An Estimate of the Global Burden of Anthropogenic Ozone and Fine Particulate Matter on Premature Human Mortality Using Atmospheric Modeling

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BACKGROUND: Ground-level concentrations of ozone (O3) and fine particulate matter (≤ 2.5 μm in aerodynamic diameter (PM2.5)) have increased since preindustrial times in urban and rural regions and are associated with cardiovascular and respiratory mortality.

OBJECTIVES: We estimated the global burden of mortality due to O3 and PM2.5 from anthropogenic emissions using global atmospheric chemical transport model simulations of preindustrial and present-day (2000) concentrations to derive exposure estimates.

METHODS: Attributable mortalities were estimated using health impact functions based on long-term relative risk estimates for O3 and PM2.5 from the epidemiology literature. Using simulated concentrations rather than previous methods based on measurements allows the inclusion of rural areas where measurements are often unavailable and avoids making assumptions for background air pollution.

RESULTS: Anthropogenic O3 was associated with an estimated 0.7 ± 0.3 million respiratory mortalitites (6.3 ± 3.0 million years of life lost) annually. Anthropogenic PM2.5 was associated with 3.5 ± 0.9 million cardiopulmonary and 220,000 ± 80,000 lung cancer mortalities (30 ± 7.6 million years of life lost) annually. Mortality estimates were reduced approximately 30% when we assumed low-concentration thresholds of 33.3 ppb for O3 and 5.8 μg/m3 for PM2.5. These estimates were sensitive to concentration thresholds and concentration–mortality relationships, often by > 50%.

CONCLUSIONS: Anthropogenic O3 and PM2.5 contribute substantially to global premature mortality. PM2.5 mortality estimates are about 50% higher than previous measurement-based estimates based on common assumptions, mainly because of methodologic differences. Specifically, we included rural populations, suggesting higher estimates; however, the coarse resolution of the global atmospheric model may underestimate urban PM2.5 exposures.

KEY WORDS: air pollution, atmospheric chemistry model, health effects of air pollution, health impact analysis, ozone, particulate matter. Environ Health Perspect 118:1189–1195 (2010). doi:10.1289/ehp.0901220 [Online 9 April 2010]

Ground-level ozone (O3) and fine particulate matter (≤ 2.5 μm in aerodynamic diameter (PM2.5)) have increased substantially since preindustrial times. Although O3 and PM2.5 concentrations have increased most in industrialized areas, observations show that background concentrations have also increased in remote regions (Akimoto 2003; Schultz et al. 2006; Staehelin et al. 2001; Vingarzan 2004; Volz and Kley 1988). O3 and PM2.5 are associated with negative health impacts, including premature mortality (e.g., Jerrett et al. 2009; Krewski et al. 2009). Cohen et al. (2004) estimated that about 800,000 annual premature deaths globally, or 1.2% of all deaths, are associated with urban outdoor PM2.5. This was considered an underestimate because it excludes O3 impacts and includes only urban areas for which econometric models trained with observations were used to predict concentrations.

We estimated the global burden of human mortality due to anthropogenic O3 and PM2.5 using a global atmospheric chemical transport model (CTM). Using an atmospheric CTM allows estimation of mortality where air quality measurements are sparse, particularly in developing nations. By simulating preindustrial concentrations, we also isolated mortality due to anthropogenic pollution and avoided making assumptions for background O3 and PM2.5 concentrations. Global CTMs have been used to estimate mortalities due to long-range transport of air pollution (Anenberg et al. 2009; Liu et al. 2009; West et al. 2009), future changes in emissions (West et al. 2006, 2007), or changes in one sector’s emissions (Corbett et al. 2007). CTMs have not been used previously to quantify the global burden of anthropogenic air pollution on human mortality.

