O ₃ not significantly linked to mortality A strong association was found between PM ₁₀ and mortality
Lipfert and Hammerstrom (1992)	Respiratory-related admissions to 79 acute-care hospitals in southern Ontario, between 1979 and 1985	COH, NO ₂ , O ₃ , SO ₂ , SO ₄ and TSP	O ₃ , SO ₂ , SO ₄ and TSP were significantly associated with respiratory admissions No single pollutant was determined to be the cause There were no reported effects for COH and NO ₂
Dockery et al (1993)	Health data collected over 14-16 years (study ended in 1991) from 8,111 white persons in six cities in the U.S -- Portage, WI, Topeka, KS, Watertown, MA, Kingston, TN, St Louis, MO, and Steubenville, OH	NO ₂ , O ₃ , PM _{2.5} , PM ₁₀ , PM ₁₅ , SO ₂ , SO ₄ , TSP and aerosol acidity considered independently	Mortality more strongly linked to PM _{2.5} , PM ₁₀ , PM ₁₅ and SO ₄ than with NO ₂ , SO ₂ , TSP and aerosol acidity. O ₃ levels varied little and was not significant Air pollution linked to lung cancer and cardiopulmonary disease-related deaths
Schwartz et al (1993)	People under 65 Data collected from hospitals in Seattle from September 1, 1989 to September 30, 1990	O ₃ , PM ₁₀ and SO ₂ considered independently	O ₃ and SO ₂ not linked to asthma-related emergency room admissions PM ₁₀ significantly linked to admissions
Kinney et al (1995)	Daily counts of total deaths in Los Angeles County from January 1 1985 to December 31 1990, excluding accidents and suicides	PM ₁₀ , CO, and O ₃ independently, plus PM ₁₀ + O ₃ , and PM ₁₀ + CO	In the single-pollutant models, each of the three pollutants had relative risks just significantly different from 1.0 (p value about equal to 0.05) In PM ₁₀ + O ₃ model, PM ₁₀ risk was same as in PM ₁₀ -only model, but O ₃ risk was 1.0 (no risk) In PM ₁₀ + CO model, risk of both was less than in single-pollutant models
Moolgavkar et al (1995)	Philadelphia mortality data collected for the period 1973-1988.	O ₃ , TSP and SO ₂ considered jointly	O ₃ linked to summer time mortality SO ₂ linked to mortality in the fall, winter and spring

TABLE 11.3-10. EPIDEMIOLOGICAL STUDIES USED TO ESTIMATE THE HEALTH EFFECTS OF OZONE IN THIS ANALYSIS

Health Effect	Studies Used To Estimate Health Effects
ARD2	Krupnick et al. (1990)
Asthma Attacks	Holgun et al (1985), Whittemore and Korn (1980)
Eye Irritation	Schwartz and Zeger (1990)
Upper and Lower Respiratory Illness	Ostro et al (1993)

TABLE 11.3-11. EPIDEMIOLOGICAL STUDIES USED IN PREVIOUS ANALYSES TO ESTIMATE THE COST OF THE HEALTH EFFECTS OF OZONE

Study Of Economic Value Of Health Effects	Health Effects Evaluated	Epidemiological Study Used To Estimate Health Effects
Hall et al., 1989 ^a	MRAD ^b	Portney and Mullahy (1986)
Harrison and Nichols, 1990 ^a	Asthma Attack	Holguin et al (1985)
	Cough, Eye Irritation	Schwartz et al (1988)
	ARD2	Krupnick et al (1990)
	MRAD ^c	Ostro and Rothschild (1989)
Krupnick and Kopp, 1988 ^a	ARD2	Krupnick et al. (1990)
	Asthma Attack	Holguin et al (1985), Whittemore and Korn (1980)
	Cough, Eye Irritation	Schwartz et al (1988)
	MRAD	Portney and Mullahy (1986)
RER (1990, Phase II)	Asthma Attack	Whittemore and Korn (1980)
	Cough, Eye Irritation	Schwartz et al (1988)
	ARD2	Krupnick et al (1990)
	MRAD	Portney and Mullahy (1986), Ostro and Rothschild (1989)
Rowe et al., 1986	Asthma Attack	Whittemore and Korn (1980)
	MRAD	Portney and Mullahy (1986)

MRAD = minor restricted activity day, ARD2 = any respiratory illness MRAD and ARD2 are defined in Table 3-1.

^aThis study also estimated symptom-days, using results from clinical studies

^bPortney and Mullahy (1986) actually measured RRAD's, although they felt (1986: 36) that in fact ozone only caused minor symptoms and not bed disability days or work loss days. In effect, they measured respiratory-related MRAD's. Similarly, Ostro and Rothschild (1989: 245) did not find an effect for ozone on RRAD's, although they did find an effect for MRAD's.

^cOstro and Rothschild (1989) estimated MRAD's that are related to both respiratory and nonrespiratory ailments

TABLE 11.3-12. REPORTED CHRONIC CONDITIONS PER 1,000 PERSONS, BY AGE (TOTAL U.S. 1988-1991)

Chronic Condition	1988	1989	1990	1991
Asthma				
ALL AGES	41.2	47.7	41.9	47.2
Under 18 Years	49.9	61.0	57.6	62.5
18-44 Years	38.7	41.3	35.2	43.4
45-64 Years	34.8	41.5	38.6	40.7
65-74 Years	43.6	57.3	32.5	38.0
Over 74 Years	38.0	42.3	42.4	35.9
Chronic Bronchitis				
ALL AGES	49.4	49.2	51.1	50.5
Under 18 Years	54.3	50.5	53.3	53.1
18-44 Years	39.0	44.5	41.5	46.7
45-64 Years	56.1	53.7	57.4	53.9
65-74 Years	65.6	54.2	76.3	56.2
Over 74 Years	63.5	57.6	61.4	46.7
Chronic Sinusitis				
ALL AGES	139.7	138.3	131.3	129.3
Under 18 Years	61.4	68.9	56.7	59.6
18-44 Years	157.5	161.1	149.0	151.0
45-64 Years	188.0	173.5	181.9	171.1
65-74 Years	176.2	151.8	154.1	156.4
Over 74 Years	167.8	155.8	148.1	113.7
Emphysema				
ALL AGES	7.9	8.2	8.2	6.6
Under 18 Years	*	0.2	*	*
18-44 Years	0.6	1.2	0.7	0.6
45-64 Years	16.8	17.2	12.8	12.8
65-74 Years	35.5	32.4	44.3	32.7
Over 74 Years	40.7	42.5	47.1	31.9
Heart Disease^a				
ALL AGES	84.1	75.9	78.5	82.6
Under 18 Years	23.3	17.1	18.9	18.5
18-44 Years	39.8	36.1	38.1	38.4
45-64 Years	135.9	118.9	118.7	134.1
65-74 Years	271.8	231.6	257.1	256.4
Over 74 Years	333.6	353.0	333.9	354.3

Source. National Center For Health Statistics' *Vital and Health Statistics Current Estimates From the National Health Interview Survey*, for the years 1988-1991.

^aHeart disease includes ischemic heart disease, heart rhythm disorders and other selected diseases of the heart, excluding hypertension

TABLE 11.3-13. SUMMARY OF HEALTH EFFECTS STUDIES EXAMINING PULMONARY FUNCTIONING AND PARTICULATES

Study	Population and Study Period	Pollutants and Correlations	Results
Dodge et al (1985)	678 children living in four towns in Arizona, two of the towns had copper smelters Data collected from 1979 to 1982	SO ₂ and SO ₄	No significant differences in lung functioning between children living in areas with different levels of SO ₂ and SO ₄
Dockery et al (1989)	Elementary school students, 10-12 years old, living in six cities in the U.S Data collected during the 1980-1981 school year	NO ₂ , O ₃ , PM _{2.5} , PM ₁₅ , SO ₂ , SO ₄ and TSP considered independently	NO ₂ not linked to bronchitis, chronic cough, chest illness, persistent wheeze and asthma O ₃ linked to asthma PM ₁₅ linked to bronchitis and chronic cough
Schwartz (1989)	National survey of people aged 6-24 years old, conducted between 1976-1980	Nitric acid, NO ₂ , O ₃ , SO ₂ , sulfuric acid and TSP considered independently	NO ₂ , O ₃ and TSP significantly linked to pulmonary function (measured by FEV ₁ , FVC and peak flow)
Chestnut et al (1991)	963 nonsmoking adults (aged 25-75) from the First National Health and Nutrition Examination Survey	TSP	TSP significantly related to reduced FEV ₁ and FVC A threshold for the effects of TSP found at 60 µg/m ³ .
Xu et al (1991)	1440 adults (40-69 years of age) living in three areas of Beijing, with different pollution levels Data collected in August 1986	SO ₂ and TSP	SO ₂ and TSP both linked to significantly reduced pulmonary functioning (measured by FEV ₁ and FVC) Independent effects for each pollutant were not determined
Hoek et al (1993)	Children from four years of age to seven years of age living in three nonindustrial towns in the Netherlands Data collected from March 1989 to July 1989	NO ₂ , O ₃ , PM ₁₀ and SO ₂ considered jointly	PM ₁₀ levels reduced lung functioning (measured by FEV ₁ , FVC, MMEF and PEF) They controlled for O ₃ , which also had a significant effect With O ₃ controlled statistically, NO ₂ and SO ₂ were not significantly linked to pulmonary functioning
Pope and Kanner (1993)	624 adult (aged 35-60) smokers, with mild to moderate chronic lung disease, living in Salt Lake City, Utah	PM ₁₀	PM ₁₀ significantly linked to lower pulmonary functioning, measured by FEV ₁ and FVC

TABLE 11.3-14. SUMMARY OF HEALTH EFFECTS STUDIES EXAMINING ACUTE RESPIRATORY SYMPTOMS (EXCLUDING HOSPITAL ADMISSIONS) AND PARTICULATES

Study	Population and Study Period	Pollutants	Results
Cohen et al (1972)	Seven months of daily symptom reports kept by 20 asthmatics living in West Virginia	NO ₃ , SO ₂ , SO ₄ , soiling index (alternative particulate measure) and TSP considered independently	Individually all of the pollutants were significant contributors to asthma attacks. No single pollutant was found to be the cause of asthma attacks.
Whittemore and Korn (1980)	443 asthmatics living in southern California between 1972 and 1975	NO _x , O _x , RSP, SO _x and TSP. All except O _x were highly collinear. In the analysis, TSP was used as a "surrogate" for all of these pollutants.	O _x and TSP were both significantly related to asthma attacks, in multiple logistic regressions.
Love et al (1981)	250 families from two polluted areas in New York City and one low pollution area in Long Island. Data during the 1971-1972 school year.	SO ₂ and TSP	Families in the communities with higher SO ₂ and TSP levels had significantly more acute respiratory problems. No single pollutant found to be the cause.
Perry et al. (1983)	24 asthmatics living in Denver. Data collected from January 9, 1979 through March 28, 1979.	CO, NO ₃ , O ₃ , PM _{2.5} , PM ₁₅ , SO ₂ , SO ₄ and TSP considered independently.	Most particulate measures not linked to asthma. Only NO ₃ significantly linked to asthma.
Robertson and Lebowitz (1984)	3800 whites living in Tucson. Data collected from August 8, 1974 to May 4, 1977.	CO, NO ₂ , O ₃ and TSP considered jointly with stepwise regression.	One pollutant at most after stepwise regression for a variety of symptoms, weather and pollen variables generally more important. TSP linked to wheeze.
Dodge et al (1985)	678 children living in four towns in Arizona, two of which had copper smelters. Data collected from 1979 to 1982.	SO ₂ and SO ₄	Children living in areas with higher SO ₂ and SO ₄ ambient concentrations had a higher incidence of cough.
Portney and Mullahy (1986)	3,347 adults (aged 17 and older) from the 1979 national Health Interview Survey.	O ₃ and SO ₄ considered independently and jointly.	The two-week average of the one-hour daily maximum of O ₃ linked to minor RRADs over this same two-week interval. O ₃ not found linked to any acute illnesses in children. No significant effects found for SO ₄ .

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Study	Population and Study Period	Pollutants	Results
Lebowitz et al (1987)	204 people living in Tucson (45 asthmatics, 68 with airway obstructive disease, 62 with allergies and 29 individuals without a history of respiratory problems)	CO, NO ₂ , O ₃ and TSP considered jointly	O ₃ linked to wheeze, cough and reduced pulmonary functioning in asthmatics. No effect of pollutants on individuals without a history of respiratory problems.
Charpin et al (1988)	450 children aged 9-11 from the Gardonne coal-basin, France. Data collected between December 1983 and March 1984.	NO ₂ , respirable particulates and SO ₂ considered jointly	Respirable particulates not linked to symptoms. SO ₂ positively correlated with the incidence of cough, wheezing, runny nose and eye irritation, in communities with the highest pollution levels.
Ostro and Rothschild (1989)	Six groups of roughly 8,000 adults (aged 18-65) from the 1986-1981 national Health Interview Survey.	O ₃ and PM _{2.5} considered jointly	PM _{2.5} significantly related to RRADs in most years. O ₃ significantly linked to minor RRADs in three of the years (1976, 1979 and 1980), and negatively related in two of the years (1977 and 1981).
Krupnick et al (1990)	290 families living in Azusa, California. Data collected from September 1978 to March 1979.	COH, NO ₂ , O ₃ and SO ₂ considered jointly	COH was positively related to the incidence of respiratory symptoms in both adults and children. O ₃ significantly related to effects only in adults. NO ₂ and SO ₂ not positively linked to health effects.
Ostro et al (1991)	207 asthmatics living in Denver. Data collected from December 1987 through February 1988.	H ⁺ , nitrates, nitric acid, PM _{2.5} , SO ₂ , and SO ₄ considered independently	PM _{2.5} linked to asthma. H ⁺ linked to moderate cough, asthma and shortness of breath. No effect found for nitrates, nitric acid or SO ₂ .
Braun-Fahrlander et al (1992)	Preschool children living in four cities in Switzerland. Volunteers recruited between November 1985 and December 1986 to fill out six-week symptom diaries.	NO ₂ , O ₃ , SO ₂ and TSP considered jointly	TSP linked to the incidence of coughing and upper respiratory symptoms, TSP also linked to the duration of respiratory symptoms. NO ₂ associated with the duration of respiratory symptom episodes, but the authors concluded that this effect may be due to confounding with TSP. No effect found for SO ₂ or O ₃ .

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Study	Population and Study Period	Pollutants	Results
Pope and Dockery (1992)	60 symptomatic (with a history of mild respiratory symptoms) and 60 asymptomatic children living in the Utah Valley, between December 6, 1990 and March 15, 1991	PM ₁₀	PM ₁₀ linked to reduced pulmonary functioning and cough in both the symptomatic and asymptomatic groups. The effects on the symptomatic group were particularly strong.
Ostro et al (1993)	321 adults (18 years and older) from Azusa, California. A subset of the data used by Krupnick et al (1990, discussed above). Data collected from September 1978 to March 1979	COH, NO ₂ , O ₃ , SO ₂ and SO ₄ considered independently.	SO ₄ and O ₃ had significant effects. SO ₂ not linked to adverse respiratory effects.
Roemer et al (1993)	74 children with chronic respiratory problems from two small industrial towns in the Netherlands. Data collected from 12/17/90 to 3/17/91.	British Smoke, NO ₂ , PM ₁₀ and SO ₂ considered independently.	SO ₂ , PM ₁₀ and British Smoke linked to asthma attacks, wheeze and runny nose. However, the pollutants were considered independently, making it difficult to ascribe an independent effect to any pollutant.

TABLE 11.3-15. SUMMARY OF HEALTH EFFECTS STUDIES EXAMINING ADMISSIONS TO HOSPITALS AND PARTICULATES

Study	Population and Study Period	Pollutants	Results
Fishelson and Graves (1978)	All ages in Chicago. Data collected from September 1971 to March 1973	COH and SO ₂ considered jointly	COH found in some regressions to be significantly and <u>negatively</u> related to hospital admissions SO ₂ linked to cardiac-related emergency room admissions of people aged 40-59 and 60+.
Richards et al (1981)	Children in Los Angeles Data collected from August 1, 1979 to January 31, 1980	COH, HC, NO, NO ₂ , O ₃ , SO ₂ and SO ₄ considered independently	NO, NO ₂ , COH and HC positively linked to asthma-related emergency room admissions SO ₂ and O ₃ <u>negatively</u> linked to asthma-related emergency room visits
Samet et al (1981)	All ages in Steubenville, Ohio Population = 31,000 Data collected for the months of March, April, October and November in 1974-1977	CO, NO ₂ , O ₃ , SO ₂ and TSP considered independently Correlation between TSP and SO ₂ = 0.69.	SO ₂ and TSP weakly linked to emergency room admissions for respiratory problems
Bates and Sizto (1983)	All ages in southern Ontario Data collected from the months of January, February, July and August 1974, 1976-1978	COH, NO ₂ , O ₃ and SO ₂ considered independently	No effect found for COH SO ₂ and O ₃ increased respiratory-related admissions to hospitals during July and August
Mazumdar and Sussman (1983)	All ages in Allegheny County, Pennsylvania (including the City of Pittsburgh) Data collected from 1972-1977	COH and SO ₂ considered jointly	COH linked to both increased emergency room admissions and increased mortality SO ₂ not consistently linked to hospital emergency admissions or mortality
Pope (1989)	Hospitals admissions for respiratory-related illnesses, between April 1985 and February 1988 in the Utah Valley	PM ₁₀	PM ₁₀ strongly related to respiratory-related admissions, particularly for asthma and bronchitis PM ₁₀ had a stronger effect on children than adults
Díaz-Caneja et al (1991)	All ages living in the city of Santander, Spain. Data collected from 1979 to 1982	SO ₂ and suspended particulate matter (SPM)	SPM found to be a better predictor than SO ₂ of hospital admissions for chronic obstructive pulmonary disease (COPD) and heart failure Effects for SPM found down to at least 40 µg/m ³ , and possibly lower

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Study	Population and Study Period	Pollutants	Results
Ponka (1991)	All ages living in Helsinki, Finland. Data collected between 1987-1989	CO, NO, NO ₂ , O ₃ , SO ₂ and TSP independently and in a stepwise regression. Correlation between SO ₂ and TSP = 0.19	All of the pollutants were correlated with asthma-related hospital admissions. Asthma-related emergency room admissions were more highly correlated with SO ₂ , TSP and O ₃ . All asthma-related hospital admissions (including emergency room) were more highly correlated with CO, NO and NO ₂ .
Pope (1991)	Comparison of hospital admission rates in three counties in Utah, between April 1985 and March 1989	PM ₁₀ . The main contributor of PM ₁₀ pollution in Utah County is a steel mill. Cache County has no mill, and has substantially lower PM ₁₀ levels.	Respiratory admissions were substantially higher in Utah County than Cache County. When the steel mill closed for a year, admissions dropped substantially in Utah County, and subsequently rose when the mill reopened.
Schwartz et al (1991)	Young children in five German cities. Data collected from January 1983 to August 1985 in Duisberg, from September 1984 to April 1987 in Koln, from January 1986 to December 1987 in Stuttgart, Tubingen and Freudenstadt.	NO ₂ , SO ₂ and TSP considered jointly	Of the three pollutants, TSP is the strongest predictor of croup. SO ₂ a significant predictor of croup-related visits to doctors and hospitals, in single pollutant models. SO ₂ <u>not</u> significant when considered with either NO ₂ or TSP.
Sunyer et al (1991)	All ages in Barcelona, Spain. Data collected from 1985-1986	British Smoke, CO, NO ₂ , O ₃ and SO ₂ considered independently. Correlation between pollutants (except ozone) ranged between 0.49 and 0.67 (ozone was lower)	British Smoke a significant predictor of emergency room admissions for COPD. SO ₂ and CO also linked to admissions.
Lipfert and Hammerstrom (1992)	Respiratory-related admissions to 79 acute-care hospitals in southern Ontario, between 1979 and 1985.	COH, NO ₂ , O ₃ , SO ₂ , SO ₄ and TSP.	O ₃ , SO ₂ , SO ₄ and TSP were significantly associated with respiratory admissions. No single pollutant was determined to be the cause. There were no reported effects for COH and NO ₂ .

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Study	Population and Study Period	Pollutants	Results
Tseng et al (1992)	Four age groups (0-1, 1-4, 5-14, and 15+) were examined in this study of the population in Hong Kong. Data collected from 1983-1989.	NO ₂ , NO _x , O ₃ , respirable particulates, SO ₂ and TSP	TSP positively linked to hospital admissions for asthma for ages 1-4. SO ₂ found to be significantly and negatively correlated with hospital admissions for asthma for all age groups. It is not clear why this results occurred, perhaps it was because of the use of highly aggregated (quarterly) data.
Schwartz et al (1993)	People under 65. Data collected from hospitals in Seattle from September 1, 1989 to September 30, 1990.	O ₃ , PM ₁₀ and SO ₂ considered independently	O ₃ and SO ₂ not linked to asthma-related emergency room admissions. PM ₁₀ significantly linked to admissions. No threshold seen for PM ₁₀ down to 10 µg/m ³ .

TABLE 11.3-16. SUMMARY OF STUDIES CONCERNING CHRONIC MORBIDITY AND PARTICULATES

Study	Population and Study Period	Pollutants	Results
Chapman et al (1985)	5,623 young adults in four Utah communities, with varying pollutant levels. Health data collected by survey in 1976, pollution data collected between 1971-1975	Nitrates, SO ₂ , SO ₄ and TSP Five-year mean pollution level used in the analysis	Communities with higher SO ₂ and SO ₄ ambient concentrations had a higher incidence of persistent cough and phlegm (i.e., cough and phlegm for at least three months per year)
Euler et al (1987)	7,445 Seventh-Day Adventists, aged 25 years and older, and who had resided within 5 miles of their current residence for 10 years Health data collected in 1977	SO ₂ and TSP	TSP was significantly related to chronic obstructive pulmonary disease, significant effects also found for SO ₂
Euler et al (1988)	7,445 Seventh-Day Adventists, aged 25 years and older, and who had resided within 5 miles of their current residence for 10 years Health data collected in 1977	NO ₂ , O ₃ , SO ₂ and TSP	TSP was significantly related to chronic obstructive pulmonary disease, controlling for O ₃ and SO ₂ . No effect found for NO ₂
Dockery et al (1989)	Elementary school students, 10-12 years old, living in six cities in the U.S Data collected during the 1980-1981 school year	NO ₂ , O ₃ , PM _{2.5} , PM ₁₅ , SO ₂ , SO ₄ and TSP considered independently	Particulate measures (especially PM ₁₅ and TSP) were associated with increased bronchitis, chronic cough and chest illness TSP was consistently associated with reduced pulmonary functioning O ₃ significantly related to asthma rates
Archer (1990)	Mortality records for respiratory cancer and nonmalignant respiratory disease (NMRD) from three Utah counties Data collected from 1950-1987 for respiratory cancer and 1968-1987 for NMRD	Utah and Salt Lake counties had higher levels of CO, NO ₂ , O ₃ , SO ₂ and TSP than Cache County.	Utah and Cache counties are closely matched demographically However, Utah County has higher pollution levels, due to a steel mill built in the early 1940s Starting in 1960, Utah County had a significantly higher rate of respiratory cancer and NMRD (compared to Cache County) This difference was attributed to air pollution from the steel mill

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Study	Population and Study Period	Pollutants	Results
Portney and Mullahy (1990)	1,318 adults (over the age of 17) in a national survey in 1979	O ₃ and TSP Both the annual average pollution level and six-year average (1974-1979) were used in this analysis	Marginally significant relationship found between TSP and emphysema, chronic bronchitis and asthma. The six-year O ₃ level was significantly related to the incidence of sinusitis and hay fever. O ₃ not related to a combined measure of more severe chronic disease (emphysema, chronic bronchitis and asthma)
Abbey et al (1991)	6000 Seventh-Day Adventists living in California, and who had resided within 5 miles of their current residence for 10 years Health data collected in 1977 and 1987	O ₃ and TSP considered independently. The average pollution level from 1973-1977 was used to determine pollution exposure The average between 1966-1977 was also tried and produced similar results. The later period was used because there are data from more monitoring stations	TSP significantly linked (95% CI) with all malignant neoplasms, airway obstructive disease and asthma O ₃ linked to asthma, bronchitis and respiratory cancer at a 90% confidence interval No link found between O ₃ and total mortality.
Vedal et al (1991)	2,199 students (grades 1-6) in Port Alberni, British Columbia, from January 1989 to April 1989	PM ₁₀ , "submicronic" particles and TSP	All particulate measures (annual mean levels) were associated with chronic cough, chronic phlegm, wheezing and congestion and hospitalization for chest illness. A threshold found for TSP (45-50 µg/m ³) and PM ₁₀ (35-40 µg/m ³)
Abbey et al (1993)	3,914 nonsmoking Seventh-Day Adventists living in California in 1977, and who had resided within 5 miles of their current residence for 10 years Health data collected in 1977 and 1987	O ₃ and TSP considered jointly The average pollution level from 1973-1977 was used to determine pollution exposure in published results Estimated the average annual number of hours of pollution exposure above five thresholds	O ₃ linked to new cases of asthma and symptom severity of asthma When O ₃ modeled with TSP the results were unstable, Abbey et al concluded both O ₃ and TSP are significantly linked to new cases of asthma and symptom severity

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Study	Population and Study Period	Pollutants	Results
Abbey et al (1995)	3,914 nonsmoking Seventh-Day Adventists living in California in 1977, and who had resided within 5 miles of their current residence for 10 years. Health data collected in 1977 and 1987.	PM ₁₀ The average pollution level from 1973-1977 was used to determine pollution exposure in published results. Estimated the average annual number of hours of pollution exposure above five thresholds.	PM ₁₀ linked to new cases of airway obstructive disease (AOD)

TABLE 11.3-17. SUMMARY OF HEALTH EFFECTS STUDIES EXAMINING MORTALITY AND PARTICULATES

Study	Population and Study Period	Pollutants	Results
Mazumdar et al (1982)	Mortality records for all nontraumatic causes in London during the winters of 1958-1972	British Smoke and SO ₂ considered jointly Correlation > 0.80 in all years	A strong association was found between British smoke and mortality, regardless of whether SO ₂ was considered in the regression
Ozkaynak and Thurston (1987)	Total mortality data from 100 metropolitan areas in 1980	PM _{2.5} , PM ₁₀ , SO ₄ and TSP	SO ₄ was the best predictor of mortality, followed by PM _{2.5} PM ₁₀ and TSP were generally not significant The strength of the effect declined with increasing particle size Particulates derived from coal and metals were found to be most potent
Fairley (1990)	Mortality data by cause of death from Santa Clara County between 1980 and 1986 (1983 missing)	COH	COH linked to respiratory, circulatory and all nonaccidental deaths There is a borderline significance between COH and cancer
Schwartz and Marcus (1990)	Mortality records for all nontraumatic causes in London during the winters of 1958-1972.	British Smoke and SO ₂ Correlation > 0.80 in all years	A strong association was found between British smoke and mortality, regardless of whether SO ₂ was considered in the regression
Kinney and Ozkaynak (1991)	All nonaccidental mortality records from Los Angeles County, 1970-1979 The study also considered respiratory-related deaths and cardiovascular-related deaths	CO, KM, NO ₂ , O ₃ and SO ₂ considered jointly	CO, KM and NO ₂ each significantly related to total mortality and cardiovascular-related mortality in models with O ₃ It was not possible to determine which pollutant (besides O ₃) caused mortality SO ₂ had no effect on mortality.
Schwartz (1991)	All nonaccidental mortality records in Detroit, Michigan, from 1973 to 1982.	O ₃ , SO ₂ and TSP considered jointly TSP predicted from daily observations of the visibility extinction coefficient	TSP strongly linked to mortality, the size of the coefficient was not significantly affected by the inclusion of SO ₂ SO ₂ not significantly linked to mortality, controlling for TSP. No link between O ₃ and mortality

Dockery et al (1992)	All nonaccidental mortality records from the St Louis metropolitan statistical area and eastern Tennessee from September 1985 to August 1986	H ⁺ , NO ₂ , O ₃ , PM _{2.5} , PM ₁₀ , SO ₂ , SO ₄ considered jointly	A strong association was found between PM ₁₀ and mortality SO ₂ not significantly linked to mortality.
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Study	Population and Study Period	Pollutants	Results
Pope et al (1992)	Nonaccidental mortality in Utah County from 1985 to 1989	PM ₁₀ Levels of H ⁺ , NO ₂ , O ₃ and SO ₂ were at low levels, except during the summer when O ₃ approached the national standard of 0.12 ppm.	PM ₁₀ had the strongest effect on respiratory deaths, followed by cardiovascular and all other deaths.
Schwartz and Dockery (1992a)	All nonaccidental mortality records from Philadelphia, from 1973-1980. The mortality records were divided into decedents < 65 years old, and decedents 65 and older	SO ₂ and TSP considered jointly	TSP strongly linked to mortality, the size of the coefficient was not significantly affected by the inclusion of SO ₂ . The effect of TSP was significantly higher on people older than 65. SO ₂ not significantly linked to mortality, controlling for TSP. No interaction found between TSP and SO ₂ .
Schwartz and Dockery (1992b)	All nonaccidental mortality records from Steubenville, Ohio metropolitan area. Data collected from 1974-1984	SO ₂ and TSP considered jointly	TSP strongly linked to mortality, the size of the coefficient was not significantly affected by the inclusion of SO ₂ . SO ₂ not significantly linked to mortality, controlling for TSP.
Dockery et al (1993)	Health data collected over 14-16 years (study ended in 1991) from 8,111 white persons in six cities in the U.S. -- Portage, WI, Topeka, KS, Watertown, MA; Kingston, TN, St. Louis, MO, and Steubenville, OH.	NO ₂ , O ₃ , PM _{2.5} , PM ₁₀ , PM ₁₅ , SO ₂ , SO ₄ , TSP and aerosol acidity considered independently	Mortality more strongly linked to PM _{2.5} , PM ₁₀ , PM ₁₅ and SO ₄ than with NO ₂ , SO ₂ , TSP and aerosol acidity. O ₃ levels varied little and were not significant. Air pollution linked to lung cancer and cardiopulmonary disease-related deaths.
Xu et al (1994)	All nonaccidental mortality records in two residential areas in Beijing, China, from 1989	SO ₂ and TSP considered jointly	TSP significantly linked to mortality only in the summer, with and without the inclusion of SO ₂ . In the winter, TSP had a negative and insignificant coefficient (t=-1.54 with SO ₂ included).

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Moolgavkar et al (1995)	Philadelphia mortality data collected for the period 1973-1988	O ₃ , TSP and SO ₂ considered jointly	With all three pollutants in model, O ₃ linked to summer time mortality and SO ₂ linked to mortality in the fall, winter and spring
Pope et al (1995a)	Mortality and individual risk factors (e.g , smoking) data from 552,138 adults in 151 metropolitan statistical areas	SO ₄ and PM _{2.5}	PM _{2.5} significantly linked to cardiopulmonary deaths. SO ₄ linked to cardiopulmonary and lung cancer deaths

TABLE 11.3-18. A VARIETY OF MEASURES FOR PARTICULATES

Particulate Measure	Description
TSP	Total suspended particulates, which are generally less than 60 micrometers (m) in diameter
British Smoke	A particulate measure typically used in Britain, similar to TSP
PM ₁₀	Particulate matter less than 10 micrometers (m) in diameter This is the U.S. criteria pollutant measure for particulates
COH	Coefficient of haze, a measure based on visibility
PM _{2.5}	Particulate matter less than 2.5 micrometers (m) in diameter
SO ₄	Sulfates, which are typically 0.2 - 0.6 micrometers (m) in diameter
H ⁺	Hydrogen ion, the acidic component of particulates

Sources EPA (1982), Withey (1989: 521), Krupnick et al. (1990: 4), Ostro (1990: 422)

TABLE 11.3-19. EPIDEMIOLOGICAL STUDIES USED IN THIS ANALYSIS TO ESTIMATE THE HEALTH EFFECTS OF PARTICULATES

Health Effect Evaluated	Particulate Measure	Epidemiological Study Used To Estimate Health Effects
Asthma	TSP	Whittemore and Korn (1980)
Respiratory-Related Restricted Activity Day (RRAD)	PM _{2.5}	Ostro and Rothschild (1989)
Airway Obstructive Disease (AOD)	PM ₁₀	Abbey et al (1995)
Acute Mortality	PM ₁₀	Pope et al (1992)
Chronic Mortality	PM _{2.5}	Ozkaynak and Thurston (1987) [lower bound] Pope et al (1995a) [upper bound]

TABLE 11.3-20. EPIDEMIOLOGICAL STUDIES USED IN PREVIOUS ANALYSES TO ESTIMATE THE HEALTH EFFECTS OF PARTICULATES

Study Of Economic Value Of Health Effects	Health Effects Evaluated	Particulate Measure	Epidemiological Studies Used To Estimate Health Effects
Hall et al , 1992	RAD Mortality	PM ₁₀ ^a PM ₁₀ ^b	Ostro (1987) Evans et al (1984a)
Harrison and Nichols, 1990	RRAD Mortality	PM ₁₀ ^a PM ₁₀	Ostro and Rothschild (1989) Evans et al (1984a)
Krupnick and Portney, 1991	Morbidity ^b Mortality	SO ₄	Ozkaynak and Thurston (1987)
RER (1990, Phase II)	Any Respiratory Symptom MRAD, RRAD Mortality	COH PM _{2.5} TSP	Krupnick et al (1990) Ostro and Rothschild (1989) Lave and Seskin (1970), Lipfert (1980)

^aThe original health effect studies (Ostro, 1987, Evans et al , 1984a) cited by Hall et al. (1992) used PM_{2.5} and TSP data, respectively. To use these studies with PM₁₀ data, Hall et al (1992) assumed that PM₁₀ is 61% PM_{2.5}, and TSP is 55% PM₁₀

^bKrupnick and Portney (1991) also estimated particulate-related morbidity, but they did not state on which paper(s) they based their estimate

TABLE 11.3-21. ASSUMED PERCENTAGE OF AOD ATTRIBUTABLE TO PM₁₀ EACH YEAR

Year	Percentage of Effect
1	1
2	1
3	2
4	3
5	4
6	6
7	7
8	8
9	9
10	9
11	9
12	9
13	8
14	7
15	6
16	4
17	3
18	2
19	1
20	1

Source Our judgement

TABLE 11.3-22. RELATIVE RISK OF AOD FROM EXPOSURE TO PM₁₀

PM₁₀ Threshold Level	Annual hours of exposure^a	Relative Risk	Estimated Coefficient^b	Arithmetic Average^c	Standard Deviation^d
40 g/m ³	250	1.01	4.352 E-5	5.401 E-5	7.50 E-6
	500	1.03	6.470 E-5		
	1000	1.05	5.344 E-5		
	2500	1.13	5.375 E-5		
	5000	1.28	5.464 E-5		
50 g/m ³	250	1.01	4.352 E-5	5.791 E-5	8.55 E-6
	500	1.03	6.470 E-5		
	1000	1.06	6.386 E-5		
	2500	1.14	5.765 E-5		
	5000	1.31	5.985 E-5		
60 g/m ³	250	1.02	8.665 E-5	7.402 E-5	7.96 E-6
	500	1.03	6.470 E-5		
	1000	1.07	7.418 E-4		
	2500	1.18	7.295 E-4		
	5000	1.38	7.160 E-4		
80 g/m ³	250	1.02	8.665 E-5	1.001 E-4	9.17 E-6
	500	1.05	1.069 E-4		
	1000	1.10	1.046 E-4		
	2500	1.26	1.022 E-4		
100 g/m ³	250	1.04	1.718 E-4	1.712 E-4	2.10 E-6
	500	1.08	1.688 E-4		
	1000	1.17	1.729 E-4		

Source: Abbey et al. (1995).

^aAnnual hours of exposure above each threshold. Abbey et al. (1995 Table 3) used the average annual number of hours from 1973-1977

^bThe estimation of the estimated coefficients is explained in Section 11.2.4.2 of the text

^cThe arithmetic average of the estimated coefficients for each threshold level.

^dThe standard deviation of the estimated coefficients for each threshold level

TABLE 11.3-23. PERCENTAGE OF TOTAL NATIONAL MORTALITY BY CAUSE OF DEATH IN 1989 AND 1990

Cause of Death	1989 ^a	1990
Major Cardiovascular Diseases	43.33	42.57
Malignancies	23.07	23.40
Accidents and Adverse Effects	4.42	4.33
Chronic Obstructive Pulmonary Diseases and Allied Conditions	3.92	4.12
Pneumonia and Influenza	3.56	3.64
Suicide	1.40	1.42
Other	20.29	20.52

Source. U.S. Bureau of the Census (1992 Table 114)

^aThe percentages do not add up to 100 due to rounding error

TABLE 11.3-24. SUMMARY OF HEALTH EFFECTS STUDIES EXAMINING PULMONARY FUNCTIONING AND SULFUR DIOXIDE

Study	Population and Study Period	Pollutants	Results
Dodge et al (1985)	678 children living in four towns in Arizona; two of the towns had copper smelters Data collected from 1979 to 1982	SO ₂ and SO ₄	No significant differences in lung functioning between children living in areas with different levels of SO ₂ and SO ₄
Dockery et al (1989)	Elementary school students, 10-12 years old, living in six cities in the U.S. Data collected during the 1980-1981 school year.	NO ₂ , O ₃ , PM _{2.5} , PM ₁₅ , SO ₂ , SO ₄ and TSP considered independently Annual mean pollutant levels used in the analysis	None of the pollutants were found to have an effect on pulmonary function (measured by FEV ₁ and other measures).
Schwartz (1989)	National survey of people aged 6-24 years old, conducted between 1976-1980.	Nitric acid, NO ₂ , O ₃ , SO ₂ , sulfuric acid and TSP considered independently	SO ₂ not significantly linked to pulmonary function (measured by FEV ₁ , FVC and peak flow) Significant effects found for TSP, O ₃ and NO ₂
Xu et al (1991)	1440 adults (40-69 years of age) living in three areas of Beijing, with different pollution levels Data collected in August 1986	SO ₂ and TSP	SO ₂ and TSP both linked to significantly reduced pulmonary functioning (measured by FEV ₁ and FVC) Independent effects for each pollutant were not determined
Hoek et al (1993)	Children from four years of age to seven years of age living in three nonindustrial towns in the Netherlands. Data collected from March 1989 to July 1989.	NO ₂ , O ₃ , PM ₁₀ and SO ₂ considered jointly	SO ₂ not significantly linked to pulmonary functioning after controlling for O ₃ Daily O ₃ levels were negatively correlated with lung functioning (measured by FEV ₁ , FVC, MMEF and PEF) A small effect was found for PM ₁₀ , independent of O ₃

TABLE 11.3-25. SUMMARY OF HEALTH EFFECTS STUDIES EXAMINING ACUTE AND CHRONIC RESPIRATORY SYMPTOMS (EXCLUDING HOSPITAL ADMISSIONS) AND SULFUR DIOXIDE

Study	Population and Study Period	Pollutants	Results
Acute effects			
Cohen et al (1972)	Seven months of daily symptom reports kept by 20 asthmatics living in West Virginia	NO ₃ , SO ₂ , SO ₄ , soiling index (alternative particulate measure) and TSP considered independently Pollutants moderately correlated TSP and SO ₂ (r=0.537)	Individually all of the pollutants were significant contributors to asthma attacks. No single pollutant was found to be the cause of asthma attacks.
Love et al (1981)	250 families from two polluted areas in New York City and one low-pollution area in Long Island. Data during the 1971-1972 school year	SO ₂ and TSP	Families in the communities with higher SO ₂ and TSP levels had significantly more acute respiratory problems. No single pollutant found to be the cause.
Perry et al (1983)	24 asthmatics living in Denver. Data collected from January 9, 1979 through March 28, 1979	CO, NO ₃ , O ₃ , PM _{2.5} , PM ₁₅ , SO ₂ , SO ₄ and TSP considered independently	SO ₂ not linked to asthma. Only NO ₃ significantly linked to asthma.
Dodge et al. (1985)	678 children living in four towns in Arizona, two of the towns had copper smelters. Data collected from 1979 to 1982.	SO ₂ , and SO ₄	Children living in areas with higher SO ₂ and SO ₄ ambient concentrations had a higher incidence of cough.
Charpin et al (1988)	450 children aged 9-11 from the Gardonne coal-basin, France. Data collected between December 1983 and March 1984	NO ₂ , respirable particulates and SO ₂ considered jointly	SO ₂ positively correlated with the incidence of cough, wheezing, runny nose and eye irritation, in communities with the highest pollution levels. Respirable particulates not linked to symptoms.
Dockery et al (1989)	Elementary school students, 10-12 years old, living in six cities in the U.S. Data collected during the 1980-1981 school year	NO ₂ , O ₃ , PM _{2.5} , PM ₁₅ , SO ₂ , SO ₄ and TSP considered independently	SO ₂ not linked to bronchitis, chronic cough, chest illness, persistent wheeze and asthma. O ₃ linked to asthma. PM ₁₅ linked to bronchitis and chronic cough.

Krupnick et al (1990)	290 families living in Azusa, California Data collected from September 1978 to March 1979	COH, NO ₂ , O ₃ and SO ₂ considered jointly Correlation SO ₂ and COH = 0.57	SO ₂ not linked to respiratory symptom in adults, and even had a <u>negative</u> effect on the incidence of respiratory symptoms in children COH was positively related to the incidence of respiratory symptoms in both adults and children
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Study	Population and Study Period	Pollutants	Results
Schwartz and Zeger (1990)	Student nurses in Los Angeles Data collected November 1961 to May 1964	CO, NO ₂ , O _x and SO ₂ considered jointly	SO ₂ not linked to the incidence of cough, phlegm, sore throat, headache, chest and eye irritation The pollutants were considered simultaneously
Jaakkola et al (1991)	759 children under the age of six living in three cities in Finland Data collected from November to December 1982	SO ₂ used as a proxy for all air pollution	Children in the more polluted city had a higher rate of respiratory infection
(Ostro et al (1991)	207 asthmatics living in Denver Data collected from December 1987 through February 1988	H ⁺ , nitrates, nitric acid, PM _{2.5} , SO ₂ , and SO ₄ considered independently Correlation between SO ₂ and PM _{2.5} = 0.64 Correlation between SO ₂ and H ⁺ = 0.35	SO ₂ not linked to moderate cough, asthma or shortness of breath H ⁺ linked to moderate cough, asthma and shortness of breath PM _{2.5} linked to asthma No effect found for nitrates or nitric acid
Schwartz et al (1991)	1800 children from six cities (Watertown, Kingston-Harriman, TN, St Louis, MO, Portage, WI, Stuebenville, OH, and Topeka, KS). Diary data collected from 1984-1988	SO ₂ Other pollutants were not discussed in this methodological paper.	SO ₂ linked to the incidence of cough. The units of measurement were not given, nor whether the pollutants were considered separately or simultaneously
Braun-Fahrlander et al (1992)	Preschool children living in four cities in Switzerland Volunteers recruited collected between November 1985 and December 1986 to fill out six-week symptom diaries	NO ₂ , O ₃ , SO ₂ and TSP considered jointly	No effect found for SO ₂ or O ₃ NO ₂ associated with the duration of respiratory symptom episodes, but the authors concluded that this effect may be due to confounding with TSP TSP linked to both the incidence and duration of respiratory symptoms
Schwartz (1992)	Student nurses in Los Angeles Data collected November 1961 to May 1964.	CO, NO ₂ , O _x , and SO ₂ considered jointly	SO ₂ increased the duration of chest discomfort, primarily in asthmatics

Ostro et al. (1993)	321 adults (18 years and older) from Azusa, California A subset of the data used by Krupnick et al (1990) Data collected from September 1978 to March 1979	COH, NO ₂ , O ₃ , SO ₂ and SO ₄ considered independently Correlation between SO ₂ and COH = 0.57 Correlation between SO ₂ and SO ₄ not reported	SO ₂ not linked to lower respiratory symptoms, upper respiratory symptoms, and eye irritation SO ₄ and O ₃ had significant effects
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Study	Population and Study Period	Pollutants	Results
Roemer et al (1993)	74 children with chronic respiratory problems from two, small industrial towns in the Netherlands Data collected from December 17, 1990 to March 17, 1991	British Smoke, NO ₂ , PM ₁₀ and SO ₂ considered independently Correlation SO ₂ and PM ₁₀ = 0.65 Correlation SO ₂ and BS = 0.63	SO ₂ , PM ₁₀ and British Smoke linked to asthma attacks, wheeze and runny nose However, the pollutants were considered independently, making it difficult to ascribe an independent effect to any pollutant
Chronic Effects			
Chapman et al (1985)	5,623 young adults in four Utah communities, with varying pollutant levels Health data collected by survey in 1976, pollution data collected between 1971-1975	nitrates, SO ₂ , SO ₄ and TSP Five-year mean pollution level used in the analysis	Communities with higher SO ₂ and SO ₄ ambient concentrations had a higher incidence of persistent cough and phlegm (i.e., cough and phlegm for at least three months per year)
Euler et al. (1988)	7,445 Seventh-Day Adventists 25 years and older living in California Data collected from 1966-1976	NO ₂ , O ₃ , SO ₂ and TSP	Considered separately, SO ₂ was significantly linked to an increased risk of COPD In models with three pollutants (O ₃ , SO ₂ and TSP), only TSP was significant
Abbey et al (1993)	3,914 nonsmoking Seventh-Day Adventists living in California in 1977, and who had resided within 5 miles of their current residence for 10 years Panel data collected in 1977 and 1987	O ₃ , SO ₂ and TSP considered jointly. The average pollution level from 1973-1977 was used to determine pollution exposure in published results Estimated the average annual number of hours of pollution exposure above five thresholds	No link found between SO ₂ and chronic respiratory illness -- emphysema, chronic bronchitis and asthma

TABLE 11.3-26. SUMMARY OF HEALTH EFFECTS STUDIES EXAMINING ADMISSIONS TO HOSPITALS AND SULFUR DIOXIDE

Study	Population and Study Period	Pollutants	Results
Fishelson and Graves (1978)	All ages in Chicago Data collected from September 1971 to March 1973	COH and SO ₂ considered jointly Correlation between SO ₂ and COH not reported	This study ran a large number of regressions testing for a link between SO ₂ and COH and hospital emergency room admissions SO ₂ linked to cardiac-related emergency room admissions of people aged 40-59 and 60+ COH found in some regressions to be significantly and <u>negatively</u> related to hospital admissions
Richards et al (1981)	Children in Los Angeles Data collected from August 1, 1979 to January 31, 1980	COH, HC, NO, NO ₂ , O ₃ , SO ₂ and SO ₄ considered independently	SO ₂ and O ₃ <u>negatively</u> (and significantly) linked to asthma-related emergency room visits. Positive correlation found between COH, HC, NO and NO ₂
Samet et al (1981)	All ages in Steubenville, Ohio Population = 31,000 Data collected for the months of March, April, October and November in 1974-1977	CO, NO ₂ , O ₃ , SO ₂ and TSP considered independently Correlation between TSP and SO ₂ = 0.69	SO ₂ and TSP weakly linked to emergency room admissions for respiratory problems
Bates and Sizto (1983)	All ages in southern Ontario Data collected from the months of January, February, July and August 1974, 1976-1978	COH, NO ₂ , O ₃ and SO ₂ considered independently	SO ₂ and O ₃ increased respiratory-related admissions to hospitals during July and August.
Mazumdar and Sussman (1983)	All ages in Allegheny County, Pennsylvania (including the City of Pittsburgh) Data collected from 1972-1977	COH and SO ₂ considered jointly	SO ₂ not consistently linked to either increased mortality or hospital emergency admissions COH linked to both increased mortality and increased emergency room admissions
Goldstein and Weinstein (1986)	All ages in New York City from 1969-1972	SO ₂	SO ₂ not linked to hospital emergency room visits for asthma.

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Study	Population and Study Period	Pollutants	Results
Bates et al (1990)	All ages in Vancouver. Data collected between July 1984 and October 1986	COH, NO ₂ , O ₃ , SO ₂ and SO ₄ considered separately Correlation SO ₂ and SO ₄ = 0.46 (May-October) and = 0.54 (November-April) Correlation SO ₂ and COH = 0.341 (May-October) and = 0.64 (November-April)	SO ₂ and SO ₄ both correlated with asthma- and other respiratory-related hospital visits During November-April, only SO ₂ correlated to respiratory-related admissions for patients 60 and younger -- no significant correlation reported between this group of people and other pollutants O ₃ linked to total admissions (The results for COH were not reported)
Díaz-Caneja et al. (1991)	All ages living in the city of Santander, Spain Data collected from 1979 to 1982.	SO ₂ and suspended particulate matter (SPM)	SPM found to be a better predictor than SO ₂ of hospital admissions for chronic obstructive pulmonary disease (COPD) and heart failure
Ponka (1991)	All ages living in Helsinki, Finland Data collected between 1987-1989	CO, NO, NO ₂ , O ₃ , SO ₂ and TSP independently and in a stepwise regression Correlation between SO ₂ and TSP = 0.19	All of the pollutants were correlated with asthma-related hospital admissions Asthma-related emergency room admissions were more highly correlated with SO ₂ , TSP and O ₃ All asthma-related hospital admissions (including emergency room) were more highly correlated with CO, NO and NO ₂
Schwartz et al (1991)	Young children in five German cities Data collected from January 1983 to August 1985 in Duisberg, from September 1984 to April 1987 in Koln, from January 1986 to December 1987 in Stuttgart, Tübingen and Freudenstadt	NO ₂ , SO ₂ and TSP considered jointly	SO ₂ a significant predictor of croup-related visits to doctors and hospitals, in single-pollutant models. SO ₂ <u>not</u> significant when considered with either NO ₂ or TSP. Of the three pollutants, TSP is the strongest predictor of croup

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Study	Population and Study Period	Pollutants	Results
Sunyer et al (1991)	All ages in Barcelona, Spain Data collected from 1985-1986	British Smoke, CO, NO ₂ , O ₃ and SO ₂ considered independently Correlation between pollutants (except ozone) ranged between 0.49 and 0.67 (ozone was lower).	SO ₂ a significant predictor of emergency room admissions for COPD British Smoke and CO also linked to admissions Each pollutant was considered separately, so it is not possible to determine if SO ₂ would still be significant, controlling for other pollutants, particularly British Smoke
Cody et al (1992)	All ages living in central and northern New Jersey. Data collected from May through August in 1988 and 1989	O ₃ , PM ₁₀ and SO ₂ and visibility (as a proxy for sulfate) considered independently	SO ₂ not significantly linked to asthma-related emergency room visits A significant association found for O ₃
Lipfert and Hammerstrom (1992)	Respiratory-related admissions to 79 acute-care hospitals in southern Ontario, between 1979 and 1985	COH, NO ₂ , O ₃ , SO ₂ , SO ₄ and TSP.	O ₃ , SO ₂ , SO ₄ and TSP were significantly associated with respiratory admissions No single pollutant was determined to be the cause There were no reported effects for COH and NO ₂ .
Tseng et al. (1992)	Four age groups (0-1, 1-4, 5-14, and 15+) were examined in this study of the population in Hong Kong Data collected from 1983-1989	NO ₂ , NO _x , O ₃ , respirable particulates, SO ₂ and TSP	SO ₂ found to be significantly and <u>negatively</u> correlated with hospital admissions for asthma for all age groups It is not clear why this result occurred, perhaps it was because of the use of highly aggregated (quarterly) data TSP positively linked to hospital admissions for asthma for ages 1-4
Schwartz et al (1993)	People under 65 Data collected from hospitals in Seattle from September 1, 1989 to September 30, 1990	O ₃ , PM ₁₀ and SO ₂ considered independently	SO ₂ not linked to asthma-related emergency room admissions (The SO ₂ ambient data were from an industrial area and may poorly characterize population exposure, and thus reduce the likelihood of finding a significant effect.) PM ₁₀ significantly linked to admissions No effect found for O ₃

TABLE 11.3-27. SUMMARY OF HEALTH EFFECTS STUDIES EXAMINING MORTALITY AND SULFUR DIOXIDE

Study	Population and Study Period	Pollutants	Results
Mazumdar et al (1982)	Mortality records for all nontraumatic causes in London during the winters of 1958-1972	British Smoke and SO ₂ considered jointly Correlation > 0.80 in all years	SO ₂ not significantly linked to mortality, controlling for British smoke. A strong association was found between British smoke and mortality, regardless of whether SO ₂ was considered in the regression
Schwartz and Marcus (1990)	Mortality records for all nontraumatic causes in London during the winters of 1958-1972	British Smoke and SO ₂ considered jointly Correlation > 0.80 in all years	SO ₂ not significantly linked to mortality, controlling for British smoke. A strong association was found between British smoke and mortality, regardless of whether SO ₂ was considered in the regression
Kinney and Ozkaynak (1991)	All nonaccidental mortality records from Los Angeles County, 1970-1979. The study also considered respiratory-related deaths and cardiovascular-related deaths.	CO, KM, NO ₂ , O _x and SO ₂ considered jointly.	No effect of SO ₂ on the daily mortality rate. O ₃ most strongly related to mortality, followed by KM, CO and NO ₂
Schwartz (1991)	All nonaccidental mortality records in Detroit, Michigan, from 1973 to 1982	O ₃ , SO ₂ and TSP considered jointly. TSP predicted from daily observations of the visibility extinction coefficient	SO ₂ not significantly linked to mortality, controlling for TSP. TSP strongly linked to mortality, the size of the coefficient was not significantly affected by the inclusion of SO ₂ . No link between O ₃ and mortality
Dockery et al. (1992)	All nonaccidental mortality records from the St. Louis metropolitan statistical area and eastern Tennessee from September 1985 to August 1986	H ⁺ , NO ₂ , O ₃ , PM _{2.5} , PM ₁₀ , SO ₂ , SO ₄ considered jointly	SO ₂ not significantly linked to mortality. A strong association was found between PM ₁₀ and mortality

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Study	Population and Study Period	Pollutants	Results
Schwartz and Dockery (1992a)	All nonaccidental mortality records from Philadelphia, from 1973-1980. The mortality records were divided into decedents < 65 years old, and decedents 65 and older.	SO ₂ and TSP considered jointly.	SO ₂ not significantly linked to mortality, controlling for TSP. No interaction found between TSP and SO ₂ . TSP strongly linked to mortality, the size of the coefficient was not significantly affected by the inclusion of SO ₂ . The effect of TSP was significantly higher on people older than 65.
Schwartz and Dockery (1992b)	All nonaccidental mortality records from Steubenville, Ohio metropolitan area, from 1974-1984.	SO ₂ and TSP considered jointly.	SO ₂ not significantly linked to mortality, controlling for TSP. TSP strongly linked to mortality, the size of the coefficient was not significantly affected by the inclusion of SO ₂ .
Dockery et al (1993)	Health data collected over 14-16 years (study ended in 1991) from 8,111 white persons in six cities in the U.S. -- Portage, WI, Topeka, KS, Watertown, MA, Kingston, TN, St. Louis, MO, and Steubenville, OH.	NO ₂ , O ₃ , PM _{2.5} , PM ₁₀ , PM ₁₅ , SO ₂ , SO ₄ , TSP and aerosol acidity considered independently.	Mortality more strongly linked to PM _{2.5} , PM ₁₀ , PM ₁₅ and SO ₄ than to NO ₂ , SO ₂ , TSP and aerosol acidity. O ₃ levels varied little and were not significant. Air pollution linked to lung cancer and cardiopulmonary disease-related deaths.
Xu et al (1994)	All nonaccidental mortality records in two residential areas in Beijing, China, from 1989.	SO ₂ and TSP considered jointly.	SO ₂ linked to mortality in both the summer and the winter. The effect remained significant with and without the inclusion of TSP in the model. TSP significant only in the summer.
Moolgavkar et al (1995)	Philadelphia mortality data collected for the period 1973-1988.	O ₃ , TSP and SO ₂ considered jointly.	With all three pollutants in model, O ₃ linked to summer time mortality and SO ₂ linked to mortality in the fall, winter and spring.

