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The Impact of Individual Anthropogenic Emissions Sectors on the Global Burden of Human Mortality due to Ambient Air Pollution

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Short Title: Impact of Emissions Sectors on Global Mortality

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Abstract

Background: Exposure to ozone and fine particulate matter ($PM_{2.5}$) can cause adverse health effects, including premature mortality due to cardiopulmonary diseases and lung cancer. Recent studies quantify global air pollution mortality but not the contribution of different emissions sectors, or they focus on a specific sector.

Objectives: We estimate the global mortality burden of anthropogenic ozone and $PM_{2.5}$, and the impact of five emissions sectors, using a global chemical transport model at a finer horizontal resolution (0.67°x0.5°) than previous studies.

Methods: We perform simulations for 2005 with MOZART-4 zeroing-out all anthropogenic emissions and emissions from specific sectors (All Transportation, Land Transportation, Energy, Industry, and Residential & Commercial). We estimate premature mortality using a log-linear concentration-response function for ozone and an integrated exposure-response model for $PM_{2.5}$.

Results: We estimate 2.23 (95% CI: 1.04, 3.33) million deaths/year related to anthropogenic $PM_{2.5}$, with the highest mortality in East Asia (48%). The Residential & Commercial sector has the greatest impact globally – 675 (428, 899) thousand deaths/year – and in most regions. Land Transportation dominates in North America (32% of total anthropogenic $PM_{2.5}$ mortality), and it has almost the same impact (24%) as Residential & Commercial (27%) in Europe. Anthropogenic ozone is associated with 493 (122, 989) thousand deaths/year, with the Land Transportation sector having the greatest impact globally (16%).

Conclusions: The contributions of emissions sectors to ambient air pollution-related mortality differ among regions, suggesting region-specific air pollution control strategies. Global sector-specific actions targeting Land Transportation (ozone) and Residential & Commercial ($PM_{2.5}$) would particularly benefit human health.

1. Introduction

Rising anthropogenic emissions of air pollutants and their precursors have significantly increased ambient air pollution in many parts of the world (Cooper et al. 2014; Lamarque et al. 2010; Naik et al. 2013; Stevenson et al. 2013). Ozone and fine particulate matter ($PM_{2.5}$) are particularly important for public health. Short-term exposure to ozone is associated with respiratory morbidity and mortality (Bell et al. 2014; Gryparis et al. 2004; Levy et al. 2005; Stieb et al. 2009) and long-term exposure has been linked with premature respiratory mortality in adults (Jerrett et al. 2009) and increased risk of death in susceptible populations with chronic cardiopulmonary diseases and diabetes (Zanobetti and Schwartz 2011). Exposure to $PM_{2.5}$ can have detrimental acute and chronic health effects, including premature mortality due to cardiopulmonary diseases and lung cancer (Brook et al. 2010; Burnett et al. 2014; Hamra et al. 2014; Krewski et al. 2009; Lepeule et al. 2012).

The global burden of disease (GBD) due to ambient air pollution was first estimated for urban $PM_{2.5}$ based on surface measurements (Cohen et al. 2004). More recent studies have included urban and rural regions using output from global atmospheric models (Anenberg et al. 2010; Fang et al. 2013; Lelieveld et al. 2013, 2015; Rao et al. 2012), or global modeling output combined with observations (Evans et al. 2013; Lim et al. 2012), to estimate exposure to $PM_{2.5}$ and ozone. Our research group previously used output from an ensemble of global chemistry-climate models to estimate 2.1 million premature deaths/year associated with anthropogenic $PM_{2.5}$ and 470,000 ozone-related deaths/year (Silva et al. 2013).

Here we use a global chemical transport model at fine horizontal resolution to estimate the impact of removing anthropogenic emissions from each of five sectors (Energy, Residential &

Commercial, Industry, Land Transportation, Shipping & Aviation) on the global and regional mortality burden of anthropogenic ozone and $PM_{2.5}$.

Understanding how different sectors impact the global burden and the relative importance of each sector among regions can help prioritize national and international air pollution control strategies. Although the impact of different sectors on health has been quantified in the U.S. (Caiazzo et al. 2013; Fann et al. 2013), Europe (Andersson et al. 2009; Brandt et al. 2013), and, very recently, globally (Lelieveld et al. 2015), other previous global studies focus on one sector – Shipping (Corbett et al. 2007), Aviation (Barrett et al. 2010) and Land Transportation (Bhalla et al. 2014; Chambliss et al. 2014). Using output from the same baseline and land transportation simulations as the present study, Chambliss et al. (2014) calculated the fraction of total $PM_{2.5}$ concentrations attributable to surface transportation emissions, applied that to the total $PM_{2.5}$ concentrations of Brauer et al. (2012) to obtain country-level attributable fractions, and applied those fractions to the GBD 2010 national mortality estimates (Lim et al. 2012).

Estimates of health impacts using output from global models are limited by coarse model resolution that cannot resolve fine gradients in air pollutant concentrations. Coarse resolution estimates are expected to underestimate $PM_{2.5}$-related mortality, mostly due to the smoothing of high urban concentrations, with smaller bias for ozone-related mortality (Li et al. 2015; Punger and West 2013). We attempt to minimize these errors by conducting simulations at a finer horizontal resolution (0.67°x0.5°) than previous global modeling studies assessing health impacts (1°x1° to 2.8°x2.8°). We also quantify the bias in mortality estimates by comparing our results with those obtained using simulations at coarser resolution.

2. Methods

2.1 Modeled ozone and $PM_{2.5}$ concentrations

We simulate ozone and $PM_{2.5}$ concentrations for 2005, using the Model for Ozone and Related Chemical Tracers, version 4 (MOZART-4). MOZART-4 includes a chemical mechanism with detailed hydrocarbon chemistry and bulk aerosols, and online representations of several processes such as dry deposition, biogenic emissions of isoprene and monoterpenes, and photolysis frequencies (Emmons et al. 2010). Anthropogenic and biomass burning emissions are from the Representative Concentration Pathway 8.5 global emissions inventory for 2005 (Riahi et al. 2011) (Supplemental Material, section 1). Biogenic emissions of isoprene and monoterpenes were calculated online in MOZART-4 using the Model of Emissions of Gases and Aerosols from Nature (MEGAN) (Guenther et al. 2006). All other natural emissions were taken from Emmons et al. (2010). The model was run at a 0.67° longitude by 0.5° latitude horizontal resolution with 72 vertical hybrid (sigma and pressure) levels, driven by GEOS-5 meteorological fields. Each simulation was run for 18 months, including 6 months spin-up. Surface concentrations are from the lowest vertical level (992.5 mb at the layer midpoint).