Materials and Methods

We calculated mortalities associated with anthropogenic air pollution using health impact functions that relate changes in pollutant concentrations to changes in mortality. We defined anthropogenic air pollution as the geographically distributed difference between present-day (2000) and preindustrial O3 and PM2.5 concentrations, as simulated by a global CTM. Health impact functions for both O3 and PM2.5 are based on a log-linear relationship between relative risk (RR) and concentrations defined by epidemiology studies (e.g., Jerrett et al. 2009; Krewski et al. 2009):

\[ RR = \exp^{\beta \Delta X} . \]  

where \( \beta \) is the concentration–response factor (CRF; i.e., the estimated slope of the log-linear relation between concentration and mortality) and \( \Delta X \) is the change in concentration. The fraction of the disease burden attributable to the risk factor, the attributable fraction (AF), was defined as

\[ AF = \frac{RR - 1}{RR} = 1 - \exp^{-\beta \Delta X} . \]  

AF was multiplied by the baseline mortality rate \( (y_0) \) and size of the exposed population \( (Pop) \) to yield an estimate of the excess mortalities attributable to air pollution \( (\Delta\text{Mort}) \):

\[ \Delta\text{Mort} = y_0 (1 - \exp^{-\beta \Delta X}) Pop. \]  

Disease survival time varies among populations, and we calculated years of life lost (YLL) associated with mortalities using the baseline YLL (YLL0) per death:

\[ \Delta\text{YLL} = \Delta\text{Mort} \times \text{YLL0}/y_0. \]
For O₃, we based CRFs on the association between long-term O₃ exposure and RR of death from respiratory disease found by Jerrett et al. (2009) in an American Cancer Society (ACS) cohort study of U.S. adults ≥ 30 years of age for 1977–2000. Although many daily time-series epidemiology studies demonstrate short-term O₃-mortality impacts (e.g., Bell et al. 2004), Jerrett et al. (2009) provide the first clear evidence for long-term impacts. For the two-pollutant model that controlled for PM₂.₅, a 10-ppb increase in the seasonal (April–September) average daily 1-hr maximum O₃ (concentration range, 33.3–104.0 ppb) was associated with a 4% [95% confidence interval (CI), 1.3–6.7%] increase in RR of death from respiratory disease.

For PM₂.₅, we used RRs from Krewski et al. (2009), which is the latest reanalysis of the ACS PM₂.₅ studies (e.g., Pope et al. 2002) and has the largest population of the available PM₂.₅ cohort studies (e.g., Hoek et al. 2002; Laden et al. 2006). We used RRs for 1999–2000 from the random-effects Cox model analysis that adjusted for 44 individual-level and seven ecological covariates. A 10-µg/m³ increase in PM₁.₅ (concentration range, 5.8–22.2 µg/m³) was associated with 6% (95% CI, 4–8%), 13% (95% CI, 10–16%), and 14% (95% CI, 6–23%) increases in total, cardiopulmonary, and lung cancer mortality. The linearity of the concentration–response function was also demonstrated up to 30 µg/m³ in the 1979–1983 analysis. Krewski et al. (2009) found that PM₁.₅ was associated most strongly with risk of death from ischemic heart disease, a subset of cardiopulmonary disease, and previous studies have found that controlling for O₃ concentrations had little effect on the PM₁.₅–mortality relationships (Krewski et al. 2000). Compared with the relationships in an earlier expert elicitation (Roman et al. 2008), the total mortality RR in Krewski et al. (2009) is generally 3–14% lower per 10-µg/m³ increase with a tighter CI.

We assumed that these relationships found in the United States are valid globally.

For O₃, Jerrett et al. (2009) is the first study showing significant long-term impacts, but the short-term impact has been well documented in North America and Europe (e.g., Anderson et al. 2004; Bell et al. 2004). For PM₁.₅, similar long-term mortality results have been demonstrated in Europe (Hoek et al. 2002), but to date no PM₁.₅ cohort studies have been conducted in the developing world. Short-term O₃ and PM₁.₅ studies in developing nations demonstrate relationships that are generally comparable with short-term studies in North America and Europe (Health Effects Institute International Scientific Oversight Committee 2004). Our assumption is further supported by evidence that concentration–mortality relationships do not vary significantly by sex, age, and race (Jerrett et al. 2009; Krewski et al. 2009; Zanobetti et al. 2000), although some sensitive populations may be at a higher risk. Because global causes of death differ from those in North America and Europe, we emphasized cause-specific mortality, which may have less error than estimates of all-cause mortality across different populations.