11.4 VALUING HEALTH EFFECTS

We first estimate the health effects caused by air pollution and then place a dollar value on the effects. An alternative procedure would be to use a hedonic approach (e.g., Brucato et al., 1990) and estimate the value of a reduction in air pollution. However, the hedonic approach estimates ill health, reduced visibility, property damage and other adverse effects of air pollution, and we are interested in valuing only the ill health. Furthermore the actual effects, or damages -- days of illness, lives lost, etc. -- are of interest in themselves, apart from their monetary value.

All of our dollar estimates are in 1991 \$.

11.4.1 Acute Morbidity

Economists use several methods to estimate the cost of such things as the common cold, asthma and other illnesses which we group under the category, acute morbidity. In their review article Cropper and Freeman (1991: 166) suggested two broad approaches:

- The observed market approach. It includes "techniques that rely on demand and cost functions, market prices, and observed behavior and choices." Household production functions and cost-of-illness studies (which estimate the direct out of pocket expenses of illness) are examples.
- The hypothetical market approach. It includes techniques that directly ask people's "willingness to pay or accept compensation for a postulated change, how their behavior would change, or how they would rank alternative situations involving different combinations of health and income or consumption." Contingent valuation is an example.

Ideally, the morbidity valuation method should count:

- lost productivity due to missing work;
- lower productivity at work due to illness;
- the value of uncompensated activities, such as recreation or household chores, that must be given up because of the illness;
- the value of performing uncompensated activities less well, because of illness;
- medical expenditures for treatment of illness;
- the nonmarket costs of treating illnesses (air, water, and solid waste pollution from hospitals and from the pharmaceutical industry);

- the dollar equivalent of the physical and psychological pain and discomfort of the directly affected person;
- the dollar equivalent of the physical and psychological pain and discomfort of those who care about the directly affected person (if such concern is not included in the WTP of the directly affected person), and
- the cost of averting behavior (for example, the cost of extra preventative health care, or the value of activities that are changed or limited on account of pollution).

We briefly review the two groups of techniques.

11.4.1.1 *Observed Market Approach to Estimating the Cost of Illness*

In general, economists like to observe what people do rather than what they say would do. All else being equal, we obviously would prefer values derived from real markets ("revealed" preferences) to values derived from hypothetical markets and preferences. However, as we discuss next, all else never is equal, and there are significant shortcomings to using observed market transactions to value the health effects of air pollution.

Household Production Function Approach

Using the household production approach, an economist models the relationship between people's health, the level of air pollution and actions they take to avoid or cure ill health, such as visiting the doctor, staying indoors, or purchasing air conditioning (Freeman, 1993: Chapter 10). One can use this method to estimate the change in a pollution level, its effect on people and how much people value the change. It captures the direct monetary costs of illness, and the amount people spend on averting behavior.¹

However, there are serious methodological drawbacks to this approach. If people's averting behavior produces a joint product, such as in the case of air conditioning, which removes particulates from the air *and* cools one's house, then one can not ascribe the full cost of the air conditioner to the health outcome of interest (Cropper and Freeman, 1991: 210). The approach assumes that the marginal benefit of a mitigating measure is equal to the marginal cost, which is true in principle for measures bought frequently and in small increments (such as aspirin), but not necessarily true for measures bought infrequently in large "chunks" (such as air conditioners). For the "typical" person that buys air conditioning, the marginal benefit will exceed marginal cost (i.e., there is consumer surplus).

There are other problems with this approach. It misses lost productivity from missed work when there are institutional structures that compensate people when they are sick, and it does not capture medical expenditures covered by free insurance

¹Harrington and Portney (1987: 101) suggested including averting behavior can be "critical" when estimating the benefits of a pollution reduction program

such as Medicare and Medicaid. It is difficult to measure the cost of averting behavior. What, for example, is the cost of spending leisure time indoors rather than outdoors? (Cropper and Freeman, 1993: 201). It also does not cover nonmarket costs of illness including the pain and discomfort of those who care about the directly affected person. Furthermore, this approach excludes the nonmarket costs associated with medical care (e.g., pollution from hospitals and pharmaceutical companies). Finally, the method is costly to perform, because of the extensive data needed to properly model exposure to pollution.²

Cost of Illness Approach to Estimating the Cost of Illness

The cost of illness approach equates the cost of illness with lost productivity (measured by wages) and the medical costs associated with illness.³ This approach obviously misses many cost categories, such as the loss of productivity that is not normally compensated (e.g., housework, work of retirees), pain and suffering, and averting behavior. The true cost of illness must exceed any cost estimated by this approach.

Hedonic Studies

An alternative to our approach of estimating damages and then valuing these damages is to look at the housing market and to estimate the value that people place on air pollution by looking at housing price differentials (e.g., Brucato et al., 1990; Graves et al., 1988). If for example, people are willing to pay \$1,000 for a house in an area with low pollution levels versus an area with high pollution levels, then one might say that \$1,000 is the upper bound that a household is willing to pay to avoid the health costs of air pollution. Air pollution also causes visibility loss, vegetation damage and other effects so, the reasoning goes, the health costs of air pollution may be significantly less than \$1,000. One problem with this reasoning is that it assumes that people are aware of all of the health effects of air pollution and they have a good idea of the level of pollution that they are exposed to. This seems most unlikely as there is much that researchers do not know about the health effects of air pollution and, at best, most people only have a very rough idea of the air pollution that they are exposed to. With full information on the effects of air pollution, it is likely that the values implied by housing prices will be quite different.

Brucato et al. (1990) in a study of ozone pollution in the San Francisco area, suggest that the hedonic approach and the damage-function approach give comparable results. However, in our opinion, their conclusion is likely coincidental, and it is *unlikely* that the *full* costs of ozone (and other pollutants) are built into the price of homes.

11 4.1.2 *Contingent Valuation Approach to Estimating the Cost of Illness*

²Lacking sufficient data, Dickie and Gerking (1991b) excluded particulates, an important contributor to ill health, from their analysis and therefore biased their result unacceptably.

³Previous studies include those by Brown (1990), Dixon (1985) and Rice et al (1985)

The contingent valuation approach uses a survey to directly ask people how much they are willing to pay to avoid illness. Its strength is that it can ask about any illness that people are familiar with and is a reasonably inclusive measure of the cost of illness. Contingent valuation includes all of the private costs that people incur when sick, including the potentially large category of pain and suffering. However, it underestimates or excludes costs covered by insurance, might not include the pain and suffering of those close to the [hypothetically] sick person (although there is no reason that this cannot be included in a survey), and definitely does not include the nonmarket costs associated with medical care (e.g., pollution from hospitals and pharmaceutical companies).

Some economists criticize the hypothetical nature of surveys, arguing that people might not consider carefully the opportunity cost of their money and hence might overestimate the amount they would actually be willing to pay to avoid illness. The critics also argue that respondents might not understand what they are valuing⁴, and that the survey instrument might not properly emphasize that people should take into account the cost of the averting behavior and how they will feel after the averting behavior (such as taking aspirin to avoid a headache). However, surveys can be designed and administered to minimize these problems (Mitchell and Carson, 1989).

11.4.1.3 *Estimates of the Cost of Acute Morbidity*

Table 11.4-1 presents the results from studies (mostly contingent valuation) of the value of morbidity. Dickie and Gerking (1991a) is the only household production function study that estimated the cost of symptoms; most studies of this type valued a reduction in ambient air pollution per se. In the contingent valuation surveys there is a very large difference between the mean and median bids, which is the result of very large outlying bids. The median bids are more plausible, although they have a large variance – Tolley et al. (1986) and Dickie et al.'s (1987) estimated WTP to reduce a day of coughing differs by an order of magnitude.

Table 11.4-3 presents the values of reduced morbidity used in previous studies of the social cost of air pollution. The cost of asthma is essentially the same in all of the studies, because researchers have based their estimates on the work of Rowe and Chestnut (1985) and Krupnick and Kopp (1988). The estimated values of RADs and RRADs have been taken from cost-of-illness studies because there are no relevant contingent valuation studies. All of the estimates for mRRADs reflect Krupnick and Kopp's (1988: 2-62) rationale that the WTP to avoid a mRRAD should exceed that for a single symptom, but not necessarily the WTP for a severe symptom or combination of symptoms. To value symptoms, such as coughing and eye irritation, researchers have used the results of contingent valuation studies. The data from these studies can be interpreted differently; hence the difference in the estimates.

⁴For example, Loehman et al. (1979) mailed surveys to a cross section of the Tampa Bay population asking them to value the reduction of 90 days per year of severe symptoms – probably more suffering than most people have experienced

We agree with Krupnick and Kopp (1988: 2-59), who concluded that the imprecision of the available estimates of morbidity costs preclude a definitive (point-estimate) of the cost. We review the available primary literature (Table 11.4-1), and the assumptions used in previous studies of health effects (Table 11.4-3), and decide on a plausible range for the cost of acute morbidity. Table 11.4-4 presents the values that we use for this study.

Lower Illness Cost for Children and Unemployed?

One might assume the cost of illness for children and the unemployed should be lower than the cost for working adults who are generally more productive. Hall et al. (1992), for example, valued non-working people's RAD's at half the value of working adults'. We did not because of the uncertainty in the estimated cost of illness, the lack of information in the literature addressing this problem, and the fact that we use widely different values for the lower and upper bound values.

Declining WTP

People may not value reducing the Nth symptom day as much as the first either because of income constraints, or simply because of declining marginal utility of "consumption" of health benefits Hall et al. (1989: 5-47) -- a common assumption in economics. Reviewing the available literature, Hall et al. (1989: 5-47) found that the average WTP to avoid each of N symptom days per year (WTP_N) equals the WTP to avoid one symptom day per year (WTP_1) multiplied by one over the square root of N:

$$WTP_N = WTP_1 \cdot N^{-0.5}$$

The result, however, is based in part on a survey conducted by Loehman et al. (1979), the results of which suggest the respondents did not understand the survey. The respondents gave their willingness to pay to avoid one, seven and ninety symptom-days per year; the symptoms included shortness of breath, coughing/sneezing, and head congestion, at two severity levels -- mild and severe. They gave extremely low bids, valuing a reduction of 90 days of severe shortness of breath at a little over one dollar a day (median bid), and valuing the other symptoms even less. Few respondents probably understood what it would be like to experience 90 days per year with severe symptoms (virtually a chronic condition), and thus did not value the condition correctly⁵. Furthermore the survey had a very low response rate -- only 22%.

Nevertheless there probably *is* a decline in people's average WTP. Hall et al.'s equation gives plausible results, so we use it in deriving our results.

⁵Thompson (1986) reported that people with chronic diseases were willing to pay a significant amount of money or undergo a nontrivial increase in the probability of mortality to be free of their chronic condition

Contiguous Symptom Days

We are not aware of any evidence regarding whether people value, say, two contiguous symptom days more or less than the sum of two separate symptom days. Furthermore we do not have any way of knowing whether the effects that we are estimating are contiguous or not. Therefore, we make the simplifying assumption that the utility of all symptom-days is independent on when the last symptom-day occurred.

Multiple Symptoms During a Single Day

Do people feel that multiple symptoms over the course of a day are more or less costly than experiencing the symptoms one at a time over the course of many days? Tolley et al. (1986) reported that the cost of individual symptoms is less than the cost of a combination of symptoms. However, we do not know the likelihood that air pollution-induced symptoms such as lower and upper respiratory illness occur together. Future work should account for this and adjust the cost of the symptoms using the work of Tolley et al or others.

Related to this, is the issue of double-counting effects when estimating individual symptoms such as chest discomfort and aggregated measures such as respiratory-related restricted activity days (RRAD). The double-counting arises because chest discomfort is often a symptom associated with an RRAD. To account for this, Krupnick (1988: Chapter 7) reduced the estimated number of respiratory symptoms such as chest discomfort by using the probability that a person experiences respiratory symptoms conditional on experiencing a mild respiratory-related restricted activity day -- an aggregated health measure that includes chest discomfort and other measures. We do not use Krupnick's (1988) result in the present study because for each pollutant that we consider we do not estimate an aggregated measure such as respiratory-related restricted activity day *in conjunction with* specific symptoms such as chest discomfort.

WTP Differing by Geographic Region

WTP measures may differ by geographic region, primarily because wage rates and hence the value of work differ. For example, Hall et al. (1989: 5.45) estimated different values for RAD's for different counties in the Los Angeles area. However, we do not account for any differences in the amount people from different parts of the country are willing to pay. Most regional values should fall within the relatively large range between our upper and lower bound estimates (Table 11.4-4).

Matching Problem

In valuing health effects, we merge the results from the epidemiological literature (which links air pollution to illness) and the economic literature (which places a value on illness). Ideally we would perfectly match the effect that we are estimating with the same effect from the economic literature and ensure that we appropriately value mild, moderate and severe effects. However, this is impossible to do, and the result is that we do not value the health effects that we estimate as precisely as we would like. This is the "matching problem" discussed by Krupnick (1988: 2-6).

For example, we estimate the number of respiratory-related restricted activity days (RRAD) caused by particulates, but we do not have a good measure of the cost of an RRAD, which is a broad category of effects. Hall et al. (1992) approximated its cost using assumptions about lost productivity and medical expenditures, but this does not capture all of the costs of RRAD nor does it capture the heterogeneity of effects associated with it (which includes both severe and mild illnesses).

11.4.2 Chronic Morbidity

Particulates cause asthma, chronic bronchitis and emphysema; we refer to this group as obstructive airway disease (AOD).⁶ Krupnick and Cropper (1992) reported that people are willing to pay \$0.46 million to \$2.08 million to avoid a statistical case of chronic bronchitis, and Viscusi et al. (1991) reported \$0.46 million to \$2.29 million per statistical case of chronic bronchitis. We do not have estimates of people's WTP to completely avoid asthma or emphysema; we assume it is the same as WTP to avoid chronic bronchitis. In our lower bound case we use \$0.5 million per case of COPD or AOD, and in the upper bound case we use \$2.0 million.⁷

11.4.3 Mortality

Economists do not actually estimate the value of life per se; rather, they estimate either WTP to avoid an increase in the chance of dying, or willingness to accept compensation (WTA) for the increased risk. WTP usually is estimated by surveying people; WTA, by calculating the extra pay given to workers in risky jobs. In order to compare different estimates of the value of a change in the risk of dying -- that is, estimates based on different changes in the risk of dying -- the initially different estimates must all be normalized to the same risk level. In principle, risk values could be normalized to any level -- say, to a 10% chance of dying. But, unfortunately for the economists who have done this sort of work, it has seemed natural to express all estimates in terms of a 100% chance of dying -- "unfortunately," because the risk at the 100% chance-of-dying level usually is understood as the value of life, and lay people interpret this "valuation of life" as a hard-headed, cold-hearted calculation of the worth of a human being. But as noted above, economists value risk, not life per se; no WTP survey asks how much one would pay to avoid certain death. One therefore should not view "value of life" estimates as a measure of the worth of a human life; rather, one should view them as a way of standardizing estimates of the value of changes in risk. (Recently, Krupnick and Portney (1991: 525) expressed the value of risk reduction by assigning "a value of \$1000 to each reduction of 0.001 in annual mortality risk", rather than using a \$1 million dollar value of life.)

We considered two approaches to valuing a statistical life. The first assumes that the value of all lives is the same regardless of the number of years of living lost

⁶Another common name is chronic obstructive pulmonary disease (COPD)

⁷Krupnick and Cropper (1992) and Viscusi et al (1991) do not report the year of their estimates. Our figures are in 1991 \$

In this approach, the value of a statistical life is estimated by contingent valuation studies, wage-risk studies and consumer-market studies. The second approach estimates the number of years lost and then places a value on those lost years (Fuchs and Zeckhauser, 1987). To estimate the value of deaths estimated by cross-sectional studies, we decided to use the former because it is difficult to estimate the number of years lost and the value that people place on those years. More generally we do not think that society's willingness to pay to reduce the risk of mortality is especially sensitive to the number of years saved, although it might be sensitive to the difference between one day and one year or longer.

There is some evidence that society places a less value on saving elderly people compared to younger individuals (Cropper et al., 1994). Since air pollution has the strongest effect on the elderly (Schwartz and Dockery, 1992a), we need to take notice of this when estimating the value of a statistical life. However, we do not formally account for this because we do not have separate dose-response functions for the elderly, so we do not know precisely what fraction of the deaths are of elderly individuals. Instead we simply lower our lower and upper bound estimates of the value of a statistical life.

11.4.3.1 Estimating the Value of Life: Wage Risk Studies

Wage-risk studies estimate the value-of-life by analyzing the wage differentials between occupations that are assumed equal in all regards except for the level of risk. However, for three reasons, wage-risk studies might underestimate the true cost of risk for most people. First, laborers might not be able to move freely between jobs, and hence might not be able to choose less risky occupations. Second, they might not fully understand the difference in risks of various occupations. Third, workers in risky occupations might be less averse to risk than are most people.

The quality of the estimates from these studies depends on the source of the data. Early wage/risk studies that used actuarial data (which includes all deaths, not just occupational deaths) reported relatively low estimates of the value of life – less than \$1 million (Table 11.4-5). Other early studies used occupational deaths by industry group (rather than actuarial data) and estimated that the value of life ranged between \$4.4 to \$9.5 million – almost an order of magnitude higher than in the actuarial studies (Table 11.4-5). The industry groupings in these latter studies, however, were too broad; they did not account for differences in occupational risk within an industry or non-risk differences in wages between industries. Newer studies, correcting for these problems have found slightly lower values, ranging between \$1.9 - 6.4 million (Table 11.4-5).

11 4.3.2 Estimating the Value of Life: Consumer Market Studies

Consumer market studies are similar to wage-risk studies, insofar as they examine revealed preferences. Consumer market studies look at the tradeoffs that consumers make in their consumption decisions, such as when deciding about the purchase of smoke detectors or wearing seat belts. In these studies, it is assumed that consumers equate the marginal benefits (i.e., risk reduction) with the marginal cost of the product (Fisher et al., 1989: 95). The estimated values of a statistical life are

relatively low (less than \$1 million), probably because the benefits are underestimated: either the change in the risk is mis-estimated, or else on average the consumer benefits exceed the cost, which is possible because the goods are “lumpy” and bought infrequently (see Cropper and Freeman, 1991: 183).

11.4.3.3 Estimating the Value of Life: Contingent Valuation Studies

In the contingent valuation survey approach, researchers develop an hypothetical market and ask people's willingness to pay for alternative levels of safety with the received bid used to place a value on a statistical life. Contingent valuation studies are attractive because they include a broad range of people (not just a segment of the working population, as in wage-risk studies) and important attributes of risk, such as whether the risk is voluntary or involuntary, or the death quick (e.g., traffic accident) or slow and painful (e.g., cancer). However, some studies have been criticized because people may have difficulty understanding the risk they are asked to value, particularly if the risk is very small. The estimated values of a statistical life from contingent valuation surveys have ranged from \$1.9 to \$5.3 million – slightly lower than the estimates from the more recent wage risk studies.

11.4.3.4 Estimating the Value of Life: Conclusion

The literature presents a wide range of estimates for the value of a statistical life. We use a range of estimates that capture the variability of the estimates and still have some empirical support.⁸ We estimate two classes of deaths and use two different sets of estimates of the value of a statistical life. We assume that deaths estimated from the time-series studies (acute harvest deaths) have been hastened a relatively short amount; for our lower bound estimate we use \$10,000 and for the upper bound we use \$50,000. The deaths estimated from the overlap of time-series and cross-sectional studies (acute non-harvest deaths) have been hastened a relatively longer time, and should be valued much more. The values from our literature review suggest a plausible range of \$2 million to \$5 million. However, taking into account the fact that a significant portion of the deaths that we are estimating are of the elderly or of individuals with compromised health, we use \$1 million for our lower bound and \$4 million for our upper bound.

When estimating chronic deaths, we assume that they will occur over a twenty-year period (see Table 11.3-21) and we value them at \$1 million to \$4 million at the time they occur. We then discount the value of these future deaths with an 8% and a 2% discount rate in the lower bound and upper bounds.

11.4.4 Cancer

Toxic pollutants such as benzene and diesel particulates increase the risk that people contract cancer. Cancers often have a long latency period, and are expensive and time-consuming to treat. The chance of recovery depends on many factors,

⁸Most previous health-effects studies have assumed that the value of a statistical life ranges (roughly) between \$1.8 to 9.2 million (Table 11 4-6)

including the age of the person and the type of cancer. Ideally our estimate of the cost of cancer would account for:

- the length of time between exposure to the toxic compound and the discovery of cancer;
- the amount of pain and suffering endured, medical costs incurred, and lost productivity between the discovery of cancer and the person's recovery or death;
- the probability of death; and
- the length of time between the discovery of cancer and death.

Unfortunately there is not enough information to address these issues satisfactorily, and as a result we must make simplifying assumptions about the cost of cancer. For *fatal* cancer cases, we assign the value of a statistical life at the point at which the cancer is discovered, and ignore, on the one hand, costs incurred between the time of discovery and death, and, on the other, the time between discovery and death. The omission of post-discovery costs understates the present value of the true total cost of cancer, but the failure to consider the time lag between discovery and death overstates the present value, so that these two simplifications tend to cancel.

We estimate the cost of non-fatal cancer cases on the basis of an estimate by Rae et al. (1991), and of estimates of the cost of chronic disease. Rae et al. (1991: 3-8) estimated that the sum of the direct cost of cancer (reflecting hospitalization, medication, treatment and administrative costs) and the indirect cost of cancer (i.e., foregone earnings⁹) ranges between \$18, 246 and \$602,048, with the higher figure reflecting lost income of the rich. The mean value, which they do not report, probably is closer to the lower figure. However, as Rae et al. (1991) note, theirs is a lower bound estimate, because it does not take into account pain and suffering and other personal costs of cancer. We have not found a comprehensive estimate of medical costs, lost productivity and pain and suffering costs of non-fatal cancer cases.

Presumably, people would pay at least as much to avoid non-fatal cancer as they would pay to avoid chronic bronchitis or arthritis. Krupnick and Cropper (1992) reported that people are willing to pay \$0.46 million to \$2.08 million to avoid a statistical case of chronic bronchitis; Viscusi et al. (1991) reported values of \$0.46 million to \$2.29 million per statistical case of chronic bronchitis; and Thompson (1986) reported that people with rheumatoid arthritis are willing to pay 22% of household income (or face a 27% increase in the risk of death) to be completely free of arthritis.

On the basis of these estimates, we choose \$0.5 million as a lower bound estimate of the cost of non-fatal cancer, and \$2 million for an upper bound. The

⁹The cost to society of cancer is not foregone earnings, as suggested by Rae et al (1991 3-8), because workers frequently receive some compensation when they are sick. The relevant cost is lost productivity

lower and upper bound estimates include all costs of cancer, including medical costs, pain and suffering to both the patient and friends and lost production to society.

11.4.5 Discounting future deaths

The effect of pollution often appears many years after exposure. We discount these delayed effects back from the time of discovery of the chronic illness to the time of exposure, using a rate of 2% in the upper bound and 8% in the upper bound. We discuss our choice of discount rate in Report #2 of the social-cost series listed at the beginning of this report.

We assume that the incidence of chronic disease is approximately normally distributed (the shape of the distribution is based on our judgment), and estimate the percentage of cases that occur each year. Since the valuation procedure is a nonlinear function, one should not use the average latency period between exposure and incidence.

11.4.6 The value of life in the future

11.4.6.1 Background

The single biggest component of the social cost of motor-vehicle air pollution is the cost of mortality. The mortality cost is equal to the number of deaths due to motor-vehicle air pollution, multiplied by the value of life (VOL).

Some of the deaths due to motor-vehicle air pollution occur many years after exposure to the pollution: some motor-vehicle air pollution today kills people in, say, the year 2010. Our dose-response functions tell us the number of deaths in the future that result from exposure to pollution today. The economic literature on valuation tells us the VOL today. But what is the VOL in the future, in say the year 2010, when the life is actually lost? In this section, we argue that the VOL is related in some way to real wealth, and hence that the VOL in the future, given the VOL today, can be calculated on the basis of the annual increase in wealth.

11.4.6.2 Calculating the value of life

The VOL is calculated from either the willingness to pay (WTP) to reduce the probability of death, or the willingness-to-accept (WTA) compensation for an increase in the probability of death. As discussed above, the WTP or WTA is estimated in one of three ways: 1) by comparing the additional wages -- "the risk premium" -- demanded by workers in occupations with a known, relatively high risk of death; 2) by evaluating the actual tradeoffs that people make between money or time and the risk of dying (e.g., by comparing the "cost" of seat-belt use with the benefit of reduced risk of dying); or 3) by asking people about their WTP or WTA in hypothetical situations.

Wage/risk studies

The VOL calculated from wage/risk studies obviously is a function of real wage rates. If real wages in all occupations change by X% year, then the risk premium -- loosely put, the difference between wages in risky occupations and wages in similar but safe occupations -- will increase by X% per year as well, and hence the calculated VOL will increase by X%/year as well. (Of course, it is possible

that wages in risky occupations will change at different rates than will wages in safe occupations.)

One indication, then, of the future rate of change of the VOL is the historical rate of change of hourly full compensation. "Compensation" includes gross salary, overtime, incentives, and the money value of all benefits. (We are interested in the full compensation rate, rather than the just the hourly salary, because workers can be compensated for increased risk by higher benefits as well as by higher salary.) According to the Bureau of the Census (*Statistical Abstract of the United States, 1992, 1992*), the real hourly rate of compensation in the entire "business" sector increased 1.9%/year from 1960 to 1980, and 0.6%/year from 1970 to 1990. (Part of the difference between the 1960-to-1980 rate and the 1970-to-1990 rate is that real hourly compensation leveled off and declined after 1987.) It appears that the real hourly rate of compensation rate in all sectors increased by 0.6%/year as well: the real *annual* rate of compensation in all sectors increased by 0.6%/year from 1970 to 1990 (Table 11.4-7), and the average number of hours worked per week did not change between 1970 and 1990¹⁰. Thus, on the basis of these statistics, one might expect the VOL as calculated from wage/risk studies to increase by about 0.6%/year, in real terms.

However, the VOL calculated from a wage/risk study applies only to the types of workers studied. It does not necessarily apply to workers, in different fields, with different perceptions and valuations of risk, or to officially unemployed or retired persons. Thus, not only should one not rely exclusively on wage/risk studies in order to determine the VOL today, one should not presume that the real VOL necessarily increases at the rate of increase of real hourly compensation. To get a complete picture of the rate of change of the VOL, one must look at other measures as well.

Other measures of the value of life

We believe that the other measures of VOL – the "shadow price" in actual markets, and the proffered WTP or WTA in hypothetical markets – like the measure derived from wage/risk studies, are in some way related to personal wealth. As people get wealthier they have more to pay and more lose, in both real and hypothetical markets. Hence, another way to evaluate the rate of change of VOL over time is on the basis of the rate of change of measures of wealth (other than the hourly compensation rate).

Table 11.4-7 shows the change from 1970 to 1990 in several different measures of wealth. Wages and salaries are the gross annual paycheck earnings, including overtime but not benefits, of officially employed persons. Compensation is equal roughly to wages + benefits + employer-paid taxes, and official disposable income, is equal approximately to compensation + interest earnings - taxes. ("Official" means

¹⁰According the Current Population Survey, people worked 39.1 hours per week in 1970, and 39.2 hours in 1990 (Bureau of the Census, *Statistical Abstract of the United States, 1992, 1992*). However, weekly hours by production and other non-supervisory ("blue-collar") workers actually declined significantly, from 37.1 to 34.5 (Bureau of Labor Statistics, *Employment, Hours, and Earnings, United States, 1909-94, 1994*)

that only the compensation of officially employed persons is counted.) The last row of the table, total implicit disposable income, is our creation, and is equal to official disposable income plus our estimate of the implicit value of the household labor of the officially unemployed. We have assumed that everyone who is over 16 and not employed and not in an institution (such as a prison) earns 25% of the average wages of officially employed persons.

Each of these different measures gives us some indication of the rate of change of wealth, and hence some indication of the rate of change of the VOL.

The first column of Table 11.4-7 shows each of these four measures on a per-capita basis (i.e., total wealth divided by the total population of the U.S. in 1970 or 1990). The data indicate that wealth per capita grew by approximately 1.5% per year between 1970 and 1990. This increase in per-capita income is explained by:

- i) greater participation of women (especially married women) in the work force;
- ii) increasing real disposable income per working person, in spite of declining real hourly compensation in the blue-collar sector¹¹ (as we will see, much of the increase in real disposable income per worker has come in the form of increased benefits and reduced taxes); and
- iii) greater participation of unmarried individuals¹².

One can argue that an increase in per-capita wealth is not an accurate indication of the increase in VOL, because if the increase is due mainly to greater participation, then perhaps more people are working, but not getting wealthier. However, greater participation is in some sense equivalent to an increase in the real wage rate, because new workers must be earning more in the labor force than they do at home implicitly, or else they would not go into the labor force. If the accounts all along had been done by assigning an implicit wage to homemaking, we would see a greater growth in real income per-working capita than we see in the traditional statistics.

This is borne out by the data on wealth per working person. It is interesting to note that whereas wages have barely increased from 1970 to 1990, total implicit

¹¹Average hourly earnings for production and other non-supervisory ("blue-collar") workers declined from 8.03/hour in 1970 to 7.46/hour in 1990 (1982 dollars) (Bureau of Labor Statistics, *Employment, Hours, and Earnings, United States, 1909-94*, 1994)

¹²The participation rate of a participation group is equal to the number of people in that group who are in the civilian labor force divided by the total number of people in the group who are 16 and over and not in an institution. The following are the salient statistics on the labor participation rate from 1970 to 1990.

- Old males (65 and over) participated quite a bit less in 1990, typically down from 25% to 15%.
- Married males participated less (which is something of a surprise): 86% down to 77%. This is due mainly to lower participation by married males over 45.
- Married females participated much, much more, in all age classes: participation jumped from 41% to 58%.
- unmarried persons of both sexes participated more (by about 10 percentage points), but the change was uneven across age groups and times.

disposable income – which counts the implicit earnings of the officially unemployed – has increased substantially. This is because: a) the value of benefits has increased substantially (compare compensation to wages; compensation = wages + benefits); b) either non-wage income has gone up, or taxes have decreased, or both (compare official disposable income to compensation; official disposable income = compensation + non-wage income - taxes); and c) fewer people are remaining at home working at the relatively low implicit rate of the officially unemployed (compare total implicit disposable income to official disposable income; the former assumes that the officially unemployed implicitly earn 25% of the officially employed; when participation increases, as it did from 1970 to 1990 [see above], people move from the implicit low rate of the officially unemployed to the higher rate of the officially employed). These statistics suggest that people are becoming more productive, and hence, perhaps, more valuable.

But wealth per worker and wealth per capita still might not give a complete picture of the VOL. For example, one can argue that one also should examine wealth at the household level, on the grounds that wealth and expenses are shared within a household -- that a worker is working not just on his own behalf, but on behalf of a family or a household. Thus, Table 11.4-7 presents also wealth per household, as households are defined by the U.S. Census, and in the last columns, wealth per “related” household. In the Census, a household is any group of people living together, whether related or not. However, one might argue that wealth is not shared among unrelated people living in the same household, and hence that a better measure of the “unit” within which wealth and are expenses are shared is the number of “related” households: the number of family households plus the number of individuals in non-family households.

Wealth per Census household and wealth per related household increased from 1970 to 1990. However, the increase in wealth per official Census household generally was smaller than the increase per capita or per working person, and the increase in wealth per related household smaller still. Wealth per related household grew less than wealth per Census household because the number of people living with unrelated individuals grew faster than did the number of families – loosely put, more people were “out on their own,” spreading the total amount of wealth more thinly, and so reducing the increase per related household.

11.4.6.3 *Summary*

The illustrative statistics presented above, for the period 1970 to 1990, suggest that wealth and perhaps the VOL increased 0% to 1.8% per year. Note, though, that the annual rate of increase generally was higher from 1960 to 1980 than from 1970 to 1990, because many of the wealth measures presented in Table 11.4-7 actually declined a bit after 1987. This suggests that wealth, as it relates to the value of life, will increase at between 0.5% and 2.0% per year. In our formal analysis, we assume a narrower range of 1.0% to 1.5%.

TABLE 11.4-1. ESTIMATED COST OF ACUTE HEALTH EFFECTS (1991 \$): SUMMARY OF THE LITERATURE

Health Effect	Median \$/Day	Mean \$/Day	Study Type	Population	Study
Minor Coughing/Sneezing	5.24	55.06	CV	404 respondents to a mailed questionnaire (22% response)	Loehman et al (1979)
Severe Coughing/Sneezing	14.42	95.69			
Minor Head Congestion	7.87	68.17			
Severe Head Congestion	17.04	111.43			
Minor Shortness of Breath	10.48	102.25			
Severe Shortness of Breath	23.59	166.49			
Cough	14.42	33.03	CV	40 respondents to mail survey in Denver and Chicago	Tolley et al (1986) ^a
Eye Irritation	16.39	36.35			
Sinus Problems	18.35	45.95			
Throat Congestion	17.04	37.97			
3-Symptom Combination	39.98	n.a.			
Can Not Breath Deeply	1.31	1494.47	CV	226 non-smoking adults in Glendora and Burbank, CA in telephone survey.	Dickie et al (1987) ^a
Pain on Deep Inspiration	4.59	1250.81			
Shortness of Breath	0.00	10.33			
Wheezing	2.63	76.03			
Chest Tightness	6.55	1066.74			
Cough	1.31	465.52			
Throat Irritation	3.93	19.66			
Sinus Congestion	4.59	313.97			
Headache	1.31	233.85			
Symptom (Normal Group) ^b	n.a.	0.76	HPF	Same as Dickie et al (1987)	Dickie and Gerking (1991a)
Symptom (Impaired Group)	n.a.	1.17			
Health Effect	Low \$/Day	High \$/Day	Study Type	Population	Study
Asthma Attack	11.80	56.37	CV	64 adult asthmatics in Glendora, California.	Rowe and Chestnut (1985) ^a
Asthma Attack	1.69	47.32	COI	U S	Krupnick and Kopp (1988)
RAD	63.91	83.48	COI		Hall et al (1989 5-45)

CV = contingent valuation, HPF = household production function; COI = cost of illness, RAD=restricted activity day (see Table 11.3-1) n.a.=not available

^aThe original paper was not immediately available, so we used the values published in Krupnick and Kopp (1988) and Cropper and Freeman (1991, Table 6.3) We used the Consumer Price Index for all goods (Table 11 4-2) to update all of the estimates to 1991's \$.

^bThe "symptom" was any one of 26 symptoms, such as chest and throat problems One hundred and twenty-six of the study group reported chronic respiratory problems and were placed in the "impaired" group, the remaining 100 respondents were placed in the "normal" group

TABLE 11.4-2. U.S. CONSUMER PRICE INDEX AND MEDICAL PRICE INDEX (1982-4=100)

Year	CPI^a	Medical Care Price Index
1982	96.5	92.5
1983	99.6	100.6
1984	103.9	106.8
1985	107.6	113.5
1986	109.6	122.0
1987	113.6	130.1
1988	118.3	138.6
1989	124.0	149.3
1990	130.7	162.8
1991	136.2	177.0
1992	140.3	--

Source: U.S. Council of Economic Advisors (August 1992: 23) and Small and Kazımı (1995)

^aThe Consumer Price Index includes all consumer items

TABLE 11.4-3. ESTIMATES OF THE COST OF ACUTE ILLNESS USED IN PREVIOUS STUDIES OF THE COST OF AIR POLLUTION (1991 \$)^a

Symptom	Low	High	Study
Asthma Attack Symptom-Day (Cough, Eye Irritation, Any Symptom [ARD2]) mRRAD	11.80 3.28 14.42	53.75 13.11 39.98	Krupnick and Kopp (1988 Table 2-6)
Asthma Attack Cough Eye Irritation Lower Respiratory Illness mRRAD Nose/Throat Irritation Upper Respiratory Illness	11.80 1.73 6.55 2.00 13.06 3.01 5.07	56.37 13.66 14.22 15.20 40.30 10.72 8.93	Krupnick (1988 Table 6-9)
Asthma Attack Symptom-Day (Cough, Eye Irritation, Any Symptom [ARD2]) RAD	10.42 3.13 10.42	41.68 10.42 31.26	Krupnick and Portney (1991 526)
Asthma Attack Eye Irritation mRAD RRAD	11.77 3.25 15.55 31.10	53.62 13.08 40.31 80.60	Harrison and Nichols (1990 Tables 6, 13)
Asthma Attack Cough Eye Irritation Headache mRAD RRAD	11.54 3.29 3.29 3.29 14.28 23.06	54.92 14.28 14.28 14.28 40.09 73.04	RER (1990 Table 26)
Cough Eye Irritation Headache mRAD RAD Sore Throat	1.82 3.13 3.13 15.11 55.23 3.91	14.59 16.93 22.93 38.82 55.23 17.45	Hall et al (1992 14)

mRRAD = minor respiratory-related restricted activity day, RAD = restricted activity day, mRAD = minor restricted activity day, RRAD = respiratory-related restricted activity day (see Table 11.3-1)

^aWe used the Consumer Price Index for all goods (Table 11.4-2) to update figures to 1991's \$

TABLE 11.4-4. COST OF A DAY OF ACUTE HEALTH EFFECTS, IN THIS STUDY (1991 \$)

Health Effect	Low \$/Day ^a	High \$/Day ^a
ARD2	3 00	14.00
Asthma Attack-Day ^b	10 00	50 00
Eye Irritation-Day ^c	3 00	14 00
Headache-Day ^c	3.00	14.00
Excess Phlegm-Day ^c	3.00	14 00
Lower and Upper Respiratory Illness ^d	3 00	14.00
RRAD	20.00	70.00
Sore Throat-Day ^c	3.00	14.00

^aAs we discuss in the text, these costs apply to the entire population. We use judgmental values based in large part on the of Krupnick and Portney (1991); Krupnick (1988 Chapter 6) and Krupnick and Kopp (1988) discussed using judgemental estimates

^bHolguin et al (1985 265) defined an asthma attack to contain three parts: self-reported symptoms, an increase in asthma medication and a decrease in expiratory peak flow. They then determined the probability of an asthma attack on any given day. Since the dependent variable is the presence or absence each day of any asthma attack, the appropriate dollar value should be what society on average is willing to pay to avoid an asthma attack-day. Not only asthmatics should be included in this WTP measure; nonasthmatics may also value a reduction in the pain and suffering of asthmatics.

^cThe incidence of eye irritation, headache, phlegm, and sore throat are measured in "symptom-days." What exactly are symptom-days? Schwartz and Zeger (1990), whose results we use to estimate air pollution-caused eye irritation, headache, phlegm, and sore throat, did not define a symptom-day. In the original survey performed in the early 1960's, respondents stated whether they had a symptom on a given day and the severity of the symptom (mild, moderate or severe). (It is not reported in Hammer et al [1974] or Schwartz and Zeger [1990] what the respondents were instructed concerning the determination of symptom severity.) Hammer et al. (1974, 256), who analyzed the original diary data used by Schwartz and Zeger (1990), reported that virtually all symptoms were mild and subsequently ignored symptom severity when they calculated the incidence rate for each symptom. We assume that Schwartz and Zeger (1990) did the same thing. Thus the estimated symptom-days are days where people experience a generally mild disturbance.

^dLower respiratory symptoms include dry cough, phlegm, shortness of breath, wheezing, chest discomfort or pain, chest cold, croup, asthma, and medically diagnosed bronchitis, influenza or pneumonia. Upper respiratory symptoms include sinusitis, runny nose, dry, scratchy throat, sore throat, head cold and hay fever. In this study, the values shown are applied to respiratory illnesses excluding asthma, which is valued separately.

TABLE 11.4-5. ESTIMATES OF THE VALUE OF A STATISTICAL LIFE

Study	Mean Risk Level for the Sample (deaths/10⁴ per.)^a	Range of Estimates (10⁶ 1991\$)^b	Judgmental Best Estimate (10⁶ 1991\$)^b
Early Low-Range Wage Risk Studies^c			
Arnould and Nichols (1983)	11.0	0.86	0.86
Thaler and Rosen (1975)	11.0	0.52 - 1.00	0.76
Early High-Range Wage Risk Studies^d			
Olson (1981)	1.0	9.5	9.5
R. Smith (1976)	1.0 & 1.5	4.3 - 4.7	4.4
V.K. Smith (1983) ^e	3.0	2.3 - 6.9	4.7
Viscusi (1978)	1.2	4.9 - 6.0	5.1
Recent Wage-Risk Studies^f			
Dillingham (1985)	1.4 - 8.3	2.5 - 6.9	3.0
Gegax et al. (1985)			
all union workers	8.2	2.3	
union blue-collar workers	10.1	1.9	1.9
Marin and Psacharopoulos (1982) ^g			
manual workers	2.0	3.2 - 3.7	3.5
nonmanual workers	2.0	10.7	
Moore and Viscusi (1988a)	0.79	6.0 - 7.8	6.4
Contingent Valuation Studies			
Gerking et al. (1988) ^h	4.2 - 10.0	2.9 - 3.9	3.1
Jones-Lee et al. (1985) ⁱ	0.8 - 1.0	1.9 - 5.3	3.6
Averting Behavior Studies			
Blomquist (1979) ^j	3.0	0.45 - 1.67	0.73
Dardis (1980) ^k	0.9	0.43 - 0.67	0.55
Ippolito and Ippolito (1984) ^l	varied	0.29 - 1.50	0.62

Source: Adapted from Fisher et al. (1989 Table 1) and Cropper and Freeman (1991 Table 6.1)

^aApproximate annual deaths per 10,000 people

^bWe updated the figures to 1991's \$ using the Consumer Price Index (Table 11.4-2)

^cBased on actuarial data

^dBased on industry accident data from the U.S. Bureau of Labor Statistics

^eAssuming 0.4 percent of all injuries are fatal, as reported by Viscusi (1978) for the U.S. Bureau of Labor Statistics injury statistics, and that the risk premium for fatal injuries is 33 to 100 percent of the premium for all risks

^fThis group of studies controlled for risk by occupation, but did not control for nonfatal occupational risks

^gStudy based on occupational data from the United Kingdom. Their age-adjusted normalized risk variable is not directly comparable with the risk levels used in other studies. However, the average risk of death for the entire sample was 2 in 10,000

^hRespondents were asked how large a decrease in wages they would accept for a specified reduction in their job-related risk of death.

ⁱStudy examines people's WTP to reduce the risk of serious motor vehicle accidents in Great Britain

^jBased on people's use of seatbelts

^kBased on people's purchases of smoke detectors

^lBased on people's change in smoking habits after publication of the Surgeon General's warning about the risks of smoking

TABLE 11.4-6. ESTIMATES OF THE VALUE OF A STATISTICAL LIFE USED IN HEALTH EFFECTS STUDIES (MILLION 1991 \$)

Source ^a	Low	Mid	High
Small and Kazimi (1995)	2.0	4.7	11.0
Hall et al. (1992: 815)	1.8	4.0	9.2
Harrison et al. (1992: 33) ^b	—	2.1	4.2
Krupnick and Portney (1991: 525) ^c	—	1.0	—
Rae et al (1991: 3-9)	1.9	4.2	9.5
RER (1990: IV-13)	1.8	4.0	9.2
Rowe et al. (1986: 2-26)		2.6	
This study, slightly premature death ^d	0.01	—	0.05
This study, premature death ^e	1.0	—	4.0

^aWe use the Consumer Price Index (Table 11.4-2) to update the values in these studies to 1991\$

^bHarrison et al. (1992) first estimated the value of a year of lost life, by dividing the value of a statistical life as estimated in wage-risk studies (\$4 to \$8 million) by the number of remaining years of life at risk in wage-risk studies (estimated to be 40 years). They then multiplied the value per year by the estimated number of years lost to premature death due to particulate air pollution (21 years)

^cKrupnick and Portney (1991) assumed that most of the mortality caused by air pollution occurs among the old and sick, and that these groups have a relatively low value of a statistical life.

^dRefers to deaths that in the absence of air pollution would have occurred shortly after they actually did occur

^eRefers to deaths that in the absence of air pollution would have occurred long after they actually did occur

TABLE 11.4-7. MEASURES OF PERSONAL WEALTH, 1970 TO 1990

	Wages and salaries ^a	Compensation of employees ^b	Official disposable income ^c	Total implicit disposable income ^d
<i>1987\$/capita^e</i>				
1970	7,615	8,525	9,960	11,336
1990	9,601	11,533	14,225	15,633
1970-1990	1.012	1.015	1.018	1.016
<i>1987\$/worker^f</i>				
1970	19,161	21,452	25,062	16,556
1990	19,974	23,992	29,593	20,497
1970-1990	1.002	1.006	1.008	1.011
<i>1987\$/official HH^g</i>				
1970	24,418	27,338	31,938	36,351
1990	25,581	30,727	37,900	41,652
1970-1990	1.002	1.006	1.009	1.007
<i>1987\$/related HH^h</i>				
1970	23,461	26,266	30,687	34,927
1990	23,391	28,096	34,655	38,086
1970-1990	1.000	1.003	1.006	1.004

From the U.S. Bureau of the Census, *Statistical Abstract of the United States, 1992* (1992) HH = household See text for definition and discussion of measures

^aIncludes overtime, excludes benefits

^bEqual roughly to wages plus benefits.

^cEqual roughly to compensation plus non-wage income minus taxes

^dEqual to official disposable income plus the implicit earnings of the officially unemployed, calculated at 25% of the wage rate of the officially employed

^eThe denominator is the total U.S. population

^fThe denominator is either the total official labor force (in the case of wages and salaries, compensation, and official disposable income), or the total non-institutional population 16 years or older.

^gThe denominator is the number of households reported by the U.S. Census

^hThe denominator is the number of family households plus individuals living in non-family households

11.5 THE CONTRIBUTION OF MOTOR-VEHICLE EMISSIONS TO AMBIENT POLLUTION AND THE HEALTH EFFECTS OF AMBIENT POLLUTION

11.5.1 Introduction

To estimate the health costs of emissions from motor-vehicles, we need to estimate the change in ambient air pollution due to motor vehicle emissions, because as shown in Chapter 11.3, our dose-response functions estimate changes in human health as a function of changes in ambient air pollution:

$$\Delta E = f(\Delta P, O) = f(PI, PP, O)$$

where:

ΔE = the change in the effect of interest (human health, crop production, or visibility)

ΔP = the change in ambient air pollution

O = other variables (such as population or incidence rate)

PI = actual ambient air quality in each county in the U.S

PP = the pollution level after the change in pollution -- in this analysis, the level had there been no motor-vehicle-related emissions from all motor vehicles or a class of motor vehicles.

In this chapter, we summarize how we estimate PP , the pollution level for three scenarios: removing all anthropogenic emissions, removing 100% of motor-vehicle-related emissions, and removing 10% of motor-vehicle-related emissions; we give a detailed explanation in Report #16 in the social-cost series listed at the beginning of this report. (The initial pollution level, PI , is the actual ambient air quality in each county in the U.S. These data, and the data for any of the other variables O , such as population, are discussed in other chapters of this report.)

The contribution of an emissions source to ambient pollution at an air-quality monitor depends on the strength and location of the source relative to other sources, and the atmospheric chemistry and physics intervening between the source and the point of exposure. In principle, one could estimate the contribution of various sources to ambient air pollution by modeling emissions, dispersion and atmospheric chemistry for all emission sources. However, this method requires an enormous amount of data and computer time, and often is accurate to within only 30%. We adopt a different, simpler approach that relies on a county-level emissions inventory (EPA, 1995a, 1995b) which we adjust to account for the location of emissions sources relative to people and, in the case of particulate, takes into account the relative potency of different particulate sources.

11.5.2 General modeling

In each county, we estimate PP , the pollution level without motor-vehicle-related emissions, on the assumption that the ratio of PP to PI (initial pollution in each AQCR or county) is equal to the ratio of the *modeled* PP to *modeled* PI :

$$\text{Assume: } \frac{PP}{PI} = \frac{PP^*}{PI^*}$$

1)

$$PP = PI \cdot \frac{PP^*}{PI^*}$$

where:

PP = the estimated actual pollution level after the change in pollution
(eliminate all anthropogenic emissions, or eliminate 10% or 100% of motor-vehicle-related emissions)

PI = the actual total ambient pollution level (data from air-quality monitors; discussed in Chapter 11.2)

PP* = the modeled level of pollution after the change in pollution

PI* = the modeled level of total ambient pollution.

Thus, in order to estimate PP, we must develop a model of ambient pollution, and estimate the ratio of PP* to PI* in each county.

In general, ambient air pollution at particular time and place is a function of the amount of pollutants emitted per unit time, the physical dispersion of the emissions from the emissions source to the site where the ambient pollution is being measured, and chemical transformations of pollutants. Dispersion and chemical transformations are a function of topography, meteorology, the mix of pollutants, and other factors. Formally:

$$PI_{P^*} = f(E_{P',i}; D_{P',i}(d, h, m, t...); C_{P' \rightarrow P}(s, m, t...))$$

2)

$$PP_{P^*} = f(E_{P',i}; D_{P',i}(d, h, m, t...); C_{P' \rightarrow P}(s, m, t...))$$

where:

PI_{P*} = the modeled initial level of ambient pollution P, at a particular time and place

PP_{P*} = the modeled level of pollution P at a particular time and place, after the change in emissions

P = the ambient pollutant, measured at the ambient air-quality monitors and included in health, crop, or visibility damage functions: carbon monoxide (CO), ozone (O₃), nitrogen oxides (NO_x), total suspended particulate matter (TSP), particulate matter less than 10 microns in aerodynamic diameter (PM₁₀), and particulate matter less than 2.5 microns (PM_{2.5})

E_{P',i} = emissions of P' from source i, over some time period

p' = the emitted pollutant: CO' (-> CO), PM_{2.5-10'} (also called "coarse" PM₁₀) (-> PM₁₀), PM_{2.5'} (-> PM_{2.5}, PM₁₀), NO_{x'} (-> NO₂, O₃, PM₁₀, PM_{2.5}); volatile organic compounds (VOCs'; -> O₃, PM_{2.5}), SO_{2'} (-> PM₁₀, PM_{2.5}), ammonia (NH_{3'} -> PM₁₀, PM_{2.5})

- $D_{p',i}(d,h,m,t\dots)$ = the dispersion of emissions P' from source i , as a function of distance (d), height (h), meteorology (m ; e.g., wind, temperature), topography (t), and other factors
- $C_{p' \rightarrow p}(s,m,t\dots)$ = the chemical transformation of emissions of P' to ambient pollutant P , as a function of the mix of pollution (s), meteorology (m), topography (t), and other factors
- $E_{p',i}$ = emissions of P' from source i over some time period, *minus* the emissions that are presumed to be eliminated; in other words, the emissions of P' from source i that remain after the hypothetical change in emissions has occurred

Note that we distinguish between ambient air pollutants (P), measured at air-quality monitors, and emitted pollutants (P'), which disperse, and in some cases participate in chemical reactions, to become ambient, measured pollutants. Emitted pollutants can be the same chemical compounds as ambient pollutants (e.g., carbon monoxide [CO] is emitted, and also is an ambient pollutant), or can be involved in chemical reactions that produce ambient pollutants (e.g., volatile organic compounds [VOCs] are emitted, and are involved in the atmospheric formation of ozone).