Simulated 2005 surface concentrations show similar agreement with observations as other global models (Supplemental Material, section 3), and as previous MOZART-4 simulations at a coarser resolution using the same meteorology and emissions inputs (Fry et al. 2013). Additionally, we ran a simulation with no anthropogenic emissions to estimate the total mortality burden of present-day anthropogenic ozone and $PM_{2.5}$ (Supplemental Material, section 2). Both simulations were also run at a coarser resolution (2.5°x1.9°) to estimate the bias relative to the fine resolution.

The impact of removing emissions from each source sector is quantified using a brute-force sensitivity analysis, where five emissions sectors are zeroed-out individually: All Transportation, Land Transportation, Energy, Industry, and Residential & Commercial. Land Transportation is a subset of All Transportation; we estimate the impact of Shipping & Aviation as the difference. This zero-out method has been used in previous studies to evaluate the contribution of different regions and/or sectors to ambient air pollutant concentrations (e.g. Andersson et al. 2009; Caiazzo et al. 2013; Corbett et al. 2007; Koch et al. 2007; Li et al. 2015). Due to non-linearity in model response to changes in emissions (e.g. emission reductions may change the ozone chemical regime), estimates of the impacts of a sector using the zero-out method may differ from those using other methods (e.g., source tracking), and the sum of source sector impacts may differ from the total in the baseline simulation (Cohan et al. 2005; Koo et al. 2009; Kwok et al. 2015).

Modeled concentrations in each grid cell were processed to obtain the metrics used in the health impact assessment, consistent with the underlying epidemiological studies (Burnett et al. 2014; Jerrett et al. 2009; Krewski et al. 2009): annual average $PM_{2.5}$ and average 1-hr daily maximum ozone for the consecutive 6-month period with the highest average. $PM_{2.5}$ concentrations were estimated as a sum of modeled species (Supplemental Material, section 4).

2.2 Health impact assessment

We estimate cause-specific excess mortality due to exposure to ambient air pollution ($\Delta Mort$) in each MOZART-4 grid cell as $\Delta Mort = y_0 * AF * Pop$, where y_0 is the baseline mortality rate (for the exposed population), $AF = 1 - 1/RR$ is the attributable fraction (RR = relative risk of death attributable to a change in pollutant concentration), and *Pop* is the exposed population (adults aged 25 and over).

For ozone, $AF = 1\text{-}exp^{-\beta\Delta X}$, where $RR = exp^{\beta\Delta X}$, β is the concentration-response factor, ΔX corresponds to the change in pollutant concentrations, and *RR* = 1.040 (95% Confidence Interval, CI: 1.013, 1.067) for a 10 ppb increase in ozone concentrations from Jerrett et al. (2009), the largest study to estimate *RR* for long-term exposure to ozone. Although Jerrett et al. (2009) estimated *RR* for adults aged 30 and over, we consider adults aged 25 and over, assuming identical *RR*, to align exposed populations for ozone and $PM_{2.5}$, following Lim et al. (2012). However, we evaluate ozone mortality due to all chronic respiratory diseases (ICD-9 BTL: B347) based on Jerrett et al. (2009), as in other global studies (Anenberg et al. 2010; Fang et al. 2013; Lelieveld et al. 2013; Silva et al. 2013), while Lim et al. (2012) considered only chronic obstructive pulmonary disease (COPD) mortality (78% of global chronic respiratory disease mortality, ranging from 27% to 93% nationally).

For $PM_{2.5}$, we use the Integrated Exposure-Response (IER) model developed for GBD 2010 (Burnett et al. 2014), which is intended to better estimate mortality at high $PM_{2.5}$ concentrations:

$$RR_{IER}(z) = \begin{cases} 1, & z < z_{cf} \\ 1 + \alpha\left(1 - e^{-\gamma\left(z - z_{cf}\right)^{\delta}}\right) & z \geq z_{cf} \end{cases}$$

where z is $PM_{2.5}$ concentration and z_{cf} is the counterfactual concentration (theoretical minimum-risk exposure, assumed by Burnett et al. (2014) to have a uniform distribution: $z_{cf} \sim U[5.8, 8.8]$).

We use the RRs given by IER for mortality due to ischemic heart disease (IHD, ICD-9: 410-414), cerebrovascular disease (Stroke, ICD-9: 430-435, 437.0-437.2, 437.5-437.8), COPD (ICD-

9: 490-492.8, 494, 496) and lung cancer (LC, ICD-9 BTL: B101). We use the values for parameters alpha, gamma and delta reported by Burnett et al. (2014) for 1000 simulations (GHDx 2013). We calculate $AF=AF_1-AF_2$, where $AF_1 = 1-1/RR_{IER(z1)}$ and $AF_2 = 1-1/RR_{IER(z2)}$, z_1 = baseline concentration (simulation with all anthropogenic emissions), z_2 = concentration in control simulation (with zeroed-out emissions).

We define the mortality burden of anthropogenic air pollution as that which is controllable, using the simulation with no anthropogenic emissions to estimate ΔX for ozone, and z_2 for $PM_{2.5}$, following Anenberg et al. (2010), Fang et al. (2013), Lelieveld et al. (2013), and Silva et al. (2013). This approach differs from GBD 2010, which considers total $PM_{2.5}$ relative to z_{cf} ($AF = AF_1$). Where natural $PM_{2.5}$ is below z_{cf} ($AF_2 = 0$), our estimate of excess mortality is identical to the total $PM_{2.5}$ mortality burden. If natural $PM_{2.5}$ concentrations are above z_{cf} (e.g. dusty regions), we estimate mortality due to anthropogenic air pollution only. Whereas Giannadaki et al. (2014) quantify the contribution of desert dust to global mortality, it is considered natural $PM_{2.5}$ under our definition. Also, given the non-linearity of the IER model, we assume that the impact of removing each sector corresponds to the difference in mortality estimates for $PM_{2.5}$ concentrations in each zeroed-out simulation relative to the total $PM_{2.5}$. As a sensitivity analysis, we also use the log−linear function with RR for CPD and LC from Krewski et al. (2009), following other global health assessments (Anenberg et al. 2010; Evans et al. 2013; Fang et al. 2013; Lelieveld et al. 2013; Silva et al. 2013).