We used present-day (2000) and preindustrial O₃ and PM₁.₅ concentrations (Figure 1) simulated by Horowitz (2006) using the Model of Ozone and Related Chemical Tracers, version 2 (MOZART-2; Horowitz et al. 2003). The preindustrial simulation, which corresponds to the 1860 simulation by Horowitz (2006), represents the “background” O₃ and PM₁.₅ present in the absence of anthropogenic emissions, allowing us to isolate the anthropogenic contributions to concentrations and premature mortalities. MOZART-2 has a resolution of 2.8° latitude by 2.8° longitude with 34 vertical levels, and we used concentrations in the first vertical level as surface concentrations. Both simulations used the same meteorology from the National Center for Atmospheric Research Community Climate Model to isolate the impact of emission changes on concentration. We defined PM₁.₅ as all simulated sulfate (SO₄²⁻), nitrate (NO₃⁻), ammonium, black carbon (BC), and primary organic carbon (OC). We excluded dust, sea salt, and secondary organic aerosols, which we assumed are unchanged from preindustrial to present. We multiplied OC mass by 1.4 to account for associated species other than carbon, and assumed all SO₄ and NO₃ exists as ammonium sulfate [(NH₄)₂SO₄] and ammonium nitrate (NH₄NO₃), following Ginoux et al. (2006). For the preindustrial case, fossil fuel–burning emissions were set to zero and emissions from burning of biofuels, savannah, tropical forests, and agricultural waste were assumed to be 10% of 1990 values.

Consistent with the epidemiology studies, we used seasonal average 1-hr daily maximum concentrations for O₃ and annual average

Figure 1. Estimated change (present minus preindustrial) in seasonal average (6-month) 1-hr daily maximum O₃ concentrations (ppb; A) and annual average PM₁.₅ (µg/m³; B) from Horowitz (2006) simulations.
concentrations for PM$_{2.5}$. Because high O$_3$ occurs during different months globally, for each grid cell, we found the consecutive 6-month period with the highest average of the simulated daily 1-hr maximum O$_3$ concentrations, which we then used to calculate annual mortalities. Table 1 shows that the modeled global population-weighted seasonal average 1-hr daily maximum O$_3$ increased by 37.1 ppb (from 19.6 ppb in 1860 to 56.7 ppb in 2000), using the present population, and the global population-weighted annual average PM$_{2.5}$ increased by 15.0 µg/m$^3$ (from 1.1 µg/m$^3$ in 1860 to 16.1 µg/m$^3$ in 2000). Globally, OC, BC, NO$_x$, and SO$_4$ are 62.3%, 6.3%, 0.3%, and 31.0% of total PM$_{2.5}$ in 1860 and 45.6%, 9.1%, 4.9%, and 40.4% in 2000 [see Supplemental Material, Figure 1 (doi:10.1289/ehp.0901220)].

We compared modeled present-day surface O$_3$ concentrations with data from the National Oceanic and Atmospheric Administration (NOAA) Earth Systems Research Laboratory Global Monitoring Division (NOAA 2008) monitoring network (mean bias = 2.5 ppb) for 11 remote locations around the world and from three nonurban networks: the Clean Air Status and Trends Network (U.S. EPA 2007) for the United States (mean bias = 2.9 ppb), the European Monitoring and Evaluation Programme (Convention on Long-Range Transboundary Air Pollution 2007) for Europe (mean bias = −0.2 ppb), and the Acid Deposition Monitoring Network in East Asia (EANET 2007) for Japan (mean bias = 0.4 ppb) [see Supplemental Material, Figures 2–5 (doi:10.1289/ehp.0901220)]. Horowitz (2006) found that simulated preindustrial O$_3$ concentrations overestimate reconstructed observations from the late 19th century by approximately 5–10 ppb, with strong sensitivity to assumed biomass burning. Modeled surface PM$_{2.5}$ 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 and at nonurban stations in Europe and the United States, with a tendency to be overestimated. These comparisons show that MOZART-2 simulates surface O$_3$ and PM$_{2.5}$ well for nonurban and remote measurements in the areas compared, and it was not apparent that corrections for bias were necessary. Although simulated concentrations were not systematically biased outside of urban regions, the coarse resolution used here (grid cell area = 9.9 × 10$^6$ km$^2$, and 5.2 × 10$^3$ km$^2$ at 0°, 30°, and 60° latitude) may cause errors in mortality estimates, particularly in urban areas with strong population and concentration gradients.

