THE GLOBAL BURDEN OF ANTHROPOGENIC OZONE AND PARTICULATE MATTER AIR POLLUTION ON PREMATURE HUMAN MORTALITY

Susan Casper

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Approved by:

J. Jason West, Advisor

David Leith, Reader

Karin Yeatts, Reader

ABSTRACT
SUSAN CASPER: The Global Burden of Anthropogenic Ozone and Particulate Matter Air Pollution on Premature Human Mortality. (Under the direction of J. Jason West.)

Tropospheric ozone and fine particulate matter (PM) have increased significantly since preindustrial times and have been demonstrated to cause negative health impacts, including cardiovascular and respiratory mortality. Previous estimates of the global burden of outdoor PM on premature human mortality have been based on air quality measurements. Here, we use results from a global atmospheric chemistry and transport model simulation of ozone and PM concentrations in the preindustrial (1860) and the present (2000) to drive human mortality estimates. This method includes rural areas where measurements are often unavailable and avoids making assumptions for background air pollution. Depending on the concentration threshold applied, anthropogenic ozone is associated with about 282,000 to 362,000 global premature cardiopulmonary mortalities, with uncertainty ranging from 135,000 to 551,000 mortalities. Anthropogenic PM is associated with about 1.3 to 2.4 million global premature cardiopulmonary and lung cancer mortalities, with uncertainty ranging from 465,000 to 3.8 million mortalities.

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LIST OF ABBREVIATIONS AND SYMBOLS

AIRS	Aerometric Information Retrieval System
ACS	American Cancer Society
BC	Black carbon
CI	Confidence Interval
CRF	Concentration-Response Function
EPA	U.S. Environmental Protection Agency
GIS	Geographic Information System
HEI	Health Effects Institute
MOZART-2	Model of Ozone and Related Tracers, version 2
NAAQS	National Ambient Air Quality Standard
NCAR	National Center for Atmospheric Research
NO_3	Nitrate aerosols
NMMAPS	National Morbidity, Mortality, and Air Pollution Study
OC	Organic carbon
ORNL	Oak Ridge National Laboratory
PI	Posterior Interval
PM	Particulate Matter
ppb	Parts per billion
ppm	Parts per million
SO_4	Sulfate aerosols
UV	Ultraviolet

CHAPTER 1
INTRODUCTION

Human influence on the atmosphere has increased substantially since industrialization. The changing composition of the atmosphere resulting from human development has important consequences for the environment and human health. This research aims to quantify the impact of human-induced, or anthropogenic, outdoor air pollution on human health. Specifically, this research examines the annual global burden of mortality caused by anthropogenic ozone and fine particulate matter (PM), two of the most ubiquitous and deleterious air pollutants. By employing a global atmospheric chemistry model to simulate ozone and PM concentrations in urban and rural areas, it improves on previous methods used to estimate the global burden of disease due to monitored urban air pollution. This work also illustrates the geographical distribution of excess mortalities due to global anthropogenic air pollution, highlighting local areas where improvements to air quality may be particularly effective at reducing air pollution-related mortality. This introduction will provide general background on anthropogenic contributions to air pollution and trends over time, the health effects resulting from exposure to air pollution, and the objectives of this study. Methodology, results, and conclusions and implications will then be discussed.

1.1 Background

While ozone in the stratosphere shields damaging ultraviolet (UV) radiation from reaching the surface of the Earth, tropospheric, or ground-level, ozone causes deleterious health effects. Tropospheric ozone is not emitted directly, but is formed in the troposphere through reactions between precursors that take place in sunlight.

Emission of ozone precursors, nitrogen oxides and volatile organic compounds, can be natural or anthropogenic. While natural precursor emissions may also change throughout time as a result of changing climate, anthropogenic precursor emissions have increased substantially in the last century, leading to a large increase in ground-level ozone. The main anthropogenic sources of ozone precursors are industrial facilities and motor vehicles.

PM is both emitted directly and formed indirectly through reaction in the atmosphere. Coarse PM, PM greater than about 2.5 μm in diameter, is largely transferred to the atmosphere by natural physical processes, such as wind-produced dust and sea salt. Conversely, fine PM, PM less than 2.5 μm in diameter, is emitted by anthropogenic sources or produced by reaction of gaseous emissions, such as nitrogen and sulfur oxides, in the atmosphere. The main anthropogenic sources of fine PM are motor vehicles and power plants. Fine PM is associated with more deleterious health effects than coarse PM, because these tiny particles can penetrate deep into the lungs and may consist of more toxic materials [Pope & Dockery, 2006]. This paper, therefore, focuses on fine PM, or $PM_{2.5}$, which will hereafter be referred to as PM. Any references to coarse PM will be explicitly stated.

Ground-level concentrations of ozone and PM have increased substantially since preindustrial times [Volz & Kley, 1988; Staehelin *et al.*, 2001; Schultz *et al.*, 2006; Horowitz, 2006; Ginoux *et al.*, 2006]. While the greatest increases in ozone and PM concentrations have occurred in heavily industrialized urban areas, increases in background concentrations have also been observed in remote regions. Because the lifetime of ozone (and its precursors) is sufficiently long that it can be transported between continents, the global background concentration of ozone has roughly doubled that in preindustrial times [Volz & Kley, 1988; Akimoto, 2003; Vingarzan, 2004]. Though the lifetime of PM is shorter than that of ozone, it is long enough that it can distribute

to form elevated regional background concentrations [Akimoto, 2003].

Associations between these pollutants and human health endpoints, found by short-term and long-term epidemiology studies, form the basis for regulatory action in the US, Europe, and many other countries. In the US, both ozone and PM are regulated by National Ambient Air Quality Standards (NAAQS), which are set by the US Environmental Protection Agency (EPA) to protect human health without regard to cost. NAAQS were first established in 1970 and are continually strengthened over time to reflect new scientific understanding. In 2008, the daily 8-hr. maximum standard for ozone was lowered from 80 parts per billion (ppb) to 75 ppb. While the original NAAQS for PM was based on PM_{10}, or all PM less than 10 μm in diameter, a separate standard for fine PM was set in 1997 after linkages were found between fine PM and health problems, such as increased hospital admissions, emergency room visits, and premature death for people with lung and heart disease. The annual average PM_{10} standard was eventually revoked in 2006 because evidence does not suggest a link between long-term exposure to coarse PM and deleterious health effects, although the 24-hr. average standard of 150 $\mu g/m^3$ was retained. The current annual average standard for fine PM is 15 $\mu g/m^3$.

1.2 Health Effects of Air Pollution

Both ozone and PM have been demonstrated to cause negative health impacts, including premature mortality. Ozone has been associated with premature mortality in many city-specific daily time-series studies. Meta-analyses of these studies have found a consistent relationship between ozone and daily mortality [Bell *et al.*, 2005; Ito *et al.*, 2005; Levy *et al.*, 2005]. Recently, larger daily time-series studies have used large datasets of urban regions in the US and Europe, with consistent results [Bell *et al.*, 2004, 2005; Gryparis *et al.*, 2004]. While these studies do not strongly suggest

the cause of death, they show that the relationship between ozone concentration and mortality holds over a wide range of concentrations. On an individual level, there may be biological concentration thresholds of exposure above or below which no health effects occur. However, to date, there is no clear evidence from these studies of population-level low-concentration or high-concentration thresholds [Bell *et al.*, 2006; Gryparis *et al.*, 2004].

Daily time-series studies also show strong relationships between PM and mortality. Some studies have utilized sudden changes in PM concentrations to document associated decreases in mortality [Pope *et al.*, 1992; Clancy *et al.*, 2002; Hedley *et al.*, 2002]. Long-term cohort studies have also connected PM with mortality by following the exposure of a group of participants for years. Three long-term cohort studies in the US [Dockery *et al.*, 1993; Pope *et al.*, 2002; Laden *et al.*, 2006] and one in Europe [Hoek *et al.*, 2002] demonstrated that PM has a much greater effect on mortality in the long term than the daily time-series studies suggest. These studies show that the relationship between PM and mortality holds over a wide range of concentrations [Laden *et al.*, 2006]. Like ozone, there is no evidence to date of population-level low-concentration or high-concentration thresholds [Schwartz & Zanobetti, 2000].

1.3 Objectives

One recent study examined the global burden of mortality due to outdoor air pollution [Cohen *et al.*, 2004, 2005]. This study estimated that about 800,000 premature mortalities, or about 1.2% of all deaths, occur per year globally due to outdoor fine PM in urban areas only. Their estimate was based on PM only, ignoring ozone. In addition, since this study relied on PM measurements, it included only urban areas, where monitors are most often located, and excluded rural areas. The population included in this study was about 30% of the total global population. In cities where

no monitor data were available, PM concentrations were inferred using econometric methods. Because anthropogenic emissions have increased both ozone and PM outside of urban areas, there is reason to believe that Cohen *et al.* [2004] may substantially underestimate the global burden of anthropogenic air pollution on human mortality.

This study aims to improve upon the method used for estimating the number of premature mortalities resulting from global anthropogenic air pollution. Specifically, we use results from global atmospheric chemistry and transport model (MOZART-2) simulations of ozone and PM concentrations in 1860, the preindustrial base case, and 2000, the present day case, to drive human mortality estimates. Global air quality models are often used to simulate past, present, and future emission scenarios. Recent studies have used global air quality models to estimate radiative climate forcing of trace gases and particles [Forster *et al.*, 2007] and the health burden due to future changes in emissions [West *et al.*, 2006, 2007; Jacobson, 2008] or to changes in one sector's emissions [Corbett *et al.*, 2007]. Global air quality models have not been used previously to quantify the global burden of anthropogenic air pollution on human mortality. Here, the use of an air quality model allows estimation of mortality in regions for which ground-level concentration measurements are unavailable and omitted from estimates based on direct air quality measurements. In addition, by simulating the preindustrial base case, we are able to isolate the mortality due to anthropogenic pollution and avoid making assumptions for background ozone and PM concentrations.

CHAPTER 2

METHODOLOGY

2.1 Experimental Design

Results of a global air quality model are used to derive the anthropogenic contribution to ground-level ozone and PM concentrations. Anthropogenic pollution is defined as the geographically-distributed difference in ozone and PM concentrations simulated for the present (2000) and preindustrial (1860). Health impact functions from the epidemiological literature for both ozone and PM are used to relate pollutant concentrations with premature human mortality. Results are plotted on a world map to illustrate geographically-distributed mortality. Finally, sensitivities to varying concentration-response factors and health effect thresholds are examined.

2.2 Health Impact Functions

Health impact functions relate air pollutant concentrations with changes in incidence of human health endpoints. Epidemiology studies have found that the natural logarithm of relative risk increases roughly linearly with concentration, which is expressed mathematically by Equation 2.1.

$$RR = \exp^{\beta \Delta X} \tag{2.1}$$

where RR is relative risk and β is the concentration-response factor (fraction excess mortalities per unit change in pollutant concentration, X).