Exposed population was obtained from the Oak Ridge National Laboratory's Landscan 2011 Global Population Dataset at approximately 1 km resolution (30"x30") (Bright et al. 2012). For adults aged 25 and over, we estimated population per 5-year age group in each cell by multiplying the country-level percentage in each age group (from Landscan) by the total cell population, using ArcGIS 10.2. Cause-specific baseline mortality rates for 187 countries were obtained from the GBD 2010 mortality dataset (IHME, 2013). We estimated the number of deaths per 5-year age group per country using national population from Landscan, and gridded these using ArcGIS 10.2. The resulting population and baseline mortality per age group at 30"x30" were regridded to the resolutions of the atmospheric model (0.67°x0.5° and 2.5°x1.9°).

We conducted 1000 Monte Carlo (MC) simulations to propagate uncertainty from the RRs, baseline mortality rates, and modeled air pollutant concentrations using random sampling of the three variables simultaneously. For ozone RRs, we used the reported 95% CIs and assumed a normal distribution. For $PM_{2.5}$ RRs, we used the parameter values of Burnett et al. (2014) for 1000 simulations (GHDx 2013). Also, we considered the reported 95% CIs for baseline mortality rates, assuming lognormal distributions. Finally, for modeled ozone and $PM_{2.5}$ concentrations we used the absolute value of the coefficient of variation (= standard deviation / mean) at each grid cell for the year 2000 minus year 1850 simulations from the ACCMIP ensemble (Lamarque et al. 2013; Silva et al. 2013), regridded to 0.67°x0.5° and following a normal distribution. Uncertainty associated with population was assumed negligible. For each MC simulation, we obtained the regional and global totals which we then used to estimate the empirical mean and 95% CI of the regional and global mortality results. We estimated the contribution of uncertainty in each variable to overall uncertainty in mortality estimates using a tornado analysis.

3. Results

Global ozone and $PM_{2.5}$ surface concentrations and population-weighted averages for ten world regions, exposed population, and baseline mortality rates are shown in the Supplemental Material (section 4).

We estimate the present-day global burden of anthropogenic ozone-related respiratory mortality to be 493 (95% CI: 122, 989) thousand deaths/year (Table 1). Most mortality occurs in East Asia (35%) and India (33%) (Figure 1 and Supplemental Material, Table S7). These regions are highly populated and, together with North America, have the highest population-weighted average anthropogenic ozone concentrations. East Asia and India have 113 deaths/year per million people due to ozone, whereas the lowest premature mortality rate occurs in Africa (11 deaths/year per million people) (Supplemental Material, Table S9). For global ozone mortality, the coefficient of variation (CV = standard deviation / mean) is 46% and uncertainty in β and in ΔX have similar contributions to overall uncertainty (45% each), while uncertainty in y_0 contributes 10%.

For anthropogenic $PM_{2.5}$, we estimate a global mortality burden of 2.2 (1.0, 3.3) million deaths/year (Table 1), with contributions from IHD (926 [436, 1300] thousand), Stroke (887 [439, 1300] thousand), COPD (260 [79.2, 477] thousand) and LC (157 [29.8, 316] thousand). The greatest mortality occurs in East Asia (48%), followed by India (18%) and Europe (11%) (Figure 1 and Supplemental Material, Table S8), regions with the highest population-weighted average anthropogenic $PM_{2.5}$. The number of deaths in Australia and South America is very low because of large areas with low population density; also, these regions have the lowest average concentrations (Supplemental Material, Table S4) that are below the threshold of the IER function in many grid cells. While East Asia has 683 deaths/year per million people due to anthropogenic $PM_{2.5}$, the lowest mortality rate occurs in Africa (32 deaths/year per million people) (Supplemental Material, Table S10). The global CV for $PM_{2.5}$ mortality is 25%, but global CVs are greater for COPD (40%) and LC (46%) than for IHD (25%) and Stroke (26%). Uncertainties in the RR model parameters α, γ and δ together have the greatest contribution to overall uncertainty (71.7%), followed by z_1 (23.3%), but z_2 (2.3%), y_0 (2.4%), and z_{cf} (0.2%) contribute little to overall uncertainty. When considering each disease individually, the contributions of different variables vary from those mentioned above, particularly the contributions of z_1 to IHD (33.2%), COPD (14.1%), and LC (13.0%) mortality uncertainties.

Globally, the zeroed-out sectors contribute about 57% of total anthropogenic ozone mortality (Table 1). Land Transportation has the greatest global impact (16%) and the greatest regional impact (20 to 26%) in North America, South America, Europe, FSU and Middle East (Figures 2 and 3), as it strongly influences ozone concentrations. Energy and Residential & Commercial also have strong impacts in India and all sectors have important impacts in East Asia. Among the deaths caused by each sector worldwide, the greatest impacts occur in India and East Asia, particularly for Residential & Commercial (83%), Industry (75%) and Energy (74%), reflecting the large exposed populations in these regions. Within each region, there is variability in the impact of different sectors, with a few hotspots for certain sectors (e.g. central Africa for Residential & Commercial, eastern North America and India for Energy, and eastern East Asia for Industry). The 43% of the total burden not accounted for by the five modeled sectors likely reflects sectors not zeroed-out, mainly Biomass Burning emissions, increases in methane from preindustrial times to present day, and nonlinear model responses.