We estimated global premature mortalities separately for O$_3$ and PM$_{2.5}$ by applying Equation 3 in each of the MOZART-2 surface grid cells, using the corresponding population and baseline mortality rates for each cell. To calculate mortality, we used the global 2006 population (Oak Ridge National Laboratory 2008) [see Supplemental Material, Figure 6 (doi:10.1289/ehp.0901220)], and, consistent with the ACS study population, we used the population fraction ≥ 30 years of age (Table 1), estimated in 14 world regions [World Health Organization (WHO) 2004] [see Supplemental Material, Figure 7 (doi:10.1289/ehp.0901220)]. We used baseline all-cause, cardiopulmonary, and lung cancer mortality rates for 14 world regions (WHO 2004) and 66 countries (WHO 2008a), back-calculating from regional rates where country-specific rates were unavailable [Table 1; see also Supplemental Material, Figures 8–11 (doi:10.1289/ehp.0901220)]. Country-specific mortality rates are broadly categorized with no cutoff at 30 years of age, and we used rates for the population ≥ 25 years of age, assuming that differences between the rates are insignificant. We used baseline YLL rates for the population ≥ 30 years of age in 14 world regions [global average = 7.89, 9.77, and 8.93 for cardiopulmonary disease, respiratory disease, and lung cancer; see Supplemental Material, Table 1 (doi:10.1289/ehp.0901220)], assuming a 3% discount rate and nonuniform age weighting, giving less weight to years lived at older ages (WHO 2008b). We griddered baseline mortality rates, baseline YLL, and the fraction of the population ≥ 30 years of age to the MOZART-2 grid, and for grid cells overlapping multiple countries, we calculated area-weighted averages using a geographic information system program.

We present results as means ± 1 SD, calculating uncertainty from 500 Monte Carlo simulations that randomly sampled from normal distributions of the CRF, as reported by the epidemiology studies, and modeled present-day concentrations (SD = 25% of simulated value). Although the epidemiology literature provides little evidence for low-concentration thresholds (LCTs) or high-concentration thresholds (HCTs) for either O$_3$ or PM$_{2.5}$ (Jerrett et al. 2009; Krewski et al. 2009; Schwartz and Zanobetti 2000), mortality relationships beyond measured concentrations are unknown. Therefore, we estimated mortalities with and without assuming LCTs below which O$_3$ and PM$_{2.5}$ are assumed to have no effect on mortality. For O$_3$, we applied an LCT of 3.3 ppb, the lowest measured level in Jerrett et al. (2009). When applied, this threshold replaced the natural background everywhere except in some grid cells in Asia and South America, where preindustrial concentrations exceeded the threshold (Table 1). We also examined an LCT of 56 ppb, which Jerrett et al. (2009) found to be close to statistical significance at an α-level of 5% (p = 0.0600). The 56-ppb threshold exceeded preindustrial concentrations in all cells. Because no grid cells exceeded the highest measured level (104.0 ppb) in Jerrett et al. (2009), we did not apply an HCT for O$_3$. For PM$_{2.5}$, we applied an LCT of 5.8 µg/m$^3$, the lowest measured level in Krewski et al. (2009), which exceeded preindustrial concentrations in all grid cells (Table 1), effectively replacing the natural background. Some grid cells in Europe and Asia exceeded the highest measured level (30.0 µg/m$^3$) in Krewski et al. (2009), and we examined HCTs of 30 µg/m$^3$ and 50 µg/m$^3$ in the sensitivity analysis. These thresholds applied only to our definition of PM$_{2.5}$ and would be affected by including dust, sea salt, and secondary organic aerosols.