To model the link between emissions and ambient air pollution we make several simplifications:

1). We assume that in each county c , the ambient pollution measured at the air-quality monitors is a function of:

- i) emissions in county c , and
- ii) emissions from other counties in the same Air Quality Control Region (AQCR)¹ as county c .

In essence, we model emissions from two source areas, or bands: the county of the monitor, and the band of counties around the county of the monitor. As explained next, we do this as a compromise between the impossible task of modeling emissions from every individual source and the oversimplification of having only one set of emission sources per air basin.

Recall that we estimate ambient air quality, as measured at EPA-ambient air-quality monitors, in each county. Ideally, we would model air quality in each county as a function of emissions from every source that contributes in any way to air quality in the county. This would require that we formally locate and characterize every individual emissions source, define air basins and pollution transport regions, and model air quality as a function of all effective emissions sources. Unfortunately, we do not have the data or resources to be able to do such detailed modeling for every county and air basin the U. S.

Rather than model the effect on air quality of every individual emissions source, one can define bands or regions of emissions, each with an effective "center"

¹Air quality control regions are defined in "Protection of Environment" Title 40 *Code of Federal Regulations* Part 81, 1995 ed

of emissions, and model the effect on air quality of emissions from these bands. The greater the number of bands or regions (as aggregations of emissions sources), the greater the precision, but the greater the data and analytical requirements. Our balance is to choose two emissions "bands," or areas: the county of the air-quality monitor in question, and the counties outside of this county but within the same AQCR. Within the county, we will estimate the actual effective location of different source categories (highway vehicles, power plants, off-road vehicles, construction, and so on). In the outside counties, we will assume a single effective location for all emission sources.

2). We ignore the transport of pollution from one AQCR to another, and assume that pollution within an AQCR is a function only of emissions within the AQCR. This assumption obviates the difficulties of analyzing long-range pollutant transport, and hence greatly simplifies our analysis.

3). We assume that emissions of precursor pollutants P' disperse as P' from the source to the receptor (the ambient air-quality monitor), and then at the receptor undergo any chemical transformations to produce ambient pollutant P . For example, we assume that VOC and NO_x emissions disperse as such from anywhere in the AQCR to the receptor in the county of interest, and at the receptor then are converted into ozone (O_3). We make this assumption because we cannot easily model chemical transformations as a function of the distance from the source.

4). In equation 1, we estimate the *ratio* PP^*/PI^* ; we do not estimate PI^* and PP^* individually in units of concentration ($\mu\text{g}/\text{m}^3$). We do this because there is less uncertainty in modeling dispersion from one source *relative* to another than in modeling dispersion in absolute terms. Our model estimates the dispersion of emissions from non-motor-vehicle sources relative to dispersion of emissions from light-duty motor vehicles. With this relative model of dispersion, we can estimate the ratio PP^*/PI^* , but not PP^* and PI^* individually.

5). In the cases where we model the chemical transformation of precursor emissions to ambient pollutants (VOCs , $\text{NO}_x \rightarrow \text{O}_3$; NO_x , SO_x , NH_3 , $\text{VOCs} \rightarrow \text{PM}_{10}$, $\text{PM}_{2.5}$), we ignore meteorology and topography and assume that the ambient pollution is a function only of the amount precursor emissions at the site of the monitor.

There are sophisticated models of emissions, dispersion, and atmospheric chemistry. However, it is time consuming and expensive to run all of the best models for every region in the U.S. To keep our task manageable, we will:

- use the results from the best available emissions models;
- treat dispersion very crudely;
- use an extremely simple nonlinear model of tropospheric ozone chemistry;
- greatly simplify tropospheric aerosol chemistry.

In the following sections, we discuss our simple models of ozone and aerosol chemistry. Details on these models, and on the treatment of dispersion and emissions, are given in Report #16 in the social-cost series listed at the beginning of this report.

11.5.3 Estimating the Contribution of Motor Vehicles to Ozone Formation

Ozone is not emitted as such by motor vehicles or any other source, but rather forms in the atmosphere from a series of photochemical reactions that involve NO_x , VOCs, and other compounds. The reaction rate and equilibrium depends on the relative abundance of the reactants, temperature, atmospheric mixing, and other factors. The reactions are complex and highly nonlinear, and there is no simple, universally accurate formula for determining the marginal contribution of each emission source or each precursor pollutant to ozone.

The most accurate way to estimate the contribution of each precursor or set of precursors to ozone -- i.e., to estimate $C_{p \rightarrow p}$ in equation 6 above -- is to run photochemical grid models with and without the precursor emissions from specific sources and estimate the change in the ozone level. But obviously this is very costly to do for the entire U.S. Instead, we assume a simple nonlinear relationship between VOCs (weighted according to their ozone-formation potential), NO_x , and ozone. Although this is a very crude basis for apportioning ozone damages, especially given the sophistication of regional ozone modeling, it almost certainly is not likely to be so much in error as to have a significant effect on our results.

We assume the following simple nonlinear relationship between ozone levels and VOC and NO_x emissions:

$$\text{Oxidant} = k \cdot (\text{Hydrocarbons})^A \cdot (\text{NO}_x)^B \quad (3)$$

This form has been used by others. For example, Schwing et al. (1980) used the following equation, taken from Merz et al. (1972), to estimate ozone formation in Los Angeles:

$$\text{Oxidant} = k \cdot (\text{Hydrocarbons})^{0.15} \cdot (\text{NO}_x)^{0.54}$$

Schwing et al. (1980) assumed that the estimated functional form stayed constant over all pollution levels and that it was generalizable to the rest of the cities in the U.S.

We will use equation 3 to estimate the contribution of motor-vehicle VOC and NO_x emissions to ambient ozone. Like Schwing et al. (1980), we will use a single equation for all regions and conditions in the U.S. However, we doubt that Schwing et al.'s (1980) estimates of the exponents A and B (0.15 and 0.40), developed many years ago for Los Angeles, apply to all cities in the U.S. today. We chose $A = 0.55$, and $B = 0.40$, which results in an ozone sensitivity to VOC of around 0.6, and an ozone sensitivity to NO_x of slightly less. The final form of our equation is therefore:

$$\text{Ozone} = (\text{VOCs})^{0.55} (\text{NO}_x)^{0.40}$$

Reactivity-weighted VOC emissions. The rate of ozone formation depends on the specific type of organic compound involved: some compounds, such as methane, react relatively slowly; others, such as some alkenes, react quite rapidly -- about two orders of magnitude more rapidly than does methane. Beyond this, the rates and equilibria of other reactions in the atmospheric chemistry of ozone also are determined by the specific mix of organic compounds involved. Thus, overall, the amount of ozone formed from VOC and NO_x emissions depends very much on the specific mix of individual organic compounds within the broad class "VOCs" (Nation Research Council, 1991).

Different emission-source categories emit very different mixes of organic compounds. For example, motor vehicles emit lots of relatively reactive alkenes, whereas natural-gas pipelines leak mainly unreactive alkanes. Because the mix of VOC emissions varies from source to source, and the ozone-creation potential of different VOC mixes varies widely, it is important to account for the different ozone-creation potential of different emission source categories.

In order to estimate the ozone-creation potential of different VOC-emission sources, one must: a) define a measure of ozone-creation potential; b) estimate the ozone formation potential of individual organic compounds; and c) estimate emissions of individual organic compounds from each source category. This can be a tall order, but fortunately for us, Derwent et al. (1996) have essentially done this already. They estimated the photochemical ozone-creation potential (POCP) of a large number of reactive hydrocarbons, under European conditions; estimated emissions of individual VOCs in each source category in the United Kingdom's emissions inventory; and then multiplied emissions of each compound by its POCP and summed over all VOC emissions within a source category, to produce an overall POCP-weighted VOC emission for each of the source category in the United Kingdom's emissions inventory.

The results of the Derwent et al. (1996) analysis, which we use here, are summarized in Table 11.5-1. We assume that POCPs estimated for European conditions are similar to POCPs for U. S. conditions, and that the mix of VOCs in each source category in the U. K. inventory is similar to the mix in the corresponding category in the U. S. emissions inventory.

11.5.4 Secondary Particulate Formation

Emissions of sulfur dioxide (SO₂), nitrogen oxides (NO_x), and ammonia (NH₃) interact with water vapor, hydrocarbons, dust, and other carbons to form particles of ammonium sulfate and ammonium nitrate, emissions of VOC contribute to the formation of secondary organic aerosols. Because "secondary" particulate matter can constitute a sizable fraction of the total ambient particulate matter measured at air quality monitors, and because emissions of the precursors can vary substantially from source to source, it is important to have at least a simple model of the formation of secondary particulate matter from emissions of SO₂, NO_x, NH₃, and VOC.

Unfortunately, but not surprisingly, it is not easy to model secondary aerosol chemistry. According to Herrick and Kulp (1987):

Reactions of the precursors with other chemical entities, often formed from photolysis, begin immediately on emission, and depending on emission rate, weather, and air concentration of all reactants may proceed at different rates. Some reactions will take place in minutes, others in days. In the meantime, the pollutants and their products are being transported, diluted, deposited, and augmented by new emissions along their path (p. I-4).

Nevertheless, in Report #16 in the social-cost series listed at the beginning of this report, we develop a simple model of the formation of secondary particulates that we use to estimate the contribution of motor vehicles to particulates.

11.5.4.1 *Estimating Effective PM₁₀*

As we noted in Chapter 11.3, the effect of particulates appears to depend on both its chemistry and its size. We assume that larger particles are less harmful than smaller particles, and that geologic particles are more inert and less biologically damaging than are combustion particles. We make these distinctions in order to more accurately estimate the contribution of each particulate emissions source (motor vehicles, road dust, etc.) to health effects of particulate air pollution.

Ideally, we would have a separate dose-response function for each particle size class (PM_{2.5} and coarse PM₁₀), for each health effect. Then, we would treat each particle size class as a different pollutant: we would estimate the change in the concentration of each PM type, due to a change in emissions, and then use the PM-type-specific dose-response functions to estimate the changes in health effects.

However, as presented in Chapter 11.3, we do not have separate dose-response functions for each PM size class; rather, for each health effect we have one function that relates health effects to one measure of PM pollution. Because coarse and fine PM are so closely correlated, any single measure of PM pollution – TSP, PM₁₀, or PM_{2.5} – almost certainly captures the effects of all PM pollution, unless the original epidemiological analysis isolated the effect of size, perhaps by including different size classes in the model. Thus, regardless of what the independent pollution variable in our dose-response function actually is, we can presume that it is influenced by emissions of all sizes of PM.

Our task, then, is to “apportion” the estimated PM health effects to individual PM emission sources according to the sizes (and types) of PM emitted from each source. A source that emits a kg of PM₁₀ that consists mainly of large particles contributes less to the health effects, and hence should get less of a weight, than does a source that emits a kg of PM₁₀ that consists mainly of fine particles, all else equal.

These health-effect “weights” are applied to the contribution of each size class to the $\mu\text{g}/\text{m}^3$ concentration of all PM. Thus, if coarse PM and fine PM contribute equally to the $\mu\text{g}/\text{m}^3$ concentration, but fine PM is twice as potent – that is, causes twice the effects per $\mu\text{g}/\text{m}^3$ – then fine PM is responsible for 2/3 of the total PM health effects estimated with the PM dose-response function. The point here is that the weights – the relative health-effects per $\mu\text{g}/\text{m}^3$ – are applied to the contribution to the $\mu\text{g}/\text{m}^3$ concentration, not to the emissions. Thus, we must estimate the

contribution of each particle size class to the $\mu\text{g}/\text{m}^3$ concentration. This is accomplished by the simple dispersion modeling discussed above.

TABLE 11.5-1. EMISSIONS, POCP-WEIGHTED EMISSIONS, AND POCP- ADJUSTMENT FACTORS FOR VARIOUS VOC-EMISSION SOURCES

Source category in U. K. emissions inventory	Source category in U.S. emissions inventory	Emissions in U.K. (kt/yr)	POCP-weighted emissions in U. K. (kt/yr)	POCP adjustment factor ^a
Petrol exhaust	Gasoline vehicle exhaust ^b	652	506	0.78
Petroleum refining and distribution	Petroleum and related industries	134	83	0.62
Petrol evaporation	Gasoline vehicle exhaust ^b	143	87	0.61
Solvent usage	Solvents and storage	787	461	0.59
Stationary combustion	Fuel combustion by electric utilities; Fuel combustion by industry; Fuel combustion by other; Other combustion	56	27	0.49
Diesel exhaust	Diesel vehicles; Non-road engines	175	77	0.44
Industrial and residential waste	Waste disposal and recycling	3	1	0.28
Natural gas leakage	None	34	9	0.26
Chemical processes	Chemicals and allied products; Metals processing; Other industrial processes	200	43	0.21
Biogenic emissions ^c	Biogenic emissions	n.a.	n.a.	1.1

From Derwent et al (1996), except "Source category in U S emissions inventory," which is our matching POCP = photochemical ozone-creation potential

^aEqual to the ratio of POCP-weighted emissions to unweighted emissions Derwent et al (1996) refer to this as the "sector-mean POCP"

^bOur runs of EMFAC7F (California Air Resource Board's motor vehicle emission model), and the analysis by Ross et al (1995), suggest that vehicular evaporative emissions (including refueling emissions, but not further "upstream" emissions) are about 0.4, and vehicular exhaust emissions about 0.6, of total (exhaust + evaporative) VOC emissions from motor vehicles

^cBased on POCP estimates of biogenic VOCs (e.g., terpene) from Derwent et al. (1996)

11.6 HEALTH EFFECTS CAUSED BY TOXICS

The effects of toxics from motor vehicles are difficult to estimate. There are many toxic compounds, and a large number of them are not well characterized. Toxics can have a variety of effects: they can be acute or chronic poisons, or cause cancer or birth defects or genetic mutations. It is difficult to model human exposure, and more difficult still to estimate defensible dose-response functions.

Ours, then, is not a complete estimate of all of the effects of all the toxic pollutants emitted by motor vehicles. Rather, we estimate the cancerous effect of only a fraction of the chemicals associated with motor vehicles. There are many other chemicals that may cause cancer that we do not consider, mainly because we can not estimate people's exposure or because the relevant studies have not been performed.¹ Furthermore, there may be chronic, acute, teratogenic or mutagenic effects that we do not consider.

Researchers have reported an increased risk of cancer in gasoline attendants, auto mechanics, truck drivers, and others exposed to motor vehicle-related pollutants. Grandjean and Andersen (1991) found a 12% higher rate of respiratory cancer in Danish gasoline station workers, compared to all employed males. Hayes et al. (1989) reported a higher incidence of lung cancer in occupations associated with motor vehicle exhaust (e.g., truck drivers), and Bender et al. (1989) reported higher rates of some types of cancer in Minnesota highway workers. Hoover and Fraumeni (1975), Blot et al. (1977), Blot and Fraumeni (1976), Gottlieb et al. (1982), Kaldor et al. (1984) reported a higher incidence of cancer mortality in counties with petroleum industries, and Theriault and Goulet (1979), Thomas et al. (1980), and Wong and Raabe (1989; 1990) reported higher cancer rates in petroleum workers.

Aside from increasing the risk of cancer, toxics and other chemicals adversely affect people by being unpleasant to smell, increasing morbidity (e.g., eye irritation, nausea),² and contributing to "environmental illness" or "multiple chemical sensitivity". Some people are extraordinarily sensitive to odorous chemicals, susceptible to effects at pollutant levels "orders of magnitude lower than levels known to cause symptoms by classical toxicologic or irritative mechanisms" (Shusterman, 1992: 81; see also Ziem and Davidoff, 1992). Goldsmith (1972; cited in Shusterman, 1992) reported a positive link between the intensity of odor exposure (validated by olfactometry) from petroleum refineries and self-reported cases of eye and nose irritation, dizziness, and nausea. Nevertheless, as we stated above, the data enable us to consider only the carcinogenic effects of toxics.

¹In a survey of the petrochemical industry, Cottle and Guidotti (1990) presented a long list of potentially dangerous chemicals. The International Agency for Research on Cancer (1989) also presented a long list of potentially dangerous chemicals in its discussion of the processes and chemicals involved in petroleum refining.

²The California Air Resources Board (1991a) and Altshuller (1993) reported formaldehyde causing eye irritation.

11.6.1 Cancer Estimating Procedure

Paustenbach et al. (1990) and Freudenberg (1988) discussed the difficulties in quantifying the deleterious effects of toxics and other chemicals:

- scant data on ambient concentrations of toxics and resulting exposure;
- uncertainty regarding the size of the effect;
- uncertainty (particularly in the case of cancer) as to when the effects occur; and
- uncertainty about the appropriate dose-response function (particularly when multiple chemicals may combine to have more or less of an effect than when examined separately).

We use four steps to value the increased incidence of cancer caused by motor vehicles. First, we estimate toxics exposure from motor vehicles, using the results from a model (Hazardous Air Pollutant Exposure Model for Mobile Sources, or HAPEM-MS) developed for the EPA (1993a). Second, given estimates of exposure to six toxic pollutants (acetaldehyde, 1,3-butadiene, benzene, diesel particulates, formaldehyde and gasoline particulates), we apply the standard linear cancer development model to estimate new cases of cancer associated with these toxics. Third, we value these cases of cancer, taking into account our estimate of the types of cancers caused, when they occur and the probability that the victims will survive. Finally, we apportion these damages to six motor vehicle types. We discuss these four steps in more detail below.

11.6.1.1 Exposure To Toxic Air Pollutants

To estimate toxics exposure, we use the HAPEM-MS model. The EPA (1993a) used this model to estimate cancer cases caused by cars. We use the EPA's work and a companion work by Johnson et al. (1992a) to estimate the annual average per capita exposure to acetaldehyde, benzene, 1,3-butadiene, formaldehyde, diesel particulates, and gasoline particulates in a metropolitan statistical area (MSA). HAPEM-MS is desirable because it offers a method to generate county-level exposure estimates to specific toxics associated with motor vehicles. It accounts for people's movement each day between "microenvironments" of varying pollution levels. (For example, the inside of a car is a microenvironment, with relatively high levels of toxic air pollutants; office buildings and homes are another microenvironment, with relatively low levels of toxic air pollutants.) In this model the average exposure to pollution is the sum of the weighted daily micro-environment exposures, wherein the weights are proportional to the pollutant concentration and the length of time spent in each microenvironment.

The model requires a large amount of data. It divides an MSA into exposure districts -- one district for each CO monitor that operates in the MSA -- and the population into demographic groups based on age and working status (Table 11.6-1). Each demographic group may be broken down further based on residence and place

of work, increasing the number of groups beyond the nine given in Table 11.6-1. Based on studies of what people do each day, HAPEM-MS shuttles each group or cohort between five microenvironments and keeps track of the length and severity of CO exposure.³ At the end of a day the model estimates the average exposure and at the end of the year estimates annual average exposure for each group. It then weights the average annual exposure of each group by its size and finds the average annual per capita CO exposure in the MSA.

To determine exposure to toxics, we assume (initially) that the emissions of toxics follow the same path as do CO emissions. That is, we assume that if the emission of one gram of CO results in an average exposure of $1 \mu\text{g}/\text{m}^3$, then the emission of, say, half a gram of benzene results in an exposure of $0.5 \mu\text{g}/\text{m}^3$. This assumption simplifies the analysis, but it may be inaccurate for toxic pollutants such as acetaldehyde and formaldehyde which break down quickly after emission and are created in secondary reactions from other gases in the atmosphere. Below we explain how we check the accuracy of this assumption and where necessary adjust the results to account for the destruction and secondary formation of toxic air pollutants.

Steps To Estimate Toxics Exposure

We estimate people's exposure to toxic air pollutants by dividing the U.S. into eleven urban regions and two rural regions, with the assumption that one subregion, or "model" area, in each region is representative of the rest of the region (This follows the procedure of the EPA [1993a] in estimating the effects of toxics.) Table 11.6-2 gives the population of each region we examine. Because we assume a homogeneous activity pattern within a region, we may apply the model-estimated toxics exposure in the "model" city to the other cities in the region.

Below, we review the four steps to estimate toxics exposure in all urban areas (i.e., MSAs). The same steps apply for rural areas which we define as counties outside MSAs.⁴ Our review is brief; interested readers should consult EPA (1993a) and Johnson et al. (1992a) for more details.

³The ambient CO level in each environment is assumed to be a fraction of the CO level measured at a fixed-site monitor. These fractions were determined by estimating the relationship between measurements of monitors that people wear and the measurements made at CO monitors.

The fraction of CO attributable to motor vehicles is higher inside motor vehicles and garages and alongside freeways than in office buildings. Johnson et al. (1992a) do not account for this when using HAPEM-MS, they assume that motor vehicles contribute the same percentage in each microenvironment. We can not amend this because we do not have access to HAPEM-MS and have relied on their results. Future work with HAPEM-MS should determine the contribution of CO in each environment by source.

⁴The average annual per capita exposure is estimated for each model rural area with a similar procedure: we take the HAPEM-MS results for two model rural areas (Paducah, Kentucky and Farmington, New Mexico), and assume that each model rural area is a proxy for exposure in non-model rural areas. Paducah is representative of the eastern half of the United States and Farmington is representative of the western half.

1) Johnson et al. (1992a) used HAPEM-MS to estimate the average annual CO exposure in 1988 (Table 11.6-3) in model urban areas.

2) Given exposure in model urban areas, we estimate the exposure ($\mu\text{g}/\text{m}^3$) in "non-model" MSAs:

$$CO\ Exposure_{Non-Model\ MSA} = \frac{Ambient\ CO_{Non-Model\ MSA}}{Ambient\ CO_{Model\ MSA}} CO\ Exposure_{Model\ MSA} \cdot^5$$

3) To determine the motor vehicle-related CO exposure in 1989-1991 – years for which we do not have results from HAPEM-MS – we adjust exposure from the base year (1988) to account for increased VMT (vehicle miles traveled)⁶:

$$CO\ Exposure_{1991,\ Model\ MSA} = \frac{Ambient\ CO_{1988,\ Model\ MSA}}{Ambient\ CO_{1991,\ Model\ MSA}} CO\ Exposure_{1988,\ Model\ MSA} \cdot VMT$$

4) We then estimate exposure to toxics:

$$Toxics\ Exposure_{1988,\ Model\ MSA} = \frac{CO_{grams\ per\ mile}}{Toxics_{grams\ per\ mile}} CO\ Exposure_{1988,\ Model\ MSA} \cdot$$

The gram per mile emission factors we use are in (Table 11.6-4).

Using Ambient Data to Check the HAPEM-MS Results, and to Adjust the Results for "Reactive" Toxics

We assume (initially) that toxic air emissions follow the same chemical and physical path from the tailpipe to air-quality monitor as does carbon monoxide (CO), which is relatively nonvolatile. However, if a significant amount of a toxic compound is destroyed en route – or, conversely, if a significant amount is formed from other emissions -- then this assumed relationship between toxics emissions and its subsequent concentration may not hold. That is, some toxics actually might not follow the same path as the relatively nonvolatile CO. To check this, the EPA (1993a) used observations on the ambient concentration of acetaldehyde, 1,3-butadiene, benzene, and formaldehyde and determined an independent estimate of

⁵When monitor readings are not available for a county in an MSA, we use the readings in the other counties in the MSA. When no readings at all are available for an MSA, we approximate the average exposure with the minimum estimated exposure of the MSAs in that urban region, on the assumption that MSAs without CO monitors have relatively low CO levels. We chose not to follow the EPA (1993a), which used the median estimated exposure in each region as a proxy for those MSAs without CO monitors, because we felt this might overestimate actual exposure.

⁶The EPA (1993a 4-7) assumed that the VMT adjustment factor for 1990 equals 1.031, and for 1995 the adjustment factor equals 1.123, we determine the factors for 1989 (1.0155) and 1991 (1.0465) by linear interpolation.

toxics exposure. (They did not consider diesel and gasoline particulates because direct measurements of diesel and gasoline particulates are not available.) EPA compared the new estimates of exposure with the HAPEM-MS results. Which estimates are better? The EPA assumed that the ambient-based estimates take precedence over HAPEM-MS, perhaps because the ambient observations seem more grounded in reality. In our view, though, both estimates have problems.

To obtain an ambient-based exposure estimate, the EPA first calculated the annual average for each monitoring site and the overall average of monitors in each of four separate groups of monitors (Table 11.6-6). The network averages include all toxics sources. The EPA removed the contribution of other (non-motor-vehicle) sources of toxics (Table 11.6-7), and adjusted for a population's movement through microenvironments using what EPA calls the "integrated exposure adjustment constant", which is a simplified version of the HAPEM-MS model that they want to check.

The "integrated exposure adjustment constant" combines the amount of time that people spend in each microenvironment with the pollution concentration in the environment. The EPA (1993a: 5-29) estimated that people spend 5.9% of their time outdoors, 61.9% indoors at home, 24.6% at work and 7.6% during some form of transport, and then weighted the percentages by CO level in each place or microenvironment. The CO level in each microenvironment equals the ratio of the CO concentration in the microenvironment to the CO concentration at a fixed outdoor monitor site: outdoors = 0.758, indoors residence = 0.495, indoors other = 0.619, and inside a motor vehicle = 1.554 (Table 11.6-9). With these data, the constant is calculated as:

$$5.9\% \cdot 0.758 \frac{CO_{outdoor}}{CO_{monitor}} + 61.9\% \cdot 0.495 \frac{CO_{home}}{CO_{monitor}} + 24.6\% \cdot 0.619 \frac{CO_{work}}{CO_{monitor}} + 7.6\% \cdot 1.554 \frac{CO_{auto}}{CO_{monitor}} = 0.622$$

The EPA calculated an ambient-based estimate for each pollutant for each network and for each year. For example one network of benzene monitors for benzene had an average reading of 6.80 $\mu\text{g}/\text{m}^3$ in 1987 (Table 11.6-6). Attributing 59.5% of ambient benzene to motor vehicles and multiplying the results by the integrated exposure adjustment constant (0.622), the EPA estimated the annual exposure to benzene is 2.52 $\mu\text{g}/\text{m}^3$.⁷

After comparing the adjusted ambient data with the HAPEM-MS results, the EPA decreased the HAPEM-MS results for formaldehyde by a factor of 1.18, decreased the HAPEM-MS results for 1,3-butadiene by a factor of 1.37 and increased the HAPEM-MS results for acetaldehyde by a factor of 2.09; they did not change the

⁷Note that our reported exposure (2.52 $\mu\text{g}/\text{m}^3$) differs slightly from those of EPA (1993a) for reasons we discuss in Table 11.6-6

benzene estimate.⁸ The EPA apparently used its best judgment in determining these factors.

Problems Comparing Ambient-Based Exposure Estimates And HAPEM-MS Exposure Estimates

Several problems encumber the EPA's (1993a) analysis. First the EPA determined national estimates of toxics exposure using monitor networks with as few as six observations: Bakersfield, Concord, Fremont, Richmond, San Jose and Stockton California comprise one network of monitors (EPA, 1993a: Appendix C, p.13); the HAPEM-MS results used CO monitors in 163 MSAs. There is little reason to expect the sets of results to agree. Second the EPA's method of comparing the HAPEM-MS and ambient-based national exposure estimates is not clear. Third when calculating their ambient-based exposure estimates, they used relatively simple assumptions about the length of time spent in microenvironments, and, furthermore, they calculated the integrated exposure adjustment constant for the nation as a whole, rather than for each of the eleven urban regions in the U.S.

An Alternative Comparison of HAPEM-MS and Ambient Toxics Data

Because of these shortcomings, we opted to redo the EPA's (1993a) calculations. For each MSA that has sufficient ambient toxics data, we compare our own ambient-based estimates with the HAPEM-MS results (Tables 11.6-10 to 11.6-13). We compare *individual cities*, and thereby avoid the problem of using data from a handful of cities to determine a national average. Second, we estimate the relationship between CO monitor observations and people's exposure for thirteen regions in the U.S. using HAPEM-MS rather using the integrated exposure adjustment constant as a proxy for the nation (Table 11.6-14).⁹ Third, we use

⁸The EPA (1993a: 5-29) did not change the HAPEM-MS benzene estimate "since the unit-risk estimate for benzene is an upper bound estimate, and the HAPEM-MS 1990 base control number [2.67 $\mu\text{g}/\text{m}^3$] matches the upper end of the range [2.68 $\mu\text{g}/\text{m}^3$]" However, the EPA is incorrect for three reasons.

1) the benzene unit-risk number is not an "upper bound" estimate. In fact, the EPA (p. 5-39) reported that the benzene unit-risk number is the geometric mean of 21 maximum likelihood estimates;

2) the EPA (p. 5-28) incorrectly reported that the "overall average of the averages from the 31 cities was 7.18 $\mu\text{g}/\text{m}^3$ " in the NAVOC data. Inadvertently, they reported the average of all 564 samples (weighted equally) from all 31 cities. The overall average is 6.83 $\mu\text{g}/\text{m}^3$, which in turn causes the exposure estimate to decline below the HAPEM-MS estimate, and

3) the EPA (Table 5-1) incorrectly reported that 1 ppm of benzene equals 3.25 $\mu\text{g}/\text{m}^3$ at 25° C, the correct figure is 3.19 $\mu\text{g}/\text{m}^3$

The effect of the last two errors is to reduce the exposure estimate based on the ambient toxics data, in Table 11.6-6 the adjusted ambient data range from 1.50 $\mu\text{g}/\text{m}^3$ to 2.53 $\mu\text{g}/\text{m}^3$ -- well below the HAPEM-MS estimate of 2.67 $\mu\text{g}/\text{m}^3$. To be consistent with their adjustments to the other HAPEM-MS estimates of toxic exposure, the EPA (1993a) should have reduced the HAPEM-MS benzene estimate to conform with the ambient data.

⁹Nevertheless, their constant is a reasonably good approximation -- most of our regional adjustment factors are close to the EPA's 0.622 constant (Table 11.6-14), and hence the EPA's adjusted ambient estimates are close to the HAPEM-MS estimates (Table 11.6-15).

ordinary least squares to determine if the ambient-based estimates are significantly different from HAPEM-MS results.

HAPEM-MS Exposure Estimate

We estimate toxics exposure in an MSA by taking the CO exposure estimate generated by HAPEM-MS (Johnson et al., 1992a) and multiplying it by the ratio of toxics and CO emissions:

$$\text{Toxics Exposure}_{1988, \text{MSA}} = \frac{\text{CO}_{\text{grams per mile}}}{\text{Toxics}_{\text{grams per mile}}} \text{CO Exposure}_{1988, \text{MSA}}$$

Ambient-Based Exposure Estimate

Because there is a shortage of ambient toxics observations, we combine data from several years in order to determine average ambient levels. To obtain an exposure estimate based on the overall average of the ambient data, we adjust the ambient toxics data by the proportion that is attributable to motor vehicles, and multiply by the regional exposure adjustment factor:

$$\text{Toxics Exposure} = \text{Ambient Toxics} \% \text{Toxics Emissions}_{\text{motor vehicles}} \text{Regional Exposure Factor}$$

where:

Toxics Exposure = ambient-based toxics exposure level ($\mu\text{g}/\text{m}^3$)

Ambient Toxic = average ambient toxic level ($\mu\text{g}/\text{m}^3$)¹⁰

Toxics Emissions_{motor vehicles} = the % of toxics attributable to motor vehicles
(Table 11.6-7)

Regional Exposure Factor = the HAPEM-MS average CO exposure divided by the actual annual average ambient CO level for the model city in the region (Table 11.6-14).

Results Of Our Comparison Between HAPEM-MS And Ambient Data

Tables 11.6-10 to 11.6-13 give the ambient-based exposure and HAPEM-MS exposure estimates for acetaldehyde, 1,3-butadiene, benzene and formaldehyde. For each pollutant, we regress the ambient-based estimates on those from HAPEM-MS. We interpret the regression coefficients as the factor with which to multiply the HAPEM-MS estimates of toxic exposure so that they conform with the ambient-based estimates of toxic exposure. If the coefficient is not different from one, then the HAPEM-MS estimates should not be changed; Table 11.6-16 gives the results.

Our regional factor equals the average per capita exposure estimate from HAPEM-MS, divided by the average ambient CO levels

$$\text{Exposure Factor in Region}_1 = \frac{\text{Average CO Exposure in Model MSA}_1, \text{HAPEM-MS}}{\text{Average Ambient CO in Model MSA}_1, \text{National Air Data Bank}}$$

¹⁰We take a weighted average of all annual averages in an MSA (1987-1990), with the weighting determined by the number of observations in each average. We make the assumption that the ambient level of toxic pollution should not differ between 1987 and 1990.

The coefficient for benzene, acetaldehyde and 1,3-butadiene differ significantly from one at the 95% confidence level, so it is appropriate to use the estimated coefficients to adjust the HAPEM-MS results. Thus, we multiply the HAPEM-MS acetaldehyde estimate by 2.53, the benzene estimate by 0.81 and the 1,3-butadiene estimate by 0.48. However, we do not change the formaldehyde exposure estimate.

Our revisions differ from the EPA's (1993a). They multiplied the HAPEM-MS acetaldehyde estimate by 2.09, the 1,3 butadiene estimate by 0.73, and the formaldehyde estimate by 0.85; they did not change the benzene estimate. We believe that our method is an improvement, and consequently use our own results¹¹

11.6.1.2 Linear Cancer Model

The second step in valuing the damages caused by motor vehicle toxics, is to translate toxics exposure into estimated cancer cases. We use the linear cancer model. The linear cancer model, also termed the "one-hit" model since even very small amounts of toxics cause cancer, assumes that the risk of getting cancer is linearly related to the dose. Thus, this model assumes that the per unit effect of toxics at high and low doses is the same -- not an assumption unanimously accepted with equanimity. At very low doses, no one knows for sure whether the effect is truly linear, or even if toxics have any effect at all (Cohrssen and Covello, 1989: 95).¹² Nevertheless, the common presumption in most studies, and in ours, is that the "one-hit" (linear down to zero dose) model is correct for estimating the incidence of cancer following exposure to potential carcinogens.¹³

¹¹We recognize that, for several reasons, our method is still imperfect

First, it is doubtful that the placement of the toxics monitors is comparable to the placement of the CO monitors. To the extent that the monitors are placed in different areas (e.g., residential versus central city), we expect the ambient-based and HAPEM-MS estimates to differ

Second, the estimated exposure level, based on the toxics monitoring data, is sensitive to the assumed contribution of motor vehicles to the ambient toxic concentration. In reality, the precise portion of the ambient toxics levels attributable to motor vehicles is unknown

¹²Very large experiments would help resolve whether low doses are harmful (Rall, 1978, Land, 1981), but the scale of the experiments would have to be prohibitively large to explore the effects of very low doses.

¹³An alternative to the use of the linear cancer model, at least for particulates, would have been to use epidemiological studies, which correlate people's estimated exposure to, say, particulates and reported cases of cancer, or cancer deaths. This method is desirable because it relies on *reported* cases of cancer. However, we did not find any studies that could provide useful dose-response functions. We consider, here, some of the better studies that we reviewed

Cupitt et al (1994) used diaries of 43 residents of Boise, Idaho to estimate exposure to extractable organic matter (EOM) from residential wood smoke and motor sources. They then estimated the risk to this exposure using the comparative potency method (Albert et al., 1983; Lewtas, 1991). They (p 82) found "an individual lifetime risk" of 4.7×10^{-4} from exposure to 4.7 ug/m^3 of EOM. Of this risk, 0.92×10^{-4} is attributable to residential wood smoke, meaning that the remainder, roughly 3.8×10^{-4} , is from mobile sources. However, we do not use Cupitt et al's results. They are based on a relatively small sample of diaries and the measured extractable organic matter is specific to Boise, Idaho.

In the linear model, the unit-risk number is the key to estimating the incidence of cancer.¹⁴ A unit-risk number estimates the excess risk of cancer, to one individual, from 70 years (a lifetime) of continuous exposure to 1 µg/m³.

$$\text{Unit Risk}_{\text{Toxic}} = \frac{\text{Cancer Risk}_{\text{Individual}}}{70 \text{ Years' Exposure to Toxic, at } 1 \mu\text{g} / \text{m}^3}$$

We should note that cancer unit-risk numbers are very imprecise: estimates of the unit risk for a particular compound can differ by an order of magnitude or more. For example, the EPA's official unit-risk estimate for formaldehyde differs by more than an order of magnitude from their updated (unofficial) estimate. The high degree of variability arises from the difficulty in measuring the effect of very small doses and using animals to estimate the effects on humans. Paustenbach et al. (1990) and Schneiderman (1981) discuss the difficulties.¹⁵

In a study of Seventh-Day Adventists who lived in the same residence in California for at least ten years, Abbey et al (1991) found that TSP caused an elevated risk of cancer in females. However, it is not clear why women would have a higher risk than men.

Archer (1991) compared three counties in Utah and found an elevated cancer risk in the county with a steel mill that generates 45% of the ambient particulates in the county. Archer assumed that smoking rates are the same in these counties. However, using smoking data, Blindauer et al (1993) reported that Archer's result can be explained by differences in smoking rates between the counties.

Buffler et al. (1988) found that TSP (their assumed proxy for toxics) was responsible for 1.5% of the lung cancer rate for white males aged 30-79 in Harris County, Texas -- equivalent to 1.9 extra lung cancer deaths per 100,000 people in Harris County. However, TSP is not a particularly good proxy for toxics because it is not a "well-dispersed" pollutant (Detels et al., 1981), and the coarser fraction of TSP is not normally inhaled. Moreover, some toxics, such as benzene are not necessarily attached to particulates. These factors lead to an underestimation of the true effect.

Doll and Peto (1981: 1248) estimated that at most 1% of lung cancer is attributable to air pollution, and Speizer (1983) reviewed the literature and estimated that at most 2% of lung cancer may be attributable to air pollution. The Doll and Peto study and Speizer's work simply review a literature that is no better (and often worse) than the Buffler study. Also, they do not estimate the effects of specific pollutants.

In a prospective cohort study of 151 metropolitan areas, Pope et al. (1995a) reported a link between lung cancer and sulfate particulates. They found a relative risk of 1.26 for sulfates (with a 95% confidence interval of 1.11-1.66) between the most and least polluted metropolitan areas, no significant effect was found for PM_{2.5}. This would be a useful study but for the fact that we do not have much ambient sulfate data.

¹⁴The EPA (1993a) gives more details on the evidence linking particular toxics to cancer, and the development of unit-risk numbers (both the EPA's own estimates and estimates by others).

¹⁵Concerning the numerous difficulties with using animal studies to estimate the risk of toxics to humans, Schneiderman (1981: 33) noted the sharp contrast between humans and lab animals. Lab animals are genetically homogeneous, live in a much less complicated environment than humans, are exposed to relatively few potential carcinogens, have a different metabolism and immune system than do humans, and typically are given doses orders of magnitude higher than what people and animals normally encounter.

To estimate the number of excess cancers to a population from exposure to a toxic air pollutant in a given year, we determine the number of "exposure-years" at an exposure level of $1 \mu\text{g}/\text{m}^3$. (For example, the exposure of seventy people to $1 \mu\text{g}/\text{m}^3$ for a year is equal to 70 exposure-years.) Multiplying the number of exposure-years by the unit-risk number then tells the number of cancer cases that will arise from exposure to motor vehicles. We calculate the number of excess cancers arising in a population from a year's exposure to a toxic pollutant by:

$$\text{Excess Cancers}_{\text{Year}_t} = \frac{\text{Unit Risk Number} \cdot \text{Average Population Exposure}_{\text{Year}_t} \cdot \text{Population}}{70 \text{ Years}}$$

where:

Excess Cancers_{Year_t} = increase in cancer caused by annual automotive toxics emissions

Unit-Risk Number = unit-risk number of toxic (EPA, 1993a)

Average Population Exposure_{Year_t} = annual per capita exposure estimated by HAPEM-MS

Population = county population of all individuals, of all ages (U.S. Bureau of the Census, 1992).

Given the estimated increased incidence of cancer, we still need to determine: (a) the latency period (the period between exposure and cancer development), (b) the type of cancer caused, (c) the recovery rate and the cost of treatment, (d) how to apportion the estimated damages among different vehicle types.

11.6.1.3 Latency Period, Type of Cancer and Recovery Rate

Latency Period

The development and diagnosis of cancer does not occur concurrently with the emission of toxics. There is a lag or latency period, which we assume may extend anywhere from one to fifty years. Unfortunately, there are no reliable estimates of the mean length of time between exposure and diagnosis of cancer, nor of the shape of the distribution of cancer cases over time. Concerning the mean of the distribution, Buffler et al. (1988) assumed a ten-year latency period when regressing lung cancer in year t on the TSP level in year $t-10$, and found a small positive relationship between lung cancer and TSP. Friberg and Cederlof (1978: 51) reported that the latency period between cigarette smoking and cancer may be as long as three to five decades, and added that there is little reason to expect the latency period for air pollution to be less than this. Speizer (1983: 36), reporting results from a study on roofers (who are exposed to fumes from burning fossil fuels), stated that "there is a suggestion of an exposure-response relationship with a lag of 20 years and then a doubling of the risk of lung cancer over the next 20 years." These reports suggest that most cancers do not occur quickly after exposure. For lack of definitive estimates of the latency period, we assume that, on average, cancer is discovered 25 years after exposure, and that the incidence of cases follows, roughly, a normal distribution

(Table 11.6-17). We feel that this reflects the notions currently in the literature tolerably well.

Discount Rate

When the cancer occurs is important in our analysis because of the well-accepted economic practice of discounting future costs. We use real discount rates of 2% and 8%, which bracket most estimates in the literature (e.g., Hartman, 1990; Lind, 1990). We discuss our choice of discount rate in Report #3 of the social-cost series listed at the beginning of this report.

Type of Cancer and Recovery Rate

For each toxic, we estimate the type of cancer formed and the associated probability of recovery – both are important factors in determining the cost of cancer. If a cancer victim dies we assume the cost is the value of a statistical life; if one recovers the cost of cancer equals the cost of treatment, lost productivity, and pain and suffering.

We estimate the type of cancer caused by each toxic, using the epidemiological and laboratory results cited by the EPA (1993a) in its estimates of the unit-risk (Table 11.6-18 to 6-20). Benzene is linked to an increased incidence of leukemia in studies of workers in rubber factories; hence, we estimate the mortality rate with the assumption that benzene causes leukemia. The other toxics are based on animal studies, and we assume that the types of cancer formed in rodents and humans are identical.¹⁶ The unit-risk number for gasoline is *not* directly based on laboratory studies, instead it uses the "comparative potency method", which groups an unknown chemical with chemicals of a known carcinogenicity (Albert, 1983; Lewtas et al., 1991). We assume that gasoline particulates cause lung cancer.

We estimate the recovery rate for each type of cancer from statistics provided by the American Cancer Institute. Tables 11.6-21 and 11.6-22 show that, in general, recovery rates have increased over time, but that the recovery rate for African-Americans is lower than for whites, perhaps because of later diagnoses or less effective treatment. In addition the incidence of cancer is higher for African-Americans. We weight our estimated recovery rates by the relative populations of whites and African-Americans, and by the higher incidence rate for African-Americans. Although there may be regional differences, we assume for simplicity that a single recovery rate holds across the U.S. (Tables 11.6-23 and 11.6-24).

11 6.1.4 Apportioning Toxics Damages to Vehicle Type

The EPA (1993a) estimates the contribution of all motor-vehicles to exposure to toxic air pollutants, but not the contribution of individual classes of motor-vehicles (light-duty gasoline vehicles, heavy-duty diesel vehicles, etc.) We estimate the separate contribution of each motor vehicle class as follows. For benzene, we

¹⁶Of course, it is possible that a particular toxic might cause a different cancer in humans than it does in rodents. However, it seems to us that any such assumption about different effects would be entirely ad hoc.

first assume that all diesel vehicles are responsible for 3% of the total motor-vehicle contribution to benzene, because according to Carey (1987: 43), diesel vehicles emit about 3% of the benzene emitted by all motor vehicles. We then distribute this 3% to each of the three classes of diesel vehicles (light-duty auto, light-duty truck, heavy-duty truck) in proportion to VOC emissions from each of the three classes. We do the same for the remaining 97% of total motor-vehicle benzene that is attributed to gasoline vehicles. We do this for each county in the U.S.

For all other toxic air pollutants, we distribute the total motor-vehicle share (estimated by EPA, 1993a) to each of the six vehicle classes in proportion to VOC emissions from each of the six classes, with no initial distinction between gasoline and diesel vehicles.

Adjusting HAPEM-MS to Reflect Upstream Sources of Toxics

Ideally, our estimate of the carcinogenic effects of toxics would take into account the many sources of toxic emissions associated with the use of motor vehicles:

- releases from oil production fields and petroleum refineries;
- the distribution and marketing of gasoline and diesel;
- combustion of gasoline and diesel fuel in motor vehicles;
- evaporation of gasoline and diesel from vehicles;
- evaporation of gasoline and diesel from service stations;
- materials used in the interior of vehicles;
- fluids, such as oil, used in vehicles; and
- the production of motor vehicles.

However, the HAPEM-MS model (EPA, 1993a) considers only the contribution of fuel combustion and motor vehicle evaporative losses; it does not include the above "upstream" emissions. Consequently, we do not include the cost of upstream motor-vehicle related toxics in our analysis.¹⁷

11.6.2 Acetaldehyde

Motor vehicles are an important source of acetaldehyde, accounting for roughly 40% of ambient acetaldehyde (Table 11.6-7). Acetaldehyde is a probable

¹⁷We considered adjusting the estimated number of cancer cases by the ratio of (motor vehicle VOC + upstream VOC)/(motor-vehicle VOC). However, for two reasons, we decided against this. Upstream VOCs probably comprise different toxic fractions than do motor-vehicle VOCs, and upstream emissions sources probably are not as close to people as our motor-vehicles.

human carcinogen (class B2; see Table 11.6-19) with an inhalation unit-risk value of $2.2 \cdot 10^{-6}/\mu\text{g}/\text{m}^3$.

The epidemiological evidence linking acetaldehyde to cancer in humans is weak. Bittersohl (1974) found an increase in the incidence of all types of cancer, but the sample size was small and the study did not control for smoking and exposure to other chemicals. However, a significant increase in cancer has been found in animals exposed to acetaldehyde. Woutersen et al. (1986) found an increased incidence of nasal and laryngeal tumors in rats, from which we assume that acetaldehyde causes cancers of the oral cavity and pharynx in humans (Tables 11.6-18 and 11.6-20).

11.6.3 Benzene

Motor vehicles contribute evaporative and exhaust benzene emissions, and account for approximately 60% of ambient benzene concentrations (Table 11.6-7).

Benzene is harmful to human health in several ways: 1) it causes leukemia; 2) it can cross the placenta, and thereby affect the health of the fetus; 3) it can disturb menstruation; and 4) it may be associated with aplastic anemia and chromosomal aberrations (IARC, 1982: 116-7, 127; National Research Council, 1985: 53). The young, the old, and other people that have immune systems that are not functioning properly, may be most susceptible to benzene's effects (Round et al., 1989: 11-39). It is not possible to quantify and value all these effects. In this analysis, we assess the increased risk of leukemia from exposure to benzene.

Benzene is a class A human carcinogen. The unit-risk number is $8.3 \cdot 10^{-6}/\mu\text{g}/\text{m}^3$. Unlike the other unit-risk numbers used in this study, which are based on an upper 95% confidence interval estimate, the unit-risk number for benzene is a maximum likelihood estimate unlike . It is based on epidemiological studies of workers, in rubber manufacturing plants, who suffer from increased nonlymphocytic leukemia, and is supported by an increased incidence of tumors in mice and rats.

11.6.4 1,3-Butadiene

Motor vehicles are responsible for about 55% of the ambient concentration of 1,3-butadiene. A recent study 1,3-butadiene production workers by Ward et al. (1995) found an increased risk of lymph cancer, and there are laboratory studies of mice and rats showing air-borne 1,3-butadiene causes cancer. It is a probable human carcinogen (class B2), with a unit-risk number of $2.8 \cdot 10^{-5}/\mu\text{g}/\text{m}^3$.

11.6.5 Diesel Particulates (as a cause of lung cancer)

Diesel particulates are dangerous because they absorb toxic, mutagenic and carcinogenic organic molecules that are emitted in the combustion process (National Research Council, 1981: 14). These particulates are less than a micron in diameter and lodge deep in the lung, for up to several hundred days; they may reach other parts of the body through the lymphatic and blood circulatory systems and cause additional damage (National Research Council, 1981: 55; Cuddihy et al., 1984: 17A).

Diesel particulates reportedly increase the incidence of lung cancer (Cuddihy et al., 1984: 18A), stomach cancer (Winkelstein, 1969) and more generalized types of cancer (Mills et al., 1991). For a recent review of diesel emissions and their effects, see *Diesel Exhaust: A Critical Analysis of Emissions, Exposure, and Health Effects* (Health Effects Institute, 1995). We assume diesel particulates cause lung cancer. The unit-risk number, based on a rat study, is $1.7 \cdot 10^{-5}/\mu\text{g}/\text{m}^3$ (EPA, 1993a: 9-34).

Note that this lung-cancer effect of diesel particulates does not double-count any of the other mortality effects of particulate matter estimated in Chapter 11.3. In Chapter 11.3, we use epidemiological studies to estimate acute respiratory deaths, acute cardiovascular deaths, and chronic cardiopulmonary deaths.

11.6.6 Formaldehyde

Motor vehicles account for roughly 35% of ambient concentrations of formaldehyde. Formaldehyde increases eye irritation and is a probable human carcinogen (class B1). The official EPA unit-risk number is $1.3 \cdot 10^{-5}/\mu\text{g}/\text{m}^3$; however, an updated, unofficial unit-risk number is 6.0×10^{-7} -- significantly lower than the official estimate. We use the updated unit-risk number.

The unit-risk number is based on the increased formation of squamous cell carcinoma in male rats that have inhaled formaldehyde. There is supporting evidence from epidemiological studies, and laboratory studies on viruses, yeast and other organisms. The epidemiological studies are considered limited primarily because they did not adequately control for exposure to other possible or known carcinogens. Still, these studies do point to an increase in lung and nasopharyngeal cancers. Furthermore, there is a possible link to leukemia and neoplasms in the brain and colon (EPA, 1992c). And, the fact that acetaldehyde, the closest aldehyde to formaldehyde, causes tumors in rats and hamsters further implicates formaldehyde as a carcinogen (EPA, 1992c).

11.6.7 Gasoline Particulates (as a cause of lung cancer)

Currently, there is no official unit-risk estimate for gasoline particulates. The EPA (1993a) relied on the comparative potency method to determine the unit-risk of $5.1 \cdot 10^{-5} \mu\text{g}/\text{m}^3$ for cars with catalysts and $1.6 \cdot 10^{-5} \mu\text{g}/\text{m}^3$ for cars without catalysts (Lewtas, 1991). The comparative potency method compares the results from mouse skin tumor initiation assays for carcinogens with known unit-risk numbers (i.e., coke oven emissions, roofing tar emissions and cigarette smoke) and gasoline particulates (Lewtas, 1991). The relative ranking found in the tumor assays is then assumed to apply to the unit-risk estimates. We assume that gasoline particulates cause lung cancer, since coke oven and roofing tar emissions and cigarette smoke are associated with lung cancer.

Whether or not a gasoline-powered car uses a catalytic converter affects the estimated toxicity of its emitted particulates. Consequently, we have separate unit risk estimates for cars with and without catalytic converters. We assume that 3% of gasoline powered vehicles do not use catalytic converters.

Note that this lung-cancer effect of gasoline particulates does not double-count any of the other mortality effects of particulate matter estimated in Chapter

11.3. In Chapter 11.33, we use epidemiological studies to estimate acute respiratory deaths, acute cardiovascular deaths, and chronic cardiopulmonary deaths.

11.6.8 Reporting the results

The cost of all six toxic air pollutants is included in Table 11.7-6, under the “toxics” column, and in Table 11.7-9, in the “toxics” row. (In these tables, gasoline and diesel particulates, as lung-cancer causing “toxic” air pollutants, are included under “toxics” rather than under “PM₁₀”). Toxic air pollutants also are included in all of the tables of the Appendix. In the Appendix tables, formaldehyde, acetaldehyde, benzene, and 1,3-butadiene are listed explicitly as ambient pollutants associated with VOC emissions. Gasoline and diesel particulates are represented by the line for the effect “lung cancer” associated with PM₁₀ emissions.

Because we do not estimate the cost of toxic emissions from any source other than motor vehicles, we do not include toxics in the tables that estimate the cost of all anthropogenic air pollution (Tables 11.7-3 and 11.7-4). Also, as mentioned above, we do not actually estimate toxic air emissions from any of the “upstream” sources, such as petroleum refineries, associated with motor-vehicle use (Tables 11.7-6, 11.7-9, and all tables in the Appendix). This omission slightly underestimates the total cost of toxic air pollution attributable to motor-vehicle use.