For anthropogenic $PM_{2.5}$, the modeled sectors contribute 70% of total global mortality (Table 1). Residential & Commercial contributes 675 (428, 899) thousand deaths/year, having the greatest impact globally (30%) and in most regions except North America, South America and Australia (Figures 4 and 5). Land Transportation dominates in North America (32% of total anthropogenic $PM_{2.5}$ mortality in this region), and in Europe it has nearly the same burden (24%) as Residential & Commercial (27%). In East Asia, Residential & Commercial contributes 21% of total mortality, followed by Industry (17%) and Energy (11%). Residential & Commercial has the greatest impact in East Asia (33%), followed by India (26%). Industry and Energy also impact East Asia the most (55% and 41%). Land Transportation has the strongest impact in Europe (27%) and East Asia (23%). The different regional impacts are associated with the impact of removing emissions from each sector on total anthropogenic $PM_{2.5}$ concentrations, and the exposed population and baseline mortality rates in each region (e.g. cardiovascular diseases in FSU). Within each region the impact of each sector varies reflecting the location of emission sources (e.g. eastern North America for Energy; and small areas in Europe, FSU, southern Africa, eastern South America, Middle East and East Asia for Energy and Industry). The 30% of total burden not accounted for by the five modeled sectors is likely associated mainly with Biomass Burning emissions.

3.1 Sensitivity analyses

3.1.1 Fine vs. coarse resolution

Using output from simulations at fine and coarse grid resolutions to directly estimate mortality, we quantify a slight negative bias of 2% for global ozone mortality and a positive bias of 16% for global $PM_{2.5}$ mortality at coarse resolution relative to fine resolution (Supplemental Material, section 7.1 and Table S13). When we regrid fine resolution modeled concentrations to the coarse resolution, following Punger and West (2013), the negative bias of the global mortality estimates for regridded ozone concentrations slightly increases to 3% (relative to original fine resolution) but the bias for $PM_{2.5}$ changes sign to a negative bias of 8% (Supplemental Material, section 7.1 and Table S14). The biases for mortality estimates obtained at original coarse resolution reflect the total effect of grid resolution on both modeled “chemistry” (e.g. Wild and Prather 2006) and “exposure” (the spatial alignment of population and concentration), while the biases estimated using concentrations reggrided to coarse resolution only capture the effect of resolution on exposure. For ozone, our total bias is very close to the “exposure” bias, suggesting a minor effect of resolution on modeled chemistry. For $PM_{2.5}$, our positive total bias at coarse resolution likely reflects a local effect of grid resolution on $PM_{2.5}$ chemistry. Our “exposure” negative bias of 8% for $PM_{2.5}$ is comparable to those estimated by Punger and West (2013) and by Li et al. (2015), showing the effect on mortality estimates of the spatial degradation of urban $PM_{2.5}$ concentrations.

3.1.2 Log-linear exposure-response function for $PM_{2.5}$

Using the log-linear model and RRs of Krewski et al. (2009), we obtain 74% of the global burden of anthropogenic $PM_{2.5}$ mortality estimated with the IER function, with marked regional differences (e.g. for North America, the log-linear estimate is 16% higher than the IER estimate). We use the RR reported for CPD for IHD, Stroke and COPD and the RR reported for LC to allow for a straightforward comparison with the IER estimate. IHD and Stroke mortality decrease by 60% and 57%, while COPD and LC mortality increase by 131% and 107%.

These differences can be explained by the non-linear shape of the IER function (Burnett et al. 2014), which gives considerably different estimates of AF for identical changes in $PM_{2.5}$

concentrations in areas with low vs. high total $PM_{2.5}$ concentrations, such as North America (8.5µg/m^3) and Middle East (27.8 µg/m^3), with the latter being on the flatter part of the IER curves. Population-weighted average anthropogenic $PM_{2.5}$ (2005 minus natural) for North America and Middle East are very close (7.1 and 7.2 µg/m^3), as are the attributable fractions for CPD (8.2% and 8.3%) and LC (9.0% and 9.1%) using the log-linear model. However, using the RRs from the IER model, AFs for IHD for North America are between 21% and 6% for all age groups, while for Middle East they are between 5% and 3%; for LC they are 2.0% (North America) and 3.9% (Middle East) for adults 25+.

4. Discussion

Our global burden estimates are comparable to those of Silva et al. (2013) using an ensemble of global models, being 5% greater for ozone mortality and 6% greater for $PM_{2.5}$ mortality, although here we use the IER model to estimate $PM_{2.5}$ mortality. For ozone mortality, our results differ from Anenberg et al. (2010) (-30%), Lim et al. (2012) (+228%), Fang et al. (2013) (+31%), Lelieveld et al. (2013) (-36%), and Lelieveld et al. (2015) (+246%). For $PM_{2.5}$, our estimates are lower than those of Anenberg et al. (2010) (-40%), Lim et al. (2012) (-30%) , Evans et al. (2013) (-18%), and Lelieveld et al. (2015) (-19%), but higher than Lelieveld et al. (2013) (+2%) and Fang et al. (2013) (+40%). We do not suggest that our estimates are better than those from these studies but we highlight differences between approaches, particularly our use of a fine resolution model and our evaluation of anthropogenic air pollution by comparing with a simulation with no anthropogenic emissions.

Our lower estimates compared to Anenberg et al. (2010) may be related to the finer resolution (vs. 2.8°x2.8°) and updates in MOZART-4 (vs. MOZART-2), but are likely due to the use of different emissions datasets, different exposure-response functions for $PM_{2.5}$, and updated population and baseline mortality rates. In comparison with Lim et al. (2012) and Lelieveld et al. (2015), we use the same exposure-response functions for $PM_{2.5}$, but we estimate anthropogenic $PM_{2.5}$ mortality, while they estimated total $PM_{2.5}$ mortality; also Lelieveld et al. (2015) used a different exposure-response function for ozone and both Lim et al. (2012) and Lelieveld et al. (2015) considered a low-concentration threshold for ozone mortality and baseline mortality rates for COPD only (whereas we considered all Chronic Respiratory diseases); differences in the spatial distributions of pollutant concentrations and exposed population may also be important. The other studies were based on model output from different global models using different inputs and definitions of anthropogenic air pollution (Fang et al. 2013; Lelieveld et al. 2013) or were based on observations and model output of total pollutant concentrations (Evans et al. 2013); their health impact assessments used the log-linear exposure-response function for $PM_{2.5}$, and different population and baseline mortality rates.