Table 1. Population ≥ 30 years of age, average baseline mortality rates, and population-weighted average and range of the seasonal average (6-month) 1-hr daily maximum O$_3$ concentrations and annual average PM$_{2.5}$ concentrations from MOZART-2 simulations of preindustrial (1860) and present-day (2000) levels.

| Country        | Population (bil.) | O$_3$ (ppb)$^a$ | PM$_{2.5}$ (µg/m$^3$)$^b$ |
|----------------|-------------------|-----------------|---------------------------|
|                | Average | Range  | Average | Range  | Average | Range  | Average | Range  |
| Africa         | 0.28    | 0.206  | 0.128  | 0.011  | 22.32  | 11.4–31.9| 54.46 | 20.2–71.5| 0.92  | 0.28–3.19| 7.50 | 0.50–12.9|
| North America  | 0.27    | 0.081  | 0.052  | 0.071  | 21.42  | 12.5–32.3| 59.75 | 27.0–83.3| 1.50  | 0.14–4.85| 8.44 | 0.31–16.6|
| Europe         | 0.44    | 0.127  | 0.122  | 0.056  | 18.26  | 15.2–27.5| 48.92 | 32.3–73.4| 0.93  | 0.11–2.98| 14.77 | 0.40–39.0|
| Asia           | 0.18    | 0.171  | 0.146  | 0.037  | 18.91  | 6.1–35.9 | 50.64 | 10.8–83.7| 1.19  | 0.23–3.06| 20.41 | 0.34–55.9|
| South America  | 0.15    | 0.121  | 0.105  | 0.025  | 18.44  | 12.3–35.8| 44.99 | 22.3–90.3| 1.00  | 0.33–3.89| 6.35  | 0.40–13.9|
| Oceania        | 0.02    | 0.074  | 0.036  | 0.035  | 13.37  | 3.7–22.8 | 26.75 | 6.1–44.4 | 0.96  | 0.22–2.27| 2.59  | 0.25–5.01|
| World          | 2.9     | 0.134  | 0.075  | 0.042  | 19.61  | 3.7–35.9 | 56.70 | 6.1–90.3 | 1.13  | 0.11–3.89| 16.11 | 0.25–55.9|

Abbreviations: CP, cardiopulmonary; LC, lung cancer; Pop, population. Data are average and range for the highest and lowest individual grid cells.

$^a$Simulated by Horowitz (2006). $^b$Population ≥ 30 years of age for the year 2006 from the LandScan database (Oak Ridge National Laboratory 2008). $^c$Baseline mortality rates are country specific for the latest year after 2000 with data available (WHO 2008a). Where country-specific rates after the year 2000 were not available, we back-calculated country-specific rates from regional rates for the year 2002.
Results

With no upper or lower concentration threshold, anthropogenic O\(_3\) was estimated to result in about 0.7 ± 0.3 million respiratory mortalities annually worldwide (Table 2), corresponding to 6.3 ± 3.0 million YLL (Table 3). Estimated global respiratory mortalities were reduced by approximately 33% when we assumed an LCT of 33.3 ppb, the lowest measured level in Jerrett et al. (2009). Regardless of threshold assumption, >75% of O\(_3\) mortalities were estimated to occur in Asia, which is densely populated and highly polluted, whereas only approximately 5% occurred in North America. Estimated excess O\(_3\) mortalities were densest in highly populated areas but were distributed more evenly across the globe when divided (normalized) by population size (Figure 2).