TABLE 11.6-1. DEMOGRAPHIC GROUPS USED IN HAPEM-MS

Demographic Groups
Children 0 to 5
Children 6 to 13
Children 14 to 18
Workers with a Low Probability of Outdoor Work
Workers with a Moderate Probability of Outdoor Work
Workers with a High Probability of Outdoor Work
Nonworking Adults Under 35
Nonworking Adults 35 to 54
Nonworking Adults 55+

Source Johnson et al. (1992a Table 4)

TABLE 11.6-2. POPULATION OF THE ELEVEN "MODEL" URBAN AND TWO "MODEL" RURAL REGIONS USED TO ESTIMATE THE CANCEROUS EFFECTS OF TOXICS BY YEAR (MILLIONS)

Model MSA ^a	Urbanized Population of Associated Region			
	1988	1989	1990	1991
Boston	14 015	14.135	14.256	14 377
Denver	4.385	4.398	4.411	4 424
Houston	38 188	38 491	38 794	39.097
Los Angeles	27.930	28.700	29 330	30 030
Minn./St. Paul	19.756	19.775	19 794	19 813
New York City	17 168	17 147	17.126	17 105
Philadelphia	12 145	12 104	12 063	12.022
Phoenix	4 713	4.838	4 962	5 086
St Louis	29 059	29.100	29 142	29.183
Spokane	7.547	7.703	7.859	8.015
Washington, DC	14 961	15 169	15.377	15.585
Subtotal Urban	189.867	191.491	193.115	194.738
Farmington, NM	21.598	21.529	21 460	21.391
Paducah, KY	34.338	34.237	34 136	34.034
Total U.S.	245.803	247.257	248.710	250.163

Source U.S Bureau of the Census (1994).

^aAs discussed in the text, we estimate exposure to toxic air pollutants on the basis of exposure to CO. Ideally, we would model CO pollution levels and exposure to CO in every city or county of the U.S. We do have data on CO levels by county, but we cannot model exposure to CO in every city or county. Instead, we use a "model" (or "representative") MSA to represent a *group* of MSAs that presumably have similar population characteristics. For example, the movement of people and the subsequent pattern of CO exposure in Anchorage, Alaska, Portland, Oregon and other MSAs in the northwestern United States is assumed to be the same as that in Spokane, Washington.

Because we assume the a population's movement and exposure pattern is the same within a given region represented by a model city, it follows that the difference between the annual average CO exposure in a model MSA and the annual average CO exposure in any other MSA that is represented by the model MSA, is determined using the ratio of the annual average ambient CO concentrations. The equations that we are use are given in the text.

TABLE 11.6-3. AVERAGE ANNUAL PER CAPITA CO EXPOSURE IN THE ELEVEN MODEL METROPOLITAN STATISTICAL AREAS (MSA) AND TWO MODEL RURAL AREAS USED IN HAPEM-MS (1988)

Model MSA	Per Capita CO Exposure ($\mu\text{g}/\text{m}^3$)^a
Boston	972.73
Denver	1,060.61
Houston	633.33
Los Angeles	1,409.09
Minneapolis	1,221.21
New York City	1,269.70
Philadelphia	766.67
Phoenix	951.52
St Louis	606.06
Spokane	1,666.67
Washington D.C.	815 15
Model Rural Area	
Farmington, New Mexico	496.97
Paducah, Kentucky	439.39

^aThese estimates of CO exposure are derived simply by reversing the calculation of Johnson et al. (1992a), who calculated benzene exposure by multiplying estimated CO exposure by 0.0033, which according to work by the American Petroleum Institute (1988) is the median ratio of benzene to CO in the exhaust from 61 late-model automobiles. Johnson et al (1992a) presented the calculated benzene exposures, but not the original detailed estimates of CO exposure with respect to which they calculated benzene exposure. Therefore, we simply divided the benzene-exposure estimates by 0.0033 to obtain the original underlying (but not presented) CO-exposure estimates used in Johnson et al (1992a)

We use the 0.0033 benzene/CO ratio only to retrieve to the original detailed CO exposure estimates of Johnson et al (1992a) We develop our own conversion factor to estimate exposure to toxics.

TABLE 11.6-4. GRAM PER MILE EMISSIONS OF CO AND TOXICS

Pollutant	Gram Per Mile Emissions
Acetaldehyde	0 0119
1,3-Butadiene	0 0156
Benzene	0 0882
Carbon Monoxide	29.6
Diesel Particulates	0 0669
Formaldehyde	0 0412
Gasoline Particulates	0 0198

Source EPA (1993a) The gram per mile emission factors are the average of emissions from gasoline and diesel vehicles weighted by the VMT of gasoline and diesel vehicles (EPA, 1993a Chapter 3)

TABLE 11.6-5. CONVERSION BETWEEN PARTS PER BILLION (PPB) AND MICROGRAMS PER METER CUBED ($\mu\text{G}/\text{M}^3$) FOR ALL POLLUTANTS (EXCEPT PARTICULATES) CONSIDERED IN THIS STUDY^a

Pollutant	Converting 1 ppb to $\mu\text{g}/\text{m}^3$
Acetaldehyde (CH_3CHO)	1 ppb = 1.802 $\mu\text{g}/\text{m}^3$
Benzene (C_6H_6)	1 ppb = 3.194 $\mu\text{g}/\text{m}^3$
1,3-Butadiene (C_4H_6)	1 ppb = 2.212 $\mu\text{g}/\text{m}^3$
Carbon Monoxide (CO)	1 ppb = 1.145 $\mu\text{g}/\text{m}^3$
Formaldehyde (HCHO)	1 ppb = 1.228 $\mu\text{g}/\text{m}^3$
Nitrogen Dioxide (NO_2)	1 ppb = 1.881 $\mu\text{g}/\text{m}^3$
Ozone (O_3)	1 ppb = 1.963 $\mu\text{g}/\text{m}^3$
Sulfur Dioxide (SO_2)	1 ppb = 2.620 $\mu\text{g}/\text{m}^3$

^aTo make the conversions, we use the ideal gas law ($PV = nRT$) We assume that $P = 1$ atmosphere, and $T = 298$ Kelvin (= 25° Celsius) $R = 0.082058$ (Liter Atmosphere)/(Kelvin Mole) $V = 1000$ Liters(= 1m^3)

Note that particulates is a class of compounds that have many different molecular weights; hence, it is not possible to determine parts per billion (ppb)

TABLE 11.6-6. AVERAGE AMBIENT CONCENTRATION OF TOXIC AIR POLLUTANTS AT MONITORING SITES, AND ESTIMATED EXPOSURE TO MOTOR-VEHICLE DERIVED TOXICS

Pollutant and Monitoring Network	Year	Average Ambient Toxic Level at Monitors ($\mu\text{g}/\text{m}^3$) ^a	EPA Estimate of Exposure to Motor-Vehicle Toxics, based on ambient data ($\mu\text{g}/\text{m}^3$) ^b	HAPEM-MS Estimate of National Exposure to Motor-Vehicle Toxics, in 1990 ($\mu\text{g}/\text{m}^3$)
<u>Acetaldehyde</u> ^c				
UATMP	1990	2.93	0.71	0.36
<u>1,3-Butadiene</u>				
AIRS ^d	1988	1.01	0.35	0.48
	1989	0.47	0.16	
	1990	0.22	0.08	
UATMP ^e	1989	0.46	0.16	
	1990	0.32	0.27	
NAVOC	1987	0.81	0.44	
<u>Benzene</u>				
AIRS	1987	6.80	2.52	2.67
	1988	4.06	1.50	
	1989	4.09	1.51	
UATMP	1989	6.01	2.22	
	1990	4.70	1.74	
TAMS	1987-1989	4.18	1.55	
NAVOC	1987	6.83	2.53	
<u>Formaldehyde</u> ^c				
UATMP	1990	5.14	1.05	1.25

Source. EPA (1993a)

AIRS = Aerometric Information Retrieval System, UATMP = Urban Air Toxics Monitoring Program, TAMS = Toxic Air Monitoring System, NAVOC = National Ambient Volatile Organic Carbon Database

^aThe ambient toxics levels for each year are the overall averages of the average ambient level found at each monitoring site

The EPA (1993a Appendix C) reported the observation in parts per billion (ppb). We convert from ppb to $\mu\text{g}/\text{m}^3$ using the factors in Table 11.6-5. Note that the conversion factor for benzene (1 ppb = 3.25 $\mu\text{g}/\text{m}^3$ at 25 °C) reported by the EPA (1993a Table 5-1) is incorrect, the correct figure is 1 ppb = 3.194 $\mu\text{g}/\text{m}^3$ at 25 °C

The EPA (1993a) unintentionally used two procedures to estimate the average toxics levels. The EPA used the overall average of the averages from all sites in the AIRS and TAMS data and

unintentionally used the average of all observations from all sites for the UATMP and NAVOC data. We report the overall average for all sites, as this appears to have been EPA's intention. EPA (1993a 5-28) reported using the "overall average of the averages for each site" when considering the UATMP data, yet in EPA (1993a Appendix C-8) all observations from all sites were averaged together.

^bThe EPA (1993a) adjusts the ambient data to account for the percentage of emissions directly attributable to motor vehicles, and the percentage of each day that people spend in the microenvironments. The percentage of ambient toxics levels attributable to motor vehicles is reported in Table 11.6-7, and the "integrated exposure adjustment constant" is 0.622. See the text for further discussion.

^cWe report only the 1990 UATMP data because only it accounts for the scavenging of formaldehyde and acetaldehyde by ozone (EPA, 1993a 6-19, 8-15), which biases down the estimated formaldehyde and acetaldehyde levels.

^dWe excluded samples collected from Houston because the EPA (1993a 7-14) reported that the monitor was close to a point source(s) of 1,3-Butadiene, and thus unrepresentative of typical exposure in Houston.

^eWe exclude samples collected from Port Neches, Texas because the EPA (1993a 7-17) reported that the monitor was close to a point source(s) of 1,3-Butadiene, and thus unrepresentative of typical exposure in Port Neches, Texas.

TABLE 11.6-7. THE ESTIMATED PERCENTAGE OF TOXICS EMISSIONS ATTRIBUTABLE TO MOTOR VEHICLES

Toxic Pollutant	EPA (1993a)	Ligocki (1993)
Acetaldehyde	39 ^a	42 ^e
Benzene	59.5 ^b	
1,3-Butadiene	55.5 ^c	
Formaldehyde	33 ^d	37.5 ^e

^aThe EPA (1993a 8-15) based its estimate of the contribution of motor vehicles to ambient acetaldehyde on a study by Ligocki and Whitten (1991 Table 3-2), who estimated emissions of ALD2 (acetaldehydes and other aldehydes) for a weekday in the Summer of 1990 in St. Louis. However, the EPA's (1993a) estimate is weak for several reasons:

Acetaldehyde and other aldehydes are considered together, which may significantly overestimate the contribution of primary emissions of acetaldehyde to ambient acetaldehyde levels; a later study by Ligocki (1993) found that primary emissions of acetaldehyde account for between only 2% and 13% of ambient acetaldehyde in Baltimore (Table 11.6-8 here).

On the other hand, Ligocki and Whitten (1991) did not account for the secondary formation of ALD2 attributable to motor vehicles. As it turns out, Ligocki (1993) reports that the secondary formation of acetaldehyde from motor vehicles is very important – contributing 30% to overall ambient acetaldehyde levels in Baltimore and Washington D.C. in 1988.

The EPA (1993a) estimate does not account for the contribution of upstream emissions, such as from refineries and gas stations; the contribution from these upstream sources is unknown.

Finally, the EPA (1993a) extrapolates the results from one city (St. Louis) to all of the U.S. There are probably significant differences across the U.S., making it impossible to know what the true average should be.

^bEPA (1993a 5-8) estimated that 85% of all benzene emissions are attributable to mobile sources, and that on-road vehicles account for 70% of mobile source emissions (59.5% of total emissions).

^cEPA (1993a 7-4) estimated that mobile sources account for 94% of all 1,3-Butadiene emissions, and that on-road mobile sources account for 59% of mobile source emissions (55.5% of total emissions).

^dEPA (1993a 6-21) cited a variety of studies in deriving this estimate. EPA concluded that 30% of ambient formaldehyde is due to primary emissions, and that 70% is formed in secondary reactions. It assumes that motor vehicles contribute 28% of primary formaldehyde (8.4% of the total), and 35% of secondary formaldehyde (24.5% of the total).

$$\% \text{Formaldehyde}_{\text{motor vehicles}} = (0.28 \cdot 0.30 + 0.35 \cdot 0.70) \cdot 100\% = 33\%$$

^eThe estimated mobile source contributions to ambient formaldehyde and acetaldehyde equal the average of the summer and winter contributions to simulated formaldehyde and acetaldehyde concentrations reported in Table 11.6-8, below.

TABLE 11.6-8. PERCENTAGE OF PRIMARY/SECONDARY AND MOTOR VEHICLE/NON-MOTOR VEHICLE CONTRIBUTIONS TO SIMULATED FORMALDEHYDE AND ACETALDEHYDE CONCENTRATIONS IN BALTIMORE/WASHINGTON IN 1988

Pollutant And Source	Summer	Winter
<u>Formaldehyde</u>		
Motor Vehicle -Primary Emissions	7	30
Motor Vehicle -Secondary Emissions	28	10
Motor Vehicle - TOTAL	35	40
Non-Motor Vehicle - Primary Emissions	13	40
Non-Motor Vehicle - Secondary Emissions	52	20
Non-Motor Vehicle TOTAL	65	60
<u>Acetaldehyde</u>		
Motor Vehicle -Primary Emissions	2	13
Motor Vehicle - Secondary Emissions	30	29
Motor Vehicle - TOTAL	32	52
Non-Motor Vehicle - Primary Emissions	3	17
Non-Motor Vehicle - Secondary Emissions	65	31
Non-Motor Vehicle - TOTAL	68	48

Source Ligocki (1993), written personal communication with Donald R. McCubbin.

TABLE 11.6-9. FIVE MICROENVIRONMENTS USED IN HAPEM-MS AND CO CONCENTRATION AS A FRACTION OF AMBIENT CO READINGS AT FIXED-SITE MONITORS

Microenvironment	Fraction of Fixed-Site Monitor
Indoors - Residence	0.495
Indoors - Other Location	0.619
Outdoors - Near Road	1.001
Outdoors - Other Locations	0.758
Inside Motor Vehicle ^a	1.554

Source Johnson et al. (1992a Table 6)

^aThe figure for inside motor vehicles is relatively low. Flachsbart et al (1987: Tables 5 and 6) found the ratio between in-vehicle ambient CO to be over four in some cases, depending upon the time and the speed of travel. We rely on the results of EPA (1993a) and can not use a higher figure for the present analysis

TABLE 11.6-10. COMPARING THE ADJUSTED AMBIENT ACETALDEHYDE MEASUREMENTS WITH THE HAPEM-MS ESTIMATE OF ACETALDEHYDE EXPOSURE

Region ^a	Metropolitan Statistical Area	Adjusted Ambient (ppb) ^b	HAPEM-MS (ppb) ^c
4	Chicago	0.47	0.14
9	Washington D.C.	0.57	0.18
10	Houston	0.16	0.14
	Baton Rouge	0.45	0.11
	Pensacola	0.19	0.15
11	Wichita	0.43	0.16

Source: Johnson et al. (1992a) and EPA (1993a).

^aFollowing the EPA (1993a), we divide metropolitan statistical areas (MSAs) into regions on the basis of geography and to a lesser extent industrial activity and use of transportation. All MSAs in a region are assumed to have activity patterns identical to those of the "model" MSA of the region.

^bThis estimate is based on the average of all annual averages (weighted by the number of observations in each average) for data from AIRS, UATMP and NAVOC. We have adjusted the ambient data to account for the proportion of time that people spend in each microenvironment, using a different adjustment factor for each of the eleven urban regions in the U.S. (Table 11.6-14).

^cThis is the average annual exposure to motor-vehicle derived acetaldehyde in 1988 estimated by HAPEM-MS.

TABLE 11.6-11. COMPARING THE ADJUSTED AMBIENT 1,3-BUTADIENE MEASUREMENTS WITH THE HAPEM-MS ESTIMATE OF 1,3-BUTADIENE EXPOSURE

Region ^a	Metropolitan Statistical Area	Adjusted Ambient (ppb) ^b	HAPEM-MS (ppb) ^c
3	Bakersfield	0.11	0.19
	San Jose	0.10	0.24
	Stockton	0.11	0.16
4	Chicago	0.05	0.15
5	Burlington	0.16	0.21
9	Washington D.C.	0.06	0.19
10	Houston	0.20	0.15
	Baton Rouge	0.13	0.12
	Dallas	0.03	0.11
	Fort Lauderdale	0.07	0.16
	Miami	0.04	0.21
	Pensacola	0.02	0.16
11	St. Louis	0.03	0.14
	Detroit	0.06	0.12
	Louisville	0.14	0.25
	Wichita	0.03	0.17

Source: Johnson et al. (1992a) and EPA (1993a)

^aFollowing the EPA (1993a), we divide metropolitan statistical areas (MSAs) into regions on the basis of geography and to a lesser extent industrial activity and use of transportation. All MSAs in a region are assumed to have activity patterns identical to those of the "model" MSA of the region.

^bThis estimate is based on the average of all annual averages (weighted by the number of observations in each average) for data from AIRS, UATMP and NAVOC. We have adjusted the ambient data to account for the proportion of time that people spend in each microenvironment, using a different adjustment factor for each of the eleven urban regions in the U.S. (Table 11.6-14)

^cThis is the average annual exposure to 1,3-butadiene in 1988 estimated by HAPEM-MS

TABLE 11.6-12. COMPARING THE ADJUSTED AMBIENT BENZENE MEASUREMENTS WITH THE HAPEM-MS ESTIMATE OF BENZENE EXPOSURE

Region ^a	Metropolitan Statistical Area	Adjusted Ambient (ppb) ^b	HAPEM-MS (ppb) ^c
1	Tacoma	0.49	1.29
2	New York	0.31	1.18
3	Los Angeles	1.35	1.31
	Bakersfield	0.83	0.75
	Fresno	1.44	0.72
	Merced	0.53	0.75
	Modesto	0.56	0.70
	Oakland	0.72	0.79
	Oxnard	0.81	0.70
	Riverside	1.09	0.99
	Sacramento	0.79	0.97
	San Diego	0.71	1.01
	San Francisco	0.54	1.17
	San Jose	0.83	0.95
	Santa Barbara	1.18	0.50
	Santa Rosa	0.82	0.93
	Stockton	0.77	0.62
Vallejo	0.90	0.85	
4	Cleveland	0.84	0.99
	Chicago	0.60	0.59
5	Boston	0.40	0.91
	Burlington	0.40	0.82
	Lowell	0.35	0.91
8	Philadelphia	0.95	0.72
9	Washington D.C.	0.67	0.77
10	Houston	0.66	0.59
	Atlanta	0.34	0.50
	Baton Rouge	0.76	0.48
	Birmingham	0.71	0.83
	Dallas	0.29	0.43
	Fort Lauderdale	0.66	0.61
	Jacksonville	0.25	0.68
	Miami	0.43	0.81
	Pensacola	0.42	0.63
11	St Louis	1.47	0.57
	Detroit	0.63	0.47
	Lansing/East Lansing	0.21	0.66
	Louisville	0.55	0.97
	Wichita	0.37	0.67

Source: Johnson et al (1992a) and EPA (1993a)

^aFollowing the EPA (1993a), we divide metropolitan statistical areas (MSAs) into regions on the basis of geography and to a lesser extent industrial activity and use of transportation. All MSAs in a region are assumed to have activity patterns identical to those of the "model" MSA of the region

^bThis estimate is based on the average of all annual averages (weighted by the number of observations in each average) for data from AIRS, UATMP and NAVOC. (The TAMS data was excluded because it did not report the number of observations in a yearly average. The TAMS data set is small it has benzene observations for four cities and formaldehyde observations for two cities. We have observations for all of these cities in the other three data sets.) We have adjusted the ambient data to account for the proportion of time that people spend in each microenvironment, using a different adjustment factor for each of the eleven urban regions in the U.S (Table 11.6-14)

^cThis is the average annual exposure to benzene in 1988 estimated by HAPEM-MS

TABLE 11.6-13. COMPARING THE ADJUSTED AMBIENT FORMALDEHYDE MEASUREMENTS WITH THE HAPEM-MS ESTIMATE OF FORMALDEHYDE EXPOSURE

Region ^a	Metropolitan Statistical Area	Adjusted Ambient (ppb) ^b	HAPEM-MS (ppb) ^c
4	Chicago	0.93	0.72
9	Washington D.C	1.47	0.92
10	Houston	0.29	0.72
	Baton Rouge	1.00	0.58
	Pensacola	0.50	0.77
11	Wichita	0.92	0.81

Source Johnson et al (1992a) and EPA (1993a)

^aFollowing the EPA (1993a), we divide metropolitan statistical areas (MSAs) into regions on the basis of geography and to a lesser extent industrial activity and use of transportation. All MSAs in a region are assumed to have activity patterns identical to those of the "model" MSA of the region.

^bThis estimate is based on the average of all annual averages (weighted by the number of observations in each average) for data from AIRS, UATMP and NAVOC (The TAMS data was excluded because it did not report the number of observations in a yearly average. The TAMS data set is small; it has benzene observations for four cities, and formaldehyde observations for two cities. We have observations for all of these cities in the other three data sets.) We have adjusted the ambient data to account for the proportion of time that people spend in each microenvironment, using a different adjustment factor for each of the eleven urban regions in the U.S. (Table 11.6-14)

^cThis is the average annual exposure to formaldehyde in 1988 estimated by HAPEM-MS

TABLE 11.6-14. URBAN REGIONAL EXPOSURE ADJUSTMENT FACTORS

Model Area	HAPEM-MS Average CO Exposure (ppm) ^a	Average Annual Ambient CO Level (ppm) ^b	Regional Exposure Adjustment Factor ^c
Boston	0.85	1.36	0.63
Denver	0.93	1.35	0.69
Houston	0.55	0.91	0.60
Los Angeles	1.23	1.79	0.69
Minn /St. Paul	1.07	1.79	0.60
New York	1.11	2.44	0.45
Philadelphia	0.67	1.09	0.61
Phoenix	0.83	1.28	0.65
St. Louis	0.53	0.90	0.59
Spokane	1.46	2.50	0.58
Washington D C	0.71	1.02	0.70

^aWe convert the per capita CO exposure levels ($\mu\text{g}/\text{m}^3$) in Table 11.6-5 to ppm using the conversion factor of 1 ppm = 1145 $\mu\text{g}/\text{m}^3$

^bSource Johnson et al (1992a Table B1)

^cThe regional exposure adjustment factor equals the HAPEM-MS average CO exposure divided by the actual annual ambient CO level (i.e., column 1 divided by column 2)

TABLE 11.6-15. COMPARISON OF THE CO EXPOSURE ESTIMATES OF HAPEM-MS WITH EPA'S ADJUSTED AMBIENT EXPOSURE ESTIMATES, BY MODEL CITY

Model Area	HAPEM-MS Average CO Exposure (ppm) ^a	Average Annual Ambient CO Level, Adjusted for Micro-Environmental Exposure (ppm) ^b	Percent Difference
Boston	0.85	0.85	0.4 %
Denver	0.93	0.84	9.3
Houston	0.55	0.57	-2.4
Los Angeles	1.23	1.11	9.5
Minn /St Paul	1.07	1.11	-4.4
New York	1.11	1.52	-36.9
Philadelphia	0.67	0.68	-1.3
Phoenix	0.83	0.80	4.2
St Louis	0.53	0.56	-5.8
Spokane	1.46	1.56	-6.9
Washington D C	0.71	0.63	10.9

^aWe convert the per capita CO exposure levels ($\mu\text{g}/\text{m}^3$) in Table 11.6-5 to ppm using the conversion factor of 1 ppm = 1145 $\mu\text{g}/\text{m}^3$.

^bThe adjusted average annual CO concentration is equal to the average annual CO level reported in Johnson et al (1992a Table B1) multiplied by 0.622, the EPA's (1993a) integrated exposure constant (see text). We do not adjust for the contribution of motor vehicles to CO exposure because the EPA's HAPEM-MS results did not account for the contribution of motor vehicles to CO exposure.

TABLE 11.6-16. RESULTS FROM REGRESSING THE AMBIENT-BASED ESTIMATE OF TOXICS EXPOSURE ON THE HAPEM-MS ESTIMATE^a

Pollutant	Coefficient	t-Statistic	Lower 95%	Upper 95%
Benzene	0.81	11.7	0.67	0.95
Formaldehyde	1.14	5.6	0.61	1.66
Acetaldehyde	2.53	5.9	1.43	3.64
1,3-Butadiene	0.48	6.5	0.33	0.64

^aWe use the data from Tables 6-10 through 6-13 to run the regressions. Note that we constrain the intercept to pass through zero. The coefficient should be interpreted as the factor with which to multiply the HAPEM-MS estimates of toxic exposure so that they conform with the estimates of toxic exposure determined from our observations of ambient toxics levels.

TABLE 11.6-17. FIFTY YEAR LATENCY PERIOD -- PERCENTAGE OF CANCER CASES ATTRIBUTABLE TO EACH YEAR^a

Year s After Exposure to Carcinogen	Percentage of Total Number of Cancer Cases Each Year
1	0.1
2	0.1
3	0.2
4	0.2
5	0.3
6	0.4
7	0.5
8	0.6
9	0.7
10	0.9
11	1.1
12	1.3
13	1.5
14	1.8
15	2.1
16	2.4
17	2.7
18	3.0
19	3.3
20	3.6
21	4.0
22	4.3
23	4.7
24	5.0
25	5.2

^a We assume that the cancer cases that we estimate will occur, will appear between 1 and 50 years after exposure, with a probability of one. The last half of this period is symmetrical with the first, so we only give the probability for each year in the first twenty-five years

TABLE 11.6-18. UNIT-RISK NUMBERS FOR TOXICS RELEASED FROM THE COMBUSTION OF GASOLINE AND DIESEL FUELS

Pollutant	Unit Risk ^a	Study Type	Cancer Found	Classification ^b
Acetaldehyde ^c	2.2×10^{-6}	rats, hamsters	nasal, nasal, trachea	B2
Benzene ^d	8.3×10^{-6}	humans	leukemia	A
1,3-Butadiene ^e	2.8×10^{-5}	mouse, rat	multiple tumors	B2
Diesel Particulates ^f	1.7×10^{-5}	rat	lung	B1
Formaldehyde ^g	1.3×10^{-5} (6.0×10^{-7})	rats, humans	squamous cell carcinoma, lung, buccal, nasopharyngeal	B1
Gasoline Particulates ^h	5.1×10^{-5} (1.6×10^{-5})	comparative potency	lung	ⁱ

^aUnit Risk = Cancer Rate/70 ppm*years/one individual/ $\mu\text{g}/\text{m}^3$

^bCarcinogenic classifications are described in Table 11 6-19

^cThe EPA (1993a 8-25) used an upper 95% confidence interval estimate, meaning that there is only a 5% chance the "true" unit risk is greater than this, they did not report a maximum likelihood estimate (MLE)

^dThe benzene unit risk estimate is a MLE (EPA, 1993a 5-39)

^eWe use the MLE for 1,3-Butadiene The 95% confidence interval estimate is an order of magnitude higher (2.8×10^{-4}) (EPA, 1993a 7-27).

^fThis is a 95% confidence interval estimate The EPA (1993a 9-34) did not report a MLE

^gThe formaldehyde unit risk number is under review, the updated (but unofficial) unit risk number is in parenthesis It is substantially lower than the official unit risk number The uncertainty in the estimated unit-risk for formaldehyde is caused, in large part, by a highly nonlinear relationship between dose and response a 2.5-fold increase in the dose of formaldehyde caused a fifty-fold increase in the number of tumors (EPA, 1993a 6-30) We use the unofficial (lower) unit-risk number

^hThe unit risk number for gasoline particulates applies to vehicles with a catalytic converter, the number in parentheses applies to vehicles without catalytic converters (EPA, 1993a 10-5) This is a 95% confidence interval estimate. The EPA did not report a MLE for gasoline particulates

ⁱGasoline particulates do not have a classification

TABLE 11.6-19. U.S. ENVIRONMENTAL PROTECTION AGENCY CANCER CLASSIFICATION SYSTEM

Classification	Description
A	Human carcinogen, with sufficient evidence from epidemiological studies
B1	Probable human carcinogen, with limited evidence from epidemiological studies
B2	Probable human carcinogen, with sufficient evidence from animal studies and inadequate evidence or no data from epidemiological studies
C	Possible human carcinogen, with limited evidence from animal studies in the absence of human data
D	Not classifiable as to human carcinogenicity, owing to inadequate human and animal evidence
E	Evidence of noncarcinogenicity for humans, with no evidence of carcinogenicity in at least two adequate animal tests in different species, or in both adequate animal and epidemiological studies

Source Cochrssen and Covello (1989 pp 49-50).

TABLE 11.6-20. TYPE OF CANCER ASSOCIATED WITH TOXICS RELEASED FROM THE COMBUSTION OF GASOLINE AND DIESEL FUELS BY MOTOR VEHICLES IN FUTURE YEARS

Pollutant	Cancer Type ^a
Acetaldehyde	Oral Cavity and Pharynx
Benzene	Leukemia
1,3-Butadiene	All Sites
Diesel Particulates	Lung
Formaldehyde	Oral Cavity and Pharynx
Gasoline Particulates	Lung

^aThe type of cancer depends on the studies on which the unit risk estimates are based

TABLE 11.6-21. TRENDS IN THE FIVE-YEAR RECOVERY RATE FOR CANCERS CAUSED BY TOXICS ASSOCIATED WITH MOTOR VEHICLES (WHITES)^a

Cancer Type	1960-63	1970-73	1974-76	1977-80	1981-87
All Sites	39	43	50	51	53
Lung and Bronchus	8	10	12	13	13
Leukemia	14	22	34	36	36
Oral Cavity and Pharynx	45	43	55	54	54

Source: American Cancer Society (1992)

^aThe five-year recovery rate is the percentage of people that are still alive five years after diagnosis, taking into account normal life expectancy (e.g., correcting for factors such as heart disease, accidents and diseases of old age)

TABLE 11.6-22. TRENDS IN THE FIVE-YEAR RECOVERY RATE FOR CANCERS CAUSED BY TOXICS ASSOCIATED WITH MOTOR VEHICLES (AFRICAN-AMERICANS)^a

Cancer Type	1960-63	1970-73	1974-76	1977-80	1981-87
All Sites ^b	27	31	39	39	38
Lung and Bronchus	5	7	11	12	11
Leukemia	--	--	31	30	29
Oral Cavity and Pharynx	--	--	35	34	31

Source American Cancer Society (1992)

^a The five-year recovery rate is the percentage of people that are still alive five years after diagnosis, taking into account normal life expectancy (e.g , correcting for factors such as heart disease, accidents and diseases of old age)

^b The five-year survival rate for all cancer sites is lower for African-Americans (38%) than for whites (53%) In part this is due to later diagnosis Also African-Americans have a 7% higher incidence of cancer

TABLE 11.6-23. FIVE-YEAR CANCER RECOVERY RATE ASSOCIATED WITH TOXICS RELEASED FROM THE COMBUSTION OF GASOLINE AND DIESEL FUELS BY MOTOR VEHICLES, AFRICAN-AMERICANS AND WHITES (1988-1992)^a

Cancer	1988	1989	1990	1991	1992
All Sites	49	49	50	50	51
Lung and Bronchus	13	13	13	13	13
Leukemia	33	33	34	35	35
Oral Cavity and Pharynx	51	51	52	52	51

Source American Cancer Society (various years)

^aThe five-year recovery rate is the percentage of people that are still alive five years after diagnosis, taking into account normal life expectancy (e.g , correcting for factors such as heart disease, accidents and diseases of old age)

TABLE 11.6-24. ESTIMATED FIVE-YEAR RECOVERY RATE OF CANCERS ASSOCIATED WITH TOXICS RELEASED FROM THE COMBUSTION OF GASOLINE AND DIESEL FUELS BY MOTOR VEHICLES IN FUTURE YEARS^a

Cancer	2000 ^b	2010	2020	2030	2040
All Sites	56	63	69	76	82
Lung and Bronchus	16	18	21	23	26
Leukemia	43	53	63	73	83
Oral Cavity and Pharynx	54	57	61	65	68

^aThe five-year recovery rate is the percentage of people that are still alive five years after diagnosis, taking into account normal life expectancy (e.g., correcting for factors such as heart disease, accidents and diseases of old age)

^bTo estimate recovery rates in future years, we regressed (linear OLS) the recovery rates on time. We use the recovery rates in Tables 6-21 and 6-22, weighted by population (12% African-Americans) and the fact that the cancer incidence is 7% higher for African-Americans than whites, we assume other minorities have incidence and recovery rates the same as whites. The estimated coefficients (and r-squared in parentheses) in the linear regression are: all sites = 0.65 (0.91), lung and bronchus = 0.25, leukemia = 1.01 (0.88), and oral cavity and pharynx = 0.36 (0.59)

Note that the data of Tables 11.6-21, 11.6-22, and 11.6-23 suggest that the recovery rate has leveled off in recent years. If so, and if this leveling off actually continues, then our linear extrapolation (via regression) overestimates recovery rates in the outer years, and hence underestimates costs. To check this, we re-estimated leukemia and lung-cancer costs assuming that recovery rates remain at their 1990 levels. The cost of each toxic increased by less than 20%, and the total social cost of air pollution changed by less than 0.01%.

11.7 RESULTS

Most of the results that we present are based on a 10% reduction in motor vehicle emissions. However, for points of reference, we also estimate the health effects associated with a 100% reduction in motor vehicle emissions and a 100% reduction in all anthropogenic emissions. We focus on a 10% reduction because pollution formation and the associated health effects are the products of nonlinear functions, and we feel that it is more useful for policy makers to consider the effect of a relatively small reduction in emissions. As it turns out, the relationship between emissions and health effects is reasonably linear, and we mostly present the effects of a 10% reduction in emissions.

The results are presented in a number of ways. We estimate the human-health cost in the entire U.S., in urban areas of the U.S., in rural areas of the U.S., and in 11 major metropolitan statistical areas (MSAs): Boston, Denver, Houston, Los Angeles, Minneapolis, New York, Philadelphia, Phoenix, St. Louis, Spokane, and Washington D. C. We estimate the total dollar costs, dollar costs per vehicle-mile of travel, and dollar costs per kg of pollutant emitted. We also estimate costs due to direct emissions from motor vehicles, emissions of road-dust particulate matter, and emissions from "upstream" fuelcycle processes.

Table 11.7-1 presents "background" statistics for the MSAs: population, vehicle miles traveled (VMT), and the annual average pollution level; it also gives population and VMT for urban and rural areas and for the nation.

Table 11.7-2 summarizes the assumptions that we use in the lower and upper bounds. These tables are especially important because they help to explain the difference between the lower-bound cost and the upper-bound cost estimates; further differences between the lower and upper bound are described in Report #16 in the social-cost series listed at the beginning of this report.

Tables 11.7-3 and 11.7-4 presents national estimates of the number of cases of adverse health and their economic cost caused by all anthropogenic pollution between 1988 and 1991.

Tables 11.4-5 and 11.4-6 give the gram-per-mile emission estimates and the cost per VMT by vehicle class and pollutant, based on a 10% reduction in motor vehicle emissions.

Table 11.7-7 gives the cost per kilogram of emission by pollutant, emissions source and geographic region, again based on a 10% reduction in motor vehicle emissions. This table is useful because it allows one to calculate the cost of emissions from sources other than motor vehicles, such as petroleum refineries, or from motor-vehicles that have emission rates different from the national-average rates used in this study (for example, future low-emitting gasoline vehicles, or alternative-fuel vehicles). A nice feature of the \$/kg estimates is that, unlike the total \$ or \$/VMT estimates, they are not affected by the uncertainty in the emissions inventory, because they are expressed per kilogram emitted. (They are, however, affected by uncertainty in the estimates of the exposure factors.) Furthermore, using the population figures in Table 11.7-1, one can use the fact that damages are

proportional to the number of people and calculate \$/kg/person. We discuss this table more below.

Finally, in the Appendix, Tables 11.A-1 through 11.A-4 give the lower and upper-bound benefit estimates of a 100% reduction of motor vehicle emissions, in dollars per one thousand VMT, for urban areas, and the nation as a whole. Tables 11.A-5 through 11.A-60 give the lower and upper-bound benefit estimates of a 10% reduction of motor vehicle emissions, in millions of dollars and in dollars per one thousand VMT, for eleven metropolitan areas, urban areas, rural areas, and the nation as a whole. In each of these tables, we show costs by pollutant, by vehicle class, and by emissions source. The 60 tables are arranged as follows:

<i>Region</i>	Millions of dollars		Dollars/1000 VMT	
	<i>Lower bound</i>	<i>Upper bound</i>	<i>Lower bound</i>	<i>Upper bound</i>
<i>100% emission reduction</i>				
All United States	Table 11.A-1	Table 11.A-2		
All urban areas	Table 11.A-3	Table 11.A-4		
<i>10% emission reduction</i>				
All United States	Table 11.A-5	Table 11.A-6	Table 11.A-33	Table 11.A-34
All urban areas	Table 11.A-7	Table 11.A-8	Table 11.A-35	Table 11.A-36
All rural areas	Table 11.A-9	Table 11.A-10	Table 11.A-37	Table 11.A-38
Boston	Table 11.A-11	Table 11.A-12	Table 11.A-39	Table 11.A-40
Denver	Table 11.A-13	Table 11.A-14	Table 11.A-41	Table 11.A-42
Houston	Table 11.A-15	Table 11.A-16	Table 11.A-43	Table 11.A-44
Los Angeles	Table 11.A-17	Table 11.A-18	Table 11.A-45	Table 11.A-46
Minneapolis	Table 11.A-19	Table 11.A-20	Table 11.A-47	Table 11.A-48
New York	Table 11.A-21	Table 11.A-22	Table 11.A-49	Table 11.A-50
Philadelphia	Table 11.A-23	Table 11.A-24	Table 11.A-51	Table 11.A-52
Phoenix	Table 11.A-25	Table 11.A-26	Table 11.A-53	Table 11.A-54
St. Louis	Table 11.A-27	Table 11.A-28	Table 11.A-55	Table 11.A-56
Spokane	Table 11.A-29	Table 11.A-30	Table 11.A-57	Table 11.A-58
Washington D. C.	Table 11.A-31	Table 11.A-32	Table 11.A-59	Table 11.A-60

11.7.1 Discussion of results

Our summary tables, we hope, are self explanatory. Rather than simply review the results, we emphasize five points.

I). Our upper-bound estimate of the health cost of motor-vehicle air pollution is enormous: \$450 billion dollars annually in the U.S. (Table 11.A-2). However, we are uneasy with this result, even as an upper bound.

Virtually all of the upper-bound cost -- 96% of it -- is damage from particulate air pollution. Most of the damage from particulates is mortality and chronic illness. In the upper bound, mortality from particulate air pollution accounts for 69% of total damages from all the pollutants -- \$312 billion annually. Of this \$312 billion, about \$235 billion are due to long-term or "chronic" deaths, as opposed to short-term or "acute" deaths.

In our upper bound, we use a recent prospective cohort study by Pope et al. (1995a; see our Chapter 11.4 here). However, as shown in Table 11.7-8 and mentioned in Chapter 11.4, the Pope et al. study estimates many more chronic deaths than does our lower-bound study, by Ozkaynak and Thurston (O&T) (1987). (Recall from Chapter 11.4, and see below, that chronic deaths are equal to total estimated deaths less acute non-harvest deaths). As shown in Table 11.7-8, had we used our lower-bound estimate of the change in the long-term mortality rate per change in particulate pollution, with all of our other upper-bound assumptions, our upper bound estimate of damages from particulate-related mortality would have been almost 50% (\$234 billion) lower (line 15A versus 15B). This dramatic dependency of the total damages on a single study is unsettling. One certainly could argue that the Pope et al. study gives implausibly large results, even as an upper bound, and should not be used pending corroboration.

We distinguish between "acute harvest" deaths (deaths that occur immediately after exposure to air pollution and would have occurred very soon anyway even if there had been no particulate air pollution) and "acute non-harvest" deaths (immediate deaths that would not have occurred soon had there been no particulate pollution) because we value the former much less than the latter (see Chapter 11.4). We distinguish between acute non-harvest deaths and "chronic" (greatly delayed) deaths because the latter occur in the future and so have a lower present value than (although the same nominal value as) the former. We assume that the number of chronic deaths is equal to the total number of cross-sectional deaths (Pope et al., 1995a, or O&T, 1987) less the amount of acute non harvest deaths.

II). The difference between our lower and our upper-bound estimates of particulates is considerable -- typically a factor of ten to twenty. This is the product of three factors: the estimated number of health effects, the value attached to the health effects, and (when comparing the lower and upper bounds for motor vehicles) the share of the damages attributable to motor vehicles. There is more uncertainty when considering motor vehicles' damages than when examining the damages of all particulates because the share of damages attributable to motor vehicles is uncertain. When considering the mortality damages of motor vehicle particulates (Table 11.7-8, lines 16A and 16B), we see the lower and upper bounds differ by a factor of almost 19. The reason for this large difference is roughly as

follows: the number of deaths differs by a factor of a little over two, the value of deaths by a factor of four to seven, and the share of motor vehicles by about 1.4.¹

Chronic illness contributes \$115.4 billion in our upper bound case, while in the lower bound it contributes only \$4.4 billion. This (factor of 26.2!) uncertainty has three sources: 1) the estimated number of health effects differs by roughly a factor of about 2.4 between the lower and upper bounds; 2) the value attached to the health effects differs by a factor of 7.4; and 3) the share of the damages attributable to motor vehicles by a factor of about 1.5. This great uncertainty, while unsettling, simply reflects the current lack of knowledge about quantifying the link between air pollution and chronic illness.

III). Ozone is the least damaging pollutant emitted directly from motor vehicles -- less damaging even than CO and NO_x. Even in Los Angeles, the lower-bound ozone damages from motor-vehicles (see Table 11.7-9) amount to only a few dollars per person per year -- an evidently trivial amount. In the upper-bound case, damages from ozone are much higher than in the lower bound case, but still far less than PM damages from any source, including again the heretofore overlooked road dust². We may conclude then, that unless ozone causes mortality (which most researchers apparently doubt) or is associated with chronic illness (which seems a more likely possibility), the dollar health damages from ozone are minor except during the most polluted times in the most polluted places.

IV). Note that the \$ and \$/VMT results apply to the per-mile emission levels of circa 1990. The Clean Air Act Amendments of 1990 mandated significant reductions in emissions from many sources, and as a result, vehicles and upstream emissions sources in the future will emit less per mile of travel and have a lower \$/VMT cost than they did in 1990. To estimate the \$/VMT cost of future, cleaner vehicles and upstream emissions sources, multiply the pertinent \$/kg factors of Table 11.7-7 by the associated estimated future kg/mi emission rates.

The \$/kg factors of Table 11.7-7 are independent of the kg/mi emission rate, and so can be applied to any future emission rate. They are, however, proportional to the *exposed* population: if you expect the exposed population to increase by 10% over 1990 levels, then you should increase the pertinent \$/kg values by 10%.

¹Table 11 7-13 suggests that difference between the motor vehicle share (including road dust and upstream sources) in the lower and upper bounds differs by a factor of 1.4. Indeed, the average motor vehicle share for the nation differs by a factor of 1.4 (In the lower bound the average share is 20% and in the upper bound it is 28%)

²We point out that this perhaps surprising prominence of road dust over ozone most definitely is not due to us having made liberal assumptions regarding road-dust damages and conservative assumptions regarding ozone damages. To the contrary, we have made a number of adjustments that significantly reduce the impact of road-dust. we have reduced the EPA's official estimate of road-dust emissions, we have assumed that road-dust particles are significantly less potent than are other particles, and we have down-weighted the impact of particles greater than 2.5 microns in diameter, which most road-dust particles are

Similarly, the \$/kg estimates are proportional to the assumed value of health effects. The \$/kg estimates also depend somewhat on the total change in pollution or emissions being considered, because some health effects are non-linearly related to pollution levels. However, the dependency is not strong: most of the major costs either vary linearly with pollution levels (in which case the \$/kg cost is independent of the pollution level), or else nearly linearly.

The \$/kg factors for different emissions sources (e.g., road dust versus motor-vehicle tailpipes) incorporate differences in the size of the population exposed, the ratio of effective exposure to emissions, the potency of particles by size class, and the potency of particles by composition. Thus, we see in Table 11.7-7 that the \$/kg cost of particulate emissions directly from motor vehicles vastly exceeds the \$/kg cost of particulates from road dust, because motor vehicle PM is more potent than is road-dust PM, and because more people are exposed to motor-vehicle PM than to road-dust PM.

V). A comparison of Tables 11.7-4, 11.7-9 and 11.7.10 reveals that, in the Los Angeles MSA, motor vehicles contribute 59% percent of anthropogenic damages in the lower bound and a rather high 86% in the upper bound. The national average is somewhat lower: 42% in the lower bound and 64% in the upper bound. These are surprisingly high figures, and are explained by the relative proximity of motor vehicles to people, particularly in urban areas, and the uncertainty regarding the emissions and potency of road dust.

VI). Two other results are worth highlighting: the nontrivial damages from upstream emissions (higher than damages from ozone attributable to direct motor-vehicle emissions), and the trivial damages from air toxics.

11.7.2. Implications of the results.

Our findings have interesting implications. Most regulations and analyses have focused on emissions of ozone precursors from motor vehicles. Yet, as we mentioned above, in our lower-bound case, ozone is the least damaging pollutant emitted directly from motor vehicles – less damaging even than CO and NO_x – and in fact is about an order of magnitude less damaging than road-dust, which no-one has even considered regulating. Ozone damages are much higher in the upper bound case, but still less far less than damages from particulates, including road dust.

Even though there is considerable uncertainty in our analysis, it is clear that, over a wide range of assumptions, damages from particulates dominate the total cost of the health effects of motor-vehicle air pollution. The summary of the damages (million \$) in Table 11.7-9 shows this dramatically. Particulates dominate mainly because they are strongly associated with mortality, which is so costly.

We do not imply that the considerable resources that have been devoted to regulating and analyzing ozone air pollution have been wasted utterly. We do suggest, however, that regulatory and analytical efforts be broadened and redirected. (This in fact is starting to happen.) Certainly, we find no basis for the presumption, too common among many analysts, that the carbon-monoxide problem now can be

considered to be "solved" (even if CO levels come down by a factor of two, CO damages still will be of the same order of magnitude as ozone damages), or that fugitive dust can continue to be ignored.

11.7.3. Comparison with Studies of Air Pollution Costs in the South Coast Air Basin

Several well-done studies have estimated the economic benefits derived from reducing air pollution in the South Coast Air Basin. We focus our comparison of results here.

11.7.3.1. Small and Kazimi (1995), Krupnick and Portney (1991) , and Hall et al. (1992): the cost of air pollution in the Los Angeles Region

Krupnick and Portney (1991), Hall et al. (1992) and Small and Kazimi (1995) have estimated the health cost of ozone and particulate pollution in the South Coast Air Quality Management District. In Table 11.7-10, we compare their estimates with our estimates for the Los Angeles region.³

The four estimates differ widely, mainly on account of differences in particulate mortality and morbidity costs. Our estimates of mortality costs are higher than those of Krupnick and Portney (1991) and Hall et al. (1992) because we estimate a larger number of deaths, and, in comparison with Krupnick and Portney (1990), a higher value of life (see Table 11.4-6). Hall et al. (1992) and Krupnick and Portney (1991) reported that reducing pollution levels to the ambient air quality standard would save 1600-2000 lives in the South Coast Air Quality Management District; we estimate that removing anthropogenic particulate pollution would save 9,700-13,100 lives in Los Angeles. Our estimate comes closest to Small and Kazimi's as they estimate that 4,000-9,000 lives are lost annually and they use a slightly higher value of life than we do (see Table 11.7-10, note "d").

There are several reasons that our estimate of the number of deaths is higher than the previous studies.

- 1) We estimate the effects of all anthropogenic pollution, whereas Krupnick and Portney and Hall et al. estimate the effects of the reductions necessary to achieve ambient air quality standards in the year 2010. We estimate the effects of all anthropogenic pollution because it appears that there are health effects at concentrations below the ambient air quality standard.
- 2) We include deaths estimated from both time-series studies and cross-sectional studies, whereas Hall et al., Krupnick and Portney, and Small and Kazimi used only cross-sectional studies. Most importantly, in our upper bound case we use a recent study (Pope et al. 1995a) that estimates more cross-sectional deaths per unit change in PM pollution.

³ The Los Angeles region that we estimate includes four counties Los Angeles, Orange, Riverside, and San Bernadino The South Coast Air Quality District includes Orange County and portions of Los Angeles, Riverside and San Bernadino counties

3) Our estimates include all of Riverside, Los Angeles, and San Bernadino Counties, whereas the other's estimate include only portions.

We estimate a higher cost of morbidity due to particulates primarily because we include an estimate of the cost of chronic illness, and the others do not. Our estimate of the cost of ozone morbidity is lower than the estimate of Hall et al. (1992) because they use laboratory studies to estimate acute morbidity damages, and in general, the laboratory results give higher estimates than do epidemiological studies.

11.7.3.2. *Small and Kazimi (1995): the cost of motor-vehicle air pollution in the Los Angeles Region*

Small and Kazimi (1995) estimated the cost of motor-vehicle particulate and ozone air pollution in the South Coast Air Quality Management District. We can compare their estimates with our estimates of the cost of motor-vehicle ozone and particulate air pollution in Los Angeles.

To estimate the impact of particulates on mortality, Small and Kazimi started by estimating motor vehicles' contribution to ambient PM₁₀ levels from *direct* PM₁₀ emissions and the atmospheric transformation of VOC, NO_x and SO_x to secondary particulates. They then estimated the health impact of these different sources, by using the fraction of the annual average ambient PM₁₀ level attributable to each source and a dose-response function by Ozkaynak and Thurston (1987). They use the results of Krupnick and Portney (1991) and Hall et al. (1992) to estimate ozone and particulate morbidity. Their cost estimates for the 1992 fleet are shown in Table 11.7-11. The results shown in Table 11.7-11 do not include the cost of upstream emissions, which they did not estimate, or the cost of road dust, which they estimated but excluded from their baseline results on account of the uncertainty in the road-dust emissions inventory. They calculated that dust from paved roads would have added 4.3 cents per mile to the cost of light-duty vehicles (Table 11.7-11). This would have resulted in a total cost of 7.6 cents per mile for light-duty gasoline vehicles, compared to 2.4 to 36.5 cents per mile in our analysis.

The estimates of Small and Kazimi (1995) fall within our ranges, but at the low end. There are two reasons that our upper-bound estimates are so much larger than Small and Kazimi's baseline estimates. First, as shown in Table 11.7-10, our upper bound estimate of the total cost of all anthropogenic ozone and particulate pollution in Los Angeles is larger than Small and Kazimi's estimate of same, mainly because we include more effects than they do: we include chronic illness from particulates and time-series as well as cross-sectional deaths from particulates. Second, in our upper bound (and on average) we attribute to motor vehicles a larger fraction of the total ozone and particulate damages (Table 11.7-12). Small and Kazimi apportion particulate damages on the basis of the contribution of particulates sources to the downtown Los Angeles monitoring station, and apportion ozone damages on the basis of the emission shares of ozone precursors. By contrast, we use exposure-weighted emission shares to apportion particulate damages, in each county in the Los Angeles region. The resulting contribution of motor vehicle

tailpipe emissions to total PM are shown in Table 11.7-12; Table 11.7-12 also presents the contribution of tailpipe emissions plus road dust and upstream sources.

Thus, in our high-cost case, we estimate greater total pollution damages, and attribute a greater fraction of this [greater] total to motor vehicles, than do Small and Kazimi (1995). Consequently, our upper-bound estimate of motor-vehicle pollution damages is quite a bit higher than Small and Kazimi's estimate. (Small and Kazimi's baseline estimate of motor-vehicle damages is higher than our lower-bound estimate of motor-vehicle damages primarily because their baseline estimate of total air pollution damages is higher than our lower-bound estimate.)

Note that we also estimate the cost of CO, NO_x, and toxic air pollution from motor vehicles, as well as the cost of O₃ and PM₁₀ pollution from vehicles. In Table 11.7-11 we have included only O₃ and PM₁₀ so that our estimates may be compared properly with those of Small and Kazimi (1995).

11.7.4. Summary of Sources of Uncertainty and Future Work Needed

The estimation of the social-cost of motor-vehicle emissions has three steps: estimating exposure to pollution, determining damages caused by exposure, and valuing the damages. Each of these steps is uncertain. In the following paragraphs we briefly summarize the key uncertainties. Most of these are discussed in more detail elsewhere in this report or other reports.

11.7.4.1 Exposure to pollution: general methodological issues

A). There are two general approaches to estimating exposure to air pollution: 1) model the movement and exposure of individuals in many micro-environments, over the course of the day, or 2) use readings from outdoor ambient air-quality monitors to represent the "average" exposure of large groups of people over wide areas and relatively long periods of time. In the first approach, the health effects of the micro-modeled exposure are estimated using dose-response functions derived from studies of the effects of exposure to air pollution in the laboratory. In the second approach, the health effects are estimated from dose-response functions derived from epidemiological studies of large populations and ambient air quality. The advantage of the first approach is that it uses a detailed, micro-model of actual individual exposure; the disadvantage is the weaknesses of the clinical studies that one must use to estimate health effects of micro-modeled exposure. The clinical studies are much less robust than are the epidemiological studies, because they apply to specific individuals under specific circumstances. Also, the endpoints of clinical studies generally are more difficult to value than are the endpoints of epidemiological studies.

In any event, the micro-exposure/clinical-study approach appears to estimate significantly higher damages than does the ambient-air-quality/epidemiological approach. Future research should aim to better understand and reduce this difference.

B). Most if not all epidemiological analyses and analyses of health costs done to date have (implicitly) weighted the readings from all monitors equally; that is, they have not tried to weight readings from monitors in proportion to the amount of human exposure associated with the pollution measured by the monitor. Future

analyses perhaps should attempt to weight monitors by exposure. We note, though, that an analysis of the health effects of ambient air pollution ought to treat monitor readings in the same way that they are treated in the epidemiological studies used to estimate dose-response functions.