A major contribution from this study is estimating sectoral contributions to the total burden of anthropogenic air pollution on mortality globally and regionally. Our estimates of almost 50,000 $PM_{2.5}$ -related deaths/year due to Shipping & Aviation are about 30% lower than the combined estimates of Corbett et al. (2007) for Shipping and Barrett et al. (2010) for Aviation, but within their confidence intervals. For Land Transportation, our estimate is 12% lower than that of Chambliss et al. (2014), reflecting a different methodology despite using identical modeled $PM_{2.5}$ concentrations. For sectors also evaluated by Lelieveld et al. (2015), our results for the sum of ozone and $PM_{2.5}$-related mortality are lower for Residential & Commercial (-27%) and Energy (-24%) and higher for Land Transportation (+79%) and Industry (+63%); these differences should be due to the methodological differences mentioned above as well as in the underlying emission inventories.

We choose not to add ozone and $PM_{2.5}$ mortality to avoid possibly double counting respiratory mortality (since we include $PM_{2.5}$ mortality associated with COPD). However, we calculated ozone respiratory mortality using RR from Jerrett et al. (2009), who controlled for $PM_{2.5}$, so double counting should be negligible due to different biological mechanisms associated with exposure to each pollutant (Anenberg et al. 2010). Our results assume that the same RRs apply worldwide, even though underlying health conditions and $PM_{2.5}$ composition vary. The RR for ozone is based on results from a US cohort (Jerrett et al. 2009), while the IER function for $PM_{2.5}$ is based on studies in North America, Western Europe and China (Burnett et al. 2014). Also, we limit our study to adults aged 25 and over, which may underestimate total and sectoral burdens. We reduce the potential for coarse resolution bias by conducting simulations at a fine horizontal resolution for a global chemical transport model; however, our results are still limited by resolution and cannot fully resolve fine concentration gradients, particularly near urban areas. For example, emissions from the Residential & Commercial sector occur where people live, and more detailed spatial analyses may suggest a greater relative impact for this sector. Our uncertainty estimates are wider than other studies, reflecting our use of the spread of modeled concentrations from the ACCMIP multi-model ensemble. These estimates of uncertainty do not account for uncertainty in emissions inventories (as the ensemble used identical emissions), nor for uncertainty in population, which is likely small.

5. Conclusions

We find regional differences in the relative importance of emissions sectors to ambient air pollution-related mortality. Globally, we estimate 493,000 deaths/year due to anthropogenic ozone and 2.2 million deaths/year due to anthropogenic $PM_{2.5}$. Land Transportation has the greatest impact on ozone respiratory mortality (80,900 deaths/year, 16% of the global burden), while the Residential & Commercial sector contributes the most to $PM_{2.5}$-related premature mortality (IHD+Stroke+COPD+LC) (675,000 deaths/year, 30%).

In East Asia, Industry has the greatest impact on ozone mortality (14%), and also has a great impact on $PM_{2.5}$ mortality (17%) following Residential & Commercial (21%). In India, Energy has the greatest impact on ozone mortality (17%), but the Residential & Commercial sector clearly dominates $PM_{2.5}$ mortality (43%). In North America, Land Transportation has the greatest impact both for ozone (23%) and $PM_{2.5}$ mortality (55%).

Uncertainty in RR and in modeled ozone concentrations have similar contributions to overall uncertainty in ozone mortality, while uncertainty in RR has the greatest impact on total $PM_{2.5}$ mortality and, especially, on COPD and LC mortality. Future epidemiological research on the long-term effects of air pollution should aim to narrow the uncertainty in RR, particularly in developing nations worldwide. Future research should also focus on improving emissions inventories for air quality modeling and reducing the bias in modeled air pollutant concentrations.

The relative impact of removing emissions from different sectors to anthropogenic ozone- and $PM_{2.5}$-related mortality in different regions suggests that location-specific air pollution control policies are appropriate. However, the development of improved emission control technologies may be pursued globally. Global actions to reduce emissions of ozone precursors from Land Transportation would be particularly beneficial for health, as would reducing $PM_{2.5}$ emissions from the Residential & Commercial sector. In East Asia, additional air pollution control strategies addressing all sectors would considerably lessen global mortality. A focus on the Energy sector and $PM_{2.5}$ emissions from Industry in India, and $PM_{2.5}$ emissions from Land Transportation in North America and Europe would yield the greatest benefit for health.

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Table 1. Global premature ozone and $PM_{2.5}$-related mortality, and impact of removing emissions from individual sectors (thousand deaths in 2005), showing the mean and 95% CI.

	All Anthropogenic	All Transportation	Land Transportation	Energy	Industry	Residential & Commercial
Ozone mortality	493 (122, 989)	115 (27.8, 244)	80.9 (17.4, 180)	65.2 (14.5, 143)	45.6 (8.7, 96.8)	53.7 (12.3, 116)
$PM_{2.5}$ mortality	2230 (1040, 3330)	261 (136, 364)	212 (114, 292)	290 (192, 386)	323 (230, 430)	675 (428, 899)

Figure Legends

Figure 1 – Premature ozone-related respiratory mortality (A) and $PM_{2.5}$-related mortality (IHD+Stroke+COPD+LC) (B) in 2005 (deaths per year per 1000 km^2), shown as the mean of 1000 Monte Carlo simulations.

Figure 2 – Impact of removing emissions from each sector (A – E) on total ozone-related respiratory mortality in 2005, shown as a ratio to the total burden in each cell. Areas shown as white have <1 ozone-related death per grid cell.