The health impact function derived by epidemiology studies and used in this study for both ozone and PM is:

$$\Delta Mort = y_0(1 - \exp^{-\beta \Delta X})Pop \tag{2.2}$$

where $\Delta Mort$ is the change in human mortalities due to a change in pollutant concentration (ΔX), y_0 is the baseline mortality rate for a given population, β is the concentration-response factor, X is the pollutant concentration, and Pop is the total population exposed. The derivation of this function and the decision to use this function over others in the literature (e.g. Ostro [2004]) is described in Appendix A.

2.2.1 Concentration-Response Factor (CRF)

Concentration-Response Factors (CRFs) relate a unit change in air pollution with relative risk, where relative risk is the ratio of the probability of health endpoint incidence in the exposed group vs. the unexposed group (Appendix A). Where the natural logarithm of relative risk increases linearly with concentration, CRFs are calculated by the following equation, which is a transformation of the equation for relative risk (Equation 2.1):

$$\beta = \frac{lnRR}{\Delta X} \tag{2.3}$$

where β is the CRF, RR is relative risk, and ΔX is the change in concentration associated with the change in relative risk.

For ozone mortality, CRFs are calculated from the 8-hour daily maximum ozone relative risks from Bell *et al.* [2004], because it was based on 95 US cities and, therefore, was not subject to publication bias that may influence meta-analyses of single-city studies (e.g. Bell *et al.* [2005]; Ito *et al.* [2005]; Levy *et al.* [2005]). Bell *et al.* [2004] calculated a national average relative rate of mortality associated with short-term exposure to ambient ozone. For each 10 ppb increase in 8-hour daily maximum

Table 2.1: Relative risks for 1979-1983, 1999-2000, and the average of the two time periods from Pope *et al.* [2002] with 95% confidence intervals (CI)

Cause of Mortality	1979-1983	1999-2000	Average
All-cause	1.04 (1.01-1.08)	1.06 (1.02-1.10)	1.06 (1.02-1.11)
Cardiopulmonary	1.06 (1.02-1.10)	1.08 (1.02-1.14)	1.09 (1.03-1.16)
Lung Cancer	1.08 (1.01-1.16)	1.13 (1.04-1.22)	1.14 (1.04-1.23)

ozone, total daily non-accidental mortality increased by 0.52% (95% posterior interval [PI], 0.27%-0.77%) and daily cardiovascular and respiratory mortality increased by 0.64% (95% PI, 0.31%-0.98%).

For PM mortality, CRFs are calculated from annual average PM relative risks from Pope *et al.* [2002]. The Pope *et al.* [2002] study was chosen because it has the largest sample size (about 500,000 adults) of the cohort studies. Associations between long-term exposure to PM and all-cause, cardiopulmonary, and lung cancer mortality were based on monitored PM data (ranging from 7.5-30 $\mu g/m^3$) along with data on individual risk factors from the American Cancer Society (ACS) Cancer Prevention II study. Relative risks associated with PM exposure were reported for 1979-1983, for 1999-2000 and for the integrated average of the two periods. While the results for 1979-1983 are reported in the abstract and conclusion, we chose to use the results from the average of 1979-1983 and 1999-2000 to calculate mortality estimates, because they were thought to be less sensitive to the specific conditions influencing each time period. For a 10 $\mu g/m^3$ increase in PM, total, cardiopulmonary, and lung cancer mortalities increased by 6% (95% CI, 2%-11%), 9% (3%-16%), and 14% (4%-23%) for the average of the two time periods (Table 2.1). We also examine the sensitivity of mortality estimates to varying CRFs (Section 3.3).

These relative risks were estimated in the US, and similar results have been demonstrated in Europe for both PM [Hoek *et al.*, 2002] and ozone [Anderson *et al.*, 2004].

To date, no long-term PM cohort study has been conducted in the developing world, but short-term PM and ozone studies in developing nations have found relationships comparable to short-term studies in North America and Europe [HEI International Scientific Oversight Committee, 2004]. However, these studies are less conclusive than those conducted in North America and Europe because they generally each focus on one city and may not be representative of the developing world as a whole. Short-term studies are further subject to uncertainty concerning the timing of exposure and resulting health endpoints. Since no evidence to the contrary yet exists, it is assumed for this study that mortality relationships found in the US are valid globally. This assumption is further supported by evidence that concentration-mortality relationships do not vary significantly by sex, age, and race [Zanobetti *et al.*, 2000; Pope *et al.*, 2002; Bell *et al.*, 2004], though some sensitive populations may be at a higher risk.

2.2.2 Pollutant Concentrations

This study uses simulations of ozone and PM concentrations produced with the Model of Ozone and Related Chemical Tracers, version 2 (MOZART-2) [Horowitz *et al.*, 2003] with meteorological inputs from the middle atmospheric version of the National Center for Atmospheric Research (NCAR) Community Climate Model (MACCM3) [Kiehl *et al.*, 1998]. MOZART-2 has a horizontal resolution of 2.8° latitude by 2.8° longitude with 34 vertical levels. It includes 63 chemical species with detailed chemistry for tropospheric ozone, nitrogen oxides, and hydrocarbons. Simulated aerosol species are sulfate, nitrate, ammonium, black carbon, organic carbon (includes primary organic carbon and secondary organic aerosols), and dust. Here, fine PM concentration is defined as the total concentration of sulfate (SO_4), nitrate (NO_3), black carbon (BC), and organic carbon (OC) aerosols. Ozone and PM concentrations in the first vertical level only were used to simulate exposure at the surface of the Earth.

Present day (2000) and preindustrial (1860) ozone and PM concentrations used for this study are simulated and described fully by Horowitz [2006]. For the preindustrial case, fossil fuel burning was set to zero, emissions from burning of biofuels, savannah, tropical forests, and agricultural waste were assumed to be 10% of 1990 values, and natural emissions (including dust) were identical for both simulations. Present day emissions are from EDGAR v2.0 [Olivier *et al.*, 1996]. Compared with the preindustrial, the present day burden of tropospheric ozone has increased by 50% and sulfate and carbonaceous PM burdens have increased by factors of 3 and 6 [Horowitz, 2006]. For this study, the anthropogenic contribution to ozone and PM was defined as the difference between the simulated present day and preindustrial concentrations.

Horowitz *et al.* [2003] and Horowitz [2006] evaluated ozone above the surface. Surface ozone concentrations simulated for 2000 were further compared with observations from one year between 1998-2004 (based on data availability) to determine whether an overall or regional bias exists (Appendix B). Modeled concentrations were compared with the Climate Modeling and Diagnostics Laboratory (CMDL) monitoring network (mean bias=2.5ppb) for remote locations around the world, Clean Air Status and Trends Network (CASTNet) (mean bias=2.9ppb) for the US, the European Monitoring and Evaluation Programme (EMEP) (mean bias=-0.2ppb) for Europe, and the Acid Deposition Monitoring Network in East Asia (EANET) (mean bias=0.4ppb) for Japan. These comparisons showed that surface ozone is well simulated by MOZART-2 and that no bias correction is necessary.

The PM concentration used in this study is the sum of the concentrations of simulated PM components (OC, BC, SO_4, and NO_3), which are assumed to be 2.5 μm or less in diameter. Modeled surface PM concentrations were compared with observations by Ginoux *et al.* [2006] and were generally found to be estimated within a factor of 2 in remote locations, Europe, and the US, with a tendency to be overestimated.

Table 2.2: The population-weighted annual average 8-hr. daily maximum ozone concentrations (ppb) and annual average PM concentrations (μg/m^3), using $\beta_{avg.}$ [Pope *et al.*, 2002] for the Horowitz [2006] simulations of 1860 and 2000.

	Ozone (ppb)		OC (μg/m^3)		BC (μg/m^3)		NO_3 (μg/m^3)		SO_4 (μg/m^3)		Total PM (μg/m^3)	
	1860	2000	1860	2000	1860	2000	1860	2000	1860	2000	1860	2000
Africa	11.64	19.75	0.36	3.18	0.05	0.57	0.00	0.07	0.32	2.10	0.73	5.92
North America	21.54	47.82	0.83	2.10	0.06	0.61	0.00	0.46	0.24	3.87	1.14	7.03
Europe	22.10	35.55	0.44	5.21	0.04	1.21	0.00	1.17	0.23	4.44	0.72	12.03
Asia	19.81	41.08	0.54	6.48	0.09	1.95	0.01	1.00	0.30	7.31	0.93	16.73
South America	13.46	25.08	0.24	2.29	0.03	0.47	0.00	0.03	0.55	2.29	0.83	5.09
Oceania	14.58	24.47	0.19	0.49	0.01	0.09	0.00	0.01	0.58	1.56	0.79	2.16
World	18.66	36.99	0.51	5.25	0.07	1.47	0.00	0.78	0.31	5.67	0.89	13.17

The change in ozone and PM concentrations from preindustrial to present day was presented by Horowitz [2006] and is shown geographically-distributed in Figure 2.1. Table 2.2 shows the population-weighted surface concentrations of ozone and PM in each of six continents and for the whole world in 1860 and 2000. The global population-weighted annual average 8-hr. daily maximum ozone increased by 28.3 ppb (from 18.7 ppb in 1860 to 37.0 ppb in 2000) and the global population-weighted annual average PM increased by 12.3 μg/m^3 (from 0.9 μg/m^3 in 1860 to 13.2 μg/m^3 in 2000).

2.2.3 Population

Spatially-distributed population is based on the 2006 population from the LandScan database [Oak Ridge National Laboratory, 2008], which apportions population to grid cells based on likelihood coefficients, such as proximity to roads, slope, land cover, and nighttime lights. Fine resolution LandScan data (30" by 30") was then mapped onto the coarse MOZART-2 grid (Appendix C). Since the Bell *et al.* [2004] included the total population and Pope *et al.* [2002] study included only individuals at least 30 years of age, it is assumed for this study that the population exposed to ozone is the whole population, while the population exposed to PM is the population aged

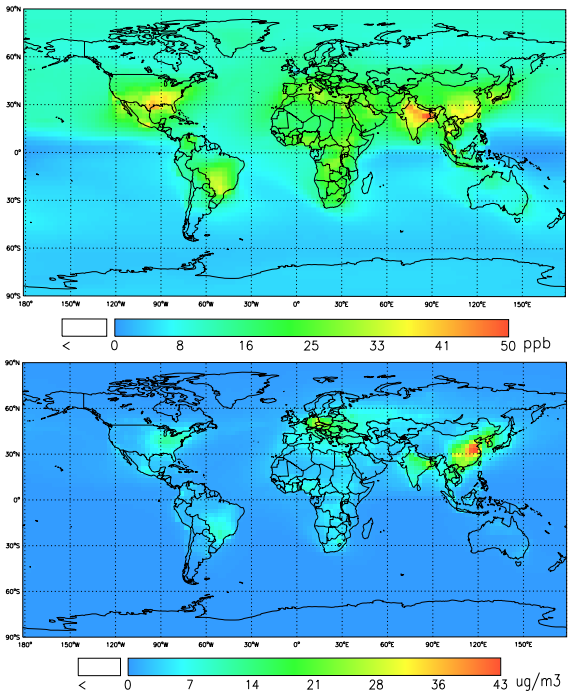

Figure 2.1: Spatial distribution of the change in (top) annual average 8-hr. daily maximum ozone concentrations (ppb) and (bottom) annual average PM (μg/m^3) from the Horowitz [2006] simulations of 1860 to 2000.