Assuming no upper or lower concentration threshold, we estimated that exposure to anthropogenic PM\(_{2.5}\) results in 3.5 ± 0.9 million cardiopulmonary mortalities and 220,000 ± 80,000 lung cancer mortalities annually (Table 2), corresponding to 28 ± 6.8 and 2.2 ± 0.8 million YLL (Table 3). With an LCT of 5.8 µg/m\(^3\), estimated cardiopulmonary and lung cancer mortalities decreased by approximately 28%. Regardless of threshold, about 75% of excess mortalities occurred in Asia because of high PM\(_{2.5}\) concentrations and dense population, followed by Europe (17%). As for O\(_3\), estimated PM\(_{2.5}\) mortalities were densest in highly populated areas but more localized because of the shorter atmospheric lifetime of PM\(_{2.5}\) compared with O\(_3\) (Figures 1, 3). The highest estimated mortalities per million people were in Europe, East Asia, and the eastern United States (Figure 3B,D), owing to baseline cardiopulmonary and lung cancer mortality rates and high PM\(_{2.5}\) concentrations.

Applying an LCT of 25 ppb for O\(_3\) resulted in approximately 14% fewer estimated respiratory mortalities than when assuming no upper or lower threshold (Table 4). With CRFs from the single-pollutant model in Jerrett et al. (2009), which did not control for PM\(_{2.5}\), O\(_3\)-mortality estimates were approximately 25% lower, corresponding to the relative magnitudes of the CRFs. Applying the 56-ppb LCT from the threshold model reduced mortality estimates by approximately 75%. For PM\(_{2.5}\), RRs from Krewski et al. (2009) are similar to the 1979–1983 and 1999–2000 average all-cause and lung cancer RR from Pope et al. (2002) but are approximately 40% higher for cardiopulmonary mortality, thus causing a corresponding increase in our estimates when applied [Table 5; see also Supplemental Material, Table 2 (doi:10.1289/ehp.0901220)]. Using RRs from Laden et al. (2006)—an extended reanalysis of the Harvard Six Cities cohort study that found significantly higher RRs than did Krewski et al. (2009)—increased estimated cardiopulmonary and lung cancer mortalities by approximately 30% and 50%, respectively. With no LCT, applying HCTs of 30 µg/m\(^3\) and 50 µg/m\(^3\) decreased estimated mortalities by approximately 10% and 1%, with larger decreases estimated for Europe and Asia, where some modeled concentrations exceeded the upper threshold values.

### Table 2. Estimated annual mortalities ± 1 SD due to anthropogenic O\(_3\) and PM\(_{2.5}\), assuming natural background only or LCTs (33.3 ppb for O\(_3\) and 5.8 µg/m\(^3\) for PM\(_{2.5}\), × 1,000).

| Region     | O\(_3\) respiratory | PM\(_{2.5}\) cardiopulmonary | PM\(_{2.5}\) lung cancer |
|------------|----------------------|-----------------------------|-------------------------|
|            | Background           | Threshold                    | Background              | Threshold              |
| Africa     | 63 ± 34              | 45 ± 30                      | 154 ± 44                | 52 ± 33                |
| North America | 35 ± 17             | 25 ± 15                      | 124 ± 57                | 65 ± 30                |
| Europe     | 41 ± 21              | 23 ± 17                      | 586 ± 149               | 393 ± 143              |
| Asia       | 543 ± 253            | 370 ± 220                    | 2,584 ± 618             | 1,991 ± 603            |
| South America | 18 ± 9              | 8 ± 6                        | 48 ± 15                 | 16 ± 9                 |
| Oceania    | 1 ± 1                | 0 ± 0                        | 2 ± 1                   | 1 ± 1                  |
| World      | 700 ± 335            | 470 ± 288                    | 3,499 ± 864             | 2,506 ± 816            | 222 ± 80 | 164 ± 68 |

### Table 3. Estimated annual YLL ± 1 SD due to anthropogenic O\(_3\) and PM\(_{2.5}\), assuming the natural background or LCTs (33.3 ppb for O\(_3\) and 5.8 µg/m\(^3\) for PM\(_{2.5}\), × 1,000).