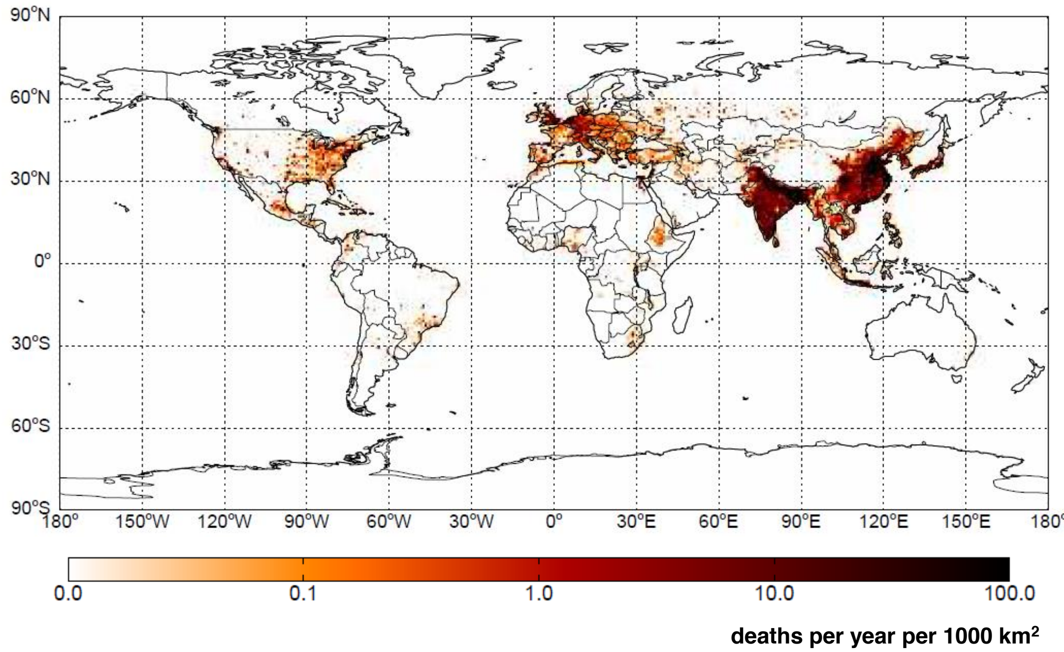
Figure 3 – Impact of removing emissions from each sector on premature ozone-related respiratory mortality in each region and globally, relative to the total burden (deaths in 2005). Numbers above each column correspond to the total burden (all anthropogenic emissions zeroed-out), and to the sum of five sectors. The ten world regions are defined in Supplemental Material (Figure S7): NA – North America, SA – South America, Europe, FSU – Former Soviet Union, (Sub-Saharan) Africa, India, East Asia, SE Asia – Southeast Asia, Aus. – Oceania, ME – Middle East (and North Africa).

Figure 4 – Impact of removing emissions from each sector (A – E) on total premature $PM_{2.5}$-related mortality (IHD+Stroke+COPD+LC) in 2005, shown as the ratio of total burden in each cell. Areas shown as white have <1 $PM_{2.5}$-related death per grid cell.

Figure 5 – Impact of removing emissions from each sector on premature $PM_{2.5}$-related mortality (IHD+Stroke+COPD+LC) in each region and globally, relative to the total burden (deaths in 2005). Numbers above each column correspond to the total burden (all anthropogenic emissions zeroed-out), and to the sum of five sectors.

Figure 1.

(A) Ozone mortality



(B) $PM_{2.5}$ mortality

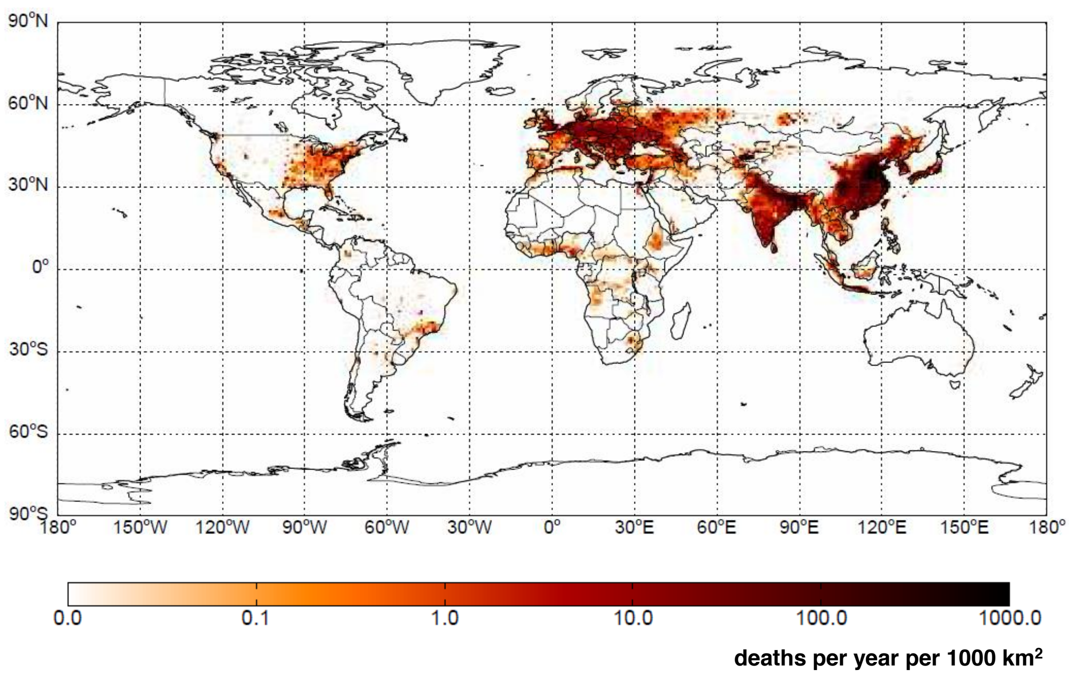
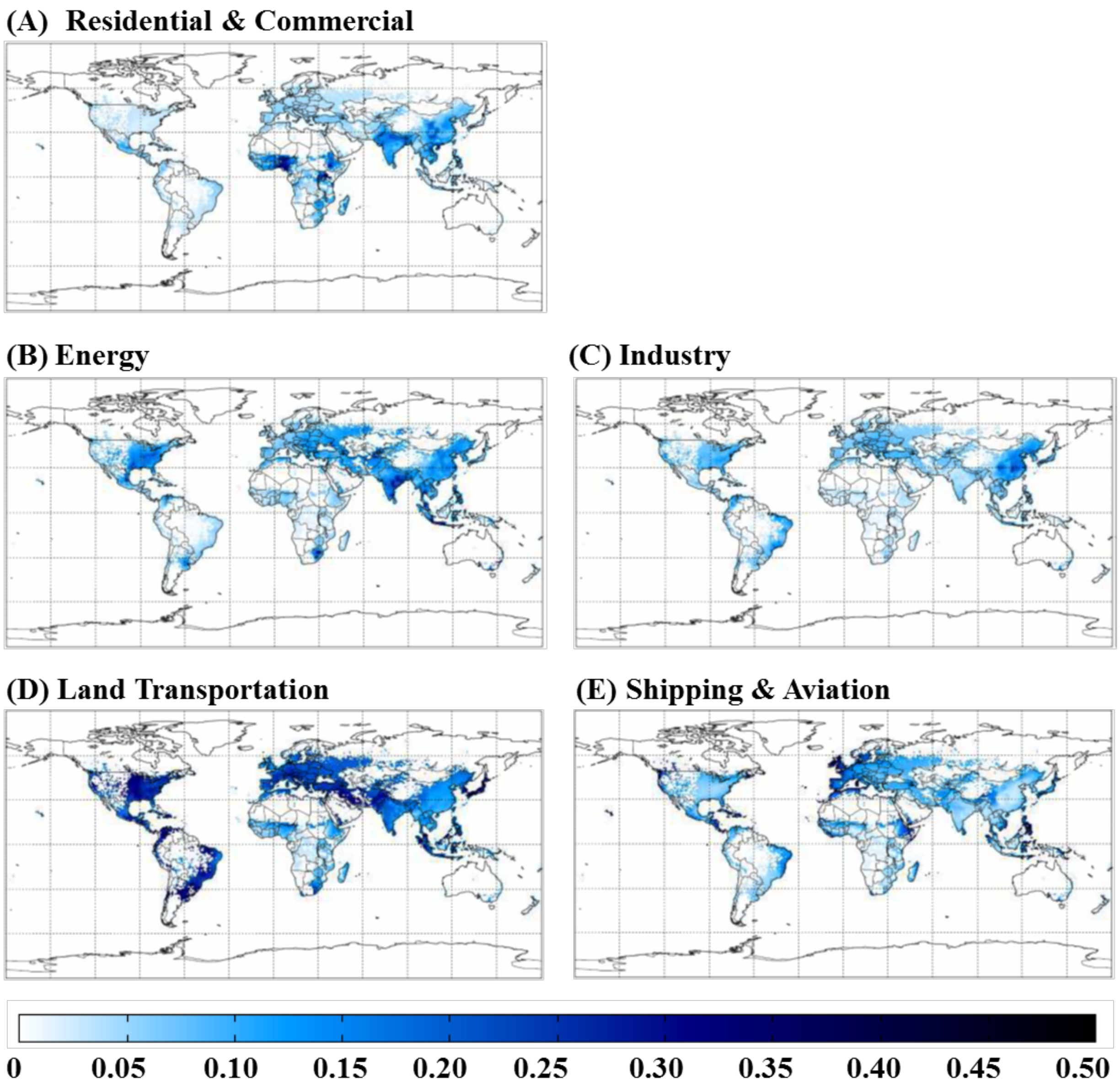