30 and older. The fraction of the population aged 30 and older was provided by the World Health Organization [World Health Organization, 2004] for 14 world regions (Appendix C). When a gridcell contained portions of more than one country within it, an area-weighted average of the fraction of the population aged 30 and older was calculated using a Geographic Information System (GIS).

2.2.4 Baseline Mortality Rates

Baseline rates of all-cause, non-accidental, cardiopulmonary, and lung cancer mortality were provided by the World Health Organization's 2004 World Health Report [World Health Organization, 2004] for 14 world regions and for 66 individual countries, and mapped onto the MOZART-2 grid. The data for individual countries are

categorized by broad age ranges with no cutoff at age 30. Therefore, mortality rates for the population aged 25 and older were used, and it was assumed that, as a whole, the population aged 25-29 reacts similarly to PM exposure as the population aged 30 and older in the Pope *et al.* [2002] study. When country-specific data was not available, population-weighted average mortality rates for the remaining countries in each of 14 world regions were back-calculated using the WHO regional baseline mortality rates. When a gridcell contained portions of more than one country within it, an area-weighted average mortality rate was calculated using a GIS. Mapped baseline mortality rates are shown in Appendix D.

2.3 Mortality Calculation

Equation 2.2 is applied in each grid cell to calculate premature mortalities separately for ozone and PM, using the exposed population of that grid cell and the corresponding country-specific baseline mortality rates. Non-accidental and cardiopulmonary mortality is calculated for ozone based on the relative risk estimates by Bell *et al.* [2004]. All-cause (includes accidental and non-accidental), cardiopulmonary, and lung cancer mortality are calculated for PM based on the relative risk estimates by Pope *et al.* [2002]. While calculated cardiopulmonary and lung cancer mortalities are additive, they are each a subset of non-accidental and all-cause mortality. Therefore, non-accidental and all-cause mortality calculations are not additive.

For ozone, premature mortalities are calculated each day based on the difference between 1860 and 2000 8-hr. daily maximum ozone concentrations. Since it is applied on each day, β values are divided by 365.25 days per year. Daily premature mortalities due to ozone are then summed to yield mortalities per year due to short-term ozone exposure. Premature mortalities due to PM are calculated based on the differences in annual average PM concentrations between 1860 and 2000.

While the epidemiological literature provides no evidence for low-concentration thresholds for either ozone or PM [Schwartz & Zanobetti, 2000], the exposure-response relationship at low concentrations are uncertain. Therefore, results are calculated assuming no low-concentration threshold and assuming a low-concentration threshold for both ozone and PM. Below these thresholds, changes in ozone and PM are assumed to have no effect on human mortality. For ozone, a threshold of 25 ppb, or roughly the current background concentration, was applied. The threshold for PM was set at 7.5 $\mu g/m^3$, the lowest value included in the Pope *et al.* [2002] study. Cohen *et al.* [2004, 2005] also used a low-concentration threshold of 7.5 $\mu g/m^3$ in the estimation of global mortality due to urban PM. The threshold in Cohen *et al.* [2004, 2005], however, applied to total $PM_{2.5}$, while here the threshold applies only to the species included in our definition of simulated $PM_{2.5}$ (sulfates, nitrates, organic carbon, and black carbon). Using a threshold replaces the simulated preindustrial concentrations, since preindustrial concentrations are almost always below these levels.

CHAPTER 3

RESULTS AND DISCUSSION

3.1 Premature Mortalities due to Anthropogenic Ozone

Premature mortalities resulting from short-term ozone exposure are shown in Table 3.1. Here, the 95% posterior intervals (PI) represent the reported uncertainty in the CRF only and do not include uncertainty from other sources, which are described in Section 4. With no concentration threshold, anthropogenic ozone is estimated to cause abut 640,000 (95% PI, 411,000-857,000) non-accidental and 362,000 (95% PI, 173,000-551,000) cardiopulmonary mortalities worldwide. When a low-concentration threshold of 25 ppb is assumed, however, global non-accidental and cardiopulmonary mortalities are reduced by about 20%. Over 60% of ozone mortalities occur in Asia, which is densely populated and highly polluted. Contrastingly, only 6% of ozone mortalities occur in North America. About 19% of non-accidental ozone mortalities but only 11% of cardiopulmonary ozone mortalities occur in Africa. This discrepancy is mainly due to the high baseline non-accidental mortality rates, but relatively low baseline cardiopulmonary mortality rates in Africa.

Figures 3.1 and 3.2 show the geographic distribution of non-accidental and cardiopulmonary mortalities due to anthropogenic ozone. Mortalities are most dense in highly populated areas, particularly in China and India. When mortalities are normalized to population, as shown on the right side of Figures 3.1 and 3.2, the ozone mortality rates per 1 million people are more evenly distributed across the globe. Non-accidental mortalities per 1 million people are highest in Africa, where baseline mortality rates are high, and northern India, where modeled ozone concentrations

Table 3.1: Annual premature non-accidental and cardiopulmonary mortalities resulting from short-term anthropogenic ozone exposure. Mortalities are based on CRFs from Bell *et al.* [2004] and 95% posterior intervals are shown next to each estimate.

	Non-Accidental (000s)		Cardiopulmonary (000s)	
threshold (ppb)	0	25	0	25
Africa	123 (79 - 165)	99 (64 - 133)	40 (19 - 60)	32 (15 - 49)
North America	40 (26 - 54)	34 (22 - 45)	23 (11 - 35)	19 (9 - 30)
Europe	61 (39 - 82)	47 (30 - 63)	44 (21 - 68)	34 (16 - 51)
Asia	396 (255 - 530)	310 (199 - 415)	244 (117 - 372)	191 (92 - 291)
South America	18 (12 - 24)	10 (7 - 14)	10 (5 - 15)	5 (3 - 8)
Oceania	1 (1 - 1)	0 (0 - 0)	1 (0 - 1)	0 (0 - 0)
World	640 (411 - 857)	500 (321 - 669)	362 (173 - 551)	282 (135 - 429)

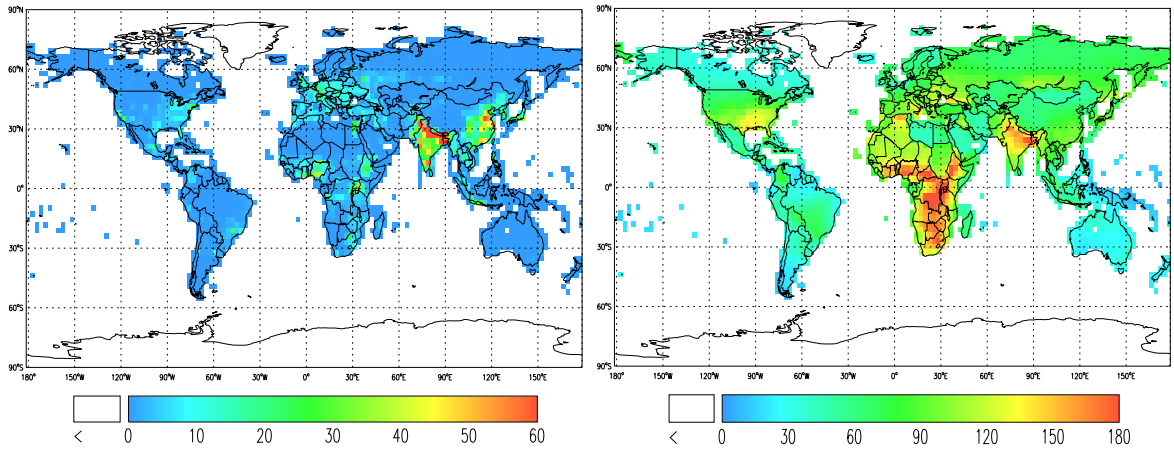

Figure 3.1: Spatial distribution of annual non-accidental mortalities per 1000 km^2 (left) and rate of non-accidental mortalities per 1E6 people (right) attributed to anthropogenic ozone, when no threshold is assumed.

are high. However, cardiopulmonary mortalities per 1 million people are highest in eastern Europe, due to high baseline cardiopulmonary mortality rates combined with about average ozone concentrations.

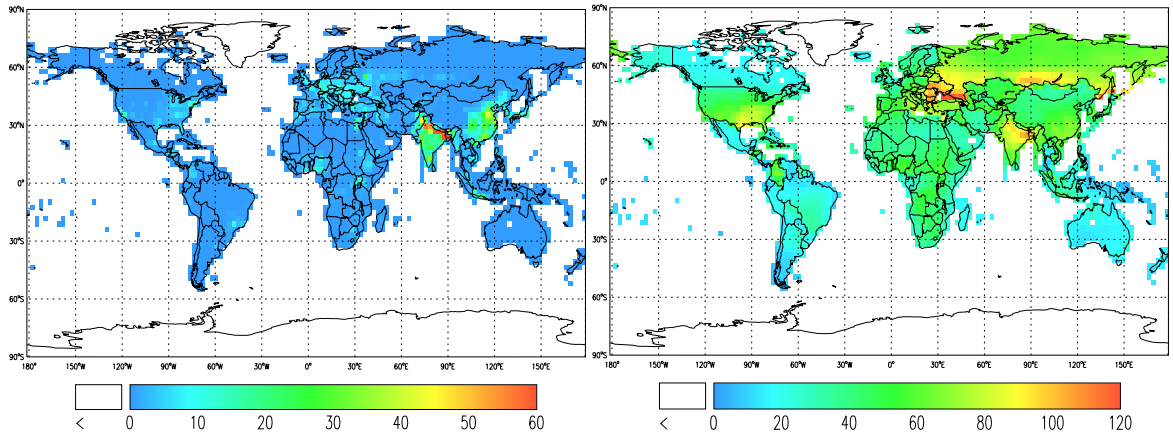

Figure 3.2: Spatial distribution of annual cardiopulmonary mortalities per 1000 km^2 (left) and rate of cardiopulmonary mortalities per 1E6 people (right) attributed to anthropogenic ozone, when no threshold is assumed.