11.7.4.2 *Exposure to pollution: modeling the contribution of motor vehicles to ambient air pollution*

We use a detailed emissions inventory, and a relatively crude air-quality model, to estimate the motor-vehicle contribution to ambient air pollution (see Report #16 in the social-cost series listed at the beginning of this report). There are several problems with this method.

A). For many pollutants, the emission inventory is uncertain, primarily because the underlying emission factors (in grams/unit of activity) are not well characterized. The emissions inventory for fugitive dust, and for many natural sources, is particularly uncertain. For example, one natural source, "dust devils" (arising in the desert) is not included in the inventory. (This makes it particularly difficult to determine the anthropogenic portion of total pollution.) The uncertainty in the emissions inventory is reflected in the span between our low and high emission "correction" factors -- the factors that we applied to the "official" EPA emissions inventory in order to produce a more accurate and properly characterized emissions inventory.

B). To estimate the contribution of motor vehicles and other sources to ambient air pollution, we use a relatively simple Gaussian plume dispersion model, in which pollutant concentration is a function of the rate of emissions, the velocity of the wind, distance from emissions source to receptor, the height of the emissions source, the height of the atmospheric region in which pollutants can mix (called the "mixing layer"); the deposition or settling of pollutants; turbulent diffusion, and many other factors. Most of these parameters are uncertain. (The parameters that characterize atmospheric turbulence, and the source-receptor distance, are especially uncertain.) Moreover, the model itself is relatively crude: it assumes that the earth's surface is a plane, the atmosphere is homogenous, vertical wind speed is zero, particles are perfectly reflected from the surface and the underside of the inversion layer, emission sources are constant over time, and the effects of turbulent diffusion in the direction of the wind are negligible in comparison to the effects of transport by the wind.

The uncertainty in the parameter values and the crudity of the model structure result in considerable uncertainty in our estimate of the contribution of one source *relative* to that of another. (We do emphasize, though, that we use the model to estimate the *relative* contribution, and that this should be much less uncertain than an estimate of the absolute concentration.) The use of more sophisticated air-quality models, and better data on source locations, can go a long way towards reducing this uncertainty.

C). Our modeling of ozone is particularly weak, especially in light of the sophisticated modeling of regional ozone levels. Still, our simplistic model probably is not a relatively large source of uncertainty, because it is unlikely that the actual contribution of motor vehicles to ambient ozone differs from the modeled

contribution by more than 50%. This potential error actually is fairly small compared to the ranges of uncertainties shown in Table 11.7-2. Moreover, if total ozone damages are closer to our lower than our upper bound, then the total damages are so low both absolutely and relative to damages from other pollutants that it matters not how the damages are apportioned. Thus, it is likely (and somewhat ironic) that, in a social-cost analysis anyway, it probably is not worthwhile to do a detailed analysis of the one problem -- contribution to ozone levels -- for which the available tools are far more sophisticated than we have used here.

D). We do account for the formation of particulates in the atmosphere from NO_x , SO_x , VOC, and NH_3 emissions, but the methods and assumptions underlying the data that we use are quite simplified. Much more work needs to be done to adequately characterize the formation of the so-called "secondary" particulates.

E). We do not address the long-range transport of pollution. A rigorous, if expensive, approach would be to perform a chemical mass balance study or tracer study in each region of interest to determine the sources of ambient pollution.

11.7.4.3 *Damage Estimation*

A). In some of the instances in which there are two or more epidemiological studies of a particular pollutant and health effect, the damage coefficient -- the estimated change in the incidence rate per unit change in pollution -- differs greatly among the studies. Unless there is a compelling reason for choosing or weighting one study over another, these differences should be acknowledged and incorporated into the analysis. This can introduce considerable uncertainty.

B). Most studies have assumed that the health effects associated with a particular pollutant depend on that pollutant only, and not on interactions between pollutants. Recent studies suggest that a suite of pollutants may interact among themselves and with environmental variables (e.g., pollen, weather) to cause adverse effects. More research and more sophisticated models are needed to address this question.

C). Particulates cause most of the estimated damages, and are the source of the greatest uncertainty in the analysis. Research on particulates has lagged research on other pollutants, and as a result several important questions still need to be answered:

1) To what extent do time-series studies and cross-sectional (including prospective cohort) studies measure the same deaths due to air pollution? The scant available evidence suggests that there is only a partial overlap between the two types of studies, and thus that the results are, to some extent, additive rather than duplicative. However, a wide range of assumptions about the degree of overlap is reasonable (it even is theoretically possible that there are types of particulate-related deaths not captured by either type of study, although there is little evidence that this is so), and this wide range of course results in a wide range of estimated costs.

2) What is the mortality coefficient -- the change in the death rate per unit change in particulate pollution? Two recent, prospective cohort studies -- probably the best studies available -- have relatively large mortality coefficients, two to four times larger than those from previous studies. We use the coefficient from one of these studies (Pope et al., 1995a), in our upper bound estimate of cross-sectional and

chronic deaths. This makes our upper-bound estimates of the total cost quite a bit higher than the estimates in other similar studies.

3). What is the effect of particulate air pollution on chronic illness? Our estimate of chronic illness is especially uncertain. Although recent work indicates that air pollution harms the growth, development and health of the respiratory system, it is difficult to quantify this effect in a way that is useful for cost analyses. More work needs to be done to determine the size of this effect and the best way to estimate people's exposure.

4). To what extent does the effect of PM depend on the size of the particle? Many researchers believe that "fine" particulate matter -- that with a diameter of 2.5 microns or less -- is more dangerous than larger particulate matter, but the functional relationship between particle size and health effects has not been quantified. On the basis of recent findings, and discussions with epidemiologists, we assume that PM_{2.5} is considerably more potent than the coarse fraction of PM₁₀, that particles larger than PM₁₀ have no effect on human health at all, and that all particles (of a given type) smaller than PM_{2.5} have the same effect. However, these are little more than our judgments, and probably wrong in detail. For example, it is possible that very fine particles (PM_{1.0}, or even smaller), cause more damage than do the particles in the PM_{1.0} to PM_{2.5} range. Ideally, one would know the continuous size distribution of particles from each emissions source, and the damages as a continuous function of size.

5). To what extent does the effect of PM depend on the chemical composition of the particles? Unfortunately, this dependence on chemistry is less well understood than is even the relatively poorly understood dependence on size. There is some indication that road dust and other soil- and mineral-based particulate matter is less damaging than is sulfate particulate matter from combustion, but the evidence is only suggestive, and certainly does not yet demonstrate that mineral-based particulates such as road dust have no adverse health effects at all. We assume in our analysis that fugitive dust PM is less dangerous than other kinds of PM, but not completely harmless.

Generally, there are many, many uncertainties regarding the characterization of, exposure to, and health effects of particulate air pollution. The July 1995 issue of the journal *Inhalation Toxicology* provides a good summary of research needs.

D). Air pollution probably has different effects on different age groups, but the differences have not been well-quantified. These differences might be important because society might wish to attach different values to damages to different age groups. Future work should determine damage coefficients by age group.

11.7.4.4 Valuing Damages

All of the values that we use to monetize air pollution damages are uncertain. As shown in Table 11.7-2, the difference between the lower and upper bounds varies by a factor of roughly two to four.⁴ Certainly more work would be helpful to further refine our estimates, particularly in regard to our estimates of morbidity and the value of life. With regards to the latter, there are at least three issues:

- 1) are the available estimates of the value of life (actually, value of risk) pertinent to the risks posed by air pollution specifically?;
- 2) is the value of life a function of the age of the individual?; and
- 3) is the value of life a function of the number of years lost?

Although further research will help answer these and other questions, and reduce the uncertainty in the valuation step, it is unlikely that society ever will agree on point estimates for damage values, and that we always will have to choose plausible lower and upper bounds.

11.7.4.5 Seasonality

Our work does little to consider the seasonal aspects of the pollution problem. Some epidemiological work has found that the effects of pollutants differ by season, perhaps because of temperature, pollen or other factors. In addition, some pollution emission sources vary by the season: fugitive dust from agricultural sources occurs primarily in the summer, and wood smoke in the winter. A more careful study will estimate damages as they occur in each season, by each pollutant (or a mix of pollutants) and then apportion the damages to the appropriate sources.

In sum, there is much that we have yet to learn about what exactly are the damages that are caused by pollution and what are the prime contributors of those damages. If nothing else, our work illustrates the considerable uncertainties in this exercise, and highlights the need for further research.

⁴We should point out that our upper bound estimate of the value of the life is much less than some values reported in the literature and assumed in other studies of health effects (see Tables 11.4-5 and 11.4-6). Consequently, we could argue that our treatment of uncertainty actually is conservative, and that the overall gap between the lower and the upper bound damages is even greater than estimated here.

ABBREVIATIONS USED IN TABLES IN CHAPTER 7

Vehicles

LDGV = light-duty gasoline vehicle
LDGT = light-duty gasoline truck
HDGT = heavy-duty gasoline truck
LDDV = light-duty diesel vehicle
LDDT = light-duty diesel truck
HDDT = heavy-duty diesel truck
VMT = vehicle miles traveled
V = all motor vehicles
U = upstream emission sources associated with motor vehicles
RD = paved road dust
RE = unpaved road dust

Pollutants

CO = carbon monoxide
NO₂ = nitrogen dioxide
NO_x = nitrogen oxides (including but not limited to NO₂)
NH₃ = ammonia
O₃ = ozone
PM = particulate matter
PM₁₀ = particulate matter of 10 microns or less aerodynamic diameter
PM_{2.5} = particulate matter of 2.5 microns or less aerodynamic diameter
Coarse PM₁₀ = particulate matter between 2.5 and 10 microns aerodynamic diameter
SO₂ = sulfur dioxide
SO_x = sulfur oxides
SOA = secondary organic aerosols
TSP = total suspended particulates
VOCs = volatile organic compounds

Health effects

AOD = airway obstructive disease (chronic illness)
ARD2 = presence of any symptom or condition
RRAD = respiratory restricted activity day

Geography

L. A. = Los Angeles
SCAQMD = South Coast Air Quality Management District (Orange County, and portions of Los Angeles, Riverside, and San Bernadino counties).
U.S. = United States

Note: all dollars are 1991 \$.

TABLE 11.7-1. POPULATION, VMT, AND POPULATION-WEIGHTED AVERAGE AIR POLLUTION FOR THE NATION, URBAN AND RURAL AREAS, AND ELEVEN METROPOLITAN STATISTICAL AREAS (1990)

Area	Population (10 ⁶)	VMT (10 ⁹)	CO (ppm)	NO ₂ (ppm)	O ₃ (ppm)	PM ₁₀ ^(a) (µg/m ³)
Nation	248.7	2,148.0	n.e.	n.e.	n.e.	n.e.
Urban	197.6	1,596.4	n.e.	n.e.	n.e.	n.e.
Rural	51.2	551.6	n.e.	n.e.	n.e.	n.e.
<i>MSAs</i>						
Boston	3.8	28.3	2.5	0.026	0.013	21.0
Denver	1.8	12.8	3.2	0.021	0.024	25.0
Houston	3.7	35.1	2.2	0.032	0.049	32.1
Los Angeles	14.5	118.2	3.5	0.063	0.064	47.1
Minneapolis	2.5	19.5	3.2	0.030	0.013	26.9
New York	17.1	93.0	3.2	0.037	0.017	27.5
Philadelphia	5.9	39.4	2.2	0.033	0.018	28.6
Phoenix	2.1	17.1	3.4	0.042	0.050	40.9
St. Louis	2.4	22.2	2.0	0.030	0.016	28.9
Spokane	0.4	2.8	4.9	0.034	0.014	41.3
Washington D. C.	3.9	29.5	2.7	0.028	0.021	26.8

The values for ozone include readings from the non-ozone season (see Table 11.2-2) Also, in all cases, where there was no monitor reading in a County, we chose the minimum of the readings from the larger EPA region (see Section 11.2). For these reasons, the values in this table might seem relatively low.

n e = not estimated

^aIncludes particulate sulfates, particulates nitrates, and organic particulates

TABLE 11.7-2. DIFFERENCES BETWEEN THE LOWER AND UPPER BOUND SCENARIOS

	Lower Bound	Upper Bound
CO natural (background) level	0.4 ppm	0.1ppm
NO ₂ natural (background) level	0.1 ppb	0.005 ppb
O ₃ natural (background) level	0.015 ppm	0.01 ppm
O ₃ effect on asthma	Whittemore & Korn (1980)	Holguin et al. (1985) (~ 3.7 times higher than in lower bound)
O ₃ effect on symptoms other than eye irritation, asthma, respiratory illness	None	Krupnick et al. (1990) (ARD2)
O ₃ effect on children's morbidity	None	Same as on adults
PM effect on children's RRAD	59% less than adults'	Same as on adults
PM effect on mortality (cross-sectional studies)	Ozkaynak and Thurston (1987)	Pope et al. (1995) (~ 2 times higher than in lower bound)
<i>Value of morbidity (1991 \$)</i>		
headache (CO)	\$3	\$14
sore throat (NO ₂)	\$3	\$14
excess phlegm (NO ₂)	\$3	\$14
eye irritation (NO ₂ , O ₃)	\$3	\$14
resp. illness excl. asthma (O ₃)	\$3	\$14
any other symptom ARD2 (O ₃)	\$3	\$14
asthma attack (O ₃ , PM)	\$10	\$50
RRAD (PM)	\$20	\$70
AOD, or chronic illness (PM)	\$0.5 million	\$2 million
<i>Value of mortality (1991 \$)</i>		
Cancer from toxics	\$0.5 million	\$2 million
Harvest death ^a	\$10,000	\$50,000
Non-harvest death ^a	\$1 million	\$4 million
Value of health: rate of increase/yr.	1%	1.5%
Discount rate	8%	2%
Estimated AOD ^b (PM)	Median of 5 estimates	Max. of 5 estimates

(continued next page)

<i>Emission correction factors^c</i>		
CO from LDGVs	1.5	1.8
NO _x from LDGVs	1.2	1.4
VOCs from LDGVs	1.1	1.3
PM ₁₀ from all vehicles	1.5	2.0
PM ₁₀ paved road dust	0.3	0.8
PM _{2.5} paved road dust	0.07	0.57
PM ₁₀ unpaved road dust	1.0	1.0
PM _{2.5} unpaved road dust	0.3	0.95
PM ₁₀ construction (except road)	0.5	0.1
PM _{2.5} construction (except road)	5.0	1.0
PM ₁₀ road construction	0.1	0.5
PM _{2.5} road construction	1.0	5.0
PM ₁₀ wind erosion	1.2	1.1
<i>The effect of PM size and composition^d</i>		
Composition effect: potency of dust from paved roads and construction relative to potency of other PM.	0.1	0.5
Composition effect: potency of dust from unpaved roads, agricultural tillage, and livestock relative to potency of other PM.	0.1	0.5
Size effect: potency of PM _{2.5} relative to potency of coarse PM ₁₀	2.0	10.0

^aWe assume that the time-series studies measure two groups of deaths – people that probably would have died anyway very shortly after they actually did die (harvest deaths) and people that would have lived much longer but for particulates (non-harvest deaths) In the lower bound we assume that 50% of the estimated time-series deaths are harvest deaths, and in the upper bound we assume 25%.

^bAbbey et al. (1995) estimated five different models of AOD, corresponding to five different exposure thresholds We estimate all five models in each county, and use the highest of the resulting estimates as our upper bound, and the median as our lower bound.

^cThese factors adjust the EPA's official county-level emission estimates to what we believe are better estimates of actual emissions See Report #16 of the social-cost series listed at the beginning of this report for details

^dPotency refers to change in health effects per change in unit concentration of the PM. As shown, we assume that fugitive-dust PM is less potent than is other PM, in any size class, and that the coarse fraction of PM₁₀ (PM_{2.5} to PM₁₀) is less potent than is PM_{2.5}, for any given particle composition.

Keep in mind that the "Low" and "High" refer to low total motor-vehicle-related damages and high total motor-vehicle-related damages Our assumptions about the potency of PM_{2.5} relative to the potency of the coarse fraction of PM₁₀ affect both the magnitude of the total damages due to motor vehicles, and the distribution of that damage between road dust PM, which is mainly but not exclusively larger than PM_{2.5}, and tailpipe PM, which is less than PM_{2.5}

Notes to Tables 11.7-3 and 11.7-4.

Shown here are the number of cases and the monetary value of the health effects of all anthropogenic CO, NO_x, O₃, and PM₁₀ air pollution. "All anthropogenic" pollution is all pollution down to natural background levels, not just pollution from motor vehicles

These tables do *not* include the health effects of so-called "toxic" air pollutants acetaldehyde, formaldehyde, benzene, 1,3-butadiene, gasoline particulates as a cause of lung cancer, and diesel particulates as a cause of lung cancer. We call these "toxic" air pollutants, and distinguish them from the other pollutants, because we estimate the health effects on the basis of unit-risk numbers (which measure cancer risk, or toxicity) and exposure to pollution in micro-environments (Chapter 11.6). By contrast, we estimate the health effects of all of the other air pollutants on the basis of human epidemiological studies and exposure to ambient air pollution (Chapters 11.2 and 11.3).

Note that in the case of particulate matter only, we estimate both toxic (lung cancer) and "non-toxic" (other mortality and morbidity) effects. We did not include in these tables the "toxic" lung-cancer effects of gasoline and diesel particulates because we did not analyze *all* sources and ambient levels of toxics. However, all of the non-lung-cancer effects of particulate matter, including other mortality effects, are included under ambient PM₁₀ in these tables. (All of the toxic air pollutants are included in Tables 11.7-6 and 11.7-9, and in the tables in the Appendix.)

TABLE 11.7-3A. SUMMARY OF THE NUMBER OF CASES OF ADVERSE HEALTH EFFECTS CAUSED BY ALL ANTHROPOGENIC POLLUTION, 1988 AND 1989 (THOUSANDS OF CASES)

Emission	Ambient pollutant	Health effect	1988		1989	
			<i>low</i>	<i>high</i>	<i>low</i>	<i>high</i>
CO	CO	headache	193,547	225,508	181,241	213,709
		hospitalization	9	25	8	24
		mortality	0.6	1.6	0.5	1.5
NO _x	NO _x	sore throat	306,511	311,479	287,253	291,937
		excess phlegm	140,000	142,350	131,418	133,636
		eye irritation	126,215	128,303	118,396	120,365
VOC+NO _x	O ₃	asthma attacks	3,877	11,824	3,655	11,334
		eye irritation	41,550	44,971	35,416	38,749
		lower respiratory illness	51,999	84,480	48,777	80,376
		upper respiratory illness	15,821	25,704	14,841	24,455
		ARD2	0	251,762	0	257,523
PM ₁₀ , SO ₂ , NO _x , VOC	PM ₁₀ ^(a)	asthma attacks	3,276	3,458	3,211	3,388
		RRAD	105,809	123,180	103,373	120,529
		chronic illness	52	111	50	112
		mortality	85	150	83	140

^aIncludes particulate sulfates, particulates nitrates, and organic particulates, as well as direct or primary particulate emissions

TABLE 11.7-3B. SUMMARY OF THE NUMBER OF CASES OF ADVERSE HEALTH EFFECTS CAUSED BY ALL ANTHROPOGENIC POLLUTION, 1990 AND 1991 (THOUSANDS OF CASES)

Emission	Ambient pollutant	Health effect	1990 ^b		1991	
			<i>low</i>	<i>high</i>	<i>low</i>	<i>high</i>
CO	CO	headache	170,385	202,416	160,020	193,213
		hospitalization	8	23	7	22
		mortality	0.5	1.5	0.5	1.4
NO _x	NO _x	sore throat	265,577	269,583	269,741	273,510
		excess phlegm	121,800	123,700	132,156	133,929
		eye irritation	109,618	111,303	112,793	114,370
VOC+NO _x	O ₃	asthma attacks	3,652	11,482	3,800	11,897
		eye irritation	33,852	37,383	36,219	39,818
		lower respiratory illness	48,584	81,089	50,680	84,220
		upper respiratory illness	14,782	24,672	15,420	25,624
		ARD2	0	276,144	0	268,403
PM ₁₀ , SO ₂ , NO _x , VOC	PM ₁₀ ^(a)	asthma attacks	3,003	3,172	2,848	3,008
		RRAD	88,673	120,133	100,636	117,459
		chronic illness	39	93	39	91
		mortality	80	137	79	132

^aIncludes particulate sulfates, particulates nitrates, and organic particulates, as well as direct or primary particulate emissions

^bIn the text we calculate an alternative upper bound for 1990, using our lower-bound estimate of long-term deaths from PM with all of the other original upper-bound assumptions. The alternative upper-bound estimate is quite a bit lower than the upper bound shown here.

TABLE 11.7-4A. SUMMARY OF HEALTH COSTS OF ALL ANTHROPOGENIC POLLUTION, 1988-1989 (1991\$ BILLION)

Emission	Ambient pollutant	Health effect	1988		1989	
			<i>low</i>	<i>high</i>	<i>low</i>	<i>high</i>
CO	CO	headache	0.5	2.8	0.5	2.7
		hospitalization	0.1	0.3	0.1	0.2
		mortality	0.6	6.5	0.5	6.1
		<i>All</i>	1.2	9.5	1.1	9.0
NO _x	NO _x	sore throat	0.7	3.4	0.7	3.2
		excess phlegm	0.4	1.9	0.4	1.8
		eye irritation	0.4	1.8	0.3	1.7
		<i>All</i>	1.5	7.1	1.4	6.7
VOC+NO _x	O ₃	asthma attacks	0.04	0.5	0.04	0.5
		eye irritation	0.1	0.6	0.1	0.5
		lower respiratory illness	0.2	1.2	0.1	1.1
		upper respiratory illness	0.05	0.4	0.04	0.3
		ARD2	0.0	2.8	0.0	3.1
<i>All</i>	0.4	5.5	0.3	5.6		
PM ₁₀ , SO ₂ , NO _x , VOC	PM ₁₀ ^(a)	asthma attacks	0.03	0.2	0.03	0.2
		RRAD	2.1	8.6	2.1	8.4
		chronic illness	13.3	210.3	12.8	213.3
		mortality	43.1	536.4	42.1	499.5
		<i>All</i>	58.6	755.5	57.0	721.4
<i>All</i>	<i>All</i>	<i>All</i>	61.7	777.7	59.8	742.7

^aIncludes particulate sulfates, particulates nitrates, and organic particulates, as well as direct or primary particulate emissions

TABLE 11.7-4B. SUMMARY OF HEALTH COSTS OF ALL ANTHROPOGENIC POLLUTION, 1990-1991 (1991\$ BILLION)

Emission	Ambient pollutant	Health effect	1990		1991	
			<i>low</i>	<i>high</i>	<i>low</i>	<i>high</i>
CO	CO	headache	0.5	2.6	0.4	2.5
		hospitalization	0.1	0.2	0.1	0.2
		mortality	0.5	5.8	0.5	5.5
		<i>All</i>	1.1	8.6	1.0	8.2
NO _x	NO _x	sore throat	0.6	3.0	0.6	3.0
		excess phlegm	0.4	1.7	0.4	1.7
		eye irritation	0.3	1.5	0.3	1.5
		<i>All</i>	1.3	6.3	1.3	6.1
VOC+NO _x	O ₃	asthma attacks	0.04	0.5	0.04	0.5
		eye irritation	0.1	0.5	0.1	0.6
		lower respiratory illness	0.1	1.1	0.2	1.2
		upper respiratory illness	0.04	0.3	0.05	0.4
		ARD2	0.0	3.3	0.0	3.2
		<i>All</i>	0.3	5.8	0.3	5.8
PM ₁₀ , SO ₂ , NO _x , VOC	PM ₁₀ ^(a)	asthma attacks	0.03	0.2	0.03	0.2
		RRAD	1.8	8.4	2.0	8.2
		chronic illness	10.1	177.1	10.0	173.8
		mortality	40.9	489.0	40.1	470.0
		<i>All</i>	52.8	674.7	52.1	652.1
<i>All</i>	<i>All</i>	<i>All</i>	55.5	695.4	54.7	672.3

^aIncludes particulate sulfates, particulates nitrates, and organic particulates, as well as direct or primary particulate emissions

^bIn the text we calculate an alternative upper bound for 1990, using our lower-bound estimate of long-term deaths from PM with all of the other original upper-bound assumptions. The alternative upper-bound estimate is quite a bit lower than the upper bound shown here.

TABLE 11.7-5. MOTOR VEHICLE POLLUTION: LOWER AND UPPER BOUND EMISSION ESTIMATES, BY MOTOR VEHICLE CLASS AND POLLUTANT (GRAM/MILE) (U.S. TOTAL, 1990)

Vehicle	Source	PM		VOC		CO		NO _x		SO ₂	SOA
		low	high	low	high	low	high	low	high		
LDGV	V	0.06	0.07	2.7	3.2	35.3	42.3	2.4	2.8	0.1	0.02
	V+U	0.1	0.1	3.9	4.4	35.5	42.5	2.6	3.0	0.5	0.03
	V+U+RD	0.6	1.4	3.9	4.4	35.5	42.5	2.6	3.0	0.5	0.03
	V+U+RD+RE	3.9	4.7	3.9	4.4	35.5	42.5	2.6	3.0	0.5	0.03
LDGT	V	0.1	0.1	4.5	5.3	52.4	62.9	3.7	4.3	0.1	0.02
	V+U	0.2	0.2	6.5	7.3	52.9	63.4	4.1	4.8	1.1	0.05
	V+U+RD	0.8	2.0	6.5	7.3	52.9	63.4	4.1	4.8	1.1	0.05
	V+U+RD+RE	5.3	6.4	6.5	7.3	52.9	63.4	4.1	4.8	1.1	0.05
HDGT	V	0.6	0.8	16.7	16.7	211.3	211.3	11.9	11.9	0.4	0.12
	V+U	0.6	0.9	22.1	22.1	211.9	211.9	12.5	12.5	1.6	0.16
	V+U+RD	2.2	5.0	22.1	22.1	211.9	211.9	12.5	12.5	1.6	0.16
	V+U+RD+RE	13.0	15.8	22.1	22.1	211.9	211.9	12.5	12.5	1.6	0.16
<i>Gasoline</i>	V	0.1	0.1	3.3	3.8	40.9	48.6	2.8	3.2	0.1	0.02
<i>Subtotal</i>	V+U	0.1	0.2	4.7	5.2	41.2	48.9	3.0	3.5	0.7	0.03
	V+U+RD	0.6	1.6	4.7	5.2	41.2	48.9	3.0	3.5	0.7	0.03
	V+U+RD+RE	4.3	5.2	4.7	5.2	41.2	48.9	3.0	3.5	0.7	0.03

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Vehicle	Source	PM		VOC		CO		NO _x		SO ₂	SOA
		low	high	low	high	low	high	low	high		
LDDV	V	0.5	0.7	0.4	0.4	1.1	1.1	1.3	1.3	0.5	0.003
	V+U	0.5	0.7	0.8	0.8	1.1	1.1	1.4	1.4	0.6	0.007
	V+U+RD	0.9	1.7	0.8	0.8	1.1	1.1	1.4	1.4	0.6	0.007
	V+U+RD+RE	3.5	4.3	0.8	0.8	1.1	1.1	1.4	1.4	0.6	0.007
LDDT	V	0.2	0.2	0.2	0.2	0.4	0.4	0.5	0.5	0.2	0.001
	V+U	0.2	0.2	0.8	0.8	0.5	0.5	0.6	0.6	0.5	0.008
	V+U+RD	0.7	1.7	0.8	0.8	0.5	0.5	0.6	0.6	0.5	0.008
	V+U+RD+RE	4.6	5.6	0.8	0.8	0.5	0.5	0.6	0.6	0.5	0.008
HDDT	V	2.5	3.3	2.2	2.2	9.0	9.0	16.0	16.0	2.3	0.05
	V+U	2.6	3.4	4.6	4.6	9.7	9.7	16.7	16.7	3.9	0.09
	V+U+RD	6.8	14.6	4.6	4.6	9.7	9.7	16.7	16.7	3.9	0.09
	V+U+RD+RE	35.7	43.6	4.6	4.6	9.7	9.7	16.7	16.7	3.9	0.09
<i>Diesel</i> <i>Subtotal</i>	V	2.0	2.7	1.8	1.8	7.1	7.1	12.6	12.6	1.9	0.04
	V+U	2.1	2.7	3.7	3.7	7.7	7.7	13.1	13.1	3.2	0.07
	V+U+RD	5.4	11.7	3.7	3.7	7.7	7.7	13.1	13.1	3.2	0.07
	V+U+RD+RE	28.5	34.7	3.7	3.7	7.7	7.7	13.1	13.1	3.2	0.07
<i>ALL</i> <i>M.V.s</i>	V	0.2	0.3	3.1	3.7	38.2	45.3	3.6	4.0	0.2	0.02
	V+U	0.3	0.4	4.6	5.1	38.6	45.6	3.8	4.3	0.9	0.04
	V+U+RD	1.0	2.4	4.6	5.1	38.6	45.6	3.8	4.3	0.9	0.04
	V+U+RD+RE	6.2	7.6	4.6	5.1	38.6	45.6	3.8	4.3	0.9	0.04

Note that the emission source category is cumulative: V includes just motor vehicle emissions, V+U includes V plus upstream emissions, V+U+RD includes V+U plus paved road dust emissions, and V+U+RD+RE includes V+U+RD plus unpaved road dust emissions. M.V. = motor vehicle

TABLE 11.7-6. COST PER MILE OF MOTOR VEHICLE TRAVEL, BASED ON A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS : LOWER AND UPPER BOUND EMISSION ESTIMATES, BY MOTOR VEHICLE CLASS AND AMBIENT POLLUTANT (CENTS/VMT) (NATION, 1990)

Vehicle	Source	PM		O ₃		CO		NO ₂		Toxics ^a		Total	
		low	high	low	high	low	high	low	high	low	high	low	high
LDGV	V	0.48	7.02	0.01	0.07	0.04	0.37	0.04	0.22	0.00	0.05	0.58	7.71
	V+U	0.56	7.50	0.01	0.07	0.04	0.37	0.04	0.22	n.e.	n.e.	0.66	8.20
	V+U+RD	0.60	10.90	0.01	0.07	0.04	0.37	0.04	0.22	n.e.	n.e.	0.69	11.60
	V+U+RD+RE	0.65	12.18	0.01	0.07	0.04	0.37	0.04	0.22	n.e.	n.e.	0.75	12.88
LDGT	V	0.74	10.70	0.01	0.11	0.06	0.53	0.06	0.32	0.00	0.09	0.88	11.72
	V+U	0.90	11.54	0.01	0.11	0.06	0.53	0.06	0.32	n.e.	n.e.	1.04	12.56
	V+U+RD	0.94	16.09	0.01	0.11	0.06	0.53	0.06	0.32	n.e.	n.e.	1.09	17.11
	V+U+RD+RE	1.02	17.80	0.01	0.11	0.06	0.53	0.06	0.32	n.e.	n.e.	1.16	18.82
HDGT	V	1.56	30.28	0.03	0.28	0.15	1.63	0.12	0.76	0.01	0.29	1.86	33.12
	V+U	1.78	31.53	0.03	0.29	0.15	1.63	0.12	0.77	n.e.	n.e.	2.09	34.38
	V+U+RD	1.90	42.55	0.03	0.29	0.15	1.63	0.12	0.77	n.e.	n.e.	2.20	45.40
	V+U+RD+RE	2.07	46.69	0.03	0.29	0.15	1.63	0.12	0.77	n.e.	n.e.	2.37	49.54
<i>Gasoline Subtotal</i>	V	0.55	8.04	0.01	0.08	0.05	0.42	0.05	0.25	0.00	0.06	0.65	8.83
	V+U	0.64	8.61	0.01	0.08	0.05	0.42	0.05	0.25	n.e.	n.e.	0.75	9.40
	V+U+RD	0.68	12.33	0.01	0.08	0.05	0.42	0.05	0.25	n.e.	n.e.	0.79	13.12
	V+U+RD+RE	0.74	13.74	0.01	0.08	0.05	0.42	0.05	0.25	n.e.	n.e.	0.85	14.53

(continued next page)

Vehicle	Source	PM		O3		CO		NO2		Toxics		Total	
		low	high	low	high	low	high	low	high	low	high	low	high
LDDV	V	1.47	18.49	0.00	0.02	0.00	0.01	0.02	0.11	0.01	0.08	1.50	18.64
	V+U	1.50	18.70	0.00	0.02	0.00	0.01	0.02	0.11	n.e.	n.e.	1.53	18.84
	V+U+RD	1.53	21.34	0.00	0.02	0.00	0.01	0.02	0.11	n.e.	n.e.	1.55	21.49
	V+U+RD+RE	1.57	22.34	0.00	0.02	0.00	0.01	0.02	0.11	n.e.	n.e.	1.60	22.48
LDDT	V	0.47	5.77	0.00	0.01	0.00	0.00	0.01	0.04	0.00	0.03	0.48	5.82
	V+U	0.52	6.14	0.00	0.01	0.00	0.00	0.01	0.04	n.e.	n.e.	0.53	6.19
	V+U+RD	0.57	10.09	0.00	0.01	0.00	0.00	0.01	0.04	n.e.	n.e.	0.58	10.14
	V+U+RD+RE	0.63	11.58	0.00	0.01	0.00	0.00	0.01	0.04	n.e.	n.e.	0.64	11.63
HDDT	V	4.18	79.93	0.02	0.19	0.01	0.07	0.15	0.98	0.02	0.33	4.35	81.19
	V+U	4.43	81.37	0.02	0.20	0.01	0.07	0.15	0.99	n.e.	n.e.	4.61	82.63
	V+U+RD	4.75	110.91	0.02	0.20	0.01	0.07	0.15	0.99	n.e.	n.e.	4.92	112.17
	V+U+RD+RE	5.21	122.01	0.02	0.20	0.01	0.07	0.15	0.99	n.e.	n.e.	5.38	123.27
<i>Diesel Subtotal</i>	V	3.48	64.86	0.01	0.15	0.00	0.05	0.12	0.78	0.01	0.27	3.62	65.85
	V+U	3.68	66.03	0.02	0.16	0.01	0.05	0.12	0.78	n.e.	n.e.	3.83	67.03
	V+U+RD	3.93	89.55	0.02	0.16	0.01	0.05	0.12	0.78	n.e.	n.e.	4.07	90.55
	V+U+RD+RE	4.30	98.39	0.02	0.16	0.01	0.05	0.12	0.78	n.e.	n.e.	4.44	99.39
ALL VEHICLES	V	0.78	12.57	0.01	0.09	0.04	0.39	0.05	0.29	0.00	0.08	0.89	13.37
	V+U	0.89	13.17	0.01	0.09	0.04	0.39	0.05	0.29	n.e.	n.e.	1.00	13.98
	V+U+RD	0.94	18.47	0.01	0.09	0.04	0.39	0.05	0.29	n.e.	n.e.	1.05	19.28
	V+U+RD+RE	1.02	20.46	0.01	0.09	0.04	0.39	0.05	0.29	n.e.	n.e.	1.14	21.27

TABLE 11.7-7A. COST PER KG OF MOTOR VEHICLE EMISSIONS, BASED ON A 10% REDUCTION IN DIRECT MOTOR-VEHICLE EMISSIONS (1991 \$, 1990 EMISSIONS)

Emission	Ambient pollutant	United States		All urban areas		Los Angeles	
		<i>low</i>	<i>high</i>	<i>low</i>	<i>high</i>	<i>low</i>	<i>high</i>
CO	CO	0.01	0.09	0.01	0.10	0.03	0.18
NO _x	nitrate PM ₁₀	1.02	16.56	1.39	22.38	6.05	75.83
	NO ₂	0.15	0.73	0.19	0.96	0.52	2.64
<i>Total for NO_x</i>		1.17	17.29	1.59	23.34	6.58	78.47
PM _{2.5}	PM _{2.5}	10.42	159.19	14.81	225.36	63.98	779.13
PM ₁₀	PM ₁₀	6.70	17.68	9.09	23.89	38.12	78.34
<i>Total for PM₁₀</i>		9.75	133.78	13.74	187.48	58.79	638.33
SO _x	sulfate PM ₁₀	6.90	65.52	9.62	90.94	34.98	226.89
VOC	organic PM ₁₀	0.10	1.15	0.13	1.45	0.51	4.34
VOC+NO _x	ozone	0.01	0.11	0.02	0.14	0.05	0.40

Each \$/kg value is equal to the total calculated health damages attributable to the pollutant and source, divided by emissions of the pollutant from the source.

TABLE 11.7-7B. COST PER KG OF MOTOR VEHICLE EMISSIONS, BASED ON A 10% REDUCTION IN DIRECT MOTOR-VEHICLE EMISSIONS AND RELATED UPSTREAM EMISSIONS (1991 \$, 1990 EMISSIONS)

Emission	Ambient pollutant	United States		All urban areas		Los Angeles	
		<i>low</i>	<i>high</i>	<i>low</i>	<i>high</i>	<i>low</i>	<i>high</i>
CO	CO	0.01	0.09	0.01	0.10	0.03	0.18
NO _x	nitrate PM ₁₀	0.96	15.53	1.31	21.17	6.02	75.11
	NO ₂	0.14	0.68	0.18	0.91	0.52	2.62
<i>Total for NO_x</i>		1.10	16.21	1.50	22.08	6.54	77.73
PM _{2.5}	PM _{2.5}	9.71	147.24	13.63	205.44	62.57	737.36
PM ₁₀	PM ₁₀	5.30	14.25	7.20	18.34	30.04	47.87
<i>Total for PM₁₀</i>		8.78	116.01	12.23	158.23	54.68	509.18
SO _x	sulfate PM ₁₀	2.80	22.60	4.40	35.28	33.53	209.88
VOC	organic PM ₁₀	0.10	0.99	0.13	1.25	0.52	4.29
VOC+NO _x	ozone	0.01	0.10	0.02	0.12	0.05	0.39

Each \$/kg value is equal to the total calculated health damages attributable to the pollutant and source, divided by emissions of the pollutant from the source.

TABLE 11.7-7C. COST PER KG OF MOTOR VEHICLE EMISSIONS, BASED ON A 10% REDUCTION IN DIRECT MOTOR-VEHICLE EMISSIONS, RELATED UPSTREAM EMISSIONS, AND PAVED-ROAD-DUST EMISSIONS (1991 \$, 1990 EMISSIONS)

Emission	Ambient pollutant	United States		All urban areas		Los Angeles	
		<i>low</i>	<i>high</i>	<i>low</i>	<i>high</i>	<i>low</i>	<i>high</i>
CO	CO	0.01	0.09	0.01	0.10	0.03	0.18
NO _x	nitrate PM ₁₀	0.96	15.53	1.31	21.17	6.02	75.11
	NO ₂	0.14	0.68	0.18	0.91	0.52	2.62
<i>Total for NO_x</i>		1.10	16.21	1.50	22.08	6.54	77.73
PM _{2.5}	PM _{2.5}	7.48	94.45	10.47	130.89	45.44	430.10
PM ₁₀	PM ₁₀	1.03	7.58	1.42	10.34	5.62	32.26
<i>Total for PM₁₀</i>		2.84	39.87	3.92	54.64	15.20	169.45
SO _x	sulfate PM ₁₀	2.80	22.60	4.40	35.28	33.53	209.88
VOC	organic PM ₁₀	0.10	0.99	0.13	1.25	0.52	4.29
VOC+NO _x	ozone	0.01	0.10	0.02	0.12	0.05	0.39

Each \$/kg value is equal to the total calculated health damages attributable to the pollutant and source, divided by emissions of the pollutant from the source

TABLE 11.7-7D. COST PER KG OF MOTOR VEHICLE EMISSIONS, BASED ON A 10% REDUCTION IN DIRECT MOTOR-VEHICLE EMISSIONS, RELATED UPSTREAM EMISSIONS, PAVED-ROAD-DUST EMISSIONS, AND UNPAVED-ROAD-DUST EMISSIONS (1991 \$, 1990 EMISSIONS)

Emission	Ambient pollutant	United States		All urban areas		Los Angeles	
		<i>low</i>	<i>high</i>	<i>low</i>	<i>high</i>	<i>low</i>	<i>high</i>
CO	CO	0.01	0.09	0.01	0.10	0.03	0.18
NO _x	nitrate	0.96	15.53	1.31	21.17	6.02	75.11
	PM ₁₀						
	NO ₂	0.14	0.68	0.18	0.91	0.52	2.62
<i>Total for NO_x</i>		1.10	16.21	1.50	22.08	6.54	77.73
PM _{2.5}	PM _{2.5}	3.22	45.22	6.53	88.79	41.93	405.29
PM ₁₀	PM ₁₀	0.27	2.95	0.63	6.20	4.69	29.77
<i>Total for PM₁₀</i>		0.60	15.13	1.45	31.69	12.35	155.58
SO _x	sulfate	2.80	22.60	4.40	35.28	33.53	209.88
	PM ₁₀						
VOC	organic	0.10	0.99	0.13	1.25	0.52	4.29
	PM ₁₀						
VOC+NO _x	ozone	0.01	0.10	0.02	0.12	0.05	0.39

Each \$/kg value is equal to the total calculated health damages attributable to the pollutant and source, divided by emissions of the pollutant from the source.

TABLE 11.7-8. ESTIMATED MORTALITY CAUSED BY PARTICULATES: COMPARISON OF UPPER AND LOWER BOUND ESTIMATES IN 1990

<i>Estimated number of deaths due to 1990 anthropogenic particulate air pollution</i>		Lower bound	Upper bound
2	Acute respiratory and cardiovascular deaths (Pope et al., 1992)	37,250	39,503
3	Fraction of acute deaths that are "harvest" deaths	0.5	0.25
4	Acute harvest deaths (3 * 2)	18,625	9,876
5	Acute non-harvest deaths (2 -4)	18,625	29,627
6A	Total cross-sectional deaths, by Ozkaynak and Thurston (O & T) (1987)	61,775	65,463
6B	Total cross-sectional deaths, by Pope et al. (1995)	119,660	126,995
7A	Chronic deaths: cross-sectional by O & T (1987) less acute non-harvest deaths (Chapter 1) (7A - 5)	43,150	35,836
7B	Chronic deaths: cross-sectional by Pope et al. (1995) less acute non-harvest deaths (Chapter 1) (7B - 5)	101,035	97,368
8A	All particulate deaths (acute plus chronic), using O & T (1987) (4+ 5 + 7A)	80,400	75,339
8B	All particulate deaths (acute plus chronic), using Pope et al. (1995) (4 + 5 + 7B)	138,285	136,871
<i>Value per estimated death (1991\$)</i>			
9	Acute harvest deaths	10,000	50,000
10	Acute death non-harvest deaths	1,000,000	4,000,000
11	Chronic death (present value)	513,000	3,800,000
<i>Total present value in 1990 of deaths due to all anthropogenic particulate air pollution (billion 1991\$)</i>			
12	Acute harvest deaths (4 * 9/1000000000)	0.19	0.49
13	Acute non-harvest deaths (5 * 10/1000000000)	18.63	118.51
14A	Chronic deaths, using O&T (1987) (7A * 11/1000000000)	22.14	136.18
14B	Chronic deaths, using Pope et al. (1995) (7B*11/1000000000)	161.47	370.00
15A	All particulate deaths, using O & T (1987) (12 + 13 + 14A) (lower bound here same as in Table 11.7-4)	40.96	255.18
15B	All particulate deaths, using Pope et al. (1995) (12 + 13 + 14B) (upper bound here same as in Table 11.7-4)	180.29	489.00

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<i>Total present value in 1990 of deaths due to motor-vehicle particulate air pollution (billion 1991\$)</i>	Lower bound	Upper bound
16A All deaths due to motor-vehicle particulate pollution (including road dust and upstream sources), using O & T (1987) (calculated by our model) (lower bound here same as in Table 11.A-1)	16.7	n.e.
16B All deaths due to motor-vehicle particulate air pollution (including road dust and upstream sources), using Pope et al. (1995) (upper bound here same as in Table 11.A-2)	n.e.	311.6

n c = not estimated.

TABLE 11.7-9. COST OF MOTOR-VEHICLE AIR POLLUTION IN THE NATION, URBAN AREAS AND LOS ANGELES MSA, BASED ON A 100% REDUCTION IN MOTOR VEHICLE EMISSIONS IN 1990 (MILLIONS OF 1991\$)

	Motor vehicles		Motor Vehicles + Upstream		Motor Vehicles + Upstream + Road Dust ^a	
	<i>lower</i>	<i>upper</i>	<i>lower</i>	<i>upper</i>	<i>lower</i>	<i>upper</i>
<i>Nation</i>						
PM ₁₀ ^(b)	16,727	266,391	18,961	279,354	21,943	432,829
O ₃	214	1,899	228	1,945	228	1,945
NO _x	1,038	5,483	1,048	5,509	1,048	5,509
CO	919	8,085	921	8,092	921	8,092
toxics	87	1,622	n.e.	n.e.	n.e.	n.e.
<i>Total</i>	18,985	283,481	21,246	296,522	24,227	449,997
<i>Urban</i>						
PM ₁₀ ^(b)	15,954	253,126	18,059	265,228	20,599	401,037
O ₃	196	1,730	209	1,771	209	1,771
NO _x	955	5,072	964	5,093	964	5,093
CO	829	7,089	831	7,094	831	7,094
toxics	76	1,411	n.e.	n.e.	n.e.	n.e.
<i>Total</i>	18,010	268,428	20,139	280,599	22,679	416,408
<i>Los Angeles MSA</i>						
PM ₁₀ ^(b)	4,203	51,869	4,291	52,564	4,633	76,260
O ₃	33	265	34	266	34	266
NO _x	137	713	137	714	137	714
CO	99	746	100	746	100	746
toxics	3	63	n.e.	n.e.	n.e.	n.e.
<i>Total^c</i>	4,476	53,656	4,565	54,353	4,968	79,033

n.e = not estimated.

^aIncludes both paved and unpaved road dust.

^bIncludes particulate sulfates, particulate nitrates, and organic particulates

**TABLE 11.7-10. ESTIMATES OF AIR POLLUTION DAMAGES IN THE LOS ANGELES REGION
(MILLIONS OF 1992 \$)**

Damage	This Study ^a		Hall et al. (1992) ^b		Krupnick and Portney (1991) ^b	Small and Kazimi (1985)
	All anthropogenic emissions in Los Angeles ^c in 1990		Emissions reductions required to achieve air quality standards in SCAQMD 2010			All emissions including biogenic in 1990
	<i>Low</i>	<i>High</i>	<i>Low</i>	<i>High</i>	<i>Best</i>	<i>Baseline</i>
<i>particulate</i>						
mortality	5,093	48,062	3,439	17,668	2,372	30,168(d)
morbidity	2,727	39,924	919	919	830	(e)
<i>ozone</i>						
morbidity	52	655	1,423	6,878	356	(e)
<i>Total</i>	7,872	88,642(f)	5,781	25,465	3,557	(e)

SCAQMD = South Coast Air Quality Management District (Orange County, and portions of Los Angeles, Riverside, and San Bernadino Counties).

^aOur original 1991 \$ estimates scaled by the 1992/1991 CPI (1.03), for comparison with the 1992 \$ estimates of Small and Kazimi (1995)

^bHall et al (1992) and Krupnick and Portney (1991) report their results in 1988 \$ We have scaled their results to 1992 \$, for comparison with the results of Small and Kazimi (1995) In their own comparison, Small and Kazimi (1995) use the 1992/1988 CPI (1.186, in their analysis) to scale the results of Hall et al. (1990) and Krupnick and Portney (1991) We do the same

^cAll of Los Angeles, Orange, San Bernadino, and Riverside Counties.

^dThe geometric mean two mortality estimates based on mortality coefficients from Ozkaynak and Thurston (1987) and Evans et al. (1984), annual average PM₁₀ of 57.8 µg/m³, and a value of life of \$4.87 million (see Small and Kazimi, 1995: footnotes 11 and 12)

Note that Small and Kazimi underestimate the lower bound coefficient (Evans et al) when they note (p. 18) "Based on the consensus that PM₁₀ rather than TSP causes health damages, we assume that among the constituents of TSP, only PM₁₀ causes mortality, then the same coefficient (0.338) applies to changes in PM₁₀ " Actually, they needed to correct for the PM₁₀ fraction of TSP, which we assume is 55%, as noted by Hall et al (1989: 4-20). The coefficient should be 0.615 (=0.338/0.55).

Finally, we estimate that anthropogenic sources contribute, on average, roughly 90% of ambient particulates. To make this estimate more comparable to our own, one should subtract 10% of their estimate to account for natural emissions

^eSmall and Kazimi (1995: 20) used the results of Hall et al. (1992) and Krupnick and Portney (1991) to estimate morbidity costs of ozone and particulates. We did not attempt to use Small and Kazimi's results to estimate the morbidity costs of all anthropogenic ozone and particulates.

TABLE 11.7-11. TWO ESTIMATES OF THE COST OF OZONE AND PARTICULATE AIR POLLUTION FROM MOTOR-VEHICLES IN THE LOS ANGELES REGION (1992 CENTS/VMT)^A

Vehicle	This Study (PM and O ₃ damages only) ^b				Small and Kazimi (1995)
	Direct Emissions		Direct + Upstream + Road Dust		Direct Emissions
	<i>Low</i>	<i>High</i>	<i>Low</i>	<i>High</i>	<i>Baseline</i>
Light-Duty Gasoline	2.1	23.2	2.4	36.5	3.3
Light-Duty Diesel	6.7	64.4	6.9	74.7	7.8
Heavy-Duty Diesel	20.5	324.0	22.9	462.8	52.7

^aSmall and Kazimi's (1995) estimates apply to the SCAQMD. Our estimates apply to the Los Angeles Metropolitan Statistical Area (MSA), which consists of Los Angeles, Riverside, Orange, San Bernadino, and Ventura Counties. Note that because here we compare cents/VMT, and not total dollar damages, the difference between the SCAQMD and the Los Angeles MSA is relatively unimportant.

^bWe have converted our estimates from 1991 \$ to 1992 \$ by multiplying by the 1992/1991 CPI of 1.03. The estimates of Small and Kazimi (1995) are in 1992 dollars.

TABLE 11.7-12. THE CONTRIBUTION OF MOTOR VEHICLE EMISSIONS TO TOTAL DAMAGES FROM PARTICULATE AIR POLLUTION IN THE COUNTIES OF THE SOUTH COAST AIR BASIN

County	The fraction of ambient PM that is from motor-vehicle tailpipe emissions of: (tailpipe plus road dust and upstream in parentheses)					
	<i>direct PM</i>		<i>Precursors of secondary ambient PM</i>			<i>all sources</i>
	Coarse ^a PM ₁₀	PM _{2.5}	NO _x	SO _x	Organic	
<i>Our upper bound</i>						
Los Angeles	0.5% (5.7%)	19.2% (40.8%)	30.5% (30.6%)	5.8% (5.9%)	1.9% (1.9%)	57.8% (84.9%)
Orange	0.4% (5.5%)	16.4% (37.1%)	27.3% (27.4%)	5.0% (5.1%)	1.7% (1.8%)	51.0% (76.9%)
Riverside	0.3% (5.1%)	14.5% (34.1%)	21.9% (21.9%)	4.3% (4.3%)	1.3% (1.3%)	42.2% (66.8%)
San Bernadino	0.3% (5.6%)	13.5% (33.8%)	21.1% (21.2%)	4.0% (4.1%)	1.2% (1.3%)	40.3% (65.9%)
<i>Our lower bound</i>						
Los Angeles	2.1% (6.0%)	13.7% (14.6%)	25.5% (25.7%)	10.4% (10.9%)	2.2% (2.4%)	53.8% (59.6%)
Orange	1.9% (6.1%)	12.7% (13.7%)	23.6% (23.8%)	9.6% (10.0%)	2.1% (2.3%)	50.0% (55.8%)
Riverside	1.6% (5.8%)	11.5% (12.4%)	19.9% (20.0%)	8.4% (8.6%)	1.7% (1.8%)	43.1% (48.6%)
San Bernadino	1.5% (5.7%)	10.5% (11.4%)	18.6% (18.7%)	7.7% (7.9%)	1.6% (1.7%)	39.9% (45.5%)
Small & Kazimi (1995)	10.6%(b)		10.5%	5.3%	4.4%	30.8%

The percentages shown are the dispersion- and potency-weighted shares of particulate pollution, the calculation of the shares is discussed in Section 11.5. In the case of Small and Kazımı (1995), this ratio also is the same as the ratio of the concentration of the particular motor-vehicle pollutant to the concentration of all particulate matter, because they weight all PM10 equally.

^aSmall and Kazımı report the contribution of PM₁₀, combining coarse PM₁₀ and PM_{2.5}.