Figure 2.



(A) Residential & Commercial
(B) Energy
(C) Industry
(D) Land Transportation
(E) Shipping & Aviation
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0.05
0.10
0.15
0.20
0.25
0.30
0.35
0.40
0.45
0.50

Figure 3.

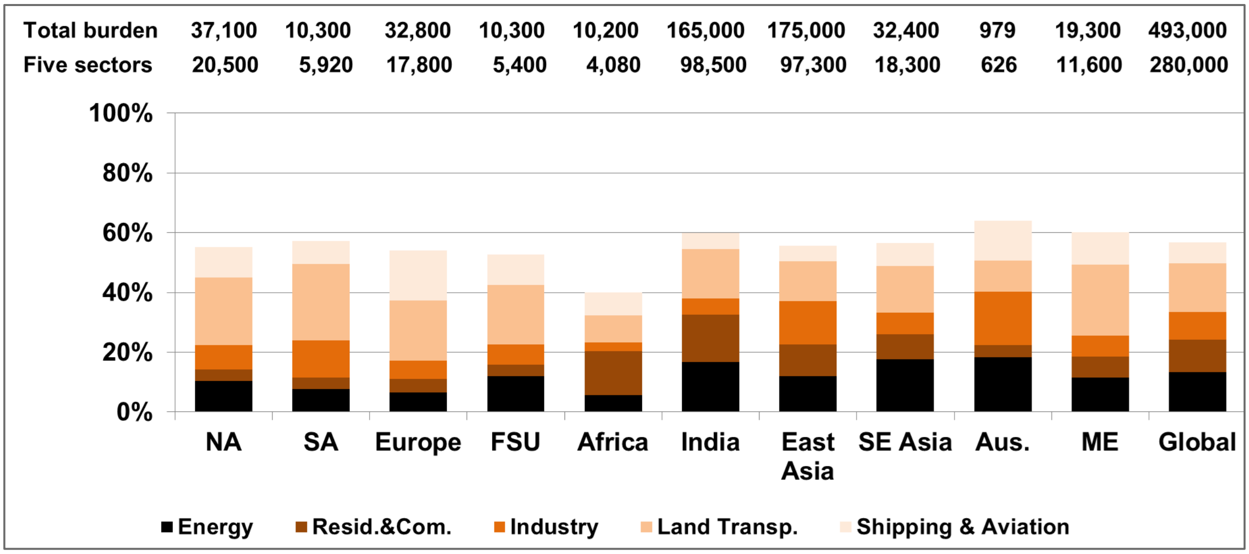
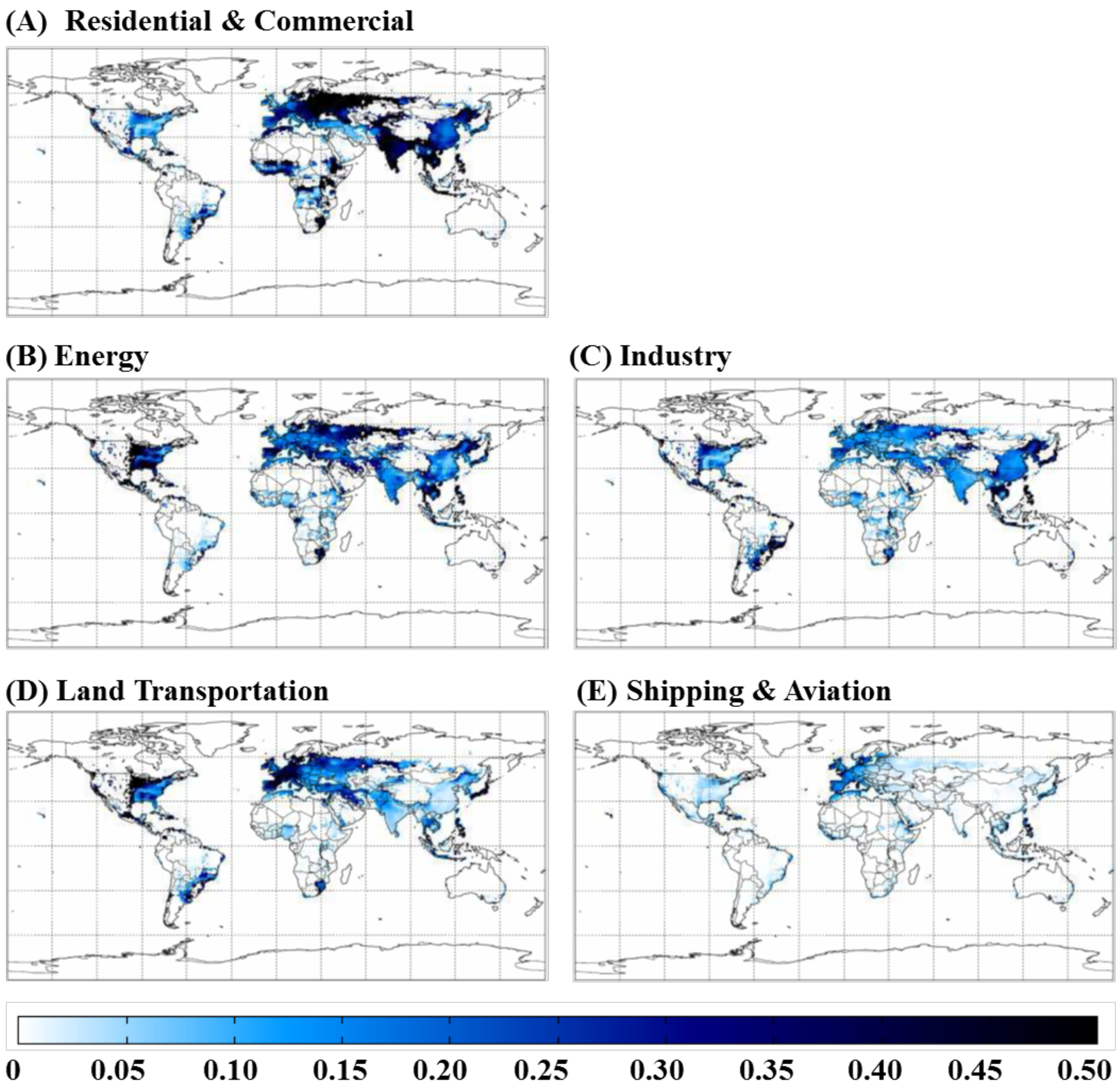