| Region     | O\(_3\) respiratory | PM\(_{2.5}\) cardiopulmonary | PM\(_{2.5}\) lung cancer |
|------------|----------------------|-----------------------------|-------------------------|
|            | Background           | Threshold                    | Background              | Threshold              |
| Africa     | 901 ± 486            | 644 ± 429                    | 1,694 ± 484             | 572 ± 363              |
| North America | 265 ± 138           | 203 ± 122                    | 804 ± 240               | 421 ± 194              |
| Europe     | 243 ± 125            | 136 ± 101                    | 4,336 ± 1,103           | 2,834 ± 1,058          |
| Asia       | 4,322 ± 2,014        | 2,945 ± 1,751                | 20,620 ± 4,932          | 15,888 ± 4,812         |
| South America | 137 ± 68           | 81 ± 46                      | 365 ± 114               | 122 ± 68               |
| Oceania    | 7 ± 7                | 0 ± 0                        | 11 ± 6                  | 0 ± 0                  |
| World      | 6,251 ± 2,992        | 4,197 ± 2,572                | 27,607 ± 6,817          | 19,772 ± 6,438         | 2,169 ± 782 | 1,602 ± 664 |

SDs reflect uncertainty in the CRF and simulated present-day concentrations (SD = 25% of simulated concentration).

### Figure 2. Estimated annual premature mortalities attributed to anthropogenic O\(_3\) when no upper or lower concentration threshold is assumed, for respiratory mortalities per 1,000 km\(^2\) (A) and rate of respiratory mortalities per 10\(^6\) people (B).
Discussion and Conclusions

We estimated the global burden of mortality due to anthropogenic O\textsubscript{3} and PM\textsubscript{2.5} using a global atmospheric CTM and health impact functions. Anthropogenic O\textsubscript{3} was associated with about 0.7 ± 0.3 million respiratory mortalities (1.1% ± 0.5% of all mortalities) and 6.3 ± 3.0 million YLL annually when we assumed no upper or lower concentration threshold. Anthropogenic PM\textsubscript{2.5} was associated with about 3.5 ± 0.9 million cardiopulmonary (5.6% ± 1.4% of all mortalities) and 220,000 ± 80,000 lung cancer mortalities (0.4% ± 0.1% of all mortalities) annually when we assumed no threshold, corresponding to 30 ± 7.6 million YLL. Global mortalities were reduced by approximately 30% when we assumed LCTs of 33.3 ppb for O\textsubscript{3} and 5.8 µg/m\textsuperscript{3} for PM\textsubscript{2.5}, the lowest measured levels in Jerrett et al. (2009) and Krewski et al. (2009). Estimated excess mortalities were densest in highly populated areas but also occurred in rural areas that have been affected by the increased regional or global background of air pollution since preindustrial times. These estimates based only on cardiopulmonary and lung cancer mortality may be conservative because O\textsubscript{3} and PM\textsubscript{2.5} may also affect other causes of mortality. In addition, to be consistent with the ACS study population, we included only the population ≥ 30 years of age, but evidence suggests that O\textsubscript{3} and PM\textsubscript{2.5} affect health negatively for all ages, including the very young [see U.S. Environmental Protection Agency (EPA) 2008 and references therein].

Estimated PM\textsubscript{2.5} mortalities were five times O\textsubscript{3} mortalities, suggesting that PM\textsubscript{2.5} is the dominant contributor to the global health burden of outdoor air pollution. To minimize double counting of mortalities, we applied long-term RRs for O\textsubscript{3} and PM\textsubscript{2.5} based on the same ACS cohort. PM\textsubscript{2.5} RRs have been shown to be independent from O\textsubscript{3} concentrations (Krewski et al. 2000), and we used O\textsubscript{3} RRs from Jerrett et al. (2009) that controlled for PM\textsubscript{2.5}. Furthermore, Jerrett et al. (2009) and Krewski et al. (2009) reported that PM\textsubscript{2.5}-related mortality was dominated by cardiovascular mortality, whereas O\textsubscript{3} was primarily associated with respiratory mortality. The independence of the exposure–response relationships and the difference in dominant biological mechanisms of mortality for each pollutant imply that double counting is unlikely to be significant. If these implications are correct, O\textsubscript{3} and PM\textsubscript{2.5} mortalities may be summed together to yield total mortalities; otherwise, summing the results would overestimate total mortalities.