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APPENDIX TABLES

In this Appendix, Tables 11.A-1 through 11.A-4 give the lower and upper-bound benefit estimates of a 100% reduction of motor vehicle emissions, in dollars per one thousand VMT, for urban areas, and the nation as a whole. Tables 11.A-5 through 11.A-60 give the lower and upper-bound benefit estimates of a 10% reduction of motor vehicle emissions, in millions of dollars and in dollars per one thousand VMT, for eleven metropolitan areas, urban areas, rural areas, and the nation as a whole. In each of these tables, we show costs by pollutant, by vehicle class, and by emissions source. The 60 tables are arranged as follows:

Region	Millions of dollars		Dollars/1000 VMT	
	Lower bound	Upper bound	Lower bound	Upper bound
<i>100% emission reduction</i>				
All United States	Table 11.A-1	Table 11.A-2		
All urban areas	Table 11.A-3	Table 11.A-4		
<i>10% emission reduction</i>				
All United States	Table 11.A-5	Table 11.A-6	Table 11.A-33	Table 11.A-34
All urban areas	Table 11.A-7	Table 11.A-8	Table 11.A-35	Table 11.A-36
All rural areas	Table 11.A-9	Table 11.A-10	Table 11.A-37	Table 11.A-38
Boston	Table 11.A-11	Table 11.A-12	Table 11.A-39	Table 11.A-40
Denver	Table 11.A-13	Table 11.A-14	Table 11.A-41	Table 11.A-42
Houston	Table 11.A-15	Table 11.A-16	Table 11.A-43	Table 11.A-44
Los Angeles	Table 11.A-17	Table 11.A-18	Table 11.A-45	Table 11.A-46
Minneapolis	Table 11.A-19	Table 11.A-20	Table 11.A-47	Table 11.A-48
New York	Table 11.A-21	Table 11.A-22	Table 11.A-49	Table 11.A-50
Philadelphia	Table 11.A-23	Table 11.A-24	Table 11.A-51	Table 11.A-52
Phoenix	Table 11.A-25	Table 11.A-26	Table 11.A-53	Table 11.A-54
St. Louis	Table 11.A-27	Table 11.A-28	Table 11.A-55	Table 11.A-56
Spokane	Table 11.A-29	Table 11.A-30	Table 11.A-57	Table 11.A-58
Washington D. C.	Table 11.A-31	Table 11.A-32	Table 11.A-59	Table 11.A-60

TABLE 11-4. COST OF AIR POLLUTION ATTRIBUTABLE TO A 100% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$ MILLION (U.S. URBAN 1990) (UPPER BOUND)

Emission	Ambient pollutant	Health effect	IDCVs			LDGs			HDCGs			IDDVs			LDDTs			HDDVs			Total diesel vehicles			Total all vehicles					
			V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD			
CO	CO	Health effect mortality	3,278	3,280	3,280	1,170	1,171	1,171	234	234	234	4,682	4,686	4,686	1	2	2	0	0	0	48	49	49	50	51	51	4,733	4,736	4,736
		hospitalization	1,810	1,810	1,810	46	46	46	9	9	9	185	185	185	0	0	0	0	0	0	2	2	2	2	2	187	187	187	
		headaches	1,618	1,619	1,619	594	595	595	120	120	120	2,152	2,154	2,154	1	1	1	0	0	0	25	25	25	26	26	26	2,169	2,170	2,170
NOx	NO2	Health effect sore throat	1,640	1,648	1,648	624	628	628	97	98	98	2,136	2,146	2,146	14	14	14	3	3	3	640	642	642	656	658	658	2,497	2,506	2,506
		excess phlegm	767	771	771	279	279	279	43	43	43	1,081	1,087	1,087	6	6	6	1	1	1	284	285	285	291	292	292	1,349	1,356	1,356
		eye irritation	696	700	700	253	255	255	39	40	40	979	984	984	6	6	6	1	1	1	289	290	290	296	297	297	1,225	1,231	1,231
NOx, NH3	nitrate PM10	Health effect mortality	52,745	52,991	52,991	19,048	19,159	19,159	2,948	2,959	2,959	74,195	74,559	74,559	414	416	416	79	80	80	19,410	19,469	19,469	19,898	19,960	19,960	93,411	93,853	93,853
		chronic illness	20,903	20,997	20,997	7,625	7,667	7,667	1,186	1,186	1,186	29,510	29,650	29,650	164	164	164	30	31	31	7,766	7,789	7,789	7,960	7,984	7,984	37,470	37,633	37,633
		asthma attacks	17	17	17	6	6	6	1	1	1	24	25	25	0	0	0	0	0	0	6	6	6	7	7	7	31	31	31
PM2.5	PM2.5	Health effect mortality	77,758	78,119	78,119	27,994	28,156	28,156	4,366	4,384	4,384	109,314	109,846	109,846	611	613	613	115	117	117	28,736	28,822	28,822	29,457	29,548	29,548	137,748	138,364	138,364
		chronic illness	9,693	10,624	10,624	4,868	5,219	5,219	1,340	1,368	1,368	15,868	17,174	17,174	1,977	1,986	1,986	345	352	352	37,407	37,580	37,580	39,690	39,878	39,878	67,349	67,737	67,737
		asthma attacks	3	4	4	2	2	2	0	0	0	6	6	6	1	1	1	1	1	1	14	14	14	13	13	13	18	19	19
Coarse PM10 coarse PM10	Coarse PM10 coarse PM10	Health effect lung cancer	184	203	203	337	337	337	26	26	26	301	308	308	38	38	38	7	7	7	14	14	14	750	754	754	1,041	1,071	1,071
		Health effect mortality	199	199	199	103	103	103	29	29	29	331	331	331	18	18	18	3	3	3	340	360	360	381	381	381	712	712	712
		Health effect chronic illness	254	375	3874	104	140	1,314	28	30	215	385	545	5,802	10	11	54	2	3	40	155	168	2,741	167	163	2,835	552	727	8,238
Total for direct PM10 emissions	SOx, NH3 sulfate PM10	Health effect mortality	14,902	16,645	80,987	7,385	8,006	29,684	2,029	2,075	5,495	24,282	26,688	115,698	2,867	2,884	2,884	89	93	93	6,105	6,105	6,105	422	453	7,456	1,382	1,713	21,478
		Health effect chronic illness	2,359	10,267	10,267	2,758	4,112	4,112	417	555	555	10,523	14,908	14,908	652	670	670	131	153	153	11,830	12,672	12,672	12,610	13,490	13,490	23,077	28,315	28,315
		Health effect asthma attacks	2,741	3,731	3,731	1,017	1,473	1,473	153	200	200	3,911	5,404	5,404	243	249	249	48	56	56	4,302	4,583	4,583	4,993	4,887	4,887	8,504	10,291	10,291
Total for SOx and NH3 emissions	VOCs organic PM10 (SOA)	Health effect mortality	136	188	188	51	75	75	8	10	10	194	272	272	12	12	12	2	3	3	218	233	233	233	248	248	425	518	518
		Health effect chronic illness	10,238	14,189	14,189	3,827	5,662	5,662	578	765	765	14,631	20,589	20,589	907	932	932	182	211	211	16,354	17,492	17,492	17,440	18,630	18,630	32,014	39,133	39,133
		Health effect asthma attacks	3,268	3,991	3,991	1,267	1,592	1,592	144	155	155	1,990	2,274	2,274	4	5	5	2	2,274	2,274	2,274	2,274	2,274	2,274	2,274	2,274	2,274	2,274	2,274
Sub-total for VOC emissions	VOCs, NOx, O3	Health effect mortality	1,290	1,522	1,522	496	597	597	144	155	155	1,990	2,274	2,274	4	5	5	2	2,274	2,274	2,274	2,274	2,274	2,274	2,274	2,274	2,274	2,274	2,274
		Health effect chronic illness	62	75	75	24	30	30	7	7	7	92	112	112	0	0	0	0	0	0	13	17	17	14	17	17	106	129	129
		Health effect asthma attacks	103	103	103	41	41	41	7	7	7	150	150	150	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Sub-total mortality effects	Subtotal cancer effects	Health effect mortality	5,087	6,056	6,056	1,972	2,293	2,293	544	589	589	7,600	9,034	9,034	14	18	18	3	9	9	1,000	1,246	1,246	1,017	1,273	1,273	8,615	10,305	10,305
		Health effect chronic illness	212	220	220	80	84	84	14	14	14	301	311	311	1	1	1	0	0	0	47	49	49	49	50	50	359	367	367
		Health effect asthma attacks	411	425	425	152	158	158	25	26	26	603	624	624	2	2	2	0	0	0	89	91	91	91	94	94	740	760	760
Subtotal chronic morbidity effects	Subtotal acute morbidity effects	Health effect mortality	266	273	273	111	115	115	20	21	21	354	362	362	1	2	2	0	0	0	27	28	28	28	29	29	225	231	231
		Health effect chronic illness	76,573	82,010	127,659	29,271	31,573	47,046	5,346	5,582	5,582	111,120	116,549	181,859	3,080	3,113	3,113	562	597	597	69,784	71,070	104,531	75,377	74,732	109,205	183,490	192,214	288,411
		Health effect asthma attacks	666	666	666	287	287	287	63	63	63	1,015	1,015	1,015	19	19	19	3	4	4	374	374	374	396	397	397	1,411	1,412	1,412
Subtotal ambient O3	Subtotal ambient NO2	Health effect mortality	7,273	7,418	8,310	2,725	2,790	3,093	475	482	500	10,025	10,212	11,440	90	91	102	17	18	27	2,779	2,813	3,461	2,884	2,920	3,587	12,560	12,787	14,627
		Health effect chronic illness	104,415	111,423	175,764	39,238	42,870	64,549	7,303	7,599	11,019	150,947	161,256	250,265	4,373	4,421	5,213	795	845	1,540	98,815	100,573	147,617	103,933	105,788	154,268	353,838	265,941	401,749
		Health effect asthma attacks	1,014	1,047	1,047	389	404	404	67	69	69	1,142	1,488	1,488	5	5	5	1	1	1	230	236	236	236	243	243	243	243	243
Total all emissions, pollutants, effects	Total all emissions, pollutants, effects	Health effect mortality	114,025	121,084	185,425	43,576	46,483	68,112	7,946	8,246	11,466	164,289	174,689	265,679	4,406	4,454	5,247	801	852	1,547	100,316	102,067	149,131	105,475	107,342	155,822	268,428	280,599	416,408
		Health effect chronic illness	29,113	30,991	46,591	10,993	11,783	17,686	2,043	2,119	3,048	42,149	44,892	69,324	1,218	1,231	1,447	219	233	422	27,381	27,830	40,765	28,818	29,294	42,633	70,967	74,186	111,957
		Health effect asthma attacks	3,108	3,119	3,119	1,155	1,162	1,162	181	181	181	4,196	4,217	4,217	25	25	25	5	5	5	1,184	1,186	1,186	1,186	1,213	1,213	1,213	1,213	1,213

TABLE 11-A COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$ MILLION (U.S. RURAL 1990) (LOWER BOUND)

Emission	Ambient pollutant	Health effect	IDGVA		LDGTS		RDGTS		Total gasoline vehicles		IDDV's		LDDT's		HDDV's		Total diesel vehicles		Total all vehicles			
			V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U
CO		morality	3	3	1	1	0	0	0	0	4	4	0	0	0	0	0	0	0	0	5	5
		hospitalization	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	1	1
		headaches	3	3	1	1	0	0	0	0	4	4	0	0	0	0	0	0	0	0	4	4
NO _x	NO ₂	sore throat	2	2	1	1	0	0	0	0	3	3	0	0	0	0	0	0	0	0	3	3
		excess phlegm	1	1	0	0	0	0	0	0	2	2	0	0	0	0	0	0	0	0	2	2
		eye irritation	1	1	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	1	1
NO _x , NEB	nitrate PM10	morality	15	15	6	6	1	1	1	1	23	23	0	0	0	0	0	0	0	0	29	29
		chronic illness	2	2	1	1	0	0	0	0	2	3	0	0	0	0	0	0	0	0	3	3
		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total for NO _x and NH ₃ emissions		RRAD	1	1	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	2	2
		morality	22	22	9	9	1	1	1	1	33	33	0	0	0	0	0	0	0	0	43	43
		chronic illness	3	3	2	2	0	0	0	0	5	5	1	1	0	0	0	0	0	0	18	18
Coarse PM10 coarse PM10		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		RRAD	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		lung cancer	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Fine PM10 coarse PM10		morality	1	1	0	0	0	0	0	1	2	22	0	0	0	0	0	0	0	0	2	2
		chronic illness	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total for direct PM10 emissions		RRAD	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		morality	4	5	2	3	1	1	1	1	7	8	37	1	1	1	1	1	1	1	24	25
		chronic illness	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
SO _x , NH ₃ sulfate PM10		morality	4	8	2	4	0	0	0	0	6	13	13	0	0	0	0	0	0	0	15	15
		chronic illness	0	1	0	1	0	0	0	0	1	2	2	0	0	0	0	0	0	0	3	3
		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total for SO _x and NH ₃ emissions		RRAD	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		morality	5	10	2	5	0	0	0	0	7	16	16	0	0	0	0	0	0	0	16	17
		chronic illness	1	2	1	1	0	0	0	0	2	3	3	0	0	0	0	0	0	0	3	3
VOCs organic PM10 (SOA)		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		RRAD	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		oral cancer	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal ambient PM10		leukemia	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		cancer all sites	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		oral cancer	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal ambient NO _x		asthma attacks	2	3	1	1	0	0	0	0	3	4	4	0	0	0	0	0	0	0	3	4
		lower resp illness	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		upper resp illness	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal ambient CO		eye irritation	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		morality	27	33	12	15	2	2	2	2	41	50	74	1	1	1	1	1	1	1	42	44
		chronic illness	1	1	0	0	0	0	0	0	1	1	1	0	0	0	0	0	0	0	1	1
Subtotal ambient NO ₂		asthma attacks	3	4	1	1	0	0	0	0	4	6	6	0	0	0	0	0	0	0	7	9
		lower resp illness	10	10	11	4	4	5	1	1	14	15	17	0	0	0	0	0	0	0	18	19
		upper resp illness	28	35	13	16	2	2	2	2	43	54	82	1	1	1	1	1	1	35	50	
Total all emissions, pollutants, effects		eye irritation	6	6	6	2	2	2	2	9	9	9	0	0	0	0	0	0	0	0	9	9
		morality	40	47	17	21	3	3	3	3	60	71	100	1	1	1	1	1	1	1	52	57
		chronic illness	97	111	37	39	37	39	37	39	111	134	154	0	0	0	0	0	0	0	97	111

TABLE 11-A.10 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$ MILLION (U.S. RURAL, 1990) (UPPER BOUND)

Emission pollutant	Health effect	LDGVs			LDGTs			HDCGs			Total gasoline vehicles			LDVVs			IDDVs			HDDVs			Total diesel vehicles			Total all vehicles		
		V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD
CO	mortality hospitalization headaches	46	47	47	18	18	18	4	4	4	68	68	68	0	0	0	0	0	0	1	1	1	1	1	1	69	69	69
		2	2	2	1	1	1	0	0	0	3	3	3	0	0	0	0	0	0	0	0	0	0	0	0	3	3	3
		19	19	19	7	7	7	1	1	1	28	28	28	0	0	0	0	0	0	0	0	0	0	0	0	28	28	28
NOx	sore throat excess phlegm eye irritation	11	11	11	5	5	5	1	1	1	16	17	17	0	0	0	0	0	0	6	6	6	6	6	6	22	22	22
		5	5	5	2	2	2	0	0	0	8	8	8	0	0	0	0	0	0	3	3	3	3	3	3	10	10	10
		5	5	5	2	2	2	0	0	0	7	7	7	0	0	0	0	0	0	2	2	2	2	2	2	9	9	9
NOx, NH3 nitrate PM10	chronic illness asthma attacks RRAD	286	289	289	122	123	123	21	21	21	428	432	432	2	2	2	0	0	0	146	147	147	149	149	149	577	581	581
		35	35	35	15	15	15	3	3	3	52	53	53	0	0	0	0	0	0	19	19	19	19	19	19	71	72	72
		0	0	0	0	0	0	0	0	0	6	6	6	0	0	0	0	0	0	2	2	2	2	2	2	9	9	9
Total for NOx and NH3 emissions PM2.5	mortality chronic illness asthma attacks RRAD lung cancer	346	349	349	147	148	148	25	25	25	518	522	522	2	2	2	3	3	3	178	178	178	181	181	181	699	704	704
		46	51	628	27	30	223	9	9	40	83	90	690	9	9	16	2	2	2	8	299	300	311	311	311	749	393	401
		6	7	74	3	4	26	1	1	5	10	12	105	0	0	0	0	0	0	1	39	39	89	41	41	92	51	52
Coarse PM10 coarse PM10	mortality chronic illness asthma attacks RRAD	1	1	9	0	0	3	0	0	1	1	13	0	0	0	0	0	0	0	4	5	11	5	5	11	6	6	24
		3	3	3	2	2	2	1	1	1	5	5	5	0	0	0	0	0	0	5	5	5	5	5	5	6	6	11
		3	4	161	1	2	55	0	0	9	5	6	225	0	0	0	0	0	0	3	3	119	3	3	123	8	9	348
Total for diesel PM10 emissions SOx, NH3 sulfate PM10	mortality chronic illness asthma attacks RRAD	37	64	64	16	30	30	3	4	4	56	99	99	3	3	3	1	1	1	101	109	109	105	113	113	180	212	212
		4	8	8	2	4	4	0	0	1	6	13	13	0	0	0	0	0	0	12	13	13	12	14	14	19	27	27
		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total for SOx and NH3 emissions VOCs	mortality chronic illness asthma attacks RRAD oral cancer leukemia cancer all sites oral cancer	41	74	74	18	35	35	3	5	5	63	114	114	4	4	4	1	1	1	114	124	124	119	129	129	181	242	242
		15	21	21	7	9	9	2	2	2	24	32	32	0	0	0	0	0	0	5	6	6	5	6	6	29	38	38
		2	2	2	1	1	1	0	0	0	3	4	4	0	0	0	0	0	0	1	1	1	1	1	1	4	5	5
Subtotal for VOC emissions VOCs, NOx, O3	asthma attacks lower resp illness upper resp illness eye irritation	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal mortality effects Subtotal cancer effects	mortality chronic illness asthma attacks RRAD oral cancer leukemia cancer all sites oral cancer	434	475	1,209	192	212	458	39	40	79	664	726	1,746	15	15	24	3	3	11	554	566	1,106	572	584	1,236	1,311	2,887	
		9	9	9	5	5	5	1	1	1	15	15	15	0	0	0	0	0	0	6	6	6	6	6	6	21	21	21
		47	53	139	21	24	53	4	5	9	73	82	201	2	2	3	0	0	0	71	72	135	73	75	140	146	157	341
Subtotal ambient PM10 Subtotal ambient O3 Subtotal ambient CO	mortality chronic illness asthma attacks RRAD	55	56	67	23	23	27	4	4	5	82	84	99	0	0	0	0	0	0	22	23	31	23	23	31	105	107	130
		443	490	1,322	199	222	501	40	43	87	683	755	1,989	17	17	27	4	4	13	638	651	1,262	659	673	1,341	1,427	3,211	
		8	8	8	3	3	3	1	1	1	11	12	12	0	0	0	0	0	0	3	3	3	3	3	3	3	14	15
Total all emissions, pollutants, effects	mortality chronic illness asthma attacks RRAD lung cancer	67	67	67	26	26	26	5	5	5	99	99	99	0	0	0	0	0	0	99	99	99	99	99	99	100	100	100
		545	593	1,424	240	264	543	48	50	94	824	907	2,561	17	18	28	4	4	13	653	667	1,278	674	688	1,318	1,508	3,378	
		6	6	6	3	3	3	1	1	1	6	6	6	0	0	0	0	0	0	6	6	6	6	6	6	7	7	7

TABLE 11-A-19 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POI MUTANT PATHWAY AND HEALTH EFFECT, \$ MILLION (MINNEAPOLIS MSA 1990) (LOWER BOU

Emission	Ambient pollutant	Health effect	LDGVs		LDG1s		HDG1s		Total gasoline vehicles		LDD1s		HDDVs		Total diesel vehicles		Total all vehicles				
			V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	
CO	CO	mortality	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
			0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
NOx	NO2	headaches	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
NOx, NH3	nitrate PM10	eye irritation	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total for NCRand NHEmissions	PM2.5	mortality	5	5	2	2	0	0	0	0	6	6	0	0	1	1	1	1	7	8	
			1	1	0	0	0	0	0	0	1	1	0	0	0	0	0	0	2	2	
Coarse PM10 coarse	PM10	chronic illness	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
			0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
Total for direct PM10emissions	SOx, NH3 sulfate PM10	mortality	1	2	4	1	2	0	0	2	3	6	0	0	2	3	4	3	5	6	
			1	2	2	0	1	1	0	0	1	3	3	0	0	1	1	1	2	4	
Subtotal for VOC emissions	VOCs	mortality	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
			0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
Subtotal mortality effects	Subtotal cancer effects	Subtotal chronic morbidity effects	7	9	11	3	4	4	0	1	10	13	16	0	0	4	4	6	4	15	
			0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
Subtotal acute morbidity effects	Subtotal ambient PM10	Subtotal ambient O3	2	2	2	1	1	1	0	0	2	3	3	0	0	1	1	1	1	3	
			9	11	14	3	4	5	0	1	12	16	19	0	0	5	6	7	5	18	
Subtotal ambient NO2	Subtotal ambient CO	Subtotal all emissions	1	1	1	0	0	0	0	1	1	1	0	0	0	0	0	0	0	1	
			1	1	1	0	0	0	0	0	1	1	1	0	0	0	0	0	0	1	
Total all emissions			10	13	15	4	5	6	0	1	15	19	22	0	0	6	8	6	20		
Total all emissions			10	13	15	4	5	6	0	1	15	19	22	0	0	6	8	6	20		

TABLE 11-A-25 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$ MILLION (PHOENIX MSA 1990) (LOWER ROUND)

Emission pollutant	Ambient pollutant	Health effect	LDGVs		LDGIs		HDGIs		Total gasoline vehicles		LDDVs		LDDIs		HDDVs		Total diesel vehicles		Total all vehicles		
			V	VAL	V	VAL	V	VAL	V	VAL	V	VAL	V	VAL	V	VAL	V	VAL	V	VAL	V
CO		mortality	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	1	1
		hospitalization	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		headaches	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	1	1
NOx		sore throat	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	1	1
		excess phlegm	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		eye irritation	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
NOx, NH3	nitrate PM10	mortality	5	5	2	2	0	0	0	6	6	0	0	0	0	1	1	1	1	8	8
		chronic illness	2	2	1	1	0	0	0	2	2	0	0	0	0	1	1	1	1	3	3
		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		RRAD	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total for NOx and NH3 emissions			7	7	3	3	0	0	0	10	10	0	0	0	0	2	2	2	2	13	13
PM2.5		mortality	1	1	0	0	0	0	0	2	2	0	0	0	0	2	2	3	3	4	4
		chronic illness	0	0	0	0	0	0	0	1	1	0	0	0	0	1	1	1	1	1	1
		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		RRAD	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		lung cancer	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Coarse PM10 coarse PM10		mortality	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		chronic illness	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		RRAD	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total for direct PM10 emissions			2	2	3	3	1	1	1	3	3	0	0	0	0	4	4	5	5	7	7
SOx, NH3 sulfate PM10		mortality	1	2	0	0	0	0	0	2	4	0	0	0	0	1	2	1	2	3	6
		chronic illness	0	1	0	0	0	0	0	1	1	0	0	0	0	1	1	1	1	2	2
		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		RRAD	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total for SOx and NH3 emissions			2	3	1	1	1	1	1	3	5	0	0	0	0	2	2	2	2	4	8
VOCs	organic PM10 (SOA)	mortality	0	1	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	1	1
		chronic illness	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		RRAD	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		oral cancer	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		leukemia	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		cancer all sites	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		oral cancer	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Sub-total for VOC emissions			1	1	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	1	1
VOCs, NOx, O3		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		lower resp illness	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		upper resp illness	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		eye irritation	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal mortality effects			8	9	10	3	4	5	0	11	14	0	0	0	0	5	6	6	6	17	20
Subtotal cancer effects			0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal chronic morbidity effects			3	3	3	1	1	2	0	4	5	0	0	0	0	2	2	2	2	6	7
Subtotal acute morbidity effects			2	2	2	1	1	1	0	3	3	0	0	0	0	1	1	1	1	3	4
Subtotal ambient PM10			10	12	14	4	6	6	1	15	19	1	1	0	0	8	8	9	10	23	28
Subtotal ambient O3			0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal ambient NO2			1	1	1	1	0	0	0	1	1	0	0	0	0	0	0	0	0	2	2
Subtotal ambient CO			1	1	1	0	0	0	0	1	1	0	0	0	0	0	0	0	0	1	1
Total all emissions, pollution, effects			13	15	16	5	7	7	1	18	22	1	1	1	0	8	8	9	10	26	31

TABLE 11-A-26 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$ MILLION (PHOENIX MSA 1990) (UPPER BOUND)

Emission	Ambient pollutant	Health effect	IDDVs		LDGTs		HDGTs		Total gasoline vehicles		IDDVs		LDGTs		HDGTs		Total diesel vehicles		Total all vehicles			
			V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U
CO	CO	fatality	6	6	2	2	1	1	1	1	8	8	0	0	0	0	0	0	0	0	9	9
		hospitalization	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		headaches	3	3	1	1	0	0	0	0	4	4	0	0	0	0	0	0	0	0	4	4
NOx	NO2	sore throat	3	3	1	1	0	0	0	0	4	4	0	0	0	0	0	0	0	0	5	5
		excess phlegm	1	1	0	0	0	0	0	0	2	2	0	0	0	0	0	0	0	0	2	2
		eye irritation	1	1	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	2	2
NOx, NH3	nitrate PM10	fatality	81	81	29	29	5	5	5	5	114	114	1	1	0	0	0	0	0	0	130	130
		chronic illness	54	54	19	19	3	3	3	3	76	76	0	0	0	0	0	0	0	0	100	100
		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total for NOx and NH3 emissions	PM2.5	RRAD	2	2	1	1	0	0	0	0	2	2	0	0	0	0	0	0	0	0	3	3
		fatality	141	141	50	50	8	8	8	8	199	199	1	1	0	0	0	0	0	0	262	262
		chronic illness	16	16	8	8	2	2	2	2	26	26	3	3	4	4	1	1	1	1	95	95
Coarse PM10 coarse PM10	PM10	fatality	10	10	5	5	18	18	2	2	17	17	2	2	3	3	0	0	0	0	63	63
		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		RRAD	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	2	2
Total for direct PM10 emissions	SOx, NH3 sulfate PM10	fatality	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		chronic illness	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1
		asthma attacks	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	2
Total for SOx and NH3 emissions	VOCs organic PM10 (SOA)	fatality	28	28	14	14	55	55	4	4	46	46	5	5	7	7	1	1	1	1	165	165
		chronic illness	9	9	3	3	4	4	1	1	12	13	1	1	1	1	0	0	0	0	28	29
		asthma attacks	6	6	2	2	0	0	0	0	8	8	0	0	0	0	0	0	0	0	18	18
Total for VOC emissions	VOCs, NOx, O3	fatality	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		chronic illness	14	15	5	6	6	6	1	1	21	21	1	1	1	1	0	0	0	0	47	48
		asthma attacks	5	6	2	2	2	2	1	1	8	10	0	0	0	0	0	0	0	0	9	11
Subtotal mortality effects	Subtotal cancer effects	fatality	4	4	1	1	2	2	0	0	5	6	0	0	0	0	0	0	0	0	6	7
		chronic illness	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal acute morbidity effects	Subtotal ambient PM10	fatality	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		chronic illness	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal ambient NO2	Subtotal ambient CO	fatality	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		chronic illness	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		asthma attacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total all emissions, pollutants, effects	Subtotal mortality effects	fatality	117	118	44	45	69	69	9	9	170	172	5	5	6	6	1	1	1	1	296	296
		chronic illness	1	1	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	1	1
		asthma attacks	74	74	28	28	44	44	6	6	107	108	3	3	4	4	1	1	1	1	189	190
Subtotal ambient NO2	Subtotal ambient CO	fatality	12	12	4	4	5	5	1	1	17	17	0	0	0	0	0	0	0	0	22	22
		chronic illness	188	190	312	312	71	72	113	114	273	276	8	8	9	9	1	1	1	1	481	484
		asthma attacks	2	2	1	1	0	0	0	0	2	2	0	0	0	0	0	0	0	0	3	3
Total all emissions, pollutants, effects	Subtotal ambient NO2	fatality	9	9	5	5	3	3	1	1	13	13	0	0	0	0	0	0	0	0	9	9
		chronic illness	203	205	327	327	77	78	119	120	295	299	8	8	8	8	10	10	1	1	506	509
		asthma attacks	203	205	327	327	77	78	119	120	295	299	8	8	8	8	10	10	1	1	506	509

TABLE 11-A-28 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$ MILLION (ST. LOUIS MSA 1990) (UPPER BOUND)

Emission pollutant	LDGVs		LDGTs		HDGTs		LDDVs		LDDTs		HDDVs		TDDVs		Total all vehicles	
	V	V+UARD	V	V+UARD	V	V+UARD	V	V+UARD	V	V+UARD	V	V+UARD	V	V+UARD	V	V+UARD
CO	4	4	1	1	0	0	0	0	0	0	0	0	0	0	5	5
	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
NOx	2	2	1	1	0	0	0	0	0	0	0	0	0	0	2	2
	1	1	0	0	0	0	0	0	0	0	0	0	0	0	1	1
NOx, NH3	58	58	21	21	3	3	3	3	0	0	18	19	19	19	97	101
	15	16	6	6	1	1	1	1	0	0	5	5	5	5	26	28
Total for NOx and NH3 emissions	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	1	1	0	0	0	0	0	0	0	0	0	0	0	0	2	2
PM2.5	76	79	27	28	4	4	4	4	1	1	25	25	25	26	132	137
	10	12	6	6	1	1	1	1	0	0	37	38	40	40	56	59
Coarse PM10	3	3	1	1	0	0	0	0	0	0	11	11	11	11	16	17
	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total for direct PM10 emissions	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1
	1	1	0	0	0	0	0	0	0	0	0	0	0	0	1	1
SOx, NH3	6	41	2	18	0	2	2	2	0	0	0	10	18	18	19	20
	2	14	6	6	0	1	1	1	0	0	3	6	6	6	6	7
Total for SOx and NH3 emissions	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
VOCs	8	56	3	24	0	3	3	3	1	1	13	25	25	26	26	109
	3	4	1	2	0	0	0	0	0	0	1	1	1	1	6	8
Subtotal for VOC emissions	1	1	0	1	0	0	0	0	0	0	0	0	0	0	2	2
	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal mortality effects	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal cancer effects	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal chronic morbidity effects	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal acute morbidity effects	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Subtotal ambient PM10	8	153	37	61	6	9	9	9	4	5	86	100	152	91	233	
	1	1	0	0	0	0	0	0	0	0	0	0	0	0	2	
Subtotal ambient O3	4	4	1	1	0	0	0	0	0	0	1	1	1	1	7	
	6	6	2	2	0	0	0	0	0	0	0	0	0	0	8	
Subtotal ambient CO	109	161	41	65	7	10	13	13	4	5	88	101	154	93	250	
	109	161	41	65	7	10	13	13	4	5	88	101	154	93	250	

TABLE 11-A-33 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$/1000 VMT (U.S. TOTAL 1990) (LOWER BOUND)

Emission pollutant	Ambient pollutant	Health effect	LDGVs			LDGTs			HDCGs			Total gasoline vehicles			LDDVs			LDDTs			HDDVs			Total diesel vehicles			Total all vehicles						
			V	VAL	VAL:RD	V	VAL	VAL:RD	V	VAL	VAL:RD	V	VAL	VAL:RD	V	VAL	VAL:RD	V	VAL	VAL:RD	V	VAL	VAL:RD	V	VAL	VAL:RD	V	VAL	VAL:RD	V	VAL	VAL:RD	
CO	CO	morality	0.19	0.19	0.19	0.26	0.26	0.26	0.69	0.69	0.69	0.22	0.22	0.22	0.01	0.01	0.01	0.00	0.00	0.00	0.03	0.03	0.03	0.02	0.02	0.02	0.20	0.20	0.20	0.03	0.03	0.03	
		hospitalization	0.03	0.03	0.03	0.04	0.04	0.04	0.11	0.11	0.11	0.03	0.03	0.03	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
		headaches	0.20	0.20	0.20	0.29	0.29	0.29	0.71	0.71	0.71	0.22	0.22	0.22	0.01	0.01	0.01	0.00	0.00	0.00	0.03	0.03	0.03	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02
NOx	NO2	sore throat	0.23	0.23	0.23	0.33	0.34	0.34	0.64	0.64	0.64	0.25	0.26	0.26	0.13	0.13	0.13	0.04	0.04	0.04	0.82	0.82	0.82	0.65	0.66	0.66	0.29	0.29	0.29	0.06	0.06	0.06	
		excess phlegm	0.10	0.10	0.10	0.15	0.15	0.15	0.28	0.28	0.28	0.11	0.11	0.11	0.06	0.06	0.06	0.02	0.02	0.02	0.36	0.37	0.37	0.29	0.29	0.29	0.13	0.13	0.13	0.03	0.03	0.03	
		eye irritation	0.09	0.09	0.09	0.13	0.14	0.14	0.26	0.26	0.26	0.10	0.10	0.10	0.05	0.05	0.05	0.02	0.02	0.02	0.33	0.33	0.33	0.26	0.27	0.27	0.12	0.12	0.12	0.01	0.01	0.01	
NOx, NH3	nitrate PM10	morality	2.20	2.22	2.22	3.18	3.22	3.22	6.07	6.13	6.13	2.45	2.47	2.47	1.26	1.27	1.27	0.41	0.42	0.42	7.78	7.84	7.84	6.22	6.27	6.27	2.75	2.77	2.77	0.66	0.66	0.66	
		chronic illness	0.61	0.62	0.62	0.86	0.89	0.89	1.69	1.71	1.71	0.68	0.69	0.69	0.36	0.36	0.36	0.11	0.12	0.12	2.15	2.17	2.17	1.72	1.74	1.74	0.76	0.77	0.77	0.00	0.00	0.00	
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
Total for NOx and NH3 emissions	PM2.5	RRAD	3.35	3.38	3.38	4.83	4.89	4.89	9.24	9.33	9.33	3.72	3.76	3.76	1.92	1.94	1.94	0.62	0.65	0.65	11.84	11.93	11.93	9.47	9.55	9.55	4.18	4.22	4.22	0.95	0.95	0.95	
		morality	0.44	0.49	0.49	0.88	0.99	0.99	1.14	1.14	1.14	0.55	0.62	0.62	0.56	0.56	0.56	0.19	0.21	0.21	1.86	1.86	1.86	1.68	1.68	1.68	1.44	1.44	1.44	0.36	0.36	0.36	
		chronic illness	0.13	0.14	0.14	0.25	0.28	0.28	0.32	0.32	0.32	0.16	0.18	0.18	0.13	0.13	0.13	0.04	0.04	0.04	0.52	0.52	0.52	0.48	0.48	0.48	0.40	0.40	0.40	0.00	0.00	0.00	
Coarse PM10 coarse PM10	PM10	asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
		RRAD	0.02	0.02	0.02	0.03	0.04	0.04	0.05	0.05	0.05	0.03	0.03	0.03	0.02	0.02	0.02	0.00	0.00	0.00	0.09	0.09	0.09	0.09	0.09	0.09	0.07	0.07	0.07	0.06	0.06	0.06	
		lung cancer	0.01	0.01	0.01	0.02	0.02	0.02	0.02	0.02	0.02	0.01	0.01	0.01	0.06	0.06	0.06	0.02	0.02	0.02	0.16	0.16	0.16	0.14	0.14	0.14	0.02	0.02	0.02	0.02	0.02	0.02	0.02
Coarse PM10 coarse PM10	PM10	morality	0.14	0.16	0.16	0.24	0.26	0.26	0.44	0.44	0.44	0.17	0.19	0.19	0.36	0.37	0.37	0.11	0.12	0.12	0.80	0.80	0.80	0.62	0.66	0.66	0.20	0.20	0.20	0.06	0.06	0.06	
		chronic illness	0.04	0.05	0.05	0.07	0.07	0.07	0.25	0.25	0.25	0.05	0.05	0.05	0.10	0.11	0.11	0.03	0.03	0.03	0.19	0.19	0.19	0.17	0.18	0.18	0.06	0.06	0.06	0.00	0.00	0.00	
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
Total for direct PM10 emissions	SOx, NH3 sulfate PM10	RRAD	0.01	0.01	0.01	0.04	0.04	0.04	0.05	0.05	0.05	0.01	0.01	0.01	0.02	0.02	0.02	0.00	0.00	0.00	0.09	0.09	0.09	0.09	0.09	0.09	0.07	0.07	0.07	0.07	0.07	0.07	
		morality	0.78	0.88	0.88	1.51	1.70	1.70	2.89	4.27	4.53	0.97	1.09	1.09	2.06	2.06	2.06	0.81	0.81	0.81	11.47	13.31	13.31	9.69	11.18	11.18	1.66	2.41	2.41	0.25	0.25	0.25	
		chronic illness	0.65	1.08	1.08	0.99	1.84	1.84	1.84	1.57	2.80	2.80	0.75	1.26	1.26	3.68	3.84	3.84	1.27	1.60	1.60	8.76	10.18	10.18	7.40	8.55	8.55	1.26	1.84	1.84	0.48	0.48	0.48
Total for SOx and NH3 emissions	VOCs	asthma attacks	0.18	0.28	0.28	0.27	0.48	0.48	0.41	0.71	0.71	0.20	0.33	0.33	1.01	1.05	1.05	0.34	0.42	0.42	2.26	2.60	2.60	1.92	2.20	2.20	0.34	0.48	0.48	0.00	0.00	0.00	
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
		RRAD	0.03	0.05	0.05	0.05	0.09	0.09	0.08	0.14	0.14	0.14	0.04	0.06	0.06	0.18	0.19	0.19	0.06	0.08	0.08	0.44	0.51	0.51	0.37	0.43	0.43	0.06	0.09	0.09	0.00	0.00	0.00
Subtotal for VOC emissions	VOCs, NOx, O3	morality	0.87	1.42	1.42	1.20	2.42	2.42	2.06	3.65	3.65	0.97	1.65	1.65	4.87	5.08	5.08	1.68	2.11	2.11	11.47	13.31	13.31	9.69	11.18	11.18	1.66	2.41	2.41	0.25	0.25	0.25	
		chronic illness	0.19	0.28	0.28	0.30	0.44	0.44	0.88	1.12	1.12	0.22	0.32	0.32	0.03	0.06	0.06	0.06	0.06	0.06	0.34	0.34	0.34	0.27	0.46	0.46	0.23	0.33	0.33	0.06	0.06	0.06	
		asthma attacks	0.05	0.07	0.07	0.08	0.12	0.12	0.25	0.30	0.30	0.06	0.06	0.06	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Subtotal mortality effects	Subtotal cancer effects	RRAD	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		lower resp illness	0.03	0.04	0.04	0.05	0.05	0.05	0.10	0.11	0.11	0.04	0.04	0.04	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
Subtotal ambient CO2	Subtotal ambient CO2	upper resp illness	0.01	0.01	0.01	0.01	0.02	0.02	0.03	0.03	0.03	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	
		eye irritation	0.03	0.04	0.04	0.05	0.06	0.06	0.11	0.12	0.12	0.04	0.04	0.04	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	
		total	3.82	4.43	4.43	5.12	5.86	5.86	7.04	7.97	14.10	16.35	4.34	5.07	5.83	10.97	11.19	11.73	3.49	3.94	4.74	31.52	33.50	33.50	26.20	27.80	27.80	6.08	8.68	8.68	7.96	9.96	9.96
Subtotal ambient CO2	Subtotal ambient CO2	asthma attacks	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	
		lower resp illness	1.02	1.16	1.32	1.54	1.84	2.05	3.24	3.66	4.17	1.15	1.33	1.50	3.11	3.16	3.29	0.97	1.08	1.26	8.51	8.99	10.36	7.10	7.49	8.58	1.62	1.82	2.07	0.53	0.53	0.53	
		total	0.92	0.96	1.00	1.36	1.44	1.49	2.85	2.98	3.10	1.03	1.08	1.10	0.84	0.85	0.88	0.27	0.30	0.34	3.34	3.47	3.79	2.72	2.82	3.08	1.16	1.22	1.28	0.11	0.11	0.11	
Subtotal ambient CO2	Subtotal ambient CO2	asthma attacks	4.84																														

TABLE 11-A-34 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$/1000 VMT (U.S. TOTAL 1990) (UPPER BOUND)

Emission CO	Ambient pollutant		Health effect		IDGVs		LDGIs		HDGIs		Total gasoline vehicles		LDVs		IDDVs		HDDVs		Total diesel vehicles		Total all vehicles			
	V	VAI	V	VAI	V	VAI	V	VAI	V	VAI	V	VAI	V	VAI	V	VAI	V	VAI	V	VAI	V	VAI	V	VAI
CO	2.40	2.40	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09
	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09
	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19
NOx	1.19	1.19	0.53	0.53	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46
	0.53	0.53	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46	0.46
	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.19
NO _x , NH ₃	36.02	36.19	13.62	13.69	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
	13.62	13.69	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
	36.02	36.19	13.62	13.69	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
Total for NO _x and NH ₃ emissions	52.53	52.79	13.62	13.69	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
	13.62	13.69	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
	52.53	52.79	13.62	13.69	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
PM _{2.5}	6.52	7.15	2.55	2.84	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	2.55	2.84	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	6.52	7.15	2.55	2.84	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Coarse PM ₁₀ coarse PM ₁₀	0.42	0.57	0.17	0.24	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	0.17	0.24	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	0.42	0.57	0.17	0.24	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Total for direct PM ₁₀ emissions	9.94	11.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	9.94	11.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
SO _x , NH ₃	4.96	7.01	1.78	2.44	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	1.78	2.44	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	4.96	7.01	1.78	2.44	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Total for SO _x and NH ₃ emissions	6.83	9.58	2.55	3.52	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	2.55	3.52	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	6.83	9.58	2.55	3.52	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
VOCs	2.19	2.69	0.84	0.99	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	0.84	0.99	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	2.19	2.69	0.84	0.99	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Subtotal for VOC emissions	3.42	4.08	1.19	1.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	1.19	1.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	3.42	4.08	1.19	1.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
VOCs, NO _x , O ₃	0.15	0.15	0.28	0.29	0.08	0.09	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
	0.28	0.29	0.08	0.09	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
	0.15	0.15	0.28	0.29	0.08	0.09	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
Subtotal mortality effects	52.52	56.02	0.49	0.49	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21
	0.49	0.49	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21
	52.52	56.02	0.49	0.49	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21
Subtotal cancer effects	18.97	20.21	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
	18.97	20.21	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
Subtotal chronic morbidity effects	5.16	5.27	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
	5.16	5.27	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
Subtotal acute morbidity effects	70.18	74.99	0.72	0.74	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22
	0.72	0.74	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22
	70.18	74.99	0.72	0.74	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22
Subtotal ambient PM ₁₀	2.20	2.22	3.69	3.69	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21
	3.69	3.69	0.21																					

TABLE 11-A 35 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$1000 VMT (URBAN U.S. TOTAL 1990) (LOWER BOUND)

Emission	Ambient pollutant	Health effect	IDGVs			LDG1s			LDG2s			HDG1s			DDV1s			LDD1s			RDDV1s			Total diesel vehicles			Total all vehicles		
			V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD
CO	CO	mortality	0.23	0.23	0.23	0.35	0.35	0.35	0.91	0.91	0.91	0.26	0.26	0.26	0.01	0.01	0.01	0.00	0.00	0.00	0.04	0.04	0.04	0.03	0.03	0.03	0.24	0.24	0.24
			0.04	0.04	0.04	0.06	0.06	0.06	0.14	0.14	0.14	0.04	0.04	0.04	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.00	0.00	0.00	0.04	0.04	0.04
NOx	NOx	headaches	0.24	0.24	0.24	0.37	0.37	0.37	0.96	0.96	0.96	0.27	0.27	0.27	0.01	0.01	0.01	0.00	0.00	0.00	0.04	0.04	0.04	0.03	0.03	0.03	0.26	0.26	0.26
			0.28	0.28	0.28	0.44	0.44	0.44	0.87	0.87	0.87	0.32	0.32	0.32	0.16	0.16	0.16	0.06	0.06	0.06	1.11	1.12	1.12	0.87	0.87	0.87	0.36	0.36	0.36
NOx	NOx	excess phlegm	0.12	0.13	0.13	0.19	0.20	0.20	0.38	0.39	0.39	0.14	0.14	0.14	0.07	0.07	0.07	0.02	0.02	0.02	0.49	0.49	0.49	0.38	0.39	0.39	0.16	0.16	0.16
			0.11	0.11	0.11	0.18	0.18	0.18	0.35	0.35	0.35	0.13	0.13	0.13	0.07	0.07	0.07	0.02	0.02	0.02	0.45	0.45	0.45	0.35	0.35	0.35	0.14	0.14	0.14
NOx	NOx, NH3	eye irritation	2.77	2.79	2.79	4.28	4.33	4.33	8.56	8.65	8.65	3.12	3.15	3.15	1.59	1.60	1.60	0.55	0.57	0.57	10.93	11.02	11.02	6.53	6.60	6.60	3.51	3.55	3.55
			0.79	0.80	0.80	1.22	1.23	1.23	2.48	2.51	2.51	0.89	0.90	0.90	0.46	0.46	0.46	0.16	0.16	0.16	3.15	3.18	3.18	2.46	2.46	2.46	1.01	1.02	1.02
Total for NOx and NH3 emissions	PM2.5	asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.00	0.00	
			0.14	0.14	0.14	0.21	0.21	0.21	0.42	0.43	0.43	0.15	0.16	0.16	0.08	0.08	0.08	0.03	0.03	0.03	0.54	0.55	0.55	0.42	0.43	0.43	0.17	0.18	0.18
Total for NOx and NH3 emissions	PM2.5	RRAD	4.22	4.26	4.26	6.51	6.59	6.59	13.08	13.21	13.21	4.75	4.80	4.80	2.43	2.45	2.45	0.84	0.87	0.87	16.68	16.81	16.81	13.02	13.13	13.13	5.35	5.40	5.40
			0.55	0.62	0.75	1.19	1.19	1.94	1.52	3.57	3.79	4.25	0.71	0.79	0.93	0.71	0.71	0.71	0.29	0.34	0.34	19.46	19.74	19.74	15.99	16.21	16.21	1.82	1.92
Coarse PM10	Coarse PM10 coarse	mortality	0.16	0.18	0.21	0.35	0.39	0.44	1.03	1.09	1.21	0.21	0.23	0.27	0.11	0.12	0.12	0.07	0.09	0.10	5.56	5.64	5.64	4.58	4.64	4.68	0.53	0.56	0.61
			0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.02	0.02	0.01	0.01	0.01	0.00	0.00	0.00
Coarse PM10	Coarse PM10 coarse	asthma attacks	0.03	0.03	0.04	0.06	0.07	0.08	0.18	0.19	0.21	0.03	0.04	0.05	0.35	0.36	0.36	0.11	0.12	0.12	0.97	0.98	0.98	0.79	0.80	0.85	0.09	0.10	0.10
			0.01	0.01	0.01	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.01	0.01	0.01	0.01	0.01	0.01	0.02	0.02	0.02	0.21	0.21	0.21	0.17	0.17	0.17	0.02	0.02
Coarse PM10	Coarse PM10 coarse	RRAD	0.18	0.20	0.24	0.32	0.36	0.42	0.92	0.97	1.11	0.21	0.24	0.28	0.45	0.46	0.46	0.14	0.16	0.16	1.01	1.08	1.08	0.85	0.90	0.90	0.26	0.29	0.31
			0.05	0.06	0.06	0.11	0.11	0.11	0.22	0.27	0.33	0.07	0.07	0.07	0.13	0.14	0.14	0.05	0.05	0.05	0.29	0.31	0.31	0.24	0.26	0.26	0.06	0.08	0.08
Coarse PM10	Coarse PM10 coarse	leukemia	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
			0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Coarse PM10	Coarse PM10 coarse	RRAD	0.99	1.11	2.11	2.05	2.30	3.74	6.08	6.05	10.13	1.25	1.40	2.51	10.27	10.31	11.09	3.29	3.38	4.63	27.57	28.03	27.98	22.69	23.05	23.05	2.82	2.94	4.57
			0.82	1.35	1.35	1.33	2.46	2.46	2.46	2.21	3.94	3.94	0.93	1.59	1.59	4.63	4.84	4.84	1.72	2.16	2.16	12.21	14.21	14.21	10.06	11.66	11.66	1.60	2.33
SOx, NH3	sulfate PM10	chronic illness	0.23	0.36	0.36	0.37	0.66	0.66	0.60	1.03	1.03	0.26	0.43	0.43	1.31	1.35	1.35	0.48	0.59	0.59	3.29	3.79	3.79	2.73	3.12	3.12	0.44	0.62	0.62
			0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.00	0.00
SOx, NH3	sulfate PM10	asthma attacks	0.04	0.07	0.07	0.07	0.12	0.12	0.11	0.19	0.19	0.05	0.08	0.08	0.23	0.24	0.24	0.09	0.11	0.11	0.61	0.71	0.71	0.71	0.71	0.71	0.08	0.11	0.11
			1.10	1.79	1.79	1.79	2.34	3.24	2.93	5.17	5.17	1.24	2.10	2.10	6.17	6.43	6.43	2.28	2.85	2.85	16.12	18.71	18.71	13.32	15.37	15.37	2.13	3.07	3.07
SOx, NH3	sulfate PM10	RRAD	0.74	0.95	0.95	0.41	0.60	0.60	1.26	1.60	1.60	0.28	0.41	0.41	0.04	0.08	0.08	0.02	0.08	0.08	0.48	0.83	0.83	0.37	0.64	0.64	0.29	0.43	0.43
			0.07	0.09	0.09	0.12	0.16	0.16	0.37	0.44	0.44	0.08	0.11	0.11	0.01	0.02	0.02	0.02	0.02	0.02	0.14	0.21	0.21	0.10	0.17	0.17	0.08	0.12	0.12
SOx, NH3	sulfate PM10	leukemia	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
			0.01	0.02	0.02	0.02	0.03	0.03	0.03	0.06	0.08	0.08	0.01	0.02	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.04	0.04	0.02	0.03	0.03	0.01	0.02
SOx, NH3	sulfate PM10	cancer all sites	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
			0.02	0.02	0.02	0.03	0.03	0.03	0.07	0.07	0.07	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
SOx, NH3	sulfate PM10	oral cancer	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
			0.02	0.02	0.02	0.03	0.03	0.03	0.07	0.07	0.07	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
SOx, NH3	sulfate PM10	oral cancer	0.35	0.48	0.48	0.58	0.83	0.83	1.77	2.21	2.21	0.41	0.57	0.57	0.06	0.10	0.10	0.02	0.11	0.11	0.64	1.09	1.09	0.30	0.85	0.85	0.41	0.59	0.59
			0.01	0.01	0.01	0.02	0.02	0.02	0.04	0.04	0.04	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.03	0.03	0.02	0.02	0.02	0.01	0.01	0.01
SOx, NH3	sulfate PM10	lower resp. illness	0.04	0.04	0.04	0.06	0.07	0.07	0.14	0.15	0.15	0.05	0.05	0.05	0.01	0.02	0.02	0.01	0.01	0.01	0.09	0.10	0.10	0.07	0.08	0.08	0.05	0.05	0.05
			0.01	0.01	0.01	0.02	0.02	0.02	0.04	0.05	0.05	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.03	0.03	0.03	0.02	0.02	0.02	0.02	0.02	0.02
SOx, NH3	sulfate PM10	upper resp. illness	0.04	0.05	0.05	0.07	0.08	0.08	0.15	0.17	0.17	0.05	0.05	0.05	0.02	0.02	0.02	0.01	0.01	0.01	0.10	0.11	0.11	0.08	0.09	0.09	0.05	0.05	0.05
			0.04	0.05	0.05	0.07	0.08	0.08	0.15	0.17	0.17	0.05	0.05	0.05	0.02	0.02	0.02	0.01	0.01	0.01	0.10	0.11	0.11	0.08	0.09	0.09	0.05	0.05	0.05
SOx, NH3	sulfate PM10	eye irritation	4.80	5.55	6.31	7.57	9.44	10.55	17.42	19.86	22.71	5.51	6.44	7.29	13.85	14.12	14.72	4.72	5.31	6.28	44.12	46.91	54.54	35.85	38.05	43.97	7.73	8.75	9.98
			0.03	0.03	0.03	0.06	0.06	0.06	0.15	0.15	0.15	0.04	0.04	0.04	0.07	0.07	0.07	0.02	0.03	0.03	0.22	0.22	0.22	0.18	0.18	0.18	0.05	0.05	0.05
SOx, NH3	sulfate PM10	asthma attacks	1.31	1.50	1.69	2.15	2.82	4.75	5.76	6.06	1.51	1.74	1.95	4.04	4.09	4.24	1.36	1.50	1.74	12.43	13.12	15.00	10.12	10.67	12.13	2.14	2.39	2.70	
			1.13	1.18	1.28	1.78	1.88	1.94	3.90	4.08	4.23																		

TABLE 11-A-38 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$/1000 VMT (RURAL U S TOTAL 1990) (UPPER BOUND)