3.2 Premature Mortality due to Anthropogenic PM

Premature all-cause, cardiopulmonary, and lung cancer mortalities resulting from long-term PM exposure are shown in Table 3.2. Assuming no concentration threshold, long-term PM exposure is estimated to result in 2.826 (95% CI, 0.996-4.852) million all-cause mortalities (includes accidental and non-accidental causes) annually, 2.172 (95% CI, 0.786-3.533) million cardiopulmonary mortalities, and 192,000 (95% CI, 63,000-282,000) lung cancer mortalities. When a low-concentration threshold of 7.5 $\mu g/m^3$ is assumed, all-cause, cardiopulmonary, and lung cancer mortalities each decrease by about half. Cardiopulmonary mortalities resulting from PM exposure are about 77% of all-cause PM mortalities. Though lung cancer CRFs are much higher than all-cause and cardiopulmonary CRFs, lung-cancer mortalities are only about 7% of all-cause PM mortalities due to low baseline incidence rates. In all cases, about three-quarters of mortalities occur in Asia. Europe has the second largest percentage of all-cause PM mortalities, mainly due to high PM concentrations and dense population.

The geographic distribution of all-cause, cardiopulmonary, and lung cancer mor-

Table 3.2: Annual all-cause, cardiopulmonary, and lung cancer mortalities resulting from long-term anthropogenic PM exposure. Mortalities are based on the average of 1979-1983 and 1999-2000 CRFs from Pope *et al.* [2002] and 95% confidence intervals are shown next to each estimate.

	All-Cause (000s)		Cardiopulmonary (000s)		Lung Cancer (000s)	
threshold ($\mu g/m^3$)	0	7.5	0	7.5	0	7.5
Africa	168 (58 - 297)	9 (3 - 16)	88 (31 - 148)	5 (2 - 9)	2 (1 - 4)	0 (0 - 0)
North America	115 (40 - 202)	29 (10 - 52)	77 (27 - 130)	20 (7 - 34)	15 (5 - 23)	4 (1 - 7)
Europe	442 (154 - 768)	187 (65 - 328)	357 (127 - 591)	153 (54 - 255)	40 (13 - 59)	18 (6 - 27)
Asia	2056 (729 - 3506)	1269 (448 - 2172)	1620 (591 - 2615)	1006 (365 - 1632)	132 (44 - 193)	88 (29 - 129)
South America	43 (15 - 76)	5 (2 - 8)	28 (10 - 48)	3 (1 - 5)	2 (1 - 3)	0 (0 - 0)
Oceania	2 (1 - 3)	0 (0 - 0)	1 (0 - 2)	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
World	2826 (996 - 4852)	1499 (528 - 2576)	2172 (786 - 3533)	1187 (429 - 1935)	192 (63 - 282)	111 (36 - 163)

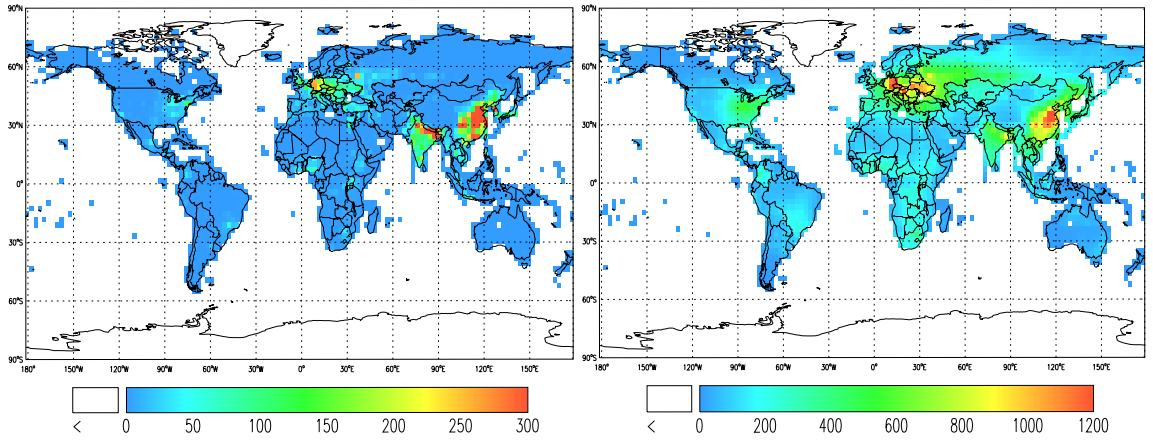

Figure 3.3: Spatial distribution of annual all-cause mortalities per 1000 km^2 (left) and rate of all-cause mortalities per 1E6 people (right) attrubuted to anthropogenic PM, when no threshold is assumed.

talities are shown in Figures 3.3, 3.4, and 3.5. Like the ozone results, these figures show that the highest numbers of mortalities occur in population-dense areas. However, when the results are population-normalized as shown on the right in Figures 3.3, 3.4, and 3.5, the highest rates of mortalities per 1 million people are found in Europe, Asia, and the eastern US. Since PM has a shorter lifetime than ozone, high PM concentrations are more localized, while ozone concentrations are more widespread (Figure 2.1). Combined with high cardiopulmonary and lung cancer mortality rates in these areas, high localized PM concentrations in Asia, Europe, and the eastern US

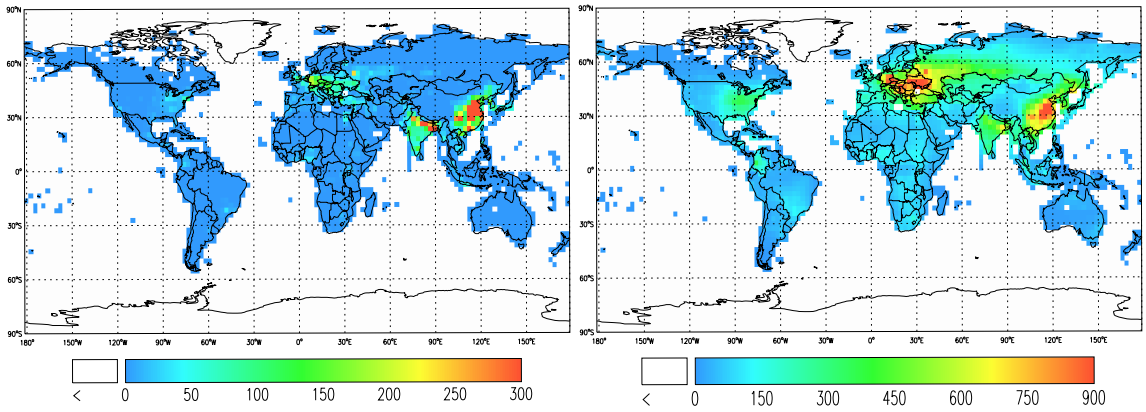

Figure 3.4: Spatial distribution of annual cardiopulmonary mortalities per 1000 km^2 (left) and rate of cardiopulmonary mortalities per 1E6 people (right) attributed to anthropogenic PM, when no threshold is assumed.

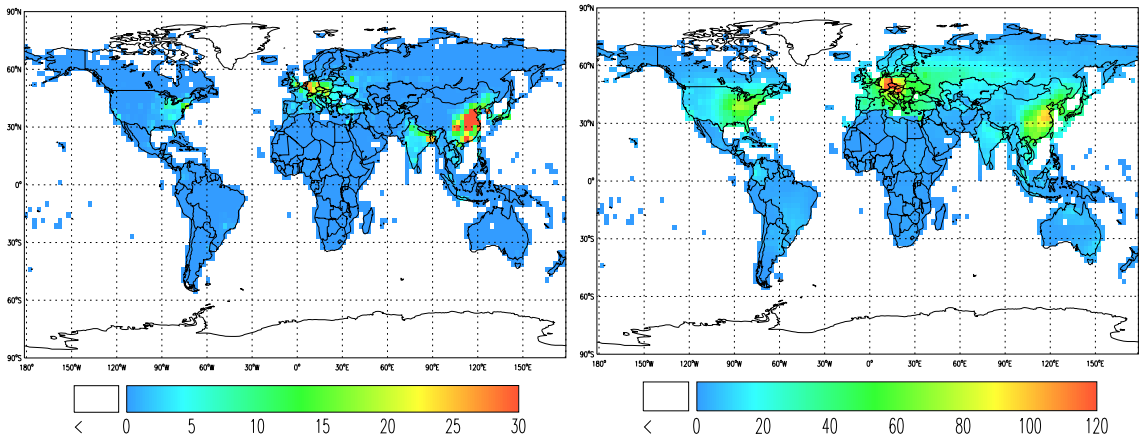

Figure 3.5: Spatial distribution of annual lung cancer mortalities per 1000 km^2 (left) and rate of lung cancer mortalities per 1E6 people (right) due to anthropogenic PM, when no threshold is assumed.

lead to high rates of PM mortality per 1 million people.

3.3 Sensitivity Analysis

We examine the sensitivity of mortality results to changing parameters, such as concentration and CRFs (Tables 3.3 and 3.4). The effect of the ozone CRF was tested by applying the average CRF from three meta-analyses of short-term ozone-mortality studies published in 2005 [Bell *et al.*, 2005; Ito *et al.*, 2005; Levy *et al.*,

2005]. The relationships between ozone and total mortality found by these studies were consistent with each other and with Bell *et al.* [2004], which was the basis for the ozone mortality results presented in Section 3.1. Applying the average of the median CRFs from the three meta-analyses increases ozone mortality by about 20% (Table 3.3). The confidence interval for the results based on the three meta-analyses is based on the lowest and highest values in the 95% confidence intervals of the CRFs found by the three studies (not averages of the low and high ends).

We also examined the effect of ozone concentrations 25% lower and higher than the concentrations simulated by MOZART-2 for both 1860 and 2000. Though modeled concentrations were compared with observations and found to be generally unbiased, we were only able to do comparisons in limited parts of the world where monitor data are available. Therefore, we tested the effect of higher and lower ozone concentrations in case the modeled concentrations are biased in some way. With no concentration threshold, ozone mortality estimates changed proportionally with the changed concentration. However, when a concentration threshold of 25 ppb was applied, a 25% change in concentration resulted in a greater than proportional change in mortalities (Table 3.3). This is likely due to the increased number of grid cells with concentrations below the threshold when simulated concentrations are reduced and the increased number of grid cells with concentrations above the threshold when simulated concentration is increased.