Mortality estimates were sensitive to concentration thresholds and concentration–mortality relationships, often changing by >50% of the estimated value under different assumptions. We assumed that the CRFs found by epidemiology studies conducted in North America apply globally, despite differences in health status, lifestyle, age structure, and medical care, and emphasize cause-specific

Figure 3. Estimated annual premature mortalities attributed to anthropogenic PM\textsubscript{2.5} when no upper or lower concentration threshold is assumed, for cardiopulmonary mortalities per 1,000 km\textsuperscript{2} (A), rate of cardiopulmonary mortalities per 10\textsuperscript{6} people (B), lung cancer mortalities per 1,000 km\textsuperscript{2} (C), and rate of lung cancer mortalities per 10\textsuperscript{6} people (D).
Table 4. Estimated annual global O₃ mortalities (mean ± 1 SD) using CRFs from the multipollutant model (in which PM₂.₅ was controlled) and single-pollutant model in Jerrett et al. (2009), and LCTS (×1,000).

| CRF Type         | O₃ Mortalities (×1,000) |
|------------------|-------------------------|
|                  | All Causes              | Cardiopulmonary | Lung Cancer |
| LCT = 7.5 ppb    | 3.049 ± 984             | 222 ± 80        |
| LCT = 25 ppb     | 2.565 ± 816             | 184 ± 68        | (–28.1%)    |
| LCT = 50 ppb     | 2.40 ± 780              | 156 ± 64        | (–34.2%)    |
| LCT = 7.5 ppb    | 2.565 ± 816             | 184 ± 68        | (–28.1%)    |
| LCT = 25 ppb     | 2.32 ± 780              | 156 ± 64        | (–34.2%)    |
| LCT = 50 ppb     | 2.14 ± 765              | 139 ± 61        | (–37.4%)    |

*Calculated using the CRF (0.00432 ppb⁻¹) and corresponding standard error (0.00121 ppb⁻¹) for respiratory mortality when a threshold of 56 ppb is included in the O₃-mortality model (Jerrett et al. 2009). Although Jerrett et al. (2009) found that no threshold model was clearly a better fit to the data than a linear representation of the overall O₃-mortality association, a threshold of 56 ppb was close to statistical significance (p = 0.06).

Table 5. Estimated annual global PM₂.₅ mortalities (mean ± 1 SD) using alternative CRFs with and without LCTS and HCTS (×1,000).

| CRF Type         | Mortality                  | All Causes | Cardiopulmonary | Lung cancer |
|------------------|---------------------------|------------|-----------------|-------------|
| Krewski et al.   | 3.38 ± 988                | 3.499 ± 984| 222 ± 80        |
| LCT = 5.8 µg/m³ | 2.37 ± 876 (–29.7%)       | 2.556 ± 816| 184 ± 68        | (–28.1%)    |
| LCT = 7.5 µg/m³ | 2.07 ± 822 (–38.6%)       | 2.201 ± 780| 164 ± 64        | (–34.2%)    |
| HCT = 30 µg/m³  | 3.05 ± 774 (–9.5%)        | 3.307 ± 676| 201 ± 68        | (–9.5%)     |
| HCT = 50 µg/m³  | 3.33 ± 940 (–1.3%)        | 3.54 ± 626 | 219 ± 78        | (–1.4%)     |
| Pope et al. (2002) | 2.33 ± 119               | 1.80 ± 742 | 139 ± 72        | (–37.4%)    |
| Laden et al. (2006) | 7.714 ± 2.736 (12.8%) | 5.454 ± 1.439| 336 ± 198       | (51.4%)     |

Data in parentheses are percentage change from estimates assuming CRFs from Jerrett et al. (2009) multipollutant model with no LCT (top row). Uncertainty is from the CRF and simulated present-day concentrations (SD = 25% of simulated concentration).

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