Emission	Ambient pollutant	Health effect	LDGVs			LDGTs			HDGTs			Total gasoline vehicles			LDGVs			LDGTs			HDDVs			Total diesel vehicles			Total all vehicles		
			V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD
CO	CO	mortality	1.24	1.25	1.36	1.59	1.59	4.24	4.24	4.24	1.37	1.38	1.38	0.01	0.03	0.03	0.01	0.01	0.01	0.20	0.20	0.20	0.16	0.17	0.17	1.26	1.26	1.26	
		hospitalization	0.05	0.06	0.06	0.06	0.06	0.17	0.17	0.17	0.05	0.05	0.05	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.05	0.05	0.05	
		headaches	0.51	0.51	0.51	0.65	0.65	1.73	1.73	1.73	0.56	0.56	0.56	0.01	0.01	0.01	0.00	0.00	0.00	0.06	0.06	0.06	0.07	0.07	0.07	0.51	0.51	0.51	
NOx	NOx	score throat	0.29	0.30	0.30	0.41	0.41	0.92	0.93	0.93	0.33	0.33	0.33	0.14	0.14	0.14	0.04	0.04	0.04	1.28	1.29	1.29	1.06	1.06	1.06	0.40	0.41	0.41	
		excess phlegm	0.14	0.14	0.14	0.19	0.19	0.43	0.43	0.43	0.15	0.15	0.15	0.06	0.06	0.06	0.02	0.02	0.02	0.59	0.59	0.59	0.49	0.49	0.49	0.19	0.19	0.19	
		eye irritation	0.12	0.12	0.12	0.17	0.17	0.38	0.39	0.39	0.14	0.14	0.14	0.06	0.06	0.06	0.02	0.02	0.02	0.53	0.53	0.53	0.44	0.44	0.44	0.17	0.17	0.17	
NOx, NH3	nitrate PM10	mortality	7.67	7.73	7.73	10.54	10.63	24.13	24.26	24.26	8.62	8.68	8.68	3.50	3.52	3.52	1.09	1.12	1.12	33.11	33.23	33.23	27.39	27.50	27.50	10.46	10.53	10.53	
		chronic illness	0.94	0.94	0.94	1.28	1.29	3.09	3.10	3.10	1.05	1.06	1.06	0.00	0.00	0.00	0.00	0.00	0.00	4.25	4.26	4.26	3.51	3.53	3.53	1.30	1.30	1.30	
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Total for NOx and NH3 emissions	RRAD	0.11	0.12	0.12	0.16	0.16	0.36	0.36	0.36	0.13	0.13	0.13	0.05	0.05	0.05	0.02	0.02	0.02	0.50	0.50	0.50	0.41	0.41	0.41	0.16	0.16	0.16		
		0.92	9.35	9.35	12.74	12.85	29.32	29.48	29.48	10.42	10.50	10.50	4.23	4.26	4.26	1.32	1.36	1.36	40.26	40.41	40.41	33.31	33.44	33.44	12.67	12.76	12.76		
		1.23	1.36	1.61	2.28	2.56	19.34	10.61	10.85	46.68	1.66	1.80	17.90	15.93	27.93	4.55	4.63	4.63	67.71	68.01	68.01	57.14	57.40	57.40	7.12	7.27	29.70		
PM2.5	PM2.5	chronic illness	0.16	0.18	0.18	0.30	0.33	2.29	1.39	1.42	5.60	0.21	0.23	2.01	2.02	3.42	0.58	0.59	2.29	8.87	8.90	20.13	7.47	7.51	16.92	0.93	0.95	3.57	
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
		RRAD	0.02	0.02	0.02	0.04	0.04	0.28	0.16	0.16	0.68	0.02	0.03	0.24	0.24	0.41	0.07	0.07	0.28	1.02	1.02	2.41	0.86	0.86	2.03	0.11	0.11	0.43	
Coarse PM10 coarse PM10	lung cancer	mortality	0.07	0.07	0.07	0.14	0.14	0.64	0.64	0.64	0.10	0.10	0.10	0.29	0.29	0.29	0.08	0.08	0.08	1.24	1.24	1.24	1.05	1.05	1.05	0.19	0.19	0.19	
		chronic illness	0.06	0.10	0.10	0.13	0.14	0.74	0.55	0.57	10.37	0.10	0.11	4.52	0.20	0.21	3.49	0.06	0.07	4.06	0.71	0.73	27.03	0.60	0.62	22.68	0.15	0.16	6.31
		asthma attacks	0.01	0.01	0.01	0.02	0.02	0.55	0.07	0.08	1.21	0.01	0.02	0.53	0.03	0.03	0.41	0.01	0.01	0.47	0.09	0.10	3.14	0.08	0.08	2.64	0.02	0.02	0.73
Total for diesel PM10 emissions	RRAD	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
		1.58	1.74	24.00	3.01	3.24	27.42	13.43	13.73	65.35	2.11	2.29	25.50	18.65	18.73	36.02	5.34	5.45	26.46	79.66	80.03	218.49	67.23	67.55	183.67	8.52	8.71	41.04	
		0.98	1.72	1.72	1.41	2.64	2.64	3.47	4.98	4.98	1.12	1.99	1.99	5.51	5.77	5.77	1.83	2.32	2.32	22.78	24.66	24.66	24.66	20.86	20.86	2.90	3.85	3.85	
SOx, NH3 sulfate PM10	chronic illness	0.11	0.22	0.22	0.16	0.37	0.37	0.41	0.63	0.93	0.13	0.26	0.26	0.66	0.66	0.66	0.21	0.27	0.27	2.71	2.98	2.98	2.27	2.52	2.52	0.34	0.46	0.46	
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
		RRAD	0.01	0.03	0.03	0.02	0.04	0.04	0.05	0.07	0.07	0.02	0.03	0.03	0.06	0.09	0.09	0.03	0.03	0.03	0.34	0.37	0.37	0.29	0.31	0.31	0.04	0.06	0.06
Total for SOx and NH3 emissions	VOCs	mortality	1.10	1.97	1.97	1.59	3.05	3.05	3.93	5.69	1.27	2.28	2.28	6.22	6.52	6.52	2.07	2.63	2.63	25.84	28.01	28.01	28.01	23.70	23.70	3.29	4.39	4.39	
		chronic illness	0.41	0.55	0.55	0.59	0.78	0.78	2.30	2.61	2.61	0.46	0.64	0.64	0.07	0.11	0.11	0.03	0.09	0.09	1.08	1.37	1.37	0.89	1.13	1.13	0.52	0.69	0.69
		asthma attacks	0.05	0.07	0.07	0.07	0.09	0.09	0.30	0.33	0.33	0.06	0.08	0.08	0.01	0.01	0.01	0.01	0.01	0.01	0.14	0.17	0.17	0.12	0.14	0.14	0.07	0.08	0.08
acetaldhyde	benzene	RRAD	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	
		oral cancer	0.00	0.00	0.00	0.01	0.01	0.01	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02
		leukemia	0.04	0.04	0.04	0.06	0.06	0.06	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
1,3-butadiene	cancer all sites	0.13	0.13	0.13	0.19	0.19	0.19	0.49	0.49	0.49	0.15	0.15	0.15	0.01	0.01	0.01	0.01	0.01	0.01	0.04	0.04	0.04	0.04	0.04	0.04	0.14	0.14	0.14	
		oral cancer	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
		fornaldehyde	0.64	0.80	0.80	0.93	1.15	1.15	3.30	3.64	3.64	0.75	0.93	0.93	0.09	0.14	0.14	0.03	0.12	0.12	1.30	1.63	1.63	1.07	1.35	1.35	0.78	0.97	0.97
Subtotal for VOC emissions	VOCs, NOx, O3	asthma attacks	0.05	0.05	0.05	0.07	0.07	0.07	0.15	0.16	0.16	0.05	0.06	0.06	0.02	0.02	0.02	0.01	0.01	0.01	0.16	0.16	0.16	0.13	0.13	0.13	0.06	0.06	0.06
		lower resp. illness	0.09	0.09	0.09	0.12	0.13	0.13	0.29	0.30	0.30	0.10	0.10	0.10	0.03	0.03	0.03	0.01	0.01	0.01	0.29	0.30	0.30	0.24	0.24	0.24	0.11	0.12	0.12
		upper resp. illness	0.03	0.03	0.03	0.04	0.04	0.04	0.09	0.09	0.09	0.03	0.03	0.03	0.01	0.01	0.01	0.01	0.01	0.01	0.09	0.09	0.09	0.07	0.07	0.07	0.03	0.04	0.04
Subtotal mortality effects	Subtotal chronic mobility effects	eye irritation	11.61	12.71	32.38	16.63	18.34	39.72	45.30	47.50	93.13	13.35	14.61	35.12	25.18	25.56	40.85	7.56	8.24	26.81	125.98	128.20	250.59	105.45	107.68	210.33	22.41	23.76	52.33
		Subtotal ambient CO	0.25	0.25	0.25	0.40	0.40	1.30	1.30	1.30	0.30	0.30	0.30	0.31	0.31	0.31	0.31	0.31	0.31	1.31	1.31	1.31	1.10	1.10	1.10	0.38	0.38	0.38	
		Subtotal ambient NO2	1.27	1.42	3.71	1.84	2.11	4.60	5.27	5.56	10.88	1.47	1.65	4.04	3.10	3.16	4.94	0.93	1.02	3.19	16.07	16.42	30.69	13.48	13.77	25.74	2.65	2.88	6.18
Subtotal ambient CO	Subtotal ambient CO	Subtotal acute mortality effects	1.47	1.50	1.79	1.98	2.02	2.34	4.92	4.94	5.65	1.68	1.96	0.72	0.73	0.96	0.22	0.24	0.51	5.07	5.13	6.93	4.22	4.27	5.78	1.90	1.94	2.35	
		Subtotal ambient PM10	11.87	13.13	35.39	17.25	19.26	43.44	47.59	50.12	101.74	13.72	15.18	38.38	28.93	29.37	46.66	8.68	9.46	30.47	144.60	147.61	286.07	121.42	123.98	240.10	24.31	25.88	58.20
		Subtotal ambient O3	0.20	0.21	0.21	0.28	0.29	0.29	0.66	0.68	0.68	0.23	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.24	0.67	0.68	0.68	0.56	0.56	0.56	0.26	0.27	0.27
Total all emissions, pollutants, effects	Total all emissions, pollutants, effects	Subtotal ambient NO2	0.55	0.56	0.56	0.76	0.77	1.73	1.75	1.75	0.62	0.63	0.63	0.15	0.15	0.15	0.08	0.08	0.08	2.40	2.41	2.41	1.99	2.00	2.00	0.75	0.76	0.76	
		Subtotal ambient CO	1.80	1.80	1.80	2.29	2.30	6.14	6.14	6.14	1.99	1.99	1.99	0.04	0.04	0.04	0.04	0.04	0.04	0.01	0.01	0.01	0.28	0.29	0.29	0.24	0.24	0.24	
		Total all emissions, pollutants, effects	14.60	15.88	38.14	20.84	22.86	47.06	56.78	59.35	110.97	16.77	18.54	41															

TABLE 11-A-41 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$1000 VMT (DENVER MSA, 1990) (LOWER BOUND)

Emission	Ambient pollutant	Health effect	IDGVs			LDGIs			RDGIs			Total gasoline vehicles			IDDVs			DDTs			HDDVs			Total diesel vehicles			Total all vehicles				
			V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD		
CO		mortality	0.27	0.27	0.27	1.92	1.92	1.92	1.70	1.70	1.70	0.37	0.37	0.37	0.01	0.01	0.01	0.02	0.02	0.02	0.08	0.08	0.08	0.06	0.06	0.06	0.35	0.35	0.35		
		hospitalization	0.04	0.04	0.04	0.30	0.30	0.30	0.27	0.27	0.27	0.06	0.06	0.06	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.06	0.06	0.06	
		headaches	0.34	0.34	0.34	2.46	2.46	2.46	2.18	2.18	2.18	0.48	0.48	0.48	0.01	0.01	0.01	0.02	0.02	0.02	0.10	0.10	0.10	0.08	0.08	0.08	0.45	0.45	0.45		
NOx		sore throat	0.14	0.14	0.14	0.84	0.84	0.84	0.47	0.47	0.47	0.19	0.19	0.19	0.09	0.09	0.09	0.12	0.12	0.12	0.82	0.82	0.82	0.63	0.64	0.64	0.21	0.21	0.21		
		excess phlegm	0.07	0.07	0.07	0.39	0.39	0.39	0.21	0.21	0.21	0.09	0.09	0.09	0.04	0.04	0.04	0.05	0.05	0.05	0.38	0.38	0.38	0.29	0.29	0.29	0.10	0.10	0.10		
		eye irritation	0.06	0.06	0.06	0.35	0.35	0.35	0.19	0.19	0.19	0.08	0.08	0.08	0.04	0.04	0.04	0.05	0.05	0.05	0.26	0.26	0.26	0.26	0.26	0.26	0.09	0.09	0.09		
NO2, NH3	nitrate PM10	mortality	1.90	1.91	1.91	11.03	11.11	11.11	6.07	6.13	6.13	2.45	2.47	2.47	1.14	1.14	1.14	1.55	1.58	1.58	10.79	10.83	10.83	8.32	8.35	8.35	2.81	2.83	2.83		
		chronic illness	0.26	0.26	0.26	1.49	1.51	1.51	0.82	0.83	0.83	0.33	0.33	0.33	0.15	0.15	0.15	0.21	0.21	0.21	1.47	1.47	1.47	1.13	1.13	1.13	0.38	0.38	0.38		
		asthma attacks	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.00	0.00		
PM2.5	nitrate PM10	RRAD	0.10	0.10	0.10	0.58	0.58	0.58	0.32	0.32	0.32	0.13	0.13	0.13	0.06	0.06	0.06	0.08	0.08	0.08	0.56	0.57	0.57	0.43	0.44	0.44	0.15	0.15	0.15		
		Total for NO2 and NH3 emissions	2.53	2.54	2.54	14.68	14.79	14.79	8.08	8.16	8.16	3.28	3.28	3.28	1.51	1.52	1.52	2.07	2.10	2.10	14.37	14.42	14.42	11.08	11.12	11.12	3.74	3.76	3.76		
		mortality	0.36	0.40	0.47	3.05	3.20	3.68	3.43	3.60	3.96	0.54	0.69	0.69	1.64	1.65	1.65	4.71	5.86	5.95	6.28	18.91	19.05	20.02	15.28	15.39	16.13	1.45	1.50	1.63	
Coarse PM10 coarse	PM10	chronic illness	0.05	0.05	0.06	0.41	0.45	0.50	0.46	0.49	0.54	0.07	0.08	0.08	0.63	0.63	0.63	0.64	0.79	0.81	0.85	2.57	2.59	2.72	2.08	2.09	2.19	0.20	0.20	0.22	
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		
		RRAD	0.02	0.02	0.02	0.16	0.17	0.19	0.18	0.19	0.21	0.03	0.03	0.04	0.24	0.24	0.24	0.25	0.31	0.33	0.33	0.99	0.99	1.04	0.80	0.80	0.84	0.08	0.08	0.08	
Coarse PM10 coarse	PM10	lung cancer	0.01	0.01	0.01	0.08	0.08	0.08	0.09	0.09	0.09	0.02	0.02	0.02	0.08	0.08	0.08	0.10	0.10	0.10	0.33	0.33	0.33	0.27	0.27	0.27	0.03	0.03	0.03		
		mortality	0.12	0.13	0.13	0.81	0.91	1.29	0.89	0.95	1.29	0.16	0.18	0.22	0.30	0.30	0.30	0.56	0.77	0.80	2.04	2.04	2.04	0.98	0.81	0.85	0.20	0.22	0.26		
		chronic illness	0.02	0.02	0.02	0.11	0.12	0.13	0.12	0.13	0.13	0.02	0.02	0.02	0.04	0.04	0.04	0.04	0.05	0.06	0.06	0.28	0.28	0.28	0.13	0.14	0.14	0.61	0.61	0.61	
Total for direct PM10 emissions	SOx, NH3 sulfate PM10	asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		
		RRAD	0.01	0.01	0.01	0.04	0.05	0.15	0.05	0.05	0.14	0.01	0.01	0.03	0.02	0.02	0.02	0.03	0.02	0.11	0.11	0.11	0.05	0.05	0.05	0.04	0.04	0.04	0.23	0.23	0.23
		mortality	0.58	0.64	1.12	4.48	5.09	7.78	5.22	5.51	8.04	0.85	0.93	1.56	5.95	5.97	6.34	7.50	7.66	10.00	23.99	24.22	31.03	19.40	19.58	24.80	1.99	2.08	2.98		
Total for SO2 and NH3 emissions	VOCs	mortality	0.45	0.70	0.70	2.85	4.60	4.60	1.77	2.97	2.97	0.60	0.94	0.94	2.54	2.64	2.64	3.68	4.48	4.48	9.90	11.09	11.09	8.04	8.98	8.98	1.05	1.43	1.43		
		chronic illness	0.06	0.10	0.10	0.39	0.44	0.44	0.24	0.42	0.42	0.08	0.13	0.13	0.34	0.36	0.36	0.50	0.61	0.61	1.35	1.52	1.52	1.09	1.23	1.23	0.14	0.20	0.20		
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
Total for SO2 and NH3 emissions	VOCs	RRAD	0.02	0.04	0.04	0.15	0.24	0.24	0.09	0.16	0.16	0.03	0.05	0.05	0.13	0.14	0.14	0.19	0.23	0.23	0.52	0.58	0.58	0.42	0.47	0.47	0.05	0.07	0.07		
		mortality	0.16	0.25	0.25	1.16	1.80	1.80	1.29	1.74	1.74	0.22	0.35	0.35	3.01	3.13	3.13	4.38	5.34	5.34	11.77	13.20	13.20	9.56	10.68	10.68	1.25	1.71	1.71		
		chronic illness	0.02	0.03	0.03	0.16	0.24	0.24	0.17	0.24	0.24	0.03	0.05	0.05	0.01	0.01	0.01	0.01	0.04	0.04	0.04	0.13	0.19	0.19	0.70	0.70	0.70	0.03	0.03	0.03	
Sub-total for VOC emissions	VOCs, NOx, O3	asthma attacks	0.01	0.01	0.01	0.07	0.08	0.08	0.05	0.06	0.06	0.01	0.02	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
		lower resp. illness	0.04	0.05	0.05	0.27	0.30	0.30	0.19	0.22	0.22	0.05	0.06	0.06	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
		upper resp. illness	0.01	0.01	0.01	0.08	0.09	0.09	0.06	0.07	0.07	0.02	0.02	0.02	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	
Subtotal mortality effects	Subtotal cancer effects	eye irritation	0.04	0.04	0.04	0.26	0.29	0.29	0.18	0.21	0.21	0.05	0.06	0.06	0.02	0.02	0.02	0.02	0.03	0.03	0.03	0.14	0.15	0.15	0.11	0.12	0.12	0.06	0.06	0.06	
		Subtotal mortality effects	3.25	3.66	4.07	20.82	23.64	25.91	15.14	17.08	19.22	4.35	4.91	5.43	8.66	8.81	9.13	11.55	12.75	14.72	41.61	43.50	49.23	33.23	34.71	39.10	6.12	6.73	7.49		
		Subtotal cancer effects	0.03	0.03	0.03	0.25	0.25	0.25	0.23	0.23	0.23	0.05	0.05	0.05	0.17	0.19	0.19	0.11	0.13	0.13	0.35	0.35	0.35	0.28	0.28	0.28	0.06	0.06	0.06		
Subtotal ambient CO3	Subtotal ambient NO2	Subtotal chronic morbidity effects	0.40	0.46	0.52	2.56	2.96	3.27	1.82	2.10	2.39	0.54	0.62	0.69	1.19	1.23	1.23	1.56	1.73	2.00	5.65	5.92	6.69	4.51	4.72	5.31	0.78	0.87	0.97		
		Subtotal acute morbidity effects	0.91	0.95	0.97	6.01	6.26	6.38	4.51	4.69	4.80	1.23	1.31	1.31	0.68	0.69	0.70	0.92	1.02	1.12	4.21	4.36	4.66	3.31	3.43	3.66	1.36	1.41	1.45		
		Subtotal ambient PM10	3.56	4.05	4.53	22.55	25.92	28.61	16.07	18.40	20.94	4.74	5.41	6.03	10.36	10.54	10.92	13.81	15.25	17.58	49.72	51.98	58.79	39.71	41.48	46.70	6.88	7.62	8.52		
Subtotal ambient CO	Subtotal ambient CO	Subtotal ambient CO3	0.10	0.11	0.11	0.67	0.76	0.76	0.46	0.55	0.55	0.14	0.15	0.15	0.04	0.04	0.04	0.04	0.04	0.04	0.06	0.09	0.09	0.35	0.40	0.40	0.15	0.16	0.16		
		Subtotal ambient NO2	0.27	0.27	0.27	1.58	1.58	1.58	0.86	0.87	0.87	0.35	0.35	0.35	0.16	0.16	0.16	0.22	0.22	0.22	1.53	1.54	1.54	1.16	1.19	1.19	0.40	0.40	0.40		
		Subtotal ambient CO	0.65	0.65	0.65	4.68	4.68	4.68	4.15	4.15	4.15	0.91	0.91	0.91	0.02	0.02	0.02	0.04	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	
Total all emissions, pollutants, effects	Total all emissions, pollutants, effects	mortality	4.60	5.10	5.59	29.64	33.11	35.80	21.70	24.11	26.65	6.16																			

TABLE 11-A-44 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$/1000 VMT (HOUSTON MSA 1990) (UPPER BOUND)

Emission pollutant	Health effect	LDGVs			LDGs			HDGs			Total gasoline vehicles			LDVs			HDVs			Total diesel vehicles			Total all vehicles					
		V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD			
CO	infectious	1.35	1.36	1.26	1.26	1.26	1.26	6.45	6.46	6.46	1.38	1.39	1.39	0.03	0.04	0.04	0.01	0.01	0.01	0.23	0.24	0.24	0.17	0.18	0.18	1.30	1.30	1.30
	hospitalization	0.05	0.05	0.05	0.05	0.05	0.05	0.26	0.26	0.26	0.05	0.05	0.05	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.05	0.05	0.05
	headaches	1.15	1.15	1.07	1.07	1.07	5.49	5.49	5.49	1.17	1.18	1.18	0.03	0.03	0.03	0.01	0.01	0.01	0.20	0.20	0.20	0.15	0.15	0.15	1.10	1.10	1.10	
NOx	sore throat	1.48	1.53	1.43	1.43	1.43	5.18	5.33	5.33	1.49	1.54	1.54	0.76	0.77	0.77	0.16	0.17	0.17	6.53	6.64	6.64	4.89	4.97	4.97	1.74	1.79	1.79	
	excess phlegm	0.65	0.68	0.63	0.63	0.63	2.28	2.35	2.35	0.66	0.68	0.68	0.34	0.34	0.34	0.07	0.08	0.08	2.88	2.93	2.93	2.19	2.19	2.19	0.77	0.79	0.79	
	eye irritation	0.60	0.62	0.56	0.56	0.56	2.09	2.15	2.15	0.60	0.62	0.62	0.31	0.31	0.31	0.06	0.07	0.07	2.64	2.68	2.68	1.97	2.01	2.01	0.70	0.72	0.72	
NOx, NH3 nitrate PM10	infectious	23.17	23.96	22.44	22.44	22.44	80.86	82.29	82.29	23.35	24.18	24.18	11.90	12.10	12.10	2.49	2.75	2.75	102.14	103.84	103.84	76.43	77.72	77.72	27.21	28.08	28.08	
	chronic illness	16.68	17.21	15.46	15.46	15.46	57.93	59.53	59.53	16.80	17.36	17.36	8.57	8.70	8.70	1.79	1.96	1.96	72.98	74.09	74.09	54.62	55.47	55.47	19.56	20.14	20.14	
	asthma attacks	0.01	0.01	0.01	0.01	0.01	0.04	0.04	0.04	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.00	0.00	0.05	0.05	0.05	0.04	0.04	0.04	0.01	0.01	0.01	
Total for NOx and NH3 emissions	RRAD	0.66	0.68	0.61	0.61	0.61	2.37	2.37	2.37	0.66	0.69	0.69	0.34	0.34	0.34	0.07	0.08	0.08	2.91	2.95	2.95	2.17	2.21	2.21	0.77	0.80	0.80	
	infectious	43.25	44.68	40.17	40.17	40.17	150.68	155.05	155.05	43.58	45.08	45.08	22.22	22.57	22.57	4.65	5.11	5.11	190.13	193.17	193.17	142.27	144.60	144.60	50.77	52.33	52.33	
	chronic illness	4.16	5.71	32.86	32.86	32.86	41.46	41.46	41.46	4.82	6.44	6.44	33.29	33.29	33.29	53.30	53.30	53.30	192.49	196.60	196.60	149.13	152.29	152.29	15.36	17.08	17.08	
PM2.5	chronic illness	3.00	4.01	22.61	22.61	22.61	37.78	37.78	37.78	3.46	4.53	4.53	38.50	38.50	38.50	53.28	53.28	53.28	140.17	140.17	140.17	106.58	108.64	108.64	236.21	11.01	12.13	
	asthma attacks	0.00	0.00	0.00	0.00	0.00	0.02	0.02	0.02	0.00	0.00	0.00	0.02	0.02	0.02	0.03	0.03	0.03	0.09	0.09	0.09	0.20	0.20	0.20	0.07	0.07	0.07	
	RRAD	0.12	0.16	0.99	0.99	0.99	1.04	1.18	1.18	0.14	0.18	0.18	1.52	1.53	1.53	2.13	2.13	2.13	5.48	5.59	5.59	12.57	12.57	12.57	4.24	4.33	4.33	
Coarse PM10 coarse PM10	lung cancer	0.29	0.29	0.37	0.37	0.37	2.65	2.65	2.65	0.34	0.34	0.34	1.70	1.70	1.70	0.33	0.33	0.33	6.43	6.43	6.43	4.97	4.97	4.97	0.68	0.68	0.68	
	infectious	0.26	0.31	7.62	7.62	7.62	1.90	2.04	2.04	2.29	2.34	2.34	7.57	7.57	7.57	0.69	0.69	0.69	2.00	2.13	2.13	68.89	1.57	1.57	51.82	0.38	0.43	
	chronic illness	0.19	0.22	5.21	5.21	5.21	1.36	1.46	1.46	1.82	1.82	1.82	5.17	5.17	5.17	0.49	0.49	0.49	1.43	1.51	1.51	47.02	1.12	1.12	35.37	0.27	0.31	
Total for direct PM10 emissions	asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
	RRAD	0.01	0.01	0.21	0.21	0.21	0.19	0.05	0.06	0.76	0.01	0.01	0.21	0.02	0.02	0.18	0.00	0.00	0.16	0.06	0.06	1.94	0.05	1.46	0.01	0.30	0.30	
	infectious	8.04	10.73	69.75	69.75	69.75	10.05	12.90	12.90	64.07	70.02	70.02	78.34	78.34	78.34	96.30	97.21	97.21	345.47	352.59	352.59	891.36	267.74	273.20	677.94	28.15	31.14	
SOx, NH3 sulfate PM10	chronic illness	2.24	4.84	2.12	4.88	4.88	8.20	16.17	16.17	2.26	4.98	4.98	12.61	13.74	13.74	2.74	4.06	4.06	43.36	51.69	51.69	33.74	40.14	40.14	4.57	7.54	7.54	
	asthma attacks	1.62	3.28	1.53	3.28	3.28	5.88	10.97	10.97	1.66	3.36	3.36	9.09	9.81	9.81	1.97	2.83	2.83	30.92	36.26	36.26	24.08	28.18	28.18	3.28	5.18	5.18	
	RRAD	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.02	0.02	0.02	0.02	0.02	0.00	0.00	0.00	
Total for SOx and NH3 emissions	oral cancer	0.02	0.02	0.02	0.02	0.02	0.08	0.08	0.08	0.02	0.02	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.05	0.06	0.06	
	leukemia	0.15	0.15	0.15	0.15	0.15	0.69	0.69	0.69	0.15	0.15	0.15	0.53	0.53	0.53	0.04	0.04	0.04	0.05	0.15	0.15	0.15	0.12	0.12	0.12	0.15	0.15	0.15
	cancer-all sites	0.01	0.01	0.01	0.01	0.01	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03
Subtotal for VOC emissions	oral cancer	3.92	8.25	3.71	8.30	8.30	14.31	27.61	27.61	3.98	8.48	8.48	22.06	23.94	23.94	4.79	7.03	7.03	75.53	89.44	89.44	58.79	69.47	69.47	7.98	12.93	12.93	
	infectious	1.49	1.77	1.77	1.77	1.77	10.47	11.36	11.36	1.57	1.87	1.87	12.61	13.74	13.74	0.07	0.16	0.16	3.50	4.29	4.29	2.60	3.20	3.20	1.65	1.97	1.97	
	chronic illness	1.07	1.27	1.27	1.27	1.27	7.55	8.16	8.16	1.13	1.34	1.34	0.19	0.25	0.25	0.05	0.11	0.11	2.51	3.06	3.06	1.86	2.28	2.28	1.19	1.41	1.41	
VOCs, NOx, O3	asthma attacks	0.04	0.05	0.05	0.05	0.05	0.30	0.32	0.32	0.04	0.05	0.05	0.01	0.01	0.01	0.00	0.00	0.00	0.10	0.12	0.12	0.07	0.09	0.09	0.05	0.06	0.06	
	upper resp. illness	0.20	0.21	0.19	0.20	0.20	0.79	0.90	0.90	0.20	0.21	0.21	0.06	0.07	0.07	0.01	0.02	0.02	0.52	0.54	0.54	0.39	0.41	0.41	0.22	0.23	0.23	
	eye irritation	0.34	0.36	0.36	0.36	0.36	1.37	1.55	1.55	0.34	0.36	0.36	0.11	0.11	0.11	0.02	0.03	0.03	0.88	0.93	0.93	0.66	0.70	0.70	0.36	0.39	0.39	
Subtotal mortality effects	asthma attacks	32.68	37.96	72.41	72.41	72.41	31.89	34.54	34.54	67.42	74.54	74.54	160.81	168.15	168.15	278.15	278.15	278.15	343.73	358.80	358.80	673.25	263.64	275.20	511.42	50.46	54.40	
	upper resp. illness	0.97	0.97	0.97	0.97	0.97	1.04	1.04	1.04	1.04	1.04	1.04	1.04	1.04	1.04	1.04	1.04	1.04	1.04	1.04	1.04	1.04	1.04	1.04	1.04	1.34	1.34	1.34
	eye irritation	21.57	25.99	49.57	49.57	49.57	21.99	25.65	25.65	46.10	50.93	50.93	109.57	109.57	109.57	189.81	189.81	189.81	245.33	255.06	255.06	470.40	188.26	195.75	357.51	35.31	39.16	
Subtotal chronic morbidity effects	asthma attacks	5.87	6.16	7.15	7.15	7.15	5.84	6.70	6.70	24.65	28.01	28.01	5.96	6.27	6.27	4.04	4.14	4.14	1.68	2.48	2.48	25.60	34.60	34.60	18.79	19.36	19.36	
	upper resp. illness	55.09	63.96	122.96	122.96	122.96	53.89	60.35	60.35	114.53	124.78	124.78	246.78	271.02	271.02	471.97	471.97	471.97	568.83	660.55	660.55	124.46	139.63	142.90	188.77	27.43	31.89	
	eye irritation	1.02	1.08	1.08	1.08	1.08	0.97	1.04	1.04	4.08	4.63	4.63	1.04	1.11	1.11	0.33	0.34	0.34	0.34	0.34	0.34	0.34	0.34	0.34	0.34	0.34	0.34	0.34
Subtotal ambient NO2	asthma attacks	2.73	2.82	2.82	2.82	2.82	2.54	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64	2.64
	upper resp. illness	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56
	eye irritation	62.08	71.08	130.11	130.11	130.11	60.45	70.08	70.08	121.26	137.33	137.33	141.50	144.81	144.81	190.67	196.82	196.82	318.89	366.15	366.15	1184.92	475.84	495.46	900.21	94.00	104.13	
Total all emissions, pollutants, effects	asthma attacks	62.08	71.08	130.11	130.11	130.11	60.45	70.08	70.08	121.26	137.33	137.33	141.50	144.81	144.81	190.67	196.82	196.82	318.89	366.15	366.15	1184.92	475.84	495.46	900.21	94.00	104.13	
	upper resp. illness	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56	2.56
	eye irritation	62.08	71.08	130.11	130.11	130.11	60.45	70.08	70.08	121.26	137.33	137.33																

TABLE 11-A-46 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$71,000 VMT (LOS ANGELES MSA 1990) (UPPER BOUND)

Emission	Ambient pollutant	Health effect	LDGVs			LDGVs			HDGVs			Total gasoline vehicles			HDDVs			Total diesel vehicles			Total all vehicles					
			V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD			
CO	CO	mortality	3.54	3.54	3.54	9.09	9.09	9.09	20.78	20.78	20.78	4.37	4.37	4.37	0.07	0.07	0.07	0.81	0.81	0.81	0.66	0.66	0.66	4.10	4.10	4.10
			0.14	0.14	0.14	0.36	0.36	0.36	0.82	0.82	0.82	0.17	0.17	0.17	0.00	0.00	0.00	0.03	0.03	0.03	0.03	0.03	0.03	0.16	0.16	0.16
NOx	NOx	headaches	2.23	2.23	2.23	5.72	5.72	5.72	13.09	13.09	13.09	2.75	2.75	2.75	0.05	0.05	0.05	0.51	0.51	0.51	0.41	0.41	0.41	2.56	2.56	2.56
			0.08	0.08	0.08	0.29	0.29	0.29	0.78	0.78	0.78	0.19	0.19	0.19	0.00	0.00	0.00	0.08	0.08	0.08	0.08	0.08	0.08	0.16	0.16	0.16
NOx, NH3	nitrate PM10	excess phlegm	1.29	1.29	1.29	3.47	3.47	3.47	6.10	6.10	6.10	1.59	1.59	1.59	0.36	0.36	0.36	0.74	0.74	0.74	0.56	0.56	0.56	4.52	4.52	4.52
			0.12	0.12	0.12	0.32	0.32	0.32	0.72	0.72	0.72	0.19	0.19	0.19	0.00	0.00	0.00	0.08	0.08	0.08	0.08	0.08	0.08	0.16	0.16	0.16
NOx, NH3	nitrate PM10	eye irritation	1.20	1.21	1.21	3.25	3.26	3.26	5.72	5.72	5.72	1.49	1.49	1.49	0.36	0.36	0.36	0.74	0.74	0.74	0.56	0.56	0.56	4.52	4.53	4.53
			0.08	0.08	0.08	0.29	0.29	0.29	0.78	0.78	0.78	0.19	0.19	0.19	0.00	0.00	0.00	0.08	0.08	0.08	0.08	0.08	0.08	0.16	0.16	0.16
NOx, NH3	nitrate PM10	chronic illness	91.53	91.76	91.76	247.51	248.22	248.22	436.81	436.81	436.81	113.01	113.30	113.30	46.72	46.87	46.87	539.66	540.86	540.86	425.84	425.84	425.84	135.34	135.67	135.67
			65.39	65.54	65.54	176.41	176.88	176.88	310.62	310.62	310.62	80.78	80.98	80.98	33.31	33.41	33.41	19.24	19.24	19.24	362.78	364.58	364.58	96.77	97.00	97.00
NOx, NH3	nitrate PM10	asthma attacks	0.04	0.04	0.04	0.10	0.10	0.10	0.18	0.18	0.18	0.05	0.05	0.05	0.02	0.02	0.02	0.01	0.01	0.01	0.02	0.02	0.02	0.06	0.06	0.06
			0.04	0.04	0.04	0.10	0.10	0.10	0.18	0.18	0.18	0.05	0.05	0.05	0.02	0.02	0.02	0.01	0.01	0.01	0.02	0.02	0.02	0.06	0.06	0.06
Total for NOx and NH3 emissions	PM2.5	RRAD	1.96	1.97	1.97	5.31	5.33	5.33	9.36	9.36	9.36	2.42	2.43	2.43	1.00	1.01	1.01	1.01	1.01	1.01	1.01	1.01	1.01	2.90	2.90	2.90
			164.48	164.88	164.88	444.34	445.58	445.58	782.00	783.39	783.39	203.12	203.62	203.62	83.90	84.16	84.16	48.49	49.35	49.35	967.75	969.85	969.85	243.26	243.85	243.85
PM2.5	PM2.5	mortality	19.39	20.86	20.86	71.02	76.40	76.40	195.68	197.70	197.70	27.49	29.18	29.18	250.01	251.10	251.10	295.99	299.99	299.99	140.16	142.84	142.84	85.45	87.18	87.18
			13.83	14.89	14.89	52.03	54.47	54.47	139.19	140.63	140.63	305.21	306.21	306.21	19.60	20.82	20.82	178.29	179.06	179.06	211.00	211.00	211.00	99.85	101.79	101.79
PM2.5	PM2.5	chronic illness	0.01	0.01	0.01	0.03	0.03	0.03	0.08	0.08	0.08	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
			0.01	0.01	0.01	0.03	0.03	0.03	0.08	0.08	0.08	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
PM2.5	PM2.5	asthma attacks	0.42	0.45	0.45	1.57	1.64	1.64	4.85	4.94	4.94	9.20	9.29	9.29	0.63	0.63	0.63	3.01	3.07	3.07	5.86	5.94	5.94	17.78	17.84	17.84
			0.09	0.09	0.09	0.36	0.36	0.36	0.95	0.95	0.95	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13
Coarse PM10 coarse PM10	Coarse PM10 coarse PM10	mortality	1.25	1.80	1.80	15.89	17.22	17.22	41.65	45.12	45.12	10.12	10.77	10.77	67.21	72.11	72.11	165.22	177.96	177.96	140.16	142.84	142.84	85.45	87.18	87.18
			0.89	1.29	1.29	7.11	7.77	7.77	16.63	18.00	18.00	4.48	4.81	4.81	29.78	31.30	31.30	19.60	20.82	20.82	178.29	179.06	179.06	99.85	101.79	101.79
Coarse PM10 coarse PM10	Coarse PM10 coarse PM10	chronic illness	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
			0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Coarse PM10 coarse PM10	Coarse PM10 coarse PM10	asthma attacks	0.03	0.04	0.04	0.34	0.36	0.36	0.94	0.97	0.97	0.28	0.29	0.29	0.12	0.12	0.12	0.54	0.57	0.57	1.28	1.28	1.28	4.00	4.00	4.00
			0.03	0.04	0.04	0.34	0.36	0.36	0.94	0.97	0.97	0.28	0.29	0.29	0.12	0.12	0.12	0.54	0.57	0.57	1.28	1.28	1.28	4.00	4.00	4.00
Total for direct PM10 emissions	SOx, NH3	sulfate PM10	35.91	39.43	39.43	133.75	141.79	141.79	357.63	382.29	382.29	86.115	90.88	90.88	442.49	459.24	459.24	509.24	539.24	539.24	246.43	252.84	252.84	152.25	156.39	156.39
			10.51	10.87	10.87	36.69	38.69	38.69	93.77	95.02	95.02	45.02	45.02	45.02	288.58	293.22	293.22	38.91	39.91	39.91	228.43	230.32	230.32	188.24	189.78	189.78
SOx, NH3	sulfate PM10	chronic illness	7.50	7.74	7.74	21.07	21.81	21.81	31.19	31.96	31.96	9.61	9.61	9.61	42.16	42.31	42.31	27.16	27.68	27.68	68.33	69.33	69.33	25.70	26.23	26.23
			0.00	0.00	0.00	0.01	0.01	0.01	0.02	0.02	0.02	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
SOx, NH3	sulfate PM10	asthma attacks	0.23	0.23	0.23	0.63	0.66	0.66	0.94	0.97	0.97	0.28	0.29	0.29	1.27	1.28	1.28	0.82	0.84	0.84	2.18	2.18	2.18	4.00	4.00	4.00
			0.23	0.23	0.23	0.63	0.66	0.66	0.94	0.97	0.97	0.28	0.29	0.29	1.27	1.28	1.28	0.82	0.84	0.84	2.18	2.18	2.18	4.00	4.00	4.00
Total for SOx and NH3 emissions	VOCs	mortality	10.24	18.85	18.85	51.30	53.18	53.18	94.91	99.91	99.91	22.65	23.41	23.41	102.57	102.57	102.57	102.57	102.57	102.57	66.13	67.43	67.43	44.55	45.45	45.45
			5.96	6.13	6.13	17.22	17.76	17.76	53.38	54.00	54.00	7.78	8.00	8.00	88.00	88.00	88.00	90.00	90.00	90.00	46.00	46.00	46.00	8.26	8.48	8.48
VOCs	(SOA)	chronic illness	4.25	4.38	4.38	12.26	12.66	12.66	38.02	38.47	38.47	5.55	5.71	5.71	6.63	6.64	6.64	3.33	3.39	3.39	13.34	13.34	13.34	15.70	15.70	15.70
			0.00	0.00	0.00	0.01	0.01	0.01	0.02	0.02	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
VOCs	(SOA)	asthma attacks	0.13	0.13	0.13	0.37	0.38	0.38	1.15	1.16	1.16	0.17	0.17	0.17	0.02	0.02	0.02	0.01	0.01	0.01	0.02	0.02	0.02	0.01	0.01	0.01
			0.01	0.01	0.01	0.02	0.02	0.02	0.03	0.03	0.03	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
VOCs	acetalddehyde	leukemia	0.05	0.05	0.05	0.14	0.14	0.14	0.28	0.28	0.28	0.06	0.06	0.06	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
			0.01	0.01	0.01	0.02	0.02	0.02	0.03	0.03	0.03	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
VOCs	1,3-butadiene	cancer all sites	0.16	0.16	0.16	0.47	0.47	0.47	0.94	0.94	0.94	0.21	0.21	0.21	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
			0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
VOCs	formaldehyde	oral cancer	10.56	10.86	10.86	30.50	31.45	31.45	93.83	94.91	94.91	24.91	25.74	25.74	360.05	361.96	361.96	417.80	417.80	417.80	66.13	67.43	67.43	44.55	45.45	45.45
			0.35	0.36	0.36	1.00																				

TABLE 11-A 47 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$/1000 VMT (MINNEAPOLIS MSA 1990) (LOWER BOUND)

Emission pollutant	Health effect	LDGVs		LDGs		HDGts		Total gasoline vehicles		LDDVs		HDDVs		Total diesel vehicles		Total all vehicles			
		V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U	V	V+U		
CO	mortality	0.19	0.19	2.26	2.30	1.24	1.26	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.25	0.25
		0.03	0.03	0.36	0.36	0.20	0.20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.04	0.04
NOx	headaches	0.21	0.22	2.30	2.54	1.37	1.39	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.27	0.28
		0.24	0.24	2.78	2.83	1.35	1.35	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.36	0.36
NOx, NH3	excess phlegm	0.11	0.11	1.25	1.27	0.59	0.61	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.14	0.16
		0.10	0.10	1.13	1.15	0.55	0.55	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.16	0.16
NOx, NH3	eye irritation	0.11	0.11	1.25	1.27	0.59	0.61	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.14	0.16
		0.10	0.10	1.13	1.15	0.55	0.55	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.16	0.16
Total for NOx and NH3 Emissions	mortality	2.47	2.51	28.72	29.27	13.99	13.87	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	3.68	3.74
		0.90	0.91	5.83	5.93	2.75	2.80	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.75	0.76
PM2.5	chronic illness	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Total for coarse PM10 emissions	asthma attacks	0.13	0.13	1.49	1.52	0.71	0.72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.19	0.19
		0.01	0.01	0.15	0.15	0.12	0.12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.19	0.19
Coarse PM10 coarse PM10	lung cancer	0.01	0.01	0.15	0.15	0.12	0.12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.03	0.03
		0.14	0.20	1.85	2.71	1.20	1.37	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.32	0.32
Total for direct PM10 emissions	mortality	0.08	0.13	1.37	1.95	2.30	1.07	1.45	1.69	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1.57	1.95
		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.32	0.40
SOx, NH3	asthma attacks	0.01	0.01	0.05	0.10	0.14	0.63	0.07	0.10	0.44	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.00
		0.01	0.01	0.05	0.10	0.14	0.63	0.07	0.10	0.44	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.00
Total for SOx and NH3 emissions	mortality	0.66	1.05	2.29	10.94	15.65	29.47	8.49	11.44	21.18	1.04	1.54	3.20	7.23	7.40	8.36	17.51	37.08	2.30
		0.52	1.16	6.36	15.39	22.97	34.55	9.49	11.44	21.18	1.04	1.54	3.20	7.23	7.40	8.36	17.51	37.08	2.30
VOCs	chronic illness	0.04	0.07	0.54	0.96	0.37	0.58	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.30	0.51
		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Subtotal for VOC emissions	asthma attacks	0.01	0.02	0.14	0.24	0.09	0.15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
VOCs, NOx, O3	oral cancer	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Subtotal for VOC emissions	leukemia	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
VOCs, NOx, O3	cancer all sites	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Subtotal for VOC emissions	oral cancer	0.28	0.46	3.61	6.07	6.07	2.44	3.69	3.69	3.69	3.69	3.69	3.69	3.69	3.69	3.69	3.69	1.87	1.87
		0.01	0.01	0.06	0.07	0.07	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.01	0.01
VOCs, NOx, O3	lower resp. illness	0.02	0.02	0.24	0.28	0.08	0.12	0.17	0.17	0.17	0.17	0.17	0.17	0.17	0.17	0.17	0.17	0.02	0.02
		0.01	0.01	0.07	0.08	0.04	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.01	0.01
Subtotal for VOC emissions	upper resp. illness	0.01	0.02	0.17	0.20	0.09	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.02	0.02
		0.01	0.02	0.17	0.20	0.09	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.02	0.02
Subtotal for VOC emissions	eye irritation	3.94	5.04	6.03	63.89	74.90	26.07	41.26	42.03	5.37	6.94	8.26	9.96	10.40	11.16	25.58	32.31	41.87	65.19
		0.76	0.98	1.18	9.41	12.45	14.68	5.03	6.67	8.24	1.35	1.62	2.03	2.11	2.27	5.19	6.53	8.47	12.20
Subtotal for VOC emissions	Subtotal cancer effects	0.93	1.00	1.05	11.01	12.06	12.65	5.62	6.24	6.65	1.35	1.43	0.78	0.81	0.85	2.08	2.47	2.98	6.36
		0.76	0.98	1.18	9.41	12.45	14.68	5.03	6.67	8.24	1.35	1.62	2.03	2.11	2.27	5.19	6.53	8.47	12.20
Subtotal for VOC emissions	Subtotal acute mortality effects	4.71	6.09	7.33	58.33	77.46	31.30	41.57	51.31	6.44	8.39	10.05	12.59	13.13	14.10	32.31	41.87	76.34	87.66
		0.04	0.05	0.05	0.54	0.63	0.63	0.28	0.38	0.38	0.38	0.38	0.38	0.38	0.38	0.38	0.38	0.38	0.38
Subtotal for VOC emissions	Subtotal ambient O3	0.44	0.45	0.45	5.16	5.26	2.46	2.51	2.51	0.96	0.96	0.96	0.96	0.96	0.96	0.96	0.96	2.95	2.95
		0.43	0.44	0.44	5.12	5.21	2.40	2.45	2.45	0.95	0.95	0.95	0.95	0.95	0.95	0.95	0.95	2.92	2.92
Subtotal for VOC emissions	Subtotal ambient CO	5.65	7.05	8.29	69.43	88.84	32.66	38.99	47.46	57.20	7.70	9.68	12.85	13.40	14.36	35.01	41.54	53.55	79.55
		0.43	0.44	0.44	5.12	5.21	2.40	2.45	2.45	0.95	0.95	0.95	0.95	0.95	0.95	0.95	0.95	0.95	0.95
Total all emissions, pollutants, effects	mortality	14.81	21.37	27.40	228.43	272.00	103.46	128.55	154.34	194.43	24.43	30.97	38.99	47.46	57.20	117.11	147.11	184.43	228.43
		14.81	21.37	27.40	228.43	272.00	103.46	128.55	154.34	194.43	24.43	30.97	38.99	47.46	57.20	117.11	147.11	184.43	228.43

TABLE 11-A-51 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$/1000 VMT (PHILADELPHIA MSA 1990) (LOWER BOUND)

Emission	Ambient pollutant	Health effect	LDGVs		LDGs		HDCGs		Total gasoline vehicles		LDDVs		LDDs		HDDVs		Total diesel vehicles		Total all vehicles			
			V	V+U+RD	V	V+U+RD	V	V+U+RD	V	V+U+RD	V	V+U+RD	V	V+U+RD	V	V+U+RD	V	V+U+RD	V	V+U+RD	V	V+U+RD
CO	CO	mortality	0.27	0.28	0.39	0.39	1.23	1.24	1.24	1.24	0.31	0.31	0.01	0.01	0.00	0.00	0.05	0.06	0.04	0.04	0.29	0.29
		hospitalization	0.04	0.04	0.06	0.06	0.19	0.20	0.20	0.20	0.05	0.05	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.05	0.05
		headaches	0.25	0.25	0.35	0.36	1.13	1.14	1.14	1.14	0.28	0.28	0.01	0.01	0.00	0.00	0.05	0.05	0.04	0.04	0.26	0.27
NOx	NO2	sores throat	0.39	0.41	0.56	0.59	1.35	1.40	1.40	1.40	0.44	0.45	0.23	0.23	0.08	0.08	1.74	1.77	1.30	1.33	0.49	0.51
		excess phlegm	0.17	0.18	0.25	0.26	0.60	0.62	0.62	0.62	0.19	0.20	0.10	0.10	0.03	0.04	0.77	0.78	0.57	0.59	0.22	0.22
		eye irritation	0.16	0.16	0.23	0.24	0.55	0.57	0.57	0.57	0.18	0.18	0.09	0.09	0.03	0.03	0.70	0.72	0.72	0.72	0.20	0.21
NOx, NH3	nitrate PM10	mortality	3.06	3.16	4.38	4.55	10.53	10.92	10.92	10.92	3.40	3.51	1.76	1.78	0.99	0.63	13.57	13.82	10.16	10.35	3.84	3.96
		chronic illness	0.64	0.66	0.91	0.94	2.17	2.25	2.25	2.25	0.71	0.73	0.37	0.37	0.12	0.13	2.79	2.84	2.09	2.12	0.60	0.62
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.00	0.00
Total for NOx and NH3 emissions	PM2.5	RRAD	1.58	1.58	2.22	2.22	5.51	5.53	5.53	5.53	1.17	1.17	0.09	0.09	0.03	0.03	6.66	6.67	5.01	5.01	0.19	0.19
		mortality	0.58	0.58	0.82	0.82	2.02	2.02	2.02	2.02	0.63	0.63	0.01	0.01	0.09	0.09	20.24	20.61	15.15	15.43	5.74	5.92
		chronic illness	0.12	0.12	0.17	0.17	0.44	0.44	0.44	0.44	0.15	0.15	0.75	0.75	0.27	0.27	24.48	24.95	19.21	20.32	1.96	2.40
Causes PM10 coarse PM10	PM2.5	asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.97	5.27	3.91	4.13	0.40	0.52
		RRAD	0.03	0.04	0.06	0.06	0.09	0.09	0.09	0.09	0.04	0.04	0.37	0.37	0.11	0.12	1.20	1.27	0.94	0.99	1.03	1.12
		lung cancer	0.01	0.01	0.02	0.02	0.08	0.08	0.08	0.08	0.01	0.01	0.09	0.09	0.03	0.03	0.28	0.28	0.22	0.22	0.03	0.03
Causes PM10 coarse PM10	PM2.5	mortality	0.19	0.23	0.32	0.36	0.91	1.15	1.32	2.99	0.22	0.27	0.48	0.50	0.80	0.14	1.27	1.53	1.02	1.21	0.22	0.33
		chronic illness	0.04	0.05	0.13	0.13	0.19	0.23	0.27	0.61	0.05	0.06	0.10	0.10	0.16	0.03	0.04	0.13	0.26	0.31	0.25	0.93
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Total for direct PM10 emissions	SOx, NH3	RRAD	0.01	0.01	0.02	0.02	0.04	0.06	0.15	0.15	0.01	0.01	0.02	0.02	0.04	0.01	0.06	0.08	0.29	0.05	0.06	0.22
		mortality	0.98	1.45	2.05	1.89	2.72	3.52	7.08	9.06	1.22	1.78	10.12	10.29	10.75	3.05	32.54	34.71	41.49	25.57	32.24	2.83
		chronic illness	1.02	1.14	1.54	1.76	2.31	3.21	4.44	5.78	1.13	1.13	7.51	7.69	7.69	1.99	17.94	29.68	14.08	23.09	2.00	5.96
Total for SOx and NH3 emissions	VOCs	asthma attacks	0.21	0.26	0.36	0.41	1.41	1.66	3.15	3.15	0.24	0.24	0.99	0.99	1.19	1.45	3.62	6.09	2.86	4.74	0.41	1.24
		chronic illness	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.02	0.01	0.02	0.00	0.00
		asthma attacks	0.05	0.20	0.20	0.20	0.33	0.33	0.16	0.74	0.06	0.23	0.28	0.34	0.34	0.10	0.21	0.87	1.45	0.69	1.13	0.10
Subtotal for VOC emissions	VOCs, NOx, O3	RRAD	1.28	5.20	5.20	1.94	8.53	6.53	19.05	19.05	1.44	6.00	7.21	8.79	8.79	2.50	22.34	37.24	17.64	28.97	2.51	7.52
		mortality	0.26	0.46	0.46	0.76	1.56	2.31	2.31	4.47	0.31	0.54	0.05	0.12	0.12	0.12	0.61	1.43	0.45	1.07	1.07	0.32
		chronic illness	0.05	0.10	0.10	0.16	0.32	0.47	0.47	0.61	0.11	0.11	0.01	0.03	0.03	0.03	0.12	0.30	0.09	0.22	0.22	0.07
Subtotal cancer effects	Subtotal cancer effects	asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		RRAD	0.01	0.02	0.02	0.04	0.04	0.08	0.11	0.11	0.01	0.03	0.00	0.01	0.01	0.01	0.03	0.07	0.07	0.02	0.05	0.02
		oral cancer	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Subtotal ambient O3	Subtotal ambient O3	leukemia	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
		cancer all sites	0.02	0.02	0.03	0.03	0.08	0.08	0.08	0.08	0.02	0.02	0.00	0.00	0.00	0.00	0.01	0.01	0.00	0.00	0.01	0.01
		oral cancer	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Subtotal ambient NO2	Subtotal ambient NO2	formaldehyde	0.36	0.61	0.61	1.00	1.00	2.07	3.01	3.01	0.41	0.71	0.07	0.16	0.16	0.77	1.81	1.81	0.57	1.35	0.42	0.75
		asthma attacks	0.01	0.02	0.02	0.02	0.05	0.07	0.07	0.07	0.02	0.02	0.01	0.01	0.01	0.01	0.04	0.04	0.03	0.03	0.03	0.02
		lower resp illness	0.05	0.06	0.06	0.10	0.10	0.21	0.28	0.28	0.06	0.07	0.02	0.02	0.02	0.01	0.14	0.17	0.11	0.13	0.13	0.06
Subtotal all emissions, pollutants, effects	Subtotal all emissions, pollutants, effects	upper resp illness	0.02	0.02	0.02	0.03	0.03	0.06	0.09	0.09	0.02	0.02	0.01	0.01	0.01	0.01	0.04	0.05	0.03	0.04	0.04	0.02
		eye irritation	0.06	0.07	0.07	0.09	0.11	0.11	0.24	0.32	0.07	0.08	0.02	0.03	0.03	0.01	0.16	0.19	0.12	0.14	0.14	0.07
		mortality	5.39	9.19	9.66	8.22	14.63	15.27	22.12	36.73	6.12	10.53	15.55	17.04	17.40	5.01	78.82	77.88	44.95	56.07	60.10	13.55
Subtotal cancer effects	Subtotal cancer effects	chronic illness	0.04	0.04	0.06	0.06	0.19	0.19	0.19	0.19	0.04	0.04	0.09	0.09	0.03	0.03	0.29	0.29	0.23	0.23	0.05	0.05
		asthma attacks	1.06	1.85	1.95	1.62	2.95	3.09	4.28	7.93	1.20	2.12	3.21	3.52	3.40	1.62	11.76	11.76	11.46	12.28	1.73	2.90
		mortality	1.42	1.66	1.68	2.06	2.46	2.49	5.43	6.44	1.26	1.66	1.25	1.34	1.36	0.41	6.52	7.38	4.96	5.62	5.82	1.81
Subtotal ambient PM10	Subtotal ambient PM10	asthma attacks	6.45	11.21	11.81	9.86	17.92	18.72	26.29	44.65	7.32	12.87	19.61	21.48	21.94	6.31	72.57	91.09	56.52	70.49	75.53	16.68
		chronic illness	0.14	0.17	0.17	0.22	0.26	0.26	0.57	0.76	0.16	0.19	0.05	0.06	0.06	0.02	0.38	0.46	0.29	0.34	0.34	0.17
		asthma attacks	0.73	0.75	0.75	1.04	1.08	1.08	2.49	2.59	0.81	0.83	0.42	0.42	0.42	0.14	3.21	3.27	2.40	2.45	2.45	0.94
Subtotal ambient CO	Subtotal ambient CO	mortality	0.57	0.57	0.57	0.80	0.81	2.56	2.57	2.57	0.63	0.64	0.02	0.02	0.02	0.11	0.11	0.12	0.12	0.08	0.09	0.60
		chronic illness	2.91	12.73	13.32	11.96	20.11	20.91	32.03	50.48	8.95	14.56	20.10	21.99	22.45	6.48	76.38	94.95	101.73	59.30	73.38	12.28
		asthma attacks	1.28	1.45	1.45	1.94	2.12	2.12	5.21	5.21	1.52	1.52	1.52	1.52	1.52	0.41	10.04	10.73	7.83	78.43	12.28	18.45