Table 3.3: Sensitivity of ozone mortality results to changing parameters

	Non-Accidental (000s)		Cardiopulmonary (000s)	
threshold (ppb)	0	25	0	25
$\beta_{\text{Bell et al. (2004)}}$	640 (411 - 857)	500 (321 - 669)	362 (173 - 551)	282 (135 - 429)
$\beta_{\text{Bell et al. (2004)}}$, 75% O_3	481 (309 - 644)	261 (168 - 350)	272 (130 - 415)	147 (70 - 225)
$\beta_{\text{Bell et al. (2004)}}$, 125% O_3	798 (514 - 1068)	717 (461 - 959)	451 (217 - 686)	404 (149 - 614)
$\beta_{\text{meta-analyses}}$	764 (440 - 1415)	597 (344 - 1106)	n/a	n/a

We also examined the sensitivity of the PM mortality results to different CRFs.

Table 3.4: Sensitivity of PM mortality results to changing parameters

	All-Cause (000s)		Cardiopulmonary (000s)		Lung Cancer (000s)	
threshold ($\mu g/m^3$)	0	7.5	0	7.5	0	7.5
$\beta_{avg.}$	2826 (996 - 4852)	1499 (528 - 2576)	2172 (786 - 3533)	1187 (429 - 1935)	192 (63 - 282)	111 (36 - 163)
$\beta_{avg.}$, 75% $PM_{2.5}$	2149 (751 - 3726)	888 (310 - 1542)	1662 (594 - 2740)	711 (253 - 1174)	148 (48 - 221)	68 (22 - 102)
$\beta_{avg.}$, 125% $PM_{2.5}$	3485 (1239 - 5925)	2150 (764 - 3658)	2661 (976 - 4273)	1684 (617 - 2711)	232 (77 - 336)	154 (51 - 223)
$\beta_{79\text{-}83}$	1937 (505 - 3668)	1027 (268 - 1947)	1508 (532 - 2381)	823 (290 - 1302)	119 (16 - 213)	68 (9 - 123)
$\beta_{Laden\ et\ al.\ (2006)}$	6635 (3253 - 9632)	3525 (1726 - 5122)	5396 (2979 - 7253)	2964 (1631 - 3994)	316 (0 - 552)	183 (0 - 322)

Relative risk estimates for 1979-1983, 1999-2000, and the average of the two time periods were reported by Pope *et al.* [2002]. Estimates for 1979-1983 are more conservative than the estimates for 1999-2000, which were used to drive the mortality results in Section 3.2. About one-third fewer mortalities are predicted when the 1979-1983 CRF is applied (Table 3.4). More recently, a reanalysis of the Harvard Six Cities adult cohort study extended mortality follow-up for 8 years in a period of reduced air pollution concentrations [Laden *et al.*, 2006]. This study found significantly higher relative risk estimates for overall mortality (1.16; 95% CI, 1.07-1.26) and mortality due to cardiopulmonary disease (1.28; 95% CI, 1.13-1.44) and lung cancer (1.27; 95% CI, 0.96-1.69) for a 10 $\mu g/m^3$ increase in PM. When relative risks from Laden *et al.* [2006] are applied, mortality results increases by a factor of 2 to 3 (Table 3.4).

For the results presented in Section 3.2, 100% of modeled sulfate, nitrate, OC, and BC was assumed to be $PM_{2.5}$, but the true $PM_{2.5}$ fraction is likely to be between 50% and 100%. We test the effect of this assumption on mortality results by changing the fraction assumed to be $PM_{2.5}$ to 75%. Since comparisons with observations showed that simulated PM was generally biased high, reducing PM by 25% everywhere also tests the impact of reducing bias. We also tested the effect of increasing simulated PM concentrations by 25% to determine the effect of limiting PM to only four species (nitrates, sulfates, organic carbon, and black carbon). With no concentration threshold, the PM mortality estimates change proportionally with the change

in concentration (Table 3.4). However, like ozone, when a low-concentration threshold is applied, a 25% change in concentration resulted in a greater than proportional change in mortality. Again, this is most likely due to the increased number of grid cells with concentrations below the threshold when simulated concentrations are reduced and the increased number of grid cells with concentrations above the threshold when simulated concentration is increased.

CHAPTER 4
SUMMARY AND CONCLUSIONS

The global burden of mortality due to anthropogenic ozone and PM was estimated using a global atmospheric chemistry model and health impact functions. Depending on the concentration threshold applied, anthropogenic ozone is associated with about 282,000 to 362,000 global premature cardiopulmonary mortalities, with uncertainty ranging from 135,000 to 551,000 mortalities. Anthropogenic PM is associated with about 1.3 to 2.4 million global premature cardiopulmonary and lung cancer mortalities, with uncertainty ranging from 465,000 to 3.8 million mortalities. Cardiopulmonary and lung cancer mortalities are presented here because they are not affected by regional differences in baseline mortality rates when extrapolating US CRFs to the global population. However, since ozone and PM may affect other causes of mortality, these estimates are likely to be conservative and lower than our estimates for non-accidental and total mortalities. The geographic distribution of results shows that while the density of mortalities is highest in population-dense areas, mortalities also occur in less populous areas that have been affected by the increased global background of air pollution since preindustrial times. Sensitivity analysis demonstrates that these results are highly dependent on the assumptions on which the health impact calculation is based.

These results are a factor of 3 higher than the 800,000 mortalities due to urban PM estimated by Cohen *et al.* [2004], which applied the more conservative 1979-1983 CRFs from Pope *et al.* [2002] and assumed a low-concentration threshold of 7.5 $\mu g/m^3$. When the same assumptions for low-concentration thresholds and CRFs are applied here, mortality estimates are still about 5% higher than those found in Cohen

et al. [2004], due to the inclusion of rural populations. The urban population included in Cohen *et al.* [2004] was about 30% of the total global population. Mortalities are likely to be underestimated due to the coarse grid size of the global atmospheric chemistry model. Peaks in ozone and PM concentrations that usually occur in urban areas are most likely subdued, due to averaging across the large grid cell. In addition, only sulfates, nitrates, organic carbon, and black carbon were included here, leaving out many other components of fine PM which contribute to urban and rural pollution. Finally, PM CRFs were applied only to the population aged 30 and older, but evidence suggests that PM affects health negatively for all ages, including the very young. For these reasons, mortality results are most likely underestimates of the total global burden of mortality due to anthropogenic air pollution.

Other limitations are associated with the assumptions made in this study. For instance, the CRFs found by epidemiological studies conducted in North America were assumed to apply to all other parts of the world. While some evidence exists to validate this assumption, no PM cohort studies have yet been conducted in developing countries. Differences in health status, lifestyle, age structure, and medical care between developing and developed countries may have substantial consequences for mortality due to anthropogenic air pollution. Until CRFs for the developing world are established, less uncertainty is associated with estimating cause-specific mortality than with estimating total mortality. Here, we calculated cardiopulmonary and lung cancer mortality in addition to total mortality, finding slightly higher results than Cohen *et al.* [2004] when using the same CRF and threshold.

Another limitation concerns the assumption that all PM species exert effects similar to PM in the aggregate. Since CRFs are available only for fine PM or coarse PM and not for specific PM components, this study aggregated black carbon, organic carbon, nitrates, and sulfates into one category for fine PM. However, evidence sug-

gests that particular PM components may be more deleterious to human health than others [Ostro *et al.*, 2007]. Since PM composition is drastically different throughout the world, component-specific CRFs should be studied in further detail by daily time-series and long-term cohort studies.

Despite these limitations, this study has important policy implications. The geographic distribution of mortalities highlights local areas and regions where improvements to air quality may be particularly effective at reducing air pollution-related mortality. As development in Asia continues at a fast pace, pollution levels are likely to continue to increase in urban areas, as well as in rural areas where background concentrations will be elevated. Furthermore, as Asia develops, major causes of mortality are shifting from infectious disease and malnutrition to chronic conditions, which are more affected by air pollution exposure. Therefore, if continued unabated, air pollution is likely to have an even larger impact on mortality in the future than is estimated for the present. The mortalities resulting from increases in both urban air pollution and rural background air pollution are significant and should be considered during national and international policymaking.

This study estimates that a greater number of premature mortalities is caused by anthropogenic air pollution than were previously calculated. Global air quality models are useful for estimating the global premature mortalities due to air pollution, despite several important limitations in the necessary assumptions and data inputs. Future studies using this method should incorporate CRFs from new studies on ozone and PM mortality relationships, including those that examine individual PM components and those that are conducted in different parts of the world. This method would also benefit greatly by using statistical methods to correct for sub-grid scale urban peak concentrations. Measurements taken in urban areas could be used to determine a distribution of concentrations for the population within the coarse model grid cell.

Population-weighted concentrations for urban grid cells could then be calculated and applied in the mortality calculation. This would result in more accurate calculation of mortalities for grid cells that surround urban areas.

APPENDIX A

DERIVATION OF HEALTH IMPACT FUNCTION

A.1 Relative Risk Estimates from the Epidemiology Literature

Relative risk is the ratio of probability of a health endpoint occurring in an exposed group versus an unexposed group. Here relative risk is determined by epidemiology studies, which assess the risk of mortality in a group exposed to various air pollution concentrations versus a group exposed to background air pollution concentrations (unexposed group). A relative risk of 1 means there is no difference in risk between the two groups. A relative risk greater than 1 indicates a greater risk of mortality for the exposed group versus the unexposed group. A relative risk less than 1 indicates a lesser risk of mortality for the exposed group versus the unexposed group.

Two methods for using relative risk to estimate the burden of disease due to air pollution have been described in the literature. These methods have been referred to differently by various studies. For simplicity, I refer to them as Method 1 and Method 2.

Method 1:

$$RR = \exp^{\beta \Delta x} \tag{A.1}$$

Method 2:

$$RR = \left[\frac{x + 0.0001}{x_0 + 0.0001} \right]^{\beta} \tag{A.2}$$

Method 1 has been called "log-linear" [Cohen *et al.*, 2004; Abt Associates, 2005] or "linear" [Ostro, 2004]. Method 2 has also been called "log-linear" [Ostro, 2004]. With no threshold, relative risk predicted by the two methods diverge greatly, with Method 2 predicting much higher than Method 1, as shown in Figure A.1. When a low concentration threshold of 7.5 μg/m^3 is applied, the curves representing Method 1 and Method 2 are closer together.