Total burden 37,100 10,300 32,800 10,300 10,200 165,000 175,000 32,400 979 19,300 493,000
Five sectors 20,500 5,920 17,800 5,400 4,080 98,500 97,300 18,300 626 11,600 280,000
100%
80%
60%
40%
20%
0%
NA
SA
Europe
FSU
Africa
India
East Asia
SE Asia
Aus.
ME
Global
Energy
Resid.&Com.
Industry
Land Transp.
Shipping & Aviation

Figure 4.



(A) Residential & Commercial
(B) Energy
(C) Industry
(D) Land Transportation
(E) Shipping & Aviation
0
0.05
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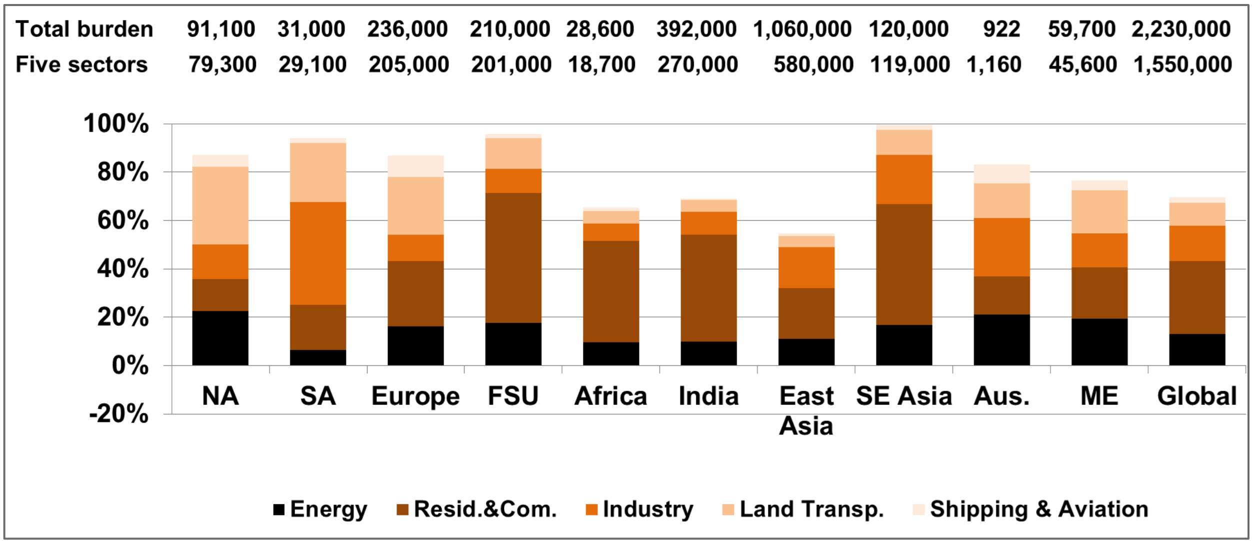
Figure 5.


Total burden 91,100 31,000 236,000 210,000 28,600 392,000 1,060,000 120,000 922 59,700 2,230,000
Five sectors 79,300 29,100 205,000 201,000 18,700 270,000 580,000 119,000 1,160 45,600 1,550,000
100%
80%
60%
40%
20%
0%
-20%
NA
SA
Europe
FSU
Africa
India
East Asia
SE Asia
Aus.
ME
Global
Energy
Resid.&Com.
Industry
Land Transp.
Shipping & Aviation