TABLE 11-A-54 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$/1000 VMT (PHOENIX MSA 1990) (UPPER BOUND)

Emission pollutant	Health effect	LDGVs			LDGIs			HDGIs			Total gasoline vehicles			LDDVs			LDDIs			HDDVs			Total diesel vehicles			Total all vehicles		
		V	V-UI-RD	V-UI-ARD	V	V-UI-RD	V-UI-ARD	V	V-UI-RD	V-UI-ARD	V	V-UI-RD	V-UI-ARD	V	V-UI-RD	V-UI-ARD	V	V-UI-RD	V-UI-ARD	V	V-UI-RD	V-UI-ARD	V	V-UI-RD	V-UI-ARD	V	V-UI-RD	V-UI-ARD
CO	mortality	5.41	5.41	3.19	3.19	3.19	3.19	26.58	26.58	26.58	4.91	4.91	4.91	0.20	0.20	0.20	0.03	0.03	0.03	0.99	0.99	0.99	0.78	0.78	0.78	4.55	4.55	4.55
		0.21	0.21	0.21	0.13	0.13	0.13	1.05	1.05	1.05	0.19	0.19	0.19	0.01	0.01	0.01	0.00	0.00	0.00	0.04	0.04	0.04	0.03	0.03	0.03	0.18	0.18	0.18
NOx	respiratory effects	2.65	2.65	1.56	1.56	1.56	1.56	13.03	13.03	13.03	2.40	2.40	2.40	0.10	0.10	0.10	0.01	0.01	0.01	0.04	0.04	0.04	0.38	0.38	0.38	2.22	2.22	2.22
		2.40	2.40	1.48	1.48	1.48	1.48	6.22	6.22	6.22	2.12	2.12	2.12	1.37	1.37	1.37	0.19	0.19	0.19	0.93	0.93	0.93	0.69	0.69	0.69	2.54	2.54	2.54
NOx, NH3	chronic illness	1.05	1.05	0.65	0.65	0.65	0.65	2.73	2.73	2.73	0.93	0.93	0.93	0.60	0.60	0.60	0.09	0.09	0.09	3.92	3.92	3.92	3.06	3.06	3.06	1.12	1.12	1.12
		0.97	0.97	0.60	0.60	0.60	0.60	2.51	2.51	2.51	0.85	0.85	0.85	0.55	0.55	0.55	0.08	0.08	0.08	3.60	3.60	3.60	2.80	2.80	2.80	1.02	1.02	1.02
PM2.5	mortality	75.81	75.81	46.67	46.67	46.67	46.67	196.34	196.34	196.34	66.94	66.94	66.94	43.16	43.16	43.16	6.15	6.15	6.15	281.82	281.82	281.82	219.73	219.73	219.73	80.37	80.37	80.37
		50.27	50.27	30.87	30.87	30.87	30.87	129.71	129.71	129.71	44.39	44.39	44.39	28.61	28.61	28.61	4.07	4.07	4.07	186.21	186.21	186.21	145.20	145.20	145.20	53.29	53.29	53.29
PM2.5	chronic illness	0.04	0.04	0.02	0.02	0.02	0.02	0.09	0.09	0.09	0.03	0.03	0.03	0.02	0.02	0.02	0.00	0.00	0.00	0.14	0.14	0.14	0.11	0.11	0.11	0.04	0.04	0.04
		1.58	1.58	1.58	0.97	0.97	0.97	4.10	4.10	4.10	1.40	1.40	1.40	0.90	0.90	0.90	0.13	0.13	0.13	5.88	5.88	5.88	4.59	4.59	4.59	1.68	1.68	1.68
PM2.5	asthma attacks	132.12	132.12	81.26	81.26	81.26	81.26	341.71	341.71	341.71	116.66	116.66	116.66	75.21	75.21	75.21	10.71	10.71	10.71	490.50	490.50	490.50	382.44	382.44	382.44	140.06	140.06	140.06
		14.71	14.71	69.88	17.68	12.69	44.68	95.01	223.09	15.11	15.11	15.11	62.90	189.83	189.83	232.72	24.56	24.56	24.56	52.38	52.38	52.38	41.56	41.56	41.56	30.81	30.81	30.81
Coarse PM10	mortality	9.76	9.76	46.15	46.15	46.15	46.15	29.49	29.49	29.49	62.82	62.82	62.82	41.56	41.56	41.56	16.27	16.27	16.27	344.54	344.54	344.54	277.26	277.26	277.26	33.63	33.63	33.63
		0.01	0.01	0.03	0.01	0.01	0.01	0.05	0.05	0.05	0.11	0.11	0.11	0.09	0.09	0.09	0.01	0.01	0.01	0.25	0.25	0.25	0.20	0.20	0.20	0.02	0.02	0.02
Coarse PM10	asthma attacks	0.31	0.31	1.46	1.46	1.46	1.46	1.98	1.98	1.98	4.66	4.66	4.66	3.31	3.31	3.31	0.51	0.51	0.51	10.88	10.88	10.88	8.75	8.75	8.75	1.06	1.06	1.06
		0.18	0.18	0.15	0.15	0.15	0.15	1.14	1.14	1.14	1.14	1.14	1.14	0.96	0.96	0.96	0.12	0.12	0.12	2.66	2.66	2.66	2.14	2.14	2.14	0.35	0.35	0.35
SOx, NiB	mortality	0.94	0.94	13.94	13.94	13.94	13.94	35.06	35.06	35.06	0.90	0.90	0.90	12.47	12.47	12.47	0.31	0.31	0.31	6.85	6.85	6.85	4.42	4.42	4.42	1.21	1.21	1.21
		0.62	0.62	9.18	9.18	9.18	9.18	3.25	3.25	3.25	23.09	23.09	23.09	8.02	8.02	8.02	0.20	0.20	0.20	4.51	4.51	4.51	2.91	2.91	2.91	0.80	0.80	0.80
SOx, NiB	asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		0.02	0.02	0.01	0.01	0.01	0.01	0.17	0.17	0.17	0.73	0.73	0.73	0.25	0.25	0.25	0.05	0.05	0.05	0.31	0.31	0.31	0.26	0.26	0.26	0.03	0.03	0.03
SOx, NiB	asthma attacks	26.54	26.54	141.11	141.11	141.11	141.11	495.09	495.09	27.15	27.15	27.15	126.43	324.81	324.81	413.83	42.00	42.00	42.00	888.95	888.95	888.95	715.35	715.35	715.35	87.91	87.91	87.91
		7.99	7.99	8.29	5.16	5.70	5.70	21.23	21.23	21.23	21.80	21.80	21.80	7.55	7.55	7.55	44.82	44.82	44.82	116.84	116.84	116.84	94.40	94.40	94.40	14.86	14.86	14.86
SOx, NiB	asthma attacks	5.30	5.30	3.42	3.42	3.42	3.42	14.04	14.04	14.04	4.74	4.74	4.74	4.75	4.75	4.75	29.73	29.73	29.73	77.10	77.10	77.10	62.30	62.30	62.30	9.83	9.83	9.83
		0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.02	0.02	0.02	0.06	0.06	0.06	0.05	0.05	0.05	0.01	0.01	0.01
SOx, NiB	asthma attacks	0.17	0.17	0.11	0.11	0.11	0.11	0.44	0.44	0.44	0.45	0.45	0.45	0.16	0.16	0.16	0.94	0.94	0.94	1.44	1.44	1.44	1.98	1.98	1.98	0.31	0.31	0.31
		13.46	13.77	13.77	8.69	9.24	9.24	35.72	36.30	36.30	36.30	36.30	36.30	12.45	12.45	12.45	75.51	75.51	75.51	112.23	112.23	112.23	158.71	159.34	159.34	25.00	25.42	25.42
VOCs	mortality	5.02	5.94	3.23	3.88	3.88	3.88	30.07	31.98	31.98	4.73	5.57	5.57	1.03	1.03	1.03	1.31	1.31	1.31	10.03	11.77	11.77	7.76	9.14	9.14	5.00	5.88	5.88
		3.33	3.94	2.14	2.57	2.57	2.57	19.93	21.19	21.19	3.14	3.69	3.69	0.68	0.68	0.68	0.87	0.87	0.87	6.64	7.78	7.78	5.14	6.05	6.05	3.31	3.90	3.90
VOCs	asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.00	0.00
		0.10	0.12	0.07	0.08	0.08	0.08	0.63	0.67	0.67	0.67	0.67	0.67	0.12	0.12	0.12	0.02	0.02	0.02	0.03	0.03	0.03	0.16	0.19	0.19	0.10	0.12	0.12
VOCs	oral cancer	0.01	0.01	0.01	0.01	0.01	0.01	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.01	0.01	0.01
		0.09	0.09	0.06	0.06	0.06	0.06	0.35	0.35	0.35	0.35	0.35	0.35	0.08	0.08	0.08	0.01	0.01	0.01	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03
VOCs	cancer all sites	0.30	0.30	0.20	0.20	0.20	0.20	1.17	1.17	1.17	1.17	1.17	1.17	0.28	0.28	0.28	0.28	0.28	0.28	0.03	0.03	0.03	0.03	0.03	0.03	0.06	0.06	0.06
		0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.00	0.00
VOCs	oral cancer	8.87	10.41	10.41	6.80	6.80	6.80	32.22	35.42	35.42	8.34	9.75	9.75	1.78	2.25	2.25	2.25	2.25	2.25	17.00	19.91	19.91	13.16	15.48	15.48	8.76	10.25	10.25
		0.31	0.32	0.19	0.20	0.20	0.20	1.00	1.02	1.02	1.02	1.02	1.02	0.28	0.28	0.28	0.28	0.28	0.28	0.10	0.10	0.10	0.46	0.47	0.47	0.29	0.30	0.30
VOCs	lower resp. illness	0.58	0.59	0.36	0.38	0.38	0.38	1.88	1.91	1.91	0.52	0.53	0.53	0.19	0.19	0.19	0.19	0.19	0.19	1.11	1.13	1.13	0.86	0.88	0.88	0.55	0.57	0.57
		0.18	0.18	0.11	0.11	0.11	0.11	0.57	0.58	0.58	0.16	0.16	0.16	0.06	0.06	0.06	0.06	0.06	0.06	0.34	0.34	0.34	0.26	0.27	0.27	0.17	0.17	0.17
VOCs	upper resp. illness	0.43	0.44	0.27	0.28	0.28	0.28	1.41	1.44	1.44	0.38	0.39	0.39	0.14	0.14	0.14	0.14	0.14	0.14	0.83	0.85	0.85	0.65	0.66	0.66	0.40	0.41	0.41
		109.88	111.11	179.27	71.60	72.81	112.34	374.13	376.62	534.85	99.75	100.99	100.99	160.03	281.44	281.74	334.73	379.90	381.17	381.17	936.62	939.16	939.16	746.64	748.64	748.64	156.78	158.09
Subtotal for VOC emissions	asthma attacks	0.58	0.58	0.41	0.41	0.41	0.41	2.71	2.71	2.71	0.55	0.55	0.55	0.10	0.10	0.10	0.10	0.10	0.10	2.77	2.77	2.77	2.23	2.23	2.23	0.70	0.70	0.70
		69.28	69.90	114.84	45.71	47.75	72.97	231.02	335.23	628.89	63.45	64.45	64.45	102.40	186.54	186.73	221.63	258.08	258.22	258.22	619.21	619.21	619.21	493.73	493.73	493.73	101.46	101.46
Subtotal for VOC emissions	lower resp. illness	11.01	11.07	12.53	6.82	6.82	6.82	37.83	37.96	41.33	9.86	9.92	9.92	1.18	1.18	1.18	1.18	1.18	1.18	39.91	39.91	39.91	31.48	31.48	31.48	11.75	11.82	13.58
		176.16	178.03	292.58	115.29	116.9																						

TABLE 11-A 55 COST OF AIR POLLUTION A TRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$/1000 VMT (ST. LOUIS MSA 1990) (LOWER BOUND)

Emission	Ambient pollutant	Health effect	IDGVs			LDGts			HDGts			Total gasoline vehicles			IDDVs			LDDTs			HDDVs			Total diesel vehicles			Total all vehicles		
			V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD
CO	CO	morality	0.17	0.17	0.17	0.26	0.26	0.26	0.67	0.67	0.67	0.19	0.19	0.19	0.01	0.01	0.01	0.00	0.00	0.00	0.03	0.03	0.03	0.02	0.02	0.02	0.18	0.18	0.18
		hospitalization	0.03	0.03	0.03	0.04	0.04	0.04	0.11	0.11	0.11	0.03	0.03	0.03	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.03	0.03	0.03
		headaches	0.16	0.16	0.16	0.24	0.25	0.25	0.62	0.63	0.63	0.18	0.18	0.18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.02	0.02	0.02	0.02	0.17	0.17	0.17
NOx	NO2	sore throat	0.24	0.26	0.26	0.37	0.41	0.41	0.69	0.77	0.77	0.27	0.29	0.29	0.14	0.14	0.14	0.06	0.06	0.06	0.87	0.91	0.91	0.68	0.72	0.72	0.30	0.33	0.33
		excess phlegm	0.11	0.12	0.12	0.17	0.18	0.18	0.31	0.34	0.34	0.12	0.13	0.13	0.06	0.06	0.06	0.02	0.02	0.02	0.39	0.41	0.41	0.31	0.32	0.32	0.13	0.15	0.15
		eye irritation	0.10	0.11	0.11	0.15	0.17	0.17	0.28	0.31	0.31	0.11	0.12	0.12	0.06	0.06	0.06	0.02	0.02	0.02	0.35	0.37	0.37	0.29	0.29	0.29	0.12	0.13	0.13
NOx, NH3	nitrate PM10	morality	1.23	1.33	1.33	1.89	2.07	2.07	3.54	3.86	3.86	1.38	1.50	1.50	0.69	0.71	0.71	0.25	0.30	0.30	4.42	4.63	4.63	3.47	3.64	3.64	1.53	1.65	1.65
		chronic illness	0.21	0.23	0.23	0.33	0.36	0.36	0.62	0.68	0.68	0.24	0.26	0.26	0.12	0.12	0.12	0.04	0.05	0.05	0.77	0.81	0.81	0.60	0.63	0.63	0.27	0.29	0.29
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Total for NOx and NH3 emissions	PM2.5	RRAD	0.06	0.06	0.06	0.09	0.10	0.10	0.17	0.18	0.18	0.07	0.07	0.07	0.03	0.03	0.03	0.01	0.01	0.01	0.21	0.22	0.22	0.17	0.17	0.17	0.07	0.08	0.08
		morality	1.95	2.11	2.11	3.00	3.29	3.29	5.62	6.15	6.15	2.18	2.37	2.37	1.09	1.13	1.13	0.39	0.47	0.47	7.01	7.36	7.36	5.51	5.78	5.78	2.43	2.63	2.63
		chronic illness	0.24	0.34	0.34	0.52	0.74	0.74	1.56	1.88	1.88	0.31	0.41	0.41	0.12	0.12	0.12	0.04	0.05	0.05	1.15	1.15	1.15	0.85	0.85	0.85	0.81	0.94	0.94
Coarse PM10 coarse PM10	PM10 coarse	morality	0.06	0.12	0.12	0.47	0.73	0.73	0.40	0.54	0.54	0.09	0.14	0.14	0.20	0.21	0.21	0.46	0.66	0.66	0.53	0.44	0.58	0.37	0.46	0.46	0.11	0.17	0.17
		chronic illness	0.02	0.03	0.03	0.05	0.15	0.15	0.08	0.12	0.12	0.02	0.03	0.03	0.04	0.04	0.04	0.10	0.10	0.10	0.09	0.13	0.13	0.08	0.10	0.10	0.02	0.04	0.04
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Total for diesel PM10 emissions	SOx, NH3 sulfate PM10	RRAD	0.00	0.01	0.01	0.02	0.02	0.02	0.02	0.03	0.03	0.01	0.01	0.01	0.01	0.01	0.01	0.02	0.02	0.02	0.02	0.03	0.03	0.02	0.02	0.02	0.15	0.01	0.01
		morality	0.41	0.59	0.59	1.11	1.24	1.24	1.98	2.49	2.49	0.51	0.74	0.74	1.32	1.47	1.47	2.12	3.14	3.14	1.40	1.40	1.40	9.39	9.89	9.89	1.17	1.42	1.42
		chronic illness	0.32	0.34	0.34	0.53	0.65	0.65	0.67	0.88	1.08	1.08	0.37	0.41	0.41	0.83	0.94	0.94	0.68	0.84	0.84	0.86	1.04	1.04	4.02	4.02	4.02	0.64	0.64
Total for SOx and NH3 emissions	VOCs	morality	0.07	0.07	0.07	0.09	0.11	0.11	0.19	0.303	0.303	0.08	0.12	0.12	0.38	0.71	0.71	0.14	0.23	0.23	0.94	0.94	0.94	0.86	0.86	0.86	0.14	0.14	0.14
		chronic illness	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		asthma attacks	0.02	0.16	0.16	0.02	0.30	0.30	0.04	0.50	0.50	0.02	0.19	0.19	0.09	0.14	0.14	0.03	0.14	0.14	0.23	0.71	0.71	0.19	0.57	0.57	0.03	0.22	0.22
Sub-total for VOC emissions	VOCs, NOx O3	morality	0.41	4.44	4.44	0.66	8.35	8.35	1.10	14.22	14.22	0.46	5.33	5.33	2.30	3.79	3.79	0.85	4.02	4.02	6.13	19.62	19.62	5.07	15.77	15.77	0.81	6.10	6.10
		chronic illness	0.11	0.18	0.18	0.35	0.35	0.35	0.53	0.77	0.77	0.12	0.22	0.22	0.02	0.04	0.04	0.04	0.05	0.05	0.20	0.48	0.48	0.16	0.37	0.37	0.13	0.23	0.23
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Subtotal cancer effects	Subtotal cancer effects	RRAD	0.00	0.01	0.01	0.01	0.02	0.02	0.03	0.04	0.04	0.01	0.01	0.01	0.01	0.01	0.01	0.02	0.02	0.02	0.02	0.03	0.03	0.02	0.02	0.02	0.01	0.01	0.01
		coral cancer	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		leukemia	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Subtotal ambient PM10	Subtotal ambient PM10	cancer all sites	0.01	0.01	0.01	0.02	0.02	0.02	0.05	0.05	0.05	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		oral cancer	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		total cancer	0.15	0.24	0.24	0.25	0.46	0.46	0.72	1.03	1.03	0.17	0.29	0.29	0.03	0.05	0.05	0.01	0.07	0.07	0.26	0.61	0.61	0.20	0.47	0.47	0.18	0.31	0.31
Subtotal ambient O3	Subtotal ambient O3	asthma attacks	0.01	0.01	0.01	0.01	0.02	0.02	0.03	0.04	0.04	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.02	0.02	0.01	0.02	0.02	0.01	0.01	0.01
		lower resp illness	0.03	0.03	0.03	0.05	0.06	0.06	0.10	0.14	0.14	0.03	0.04	0.04	0.01	0.01	0.01	0.01	0.01	0.01	0.06	0.06	0.06	0.05	0.06	0.06	0.03	0.04	0.04
		upper resp illness	0.01	0.01	0.01	0.01	0.02	0.02	0.03	0.04	0.04	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.02	0.02	0.02	0.02	0.02	0.01	0.01	0.01
Subtotal mortality effects	Subtotal mortality effects	eye irritation	0.03	0.03	0.03	0.05	0.06	0.06	0.09	0.14	0.14	0.03	0.04	0.04	0.01	0.01	0.01	0.01	0.01	0.01	0.06	0.08	0.08	0.05	0.06	0.06	0.03	0.04	0.04
		asthma attacks	2.15	5.49	5.49	5.90	10.10	10.10	7.57	16.41	16.41	2.47	6.50	6.50	5.84	7.04	7.04	2.00	4.55	4.55	18.49	29.57	29.57	15.07	23.86	23.86	3.40	7.78	7.78
		lower resp illness	0.02	0.02	0.02	0.02	0.04	0.04	0.11	0.11	0.11	0.03	0.03	0.03	0.06	0.06	0.06	0.06	0.06	0.06	0.16	0.16	0.16	0.13	0.13	0.13	0.04	0.04	0.04
Subtotal chronic morbidity effects	Subtotal chronic morbidity effects	asthma attacks	0.37	1.31	1.31	1.39	0.61	2.43	2.55	4.38	4.67	0.42	1.56	1.65	1.16	1.51	1.58	0.40	1.12	1.23	3.68	6.79	6.79	3.00	5.47	5.47	0.61	1.85	1.97
		upper resp illness	0.80	1.01	1.01	1.04	1.25	1.67	2.60	3.35	3.43	0.90	1.16	1.19	0.56	0.63	0.65	0.20	0.35	0.37	2.69	3.34	3.34	2.14	2.66	2.66	0.81	1.27	1.31
		eye irritation	2.45	6.89	6.89	7.41	13.50	13.50	8.59	23.02	24.91	2.81	8.17	8.75	7.33	8.94	9.34	2.51	5.91	5.91	23.19	37.92	37.92	18.91	30.58	34.53	4.00	9.83	10.66
Subtotal ambient NO2	Subtotal ambient NO2	asthma attacks	0.07	0.09	0.09	0.12	0.15	0.15	0.24	0.35	0.35	0.08	0.10	0.10	0.01	0.03	0.03	0.01	0.02	0.02	0.16	0.20	0.20	0.13	0.16	0.16	0.05	0.11	0.11

TABLE 11-A-57 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$/1000 VMT (SPOKANE MSA 1990) (LOWER BOUND)

Emission pollutant	Ambient pollutant	Health effect	IDCVs			LDGIs			HDGIs			Total gasoline vehicles			IDDVs			IDDVs			BDDVs			Total diesel vehicles			Total all vehicles			
			V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	V	V+U	V+U+RD	
CO	CO	morality	0.67	0.67	0.58	0.58	2.14	2.17	2.17	0.65	0.67	0.67	0.02	0.02	0.02	0.01	0.01	0.01	0.01	0.01	0.01	0.13	0.13	0.13	0.07	0.10	0.10	0.61	0.62	0.62
		hospitalization	0.11	0.11	0.09	0.09	0.34	0.34	0.34	0.10	0.11	0.11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.02	0.02	0.02	0.02	0.02	0.01	0.01	0.10
		headaches	0.64	0.65	0.56	0.56	2.07	2.09	2.09	0.63	0.64	0.64	0.02	0.02	0.02	0.01	0.01	0.01	0.01	0.01	0.01	0.09	0.12	0.12	0.07	0.09	0.09	0.59	0.60	0.60
NOx	NO2	sore throat	0.37	0.37	0.34	0.34	1.02	1.02	1.02	0.37	0.37	0.37	0.20	0.20	0.20	0.04	0.04	0.04	0.04	0.04	0.04	1.35	1.35	1.35	1.03	1.03	1.03	0.42	0.42	0.42
		excess phlegm	0.17	0.17	0.15	0.15	0.46	0.46	0.46	0.17	0.17	0.17	0.09	0.09	0.09	0.02	0.02	0.02	0.02	0.02	0.02	0.60	0.60	0.60	0.46	0.46	0.46	0.19	0.19	0.19
		eye irritation	0.15	0.15	0.14	0.14	0.42	0.42	0.42	0.15	0.15	0.15	0.08	0.08	0.08	0.02	0.02	0.02	0.02	0.02	0.02	0.55	0.55	0.55	0.42	0.42	0.42	0.17	0.17	0.17
NOx, NH3	nitrate PM10	morality	2.59	2.59	2.32	2.32	7.06	7.07	7.07	2.56	2.56	2.56	1.41	1.41	1.41	0.29	0.29	0.29	0.29	0.29	0.29	9.33	9.33	9.33	7.12	7.12	7.12	2.91	2.91	2.91
		chronic illness	1.00	1.00	0.89	0.89	2.72	2.73	2.73	0.99	0.99	0.99	0.54	0.54	0.54	0.11	0.11	0.11	0.11	0.11	0.11	3.59	3.60	3.60	2.75	2.75	2.75	1.12	1.12	1.12
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.00	0.00
Total for NOx and NH3 emissions	PM2.5	READ	0.12	0.12	0.10	0.10	0.32	0.32	0.32	0.11	0.12	0.12	0.06	0.06	0.06	0.01	0.01	0.01	0.01	0.01	0.01	0.42	0.42	0.42	0.32	0.32	0.32	0.13	0.13	0.13
		morality	4.39	4.40	3.94	3.94	12.00	12.02	12.02	4.35	4.36	4.36	2.39	2.39	2.39	0.50	0.50	0.50	0.50	0.50	0.50	15.84	15.85	15.85	12.10	12.11	12.11	4.95	4.95	4.95
		chronic illness	0.45	0.50	0.69	0.66	2.77	2.91	2.91	0.51	0.58	0.76	0.76	0.76	0.18	0.18	0.18	0.08	0.08	0.08	0.08	0.08	16.34	16.53	16.53	12.98	13.12	13.12	14.71	15.4
Coarse PM10 coarse PM10	PM10	morality	0.17	0.19	0.27	0.25	0.31	1.07	1.12	1.35	0.20	0.22	0.29	0.21	0.21	0.21	0.22	0.22	0.22	0.22	0.22	6.30	6.36	6.99	5.01	5.06	5.53	0.7	0.60	0.70
		chronic illness	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.02	0.02	0.02	0.02	0.02	0.00	0.00	0.00
		asthma attacks	0.02	0.02	0.03	0.03	0.04	0.12	0.13	0.16	0.02	0.03	0.03	0.26	0.26	0.26	0.05	0.05	0.05	0.05	0.05	0.73	0.74	0.81	0.58	0.59	0.64	0.07	0.07	0.08
Total for direct PM10 emissions	SOx, NH3 sulfate PM10	lung cancer	0.01	0.01	0.01	0.01	0.06	0.06	0.06	0.01	0.01	0.01	0.07	0.07	0.07	0.01	0.01	0.01	0.01	0.01	0.01	0.19	0.19	0.19	0.15	0.15	0.15	0.02	0.02	0.02
		morality	0.14	0.17	1.20	1.19	1.02	0.72	0.78	3.96	0.15	0.18	1.18	0.36	0.37	1.17	0.07	0.08	0.08	0.08	0.08	0.85	0.93	9.46	0.69	0.75	7.27	0.19	0.23	1.65
		chronic illness	0.06	0.06	0.46	0.46	0.39	0.28	0.30	1.53	0.06	0.07	0.46	0.14	0.14	0.45	0.03	0.03	0.03	0.03	0.03	0.33	0.36	3.66	0.26	0.29	2.81	0.07	0.09	0.64
Total for direct PM10 emissions	SOx, NH3 sulfate PM10	asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		READ	0.01	0.01	0.05	0.01	0.05	0.03	0.18	0.01	0.01	0.05	0.02	0.02	0.05	0.00	0.00	0.00	0.00	0.00	0.00	0.04	0.04	0.04	0.03	0.03	0.03	0.01	0.01	0.07
		morality	0.85	0.97	2.72	1.04	1.23	2.63	5.06	0.96	1.10	2.79	0.79	0.80	10.17	1.66	1.69	2.91	24.81	25.20	29.72	19.73	20.02	31.10	19.73	20.02	31.10	2.40	2.56	4.97
Total for SOx and NH3 emissions	VOCs	morality	0.20	0.34	0.34	0.34	0.34	0.88	1.28	1.28	0.20	0.35	0.35	0.04	0.08	0.08	0.01	0.05	0.05	0.05	0.05	0.38	0.69	0.69	0.29	0.53	0.53	0.21	0.37	0.37
		chronic illness	0.08	0.13	0.13	0.13	0.13	0.24	0.49	0.49	0.08	0.14	0.14	0.01	0.03	0.03	0.02	0.02	0.02	0.02	0.02	0.15	0.27	0.27	0.11	0.20	0.20	0.08	0.14	0.14
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Subtotal for VOC emissions	VOCs, NOx, O3	READ	0.01	0.02	0.02	0.02	0.04	0.06	0.06	0.01	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.03	0.03	0.03	0.01	0.02	0.02	0.01	0.02	0.02
		oral cancer	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
		leukemia	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
Subtotal for VOC emissions	VOCs, NOx, O3	cancer all sites	0.02	0.02	0.02	0.02	0.06	0.06	0.06	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02
		oral cancer	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		morality	0.21	0.31	0.51	0.30	0.50	1.34	1.91	1.91	0.32	0.53	0.53	0.06	0.11	0.11	0.01	0.07	0.07	0.07	0.07	0.56	1.00	1.00	0.42	0.77	0.77	0.32	0.54	0.54
Subtotal mortality effects	Subtotal cancer effects	asthma attacks	0.01	0.01	0.01	0.01	0.02	0.02	0.02	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.02	0.02	0.02	0.02	0.02	0.01	0.01	0.01	0.01
		lower resp. illness	0.02	0.03	0.03	0.02	0.02	0.07	0.07	0.02	0.03	0.03	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.06	0.06	0.06	0.05	0.05	0.05	0.03	0.03	0.03
		upper resp. illness	0.01	0.01	0.01	0.01	0.02	0.02	0.02	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.02	0.02	0.01	0.01	0.01	0.01	0.01	0.01
Subtotal acute mortality effects	Subtotal ambient PM10	eye irritation	0.02	0.02	0.02	0.02	0.05	0.06	0.06	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02
		morality	4.59	5.01	6.23	4.32	5.06	6.04	15.01	16.08	19.85	4.64	5.15	6.34	10.89	10.75	11.70	2.15	2.25	3.10	3.10	35.57	36.80	46.93	28.00	28.93	36.66	6.44	6.99	8.67
		chronic illness	0.03	0.03	0.03	0.03	0.03	0.14	0.14	0.14	0.03	0.03	0.03	0.07	0.07	0.07	0.01	0.02	0.02	0.02	0.02	0.20	0.20	0.20	0.16	0.16	0.16	0.04	0.04	0.04
Subtotal ambient CO	Subtotal ambient NO2	asthma attacks	1.52	1.67	2.14	1.45	1.73	2.10	4.96	5.37	6.82	1.54	1.73	2.19	4.12	4.14	4.50	0.83	0.87	1.19	1.19	13.68	14.15	18.06	10.77	11.12	14.11	2.25	2.46	3.11
		upper resp. illness	1.67	1.71	1.76	1.48	1.54	1.58	5.05	5.14	5.31	1.66	1.70	1.75	0.91	0.92	0.96	0.19	0.19	0.23	0.23	4.38	4.48	4.95	3.39	3.46	3.62	1.79	1.83	1.91
		morality	5.64	6.21	7.97	5.39	6.42	7.82	18.48	19.98	25.39	5.72	6.44	8.13	15.34	15.43	16.79	3.08	3.22	4.44	4.4									

TABLE 11-A-56 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$/1,000 VMT (SPOKANE MSA 1990) (UPPER BOUND)

Emission	Ambient pollutant	Health effect	LDGVA		LDGTS		HDGTS		Total gasoline vehicles		LDDTs		HDDVs		Total diesel vehicles		Total all vehicles			
			V	V/1000	V	V/1000	V	V/1000	V	V/1000	V	V/1000	V	V/1000	V	V/1000	V	V/1000	V	V/1000
CO	CO	moribidity	8.50	8.51	7.19	7.22	37.14	37.19	37.19	8.45	8.47	8.47	0.05	0.05	1.65	1.70	1.30	1.30	7.90	7.92
		hospitalization	0.34	0.34	0.29	0.29	1.47	1.47	1.47	0.33	0.34	0.34	0.00	0.00	0.07	0.07	0.05	0.05	0.05	0.05
		headaches	3.97	3.98	3.36	3.38	17.39	17.41	17.41	3.95	3.96	3.96	0.02	0.02	0.77	0.80	0.59	0.61	3.69	3.70
NOx	NOx	sore throat	1.94	1.94	1.73	1.73	7.17	7.17	7.17	1.94	1.94	1.94	0.01	0.01	9.43	9.43	7.12	7.12	2.34	2.34
		excess phlegm	0.87	0.87	0.77	0.77	3.20	3.20	3.20	0.87	0.87	0.87	0.01	0.01	4.21	4.21	3.18	3.18	1.04	1.04
		eye irritation	0.79	0.79	0.70	0.70	2.91	2.91	2.91	0.79	0.79	0.79	0.02	0.02	3.33	3.33	2.89	2.89	0.95	0.95
NOx, NH3	nitrate PM10	moribidity	53.98	53.99	47.86	47.87	198.83	198.85	198.85	53.89	53.90	53.90	5.23	5.23	261.54	261.56	197.52	197.54	64.93	64.94
		chronic illness	19.93	19.94	17.66	17.67	73.37	73.38	73.38	19.91	19.91	19.91	0.00	0.00	96.54	96.55	72.91	72.92	23.99	24.00
		asthma attacks	0.03	0.03	0.02	0.02	0.10	0.10	0.10	0.03	0.03	0.03	0.00	0.00	0.13	0.13	0.09	0.09	0.03	0.03
Total for NChond NHEmissions	RRAD	RRAD	0.90	0.90	0.79	0.79	3.30	3.30	3.30	0.89	0.89	0.89	0.42	0.42	4.34	4.34	3.28	3.28	1.08	1.08
		RRAD	78.44	78.45	69.54	69.55	288.86	288.91	288.91	78.31	78.32	78.32	36.81	36.82	368.82	368.84	287.00	287.02	94.36	94.38
		RRAD	8.74	8.88	10.92	11.16	44.65	44.65	44.65	10.29	10.46	10.46	112.22	112.22	144.81	144.81	141.94	141.94	239.69	239.69
PM2.5	PM2.5	moribidity	3.22	3.28	4.03	4.12	16.48	16.48	16.48	3.80	3.86	3.86	7.76	7.77	18.51	18.51	141.94	141.94	14.44	14.51
		chronic illness	0.00	0.00	0.01	0.01	0.02	0.02	0.04	0.01	0.02	0.02	0.01	0.01	0.02	0.02	0.01	0.01	0.02	0.02
		asthma attacks	0.15	0.15	0.84	0.84	3.18	3.18	3.18	0.17	0.17	0.17	0.35	0.35	0.83	0.83	6.37	6.38	10.76	10.76
Coarse PM10 coarse PM10	lung cancer	lung cancer	0.18	0.18	0.22	0.22	0.22	0.22	0.22	0.21	0.21	0.21	0.16	0.16	3.70	3.70	2.87	2.87	0.41	0.41
		lung cancer	0.56	0.57	11.24	11.24	4.41	4.41	4.41	0.62	0.62	0.62	1.41	1.41	9.71	9.71	4.04	4.07	71.45	71.45
		lung cancer	0.21	0.21	0.22	0.22	1.63	1.63	1.63	0.22	0.22	0.22	0.52	0.52	3.58	3.58	1.49	1.50	26.37	26.37
Total for all PM10 emissions	RRAD	RRAD	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		RRAD	13.06	13.26	16.17	16.52	74.75	74.75	74.75	15.21	15.56	15.56	62.02	62.02	158.36	158.36	215.01	215.01	697.79	697.79
		RRAD	4.72	4.93	4.42	4.56	19.05	19.05	19.05	4.00	4.11	4.11	26.50	26.51	26.51	26.51	26.51	26.51	87.85	87.85
VOCs	organic PM10 (SOA)	chronic illness	1.07	1.36	1.05	1.33	7.64	7.64	7.64	1.14	1.43	1.43	0.23	0.23	3.28	3.69	2.46	2.94	1.24	1.55
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		RRAD	0.05	0.06	0.05	0.06	0.34	0.34	0.34	0.05	0.06	0.06	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
Subtotal for VOC emissions	oral cancer	oral cancer	0.01	0.01	0.01	0.01	0.05	0.05	0.05	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
		leukemia	0.09	0.09	0.09	0.09	0.42	0.42	0.42	0.09	0.09	0.09	0.01	0.01	0.04	0.04	0.04	0.04	0.09	0.09
		cancer all sites	0.31	0.31	0.30	0.30	1.42	1.42	1.42	0.32	0.32	0.32	0.03	0.03	0.12	0.12	0.09	0.09	0.30	0.30
Subtotal for VOC emissions	oral cancer	oral cancer	0.00	0.00	0.00	0.00	0.02	0.02	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		oral cancer	4.44	5.51	4.34	5.39	20.69	20.69	20.69	4.72	5.81	5.81	11.2	11.2	11.2	11.2	11.2	11.2	5.08	6.22
		oral cancer	0.11	0.12	0.10	0.11	0.45	0.46	0.46	0.11	0.12	0.12	0.04	0.04	0.04	0.04	0.04	0.04	0.13	0.13
Subtotal ambient O3	asthma attacks	asthma attacks	0.21	0.22	0.19	0.20	0.83	0.85	0.85	0.21	0.22	0.22	0.02	0.02	0.72	0.73	0.55	0.55	0.24	0.25
		asthma attacks	0.06	0.07	0.06	0.06	0.26	0.26	0.26	0.06	0.07	0.07	0.01	0.01	0.01	0.01	0.01	0.01	0.07	0.08
		asthma attacks	0.11	0.11	0.10	0.11	0.44	0.45	0.45	0.11	0.12	0.12	0.04	0.04	0.04	0.04	0.04	0.04	0.13	0.13
Subtotal mortality effects	Subtotal mortality effects	Subtotal mortality effects	79.39	80.35	133.15	133.15	73.81	75.40	75.40	81.14	82.44	82.44	32.77	32.77	692.77	692.77	692.77	692.77	127.33	128.71
		Subtotal mortality effects	0.59	0.59	0.63	0.63	3.63	3.63	3.63	0.64	0.64	0.64	0.88	0.88	0.88	0.88	0.88	0.88	0.82	0.82
		Subtotal mortality effects	26.18	26.60	46.02	46.02	24.59	25.16	25.16	26.84	27.32	27.32	61.35	61.35	76.44	76.44	119.6	120.07	25.54	25.54
Subtotal ambient NO2	Subtotal ambient NO2	Subtotal ambient NO2	9.63	9.66	8.44	8.50	39.73	39.73	39.73	9.62	9.67	9.67	10.54	10.54	4.82	4.82	5.52	5.52	26.80	26.80
		Subtotal ambient NO2	98.46	100.04	175.97	175.97	92.57	94.73	94.73	100.98	102.76	102.76	45.09	45.09	96.11	96.11	1,228.30	1,232.36	1,408.17	1,412.22
		Subtotal ambient NO2	0.50	0.52	0.46	0.47	1.97	2.02	2.02	0.50	0.52	0.52	0.18	0.18	0.04	0.04	0.04	0.04	1.31	1.31
Subtotal ambient CO	Subtotal ambient CO	Subtotal ambient CO	3.60	3.60	3.60	3.60	13.28	13.28	13.28	3.60	3.60	3.60	0.35	0.35	17.47	17.47	13.19	13.19	4.33	4.33
		Subtotal ambient CO	12.80	12.83	12.83	12.83	56.01	56.01	56.01	12.74	12.77	12.77	0.33	0.33	0.07	0.07	2.48	2.57	11.90	11.90
		Subtotal ambient CO	115.78	117.80	190.33	190.33	107.16	109.68	109.68	118.25	120.07	120.07	232.22	232.22	232.22	232.22	232.22	232.22	96.61	96.61
Total all emissions, pollutants, effects	Total all emissions, pollutants, effects	Total all emissions, pollutants, effects	115.78	117.80	190.33	190.33	107.16	109.68	109.68	118.25	120.07	120.07	232.22	232.22	232.22	232.22	232.22	232.22	96.61	96.61
		Total all emissions, pollutants, effects	115.78	117.80	190.33	190.33	107.16	109.68	109.68	118.25	120.07	120.07	232.22	232.22	232.22	232.22	232.22	232.22	96.61	96.61
		Total all emissions, pollutants, effects	115.78	117.80	190.33	190.33	107.16	109.68	109.68	118.25	120.07	120.07	232.22	232.22	232.22	232.22	232.22	232.22	96.61	96.61

TABLE 11-A-59 COST OF AIR POLLUTION ATTRIBUTABLE TO A 10% REDUCTION IN MOTOR VEHICLE EMISSIONS, BY EMISSION-POLLUTANT PATHWAY AND HEALTH EFFECT, \$/1000 VMT (WASHINGTON D C MSA 1990) (LOWER E

Emission	Ambient pollutant	Health effect	LDGVs			LDGIs			HDGIs			Total gasoline vehicles			LDGVs			LDGIs			HDGIs			Total diesel vehicles			Total all vehicles		
			V	V+U+RD	V+U+RD	V	V+U+RD	V+U+RD	V	V+U+RD	V+U+RD	V	V+U+RD	V+U+RD	V	V+U+RD	V+U+RD	V	V+U+RD	V+U+RD	V	V+U+RD	V+U+RD	V	V+U+RD	V+U+RD	V	V+U+RD	V+U+RD
CO	CO	increased mortality	0.22	0.22	0.22	0.32	0.32	0.32	1.04	1.04	1.04	0.24	0.24	0.24	0.01	0.01	0.01	0.00	0.00	0.00	0.04	0.04	0.04	0.03	0.03	0.03	0.23	0.23	0.23
		hospitalization	0.03	0.03	0.03	0.05	0.05	0.05	0.16	0.16	0.16	0.04	0.04	0.04	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.00	0.00	0.00	0.04	0.04	0.04
		headaches	0.26	0.26	0.26	0.39	0.39	0.39	1.27	1.27	1.27	0.30	0.30	0.30	0.01	0.01	0.01	0.00	0.00	0.00	0.05	0.05	0.05	0.04	0.04	0.04	0.28	0.28	0.28
NOx	NO2	sore throat	0.31	0.31	0.31	0.46	0.46	0.46	1.10	1.10	1.10	0.35	0.35	0.35	0.18	0.18	0.18	0.06	0.06	0.06	1.37	1.37	1.37	1.02	1.02	1.02	0.39	0.39	0.39
		excess phlegm	0.14	0.14	0.14	0.20	0.20	0.20	0.49	0.49	0.49	0.15	0.15	0.15	0.08	0.08	0.08	0.03	0.03	0.03	0.61	0.61	0.61	0.45	0.45	0.45	0.17	0.17	0.17
		eye irritation	0.13	0.13	0.13	0.19	0.19	0.19	0.45	0.45	0.45	0.14	0.14	0.14	0.07	0.07	0.07	0.02	0.02	0.02	0.55	0.55	0.55	0.41	0.41	0.41	0.16	0.16	0.16
NOx, NH3	nitrate PM10	mortality	2.39	2.39	2.39	3.52	3.52	3.52	8.47	8.47	8.47	2.67	2.67	2.67	1.37	1.37	1.37	0.46	0.46	0.46	10.54	10.54	10.54	7.85	7.85	7.85	2.99	2.99	3.00
		chronic illness	0.38	0.38	0.38	0.55	0.55	0.55	1.34	1.34	1.34	0.42	0.42	0.42	0.22	0.22	0.22	0.07	0.07	0.07	1.67	1.67	1.67	1.24	1.24	1.24	0.47	0.48	0.48
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.00	0.00
Total for NOx and NH3 emissions	RRAD	mortality	0.13	0.13	0.13	0.19	0.19	0.19	0.45	0.45	0.45	0.14	0.14	0.14	0.07	0.07	0.07	0.02	0.02	0.02	0.57	0.57	0.57	0.42	0.42	0.42	0.16	0.16	0.16
		chronic illness	0.47	0.47	0.47	0.68	0.68	0.68	1.62	1.62	1.62	0.57	0.57	0.57	0.31	0.31	0.31	0.12	0.12	0.12	15.36	15.36	15.36	11.36	11.40	11.40	4.35	4.37	4.37
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.00	0.00
PM2.5	PM2.5 coarse PM10	mortality	0.47	0.50	0.56	0.97	1.05	1.13	3.67	3.77	4.04	0.60	0.64	0.71	0.60	0.68	0.71	0.18	0.18	0.18	19.97	19.97	20.13	15.56	15.67	16.20	1.56	1.61	1.71
		chronic illness	0.08	0.08	0.09	0.16	0.17	0.19	0.59	0.61	0.65	0.10	0.11	0.12	0.09	0.09	0.09	0.30	0.30	0.30	3.24	3.24	3.24	2.51	2.53	2.61	0.25	0.26	0.28
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.02	0.02	0.02	0.02	0.02	0.01	0.01	0.01
Coarse PM10 coarse PM10	RRAD	mortality	0.01	0.01	0.01	0.01	0.01	0.01	0.05	0.05	0.05	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
		chronic illness	0.15	0.16	0.16	0.26	0.28	0.28	0.68	0.71	0.73	0.18	0.19	0.21	0.18	0.19	0.21	0.12	0.12	0.12	1.04	1.04	1.04	0.82	0.86	0.86	0.22	0.23	0.23
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Total for direct PM10 emissions	SOx, NH3 sulfate PM10	mortality	0.77	0.81	1.24	1.50	1.63	2.22	5.65	5.82	7.73	0.96	1.02	1.50	0.91	0.95	1.01	0.05	0.05	0.05	25.68	25.68	25.93	20.21	20.21	23.97	2.19	2.26	2.94
		chronic illness	0.92	1.09	1.09	1.26	1.87	1.87	2.65	3.65	3.65	0.93	1.27	1.27	0.75	0.77	0.77	0.27	0.27	0.27	14.57	14.57	15.88	11.41	12.39	12.39	1.60	1.98	1.98
		asthma attacks	0.13	0.18	0.18	0.21	0.31	0.31	0.43	0.60	0.60	0.15	0.21	0.21	0.15	0.15	0.15	0.00	0.00	0.00	2.35	2.35	2.57	1.84	2.01	2.01	0.26	0.32	0.32
Total for SOx and NH3 emissions	VOCs organic PM10 (SOA)	mortality	0.04	0.06	0.06	0.07	0.10	0.10	0.14	0.19	0.19	0.05	0.07	0.07	0.25	0.25	0.25	0.09	0.09	0.09	0.78	0.78	0.84	0.61	0.66	0.66	0.09	0.10	0.10
		chronic illness	0.20	0.32	0.32	0.55	0.55	0.55	1.24	1.73	1.73	0.23	0.38	0.38	0.04	0.06	0.06	0.02	0.02	0.02	17.70	17.70	19.30	13.87	15.06	15.06	1.95	2.41	2.41
		asthma attacks	0.03	0.05	0.05	0.05	0.09	0.09	0.20	0.28	0.28	0.04	0.06	0.06	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Subtotal ambient PM10	acetone/aldehyde benzene 1,3-butadiene formaldehyde	mortality	0.01	0.02	0.02	0.02	0.03	0.03	0.07	0.09	0.09	0.01	0.02	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		chronic illness	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		asthma attacks	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Subtotal ambient VOCs, NOx O3	upper resp. illness eye irritation	mortality	0.25	0.41	0.41	0.41	0.70	0.70	1.57	2.17	2.17	0.30	0.48	0.48	0.05	0.10	0.10	0.02	0.02	0.02	1.22	1.22	1.22	0.43	0.43	0.43	0.31	0.51	0.51
		chronic illness	0.01	0.01	0.01	0.02	0.02	0.02	0.05	0.06	0.06	0.01	0.02	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.02	0.02
		asthma attacks	0.05	0.05	0.05	0.08	0.08	0.08	0.20	0.22	0.22	0.06	0.06	0.06	0.02	0.02	0.02	0.00	0.00	0.00	0.04	0.04	0.04	0.03	0.03	0.03	0.02	0.02	0.02
Subtotal mortality effects	Subtotal chronic morbidity effects	mortality	4.25	4.68	5.04	6.63	7.60	8.08	17.98	19.61	21.22	4.84	5.39	5.79	12.51	12.65	12.93	4.09	4.41	4.83	46.63	46.63	48.70	36.00	37.51	40.64	6.85	7.46	8.03
		chronic illness	0.65	0.72	0.78	1.01	1.18	1.25	2.71	2.99	3.24	0.74	0.83	0.89	2.03	2.05	2.09	0.66	0.72	0.78	7.47	7.83	8.49	5.77	6.04	6.52	1.06	1.17	1.26
		asthma attacks	1.22	1.26	1.28	1.83	1.91	1.93	4.92	5.06	5.15	1.37	1.42	1.44	1.06	1.07	1.09	0.35	0.38	0.40	5.41	5.57	5.80	4.17	4.21	4.38	1.55	1.60	1.63
Subtotal ambient PM10	Subtotal ambient O3	mortality	4.90	5.44	5.87	7.68	8.86	9.45	20.61	22.65	24.35	5.99	6.27	6.74	15.25	15.62	15.76	4.98	5.37	5.88	56.74	56.74	59.27	43.80	45.68	49.45	8.06	8.81	9.49
		chronic illness	0.13	0.14	0.14	0.20	0.22	0.22	0.53	0.58	0.58	0.16	0.16	0.16	0.31	0.35	0.35	0.02	0.02	0.02	2.31	2.35	2.54	1.88	1.89	2.07	0.15	0.17	0.17
		asthma attacks	0.58	0.58	0.58	0.85	0.85	0.85	2.04	2.04	2.04	0.64	0.65	0.65	0.33	0.33	0.33	0.11	0.11	0.11	2.53	2.54	2.54	1.88	1.89	2.07	0.72	0.72	0.72
Subtotal ambient NO2	Subtotal ambient CO	mortality	0.51	0.51	0.51	0.76	0.76	0.76	2.47	2.47	2.47	0.58	0.58	0.58	0.02	0.02	0.02	0.02	0.02	0.02	0.01	0.01	0.01	0.01	0.01	0.01	0.55	0.55	0.55
		chronic illness	0.13	0.13	0.13	0.19	0.19	0.19	0.47	0.47	0.47	0.14	0.14	0.14	0.07	0.07	0.07	0.02	0.02	0.02	0.55	0.55	0.55	0.41	0.41	0.41	0.16	0.16	0.16
		asthma attacks	0.61	0.61	0.61	0.85	0.85	0.85	2.04	2.04	2.04	0.64	0.65	0.65	0.33	0.33	0.33	0.11	0.11	0.11	2.53	2.54	2.54	1.88	1.89	2.07	0.72	0.72	0.72
Total all emissions, pollutants, effects	Subtotal ambient CO	mortality	6.13	6.68	7.11	9.50	10.71	11.30	25.71	27.80	29.71	6.97	7.67	8.14	15.65	15.82	16.16	5.12	5.52	6.03	59.68	59.68	62.26	45.99					