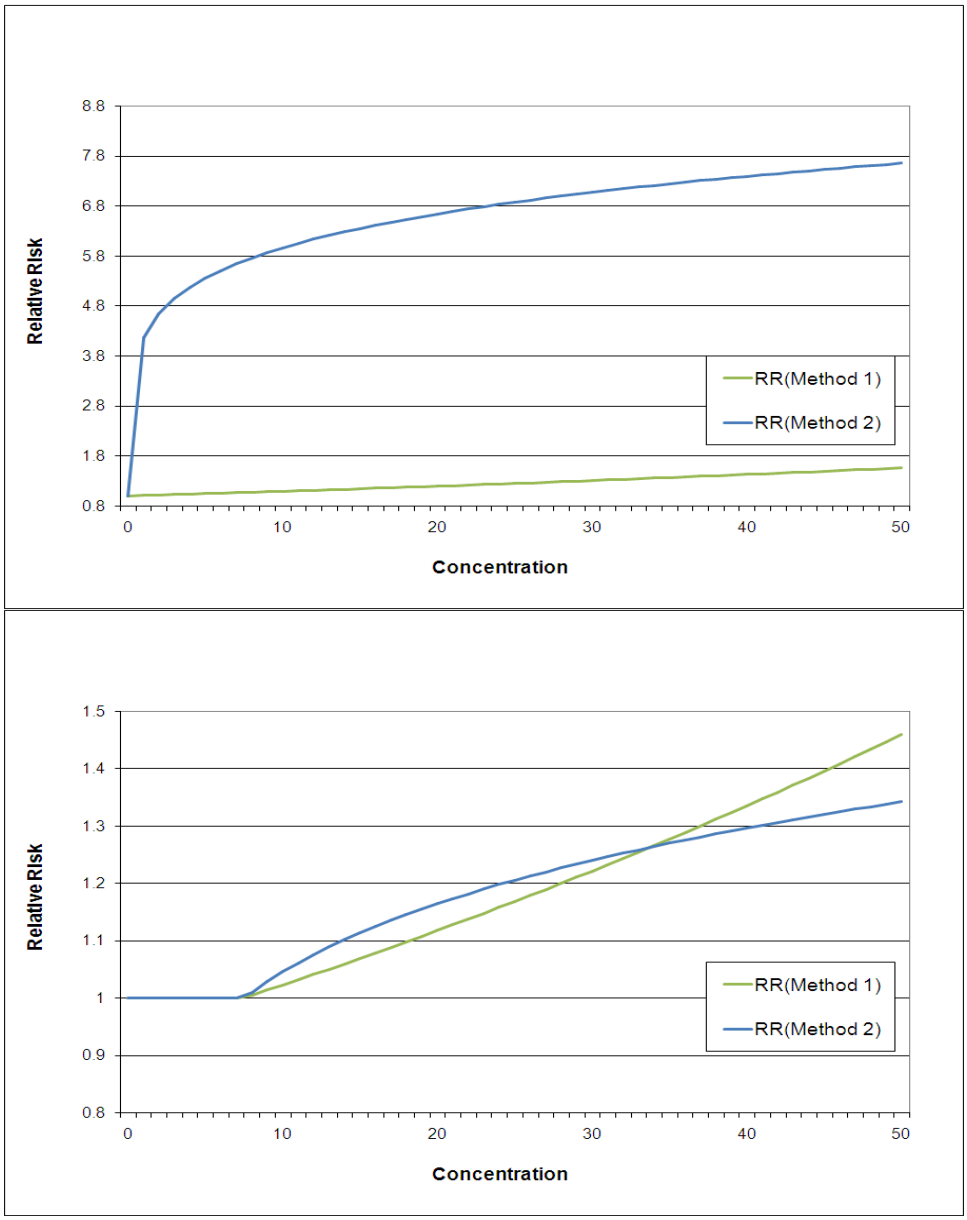

Figure A.1: Comparison of mortality calculation methods with no threshold applied (top) and with a threshold of 7.5 μg/m^3 (bottom)

Since mortalities are calculated here with no threshold applied, the choice of health impact functions is significant. Method 1 is commonly used by the US Environmental Protection Agency and health impact researchers. Method 2 is recommended by Ostro [2004] and applied in a study estimating mortality due to ship emissions [Corbett *et al.*, 2007]. For this analysis, Method 1 was chosen because it is based on the

concentration-response relationships found by Pope *et al.* [2002]. Pope *et al.* [2002] plotted the natural logarithm of relative risk against $PM_{2.5}$ concentration, showing a nearly linear relationship. These results imply a log-linear relationship between relative risk and $PM_{2.5}$ concentration, as described by Method 1.

A.2 Derivation of the Health Impact Function

Attributable fraction is the fraction of the disease burden attributable to the risk factor. It is calculated by Equation A.3.

$$AF = \frac{RR-1}{RR} = \frac{\exp^{\beta\Delta X}-1}{\exp^{\beta\Delta X}} = 1 - \frac{1}{\exp^{\beta\Delta X}} = 1 - \exp^{-\beta\Delta X} \tag{A.3}$$

where RR is relative risk and 1 denotes the relative risk of the unexposed population.

To calculate excess mortalities, the attributable fraction must then be multiplied by the baseline mortality rate and exposed population to yield the number of mortalities attributable to air pollution (Equation A.4).

$$\Delta Y = Y_o(1 - \exp^{-\beta\Delta X})Pop \tag{A.4}$$

where ΔY is excess mortalities, Y_o is the baseline mortality rate, β is the concentration-response factor, ΔX is the change in concentration, and Pop is the exposed population.

APPENDIX B

COMPARISONS OF MOZART-2 SIMULATED OZONE CONCENTRATIONS WITH OBSERVED CONCENTRATIONS

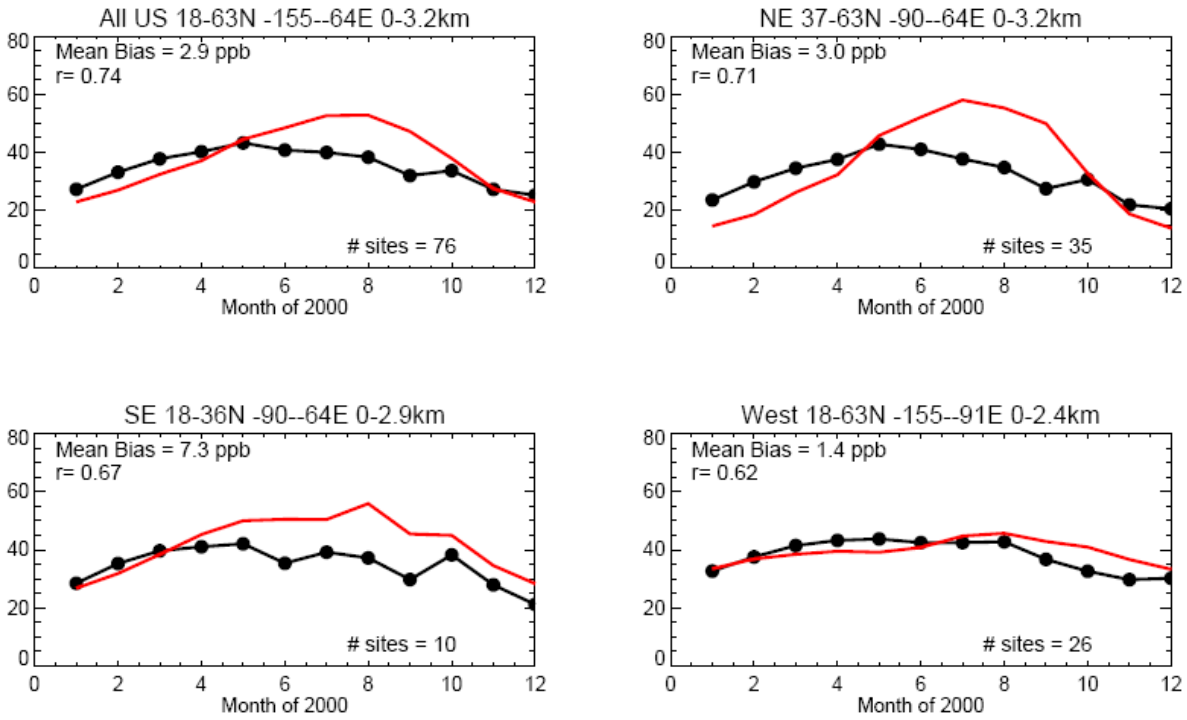

Figure B.1: Comparison of simulated ozone (red) and CASTNet observations (black) in 2000

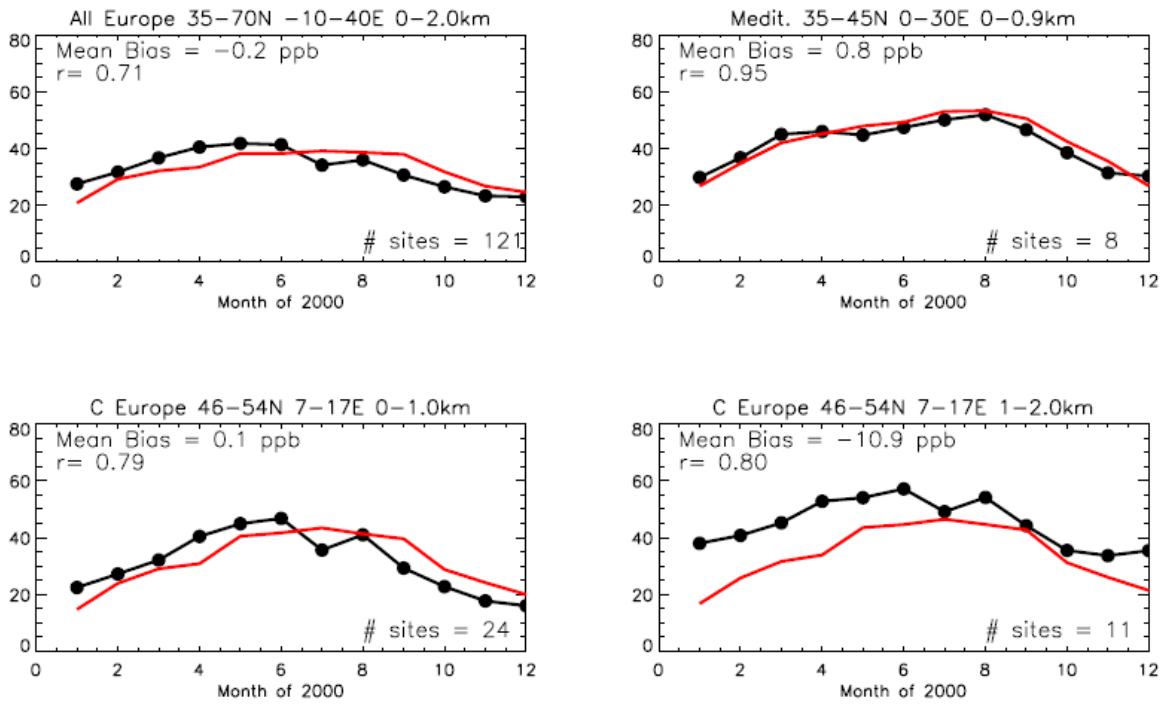

Figure B.2: Comparison of simulated ozone (red) and EMEP observations (black) in 2000

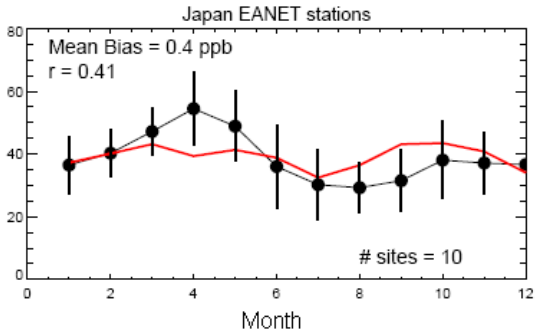

Figure B.3: Comparison of simulated ozone in 2000 (red) and EANET observations (black) in 2001

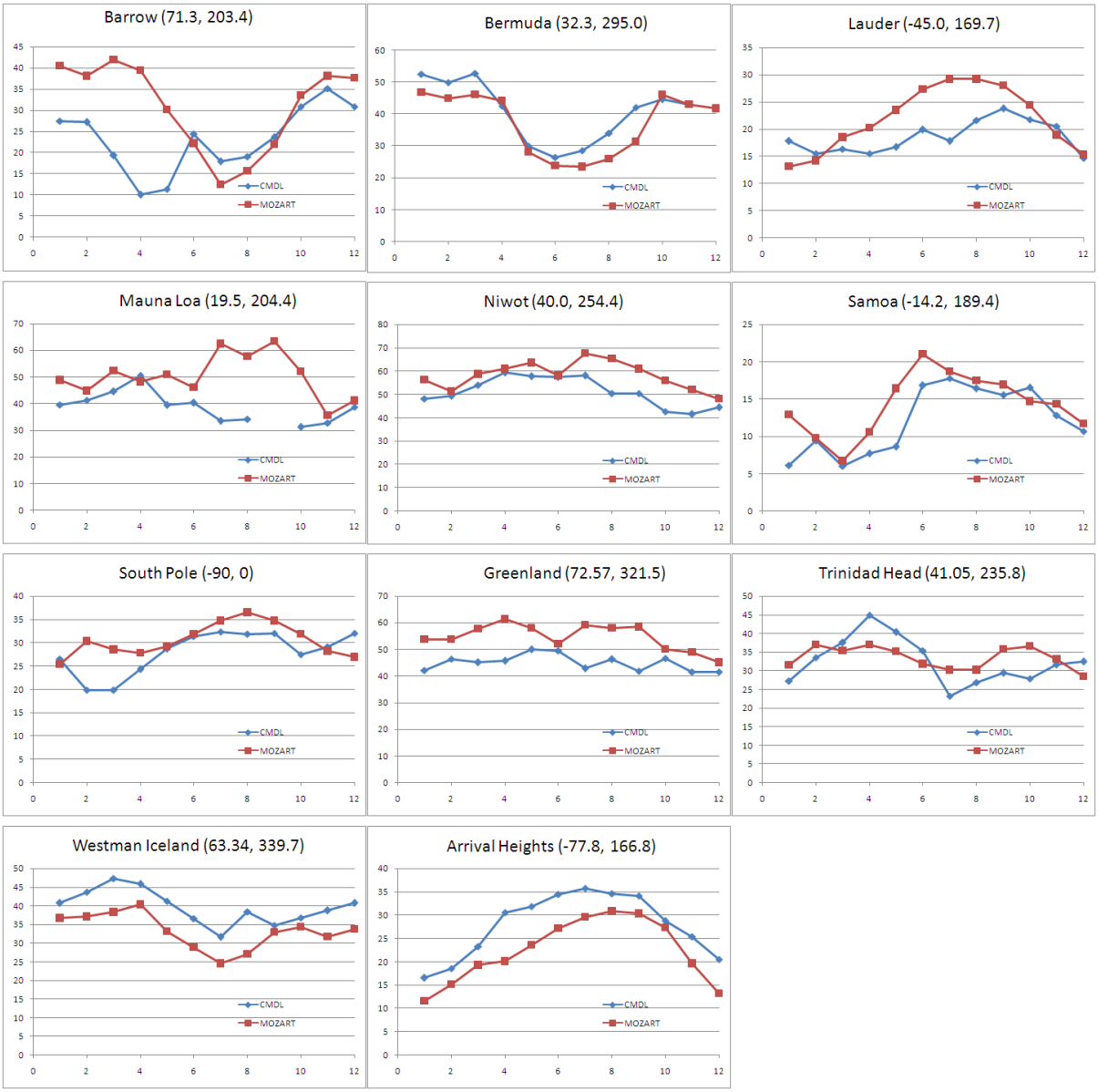

Figure B.4: Comparison of simulated ozone in 2000 (red) and CMDL observations (blue) from years 1998-2004

APPENDIX C

POPULATION

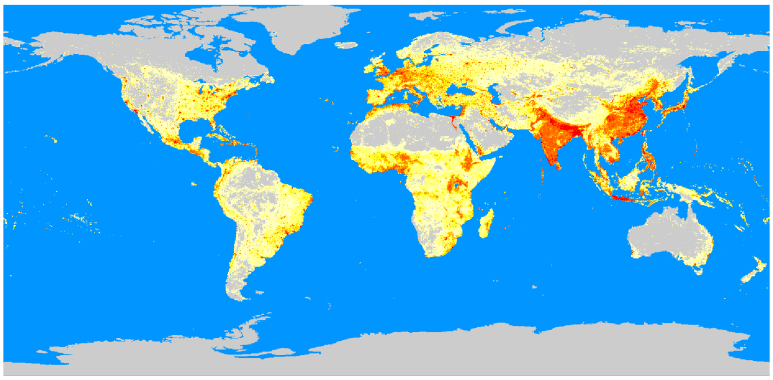

Figure C.1: Spatial distribution of 2006 population from Landscan database [Oak Ridge National Laboratory, 2008]

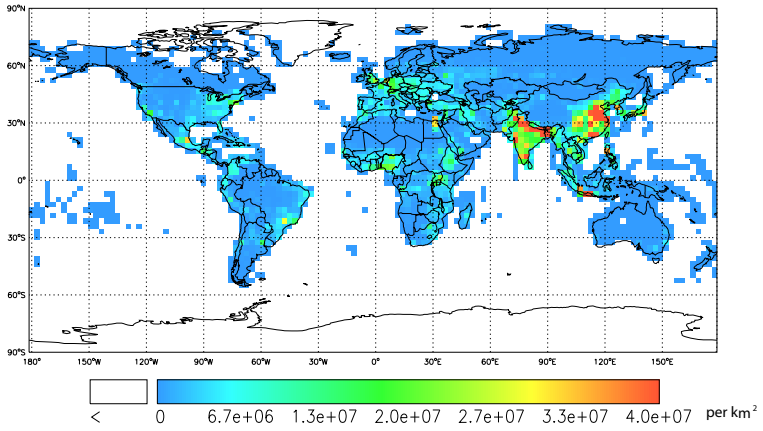

Figure C.2: Spatial distribution of 2006 population from Landscan database [Oak Ridge National Laboratory, 2008] summed to MOZART-2 grid

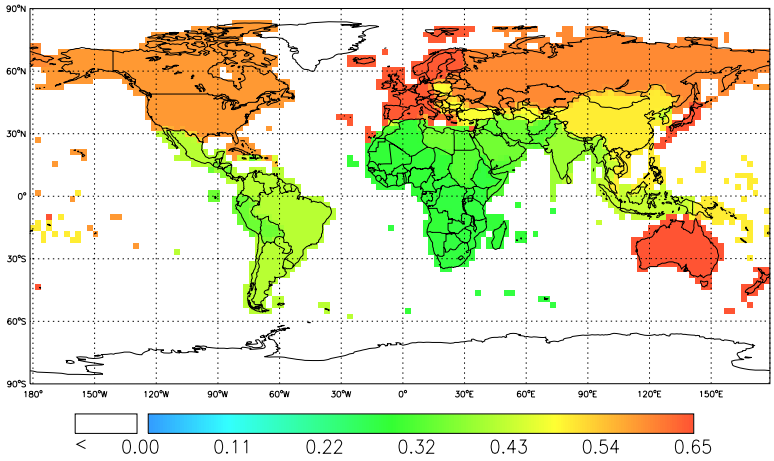

Figure C.3: Fraction of population aged 30 and older from World Health Organization [2004] mapped onto MOZART-2 grid

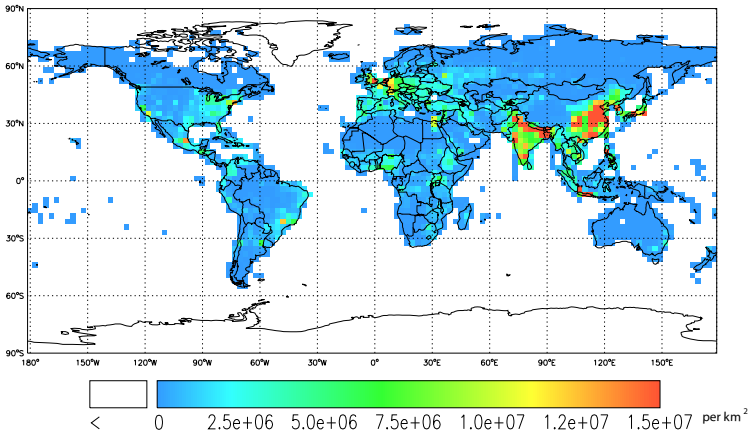

Figure C.4: Population aged 30 and older calculated from Landscan population [Oak Ridge National Laboratory, 2008] and fraction of population aged 30 and older from World Health Organization [2004] mapped onto MOZART-2 grid

APPENDIX D

BASELINE MORTALITY RATES

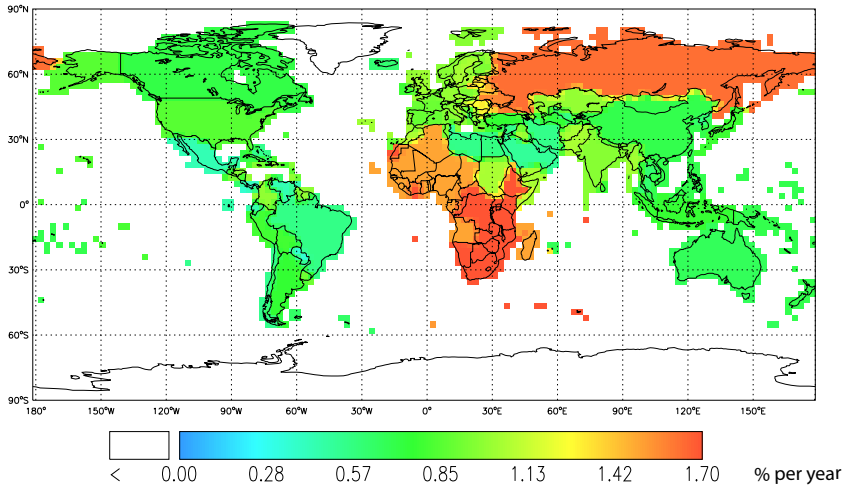

Figure D.1: Baseline all-cause mortality rates mapped onto MOZART-2 grid

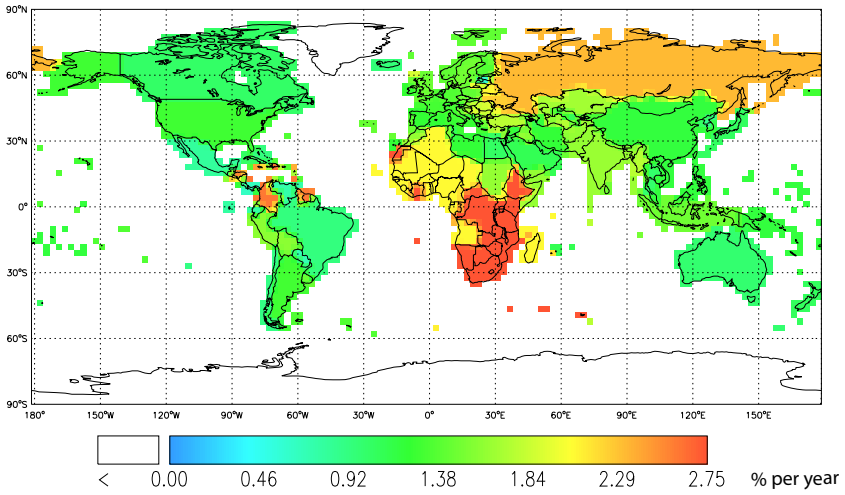

Figure D.2: Baseline all-cause mortality rates for population aged 30 and older mapped onto MOZART-2 grid

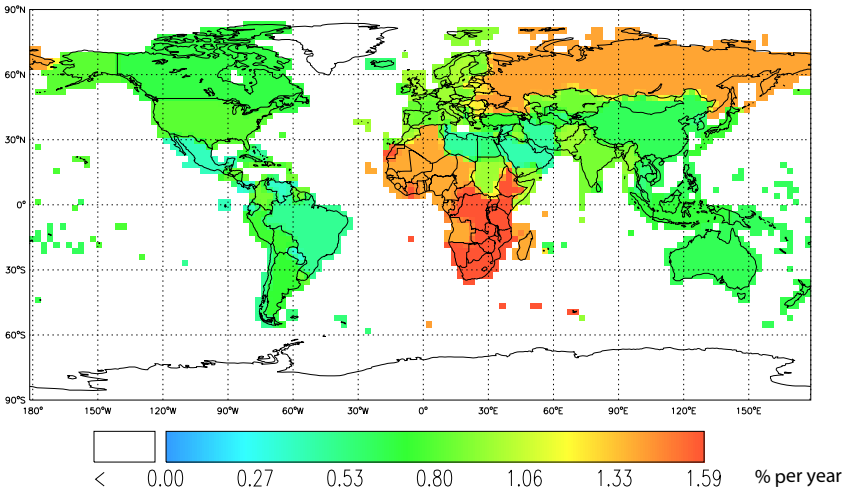

Figure D.3: Baseline non-accidental mortality rates mapped onto MOZART-2 grid

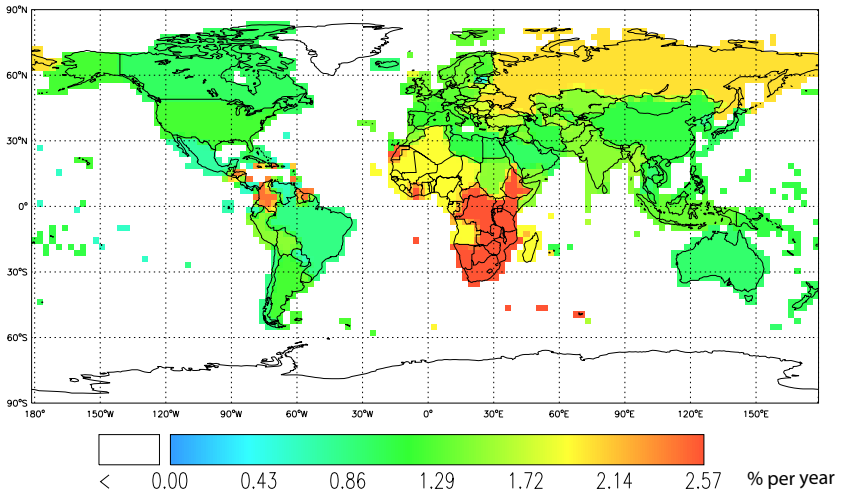

Figure D.4: Baseline non-accidental mortality rates for population aged 30 and older mapped onto MOZART-2 grid

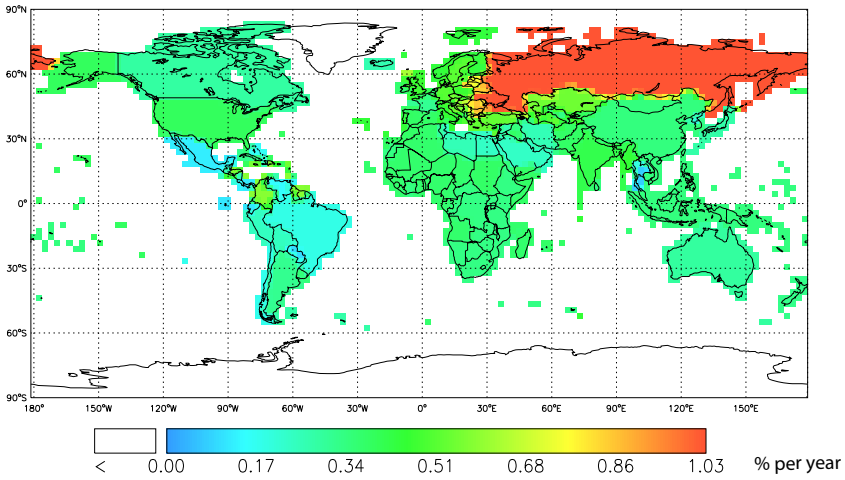

Figure D.5: Baseline cardiopulmonary mortality rates mapped onto MOZART-2 grid

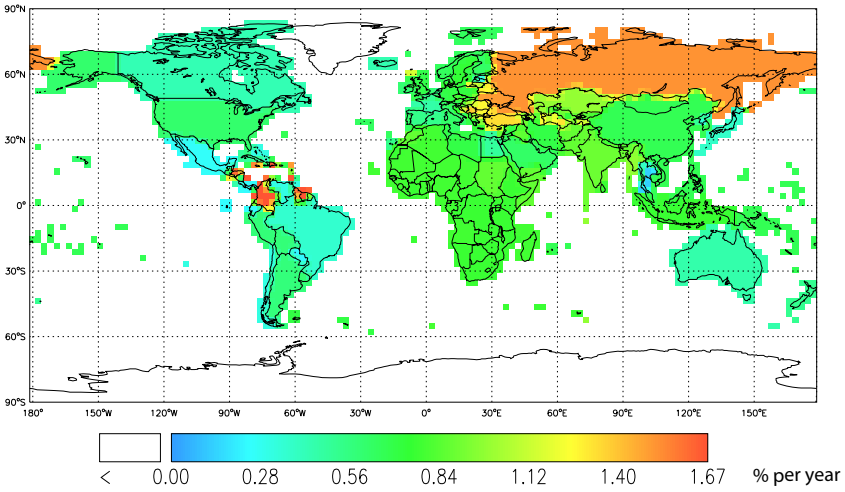

Figure D.6: Baseline cardiopulmonary mortality rates for population aged 30 and older mapped onto MOZART-2 grid

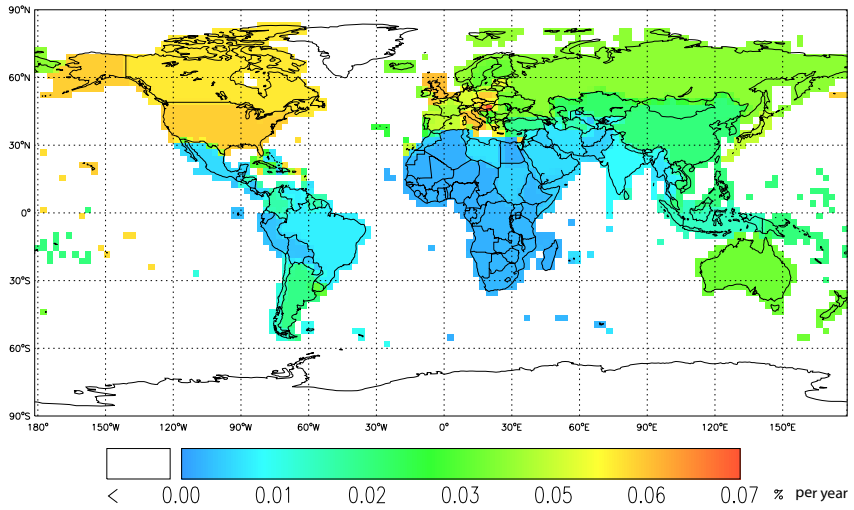

Figure D.7: Baseline lung cancer mortality rates mapped onto MOZART-2 grid

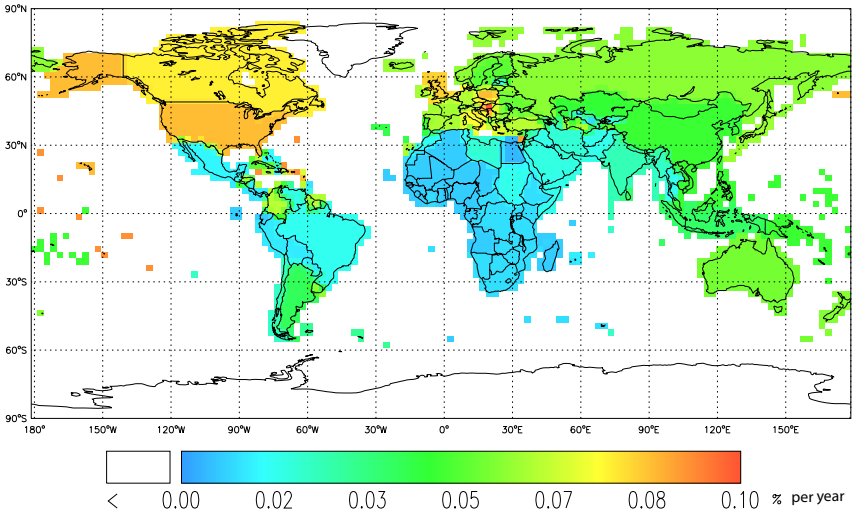

Figure D.8: Baseline lung cancer mortality rates for population aged 30 and older mapped onto MOZART-2 grid

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