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2016 Environ. Res. Lett. 11 124022

(http://iopscience.iop.org/1748-9326/11/12/124022)

Environmental Research Letters

LETTER

Global burden of mortalities due to chronic exposure to ambient $PM_{2.5}$ from open combustion of domestic waste

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Keywords: aerosol, environmental health, smoke, ambient $PM_{2.5}$

Supplementary material for this article is available online

OPEN ACCESS

RECEIVED
28 June 2016

REVISED
9 November 2016

ACCEPTED FOR PUBLICATION
11 November 2016

PUBLISHED
12 December 2016

Abstract

Uncontrolled combustion of domestic waste has been observed in many countries, creating concerns for air quality; however, the health implications have not yet been quantified. We incorporate the Wiedinmyer *et al* (2014 *Environ. Sci. Technol.* **48** 9523–30) emissions inventory into the global chemical-transport model, GEOS-Chem, and provide a first estimate of premature adult mortalities from chronic exposure to ambient $PM_{2.5}$ from uncontrolled combustion of domestic waste. Using the concentration-response functions (CRFs) of Burnett *et al* (2014 *Environ. Health Perspect.* **122** 397–403), we estimate that waste-combustion emissions result in 270 000 (5th–95th: 213 000–328 000) premature adult mortalities per year. The confidence interval results only from uncertainty in the CRFs and assumes equal toxicity of waste-combustion $PM_{2.5}$ to all other $PM_{2.5}$ sources. We acknowledge that this result is likely sensitive to choice of chemical-transport model, CRFs, and emission inventories. Our central estimate equates to 9% of adult mortalities from exposure to ambient $PM_{2.5}$ reported in the Global Burden of Disease Study 2010. Exposure to $PM_{2.5}$ from waste combustion increases the risk of premature mortality by more than 0.5% for greater than 50% of the population. We consider sensitivity simulations to uncertainty in waste-combustion emission mass, the removal of waste-combustion emissions, and model resolution. A factor-of-2 uncertainty in waste-combustion $PM_{2.5}$ leads to central estimates ranging from 138 000 to 518 000 mortalities per year for factors-of-2 reductions and increases, respectively. Complete removal of waste combustion would only avoid 191 000 (5th–95th: 151 000–224 000) mortalities per year (smaller than the total contributed premature mortalities due to nonlinear CRFs). Decreasing model resolution from $2^\circ \times 2.5^\circ$ to $4^\circ \times 5^\circ$ results in 16% fewer mortalities attributed to waste-combustion $PM_{2.5}$, and over Asia, decreasing resolution from $0.5^\circ \times 0.666^\circ$ to $2^\circ \times 2.5^\circ$ results in 21% fewer mortalities attributed to waste-combustion $PM_{2.5}$. Owing to coarse model resolution, our global estimates of premature mortality from waste-combustion $PM_{2.5}$ are likely a lower bound.

1. Introduction

Open, uncontrolled combustion of domestic waste (i.e. trash burning) occurs on a global scale, emitting particulate matter (PM) and toxic gaseous and particulate compounds (Christian *et al* 2010, Wiedinmyer *et al* 2014). Domestic waste can include food and agricultural products, containers and packaging, and other sources of residential trash. Combustion occurs both in homes and at community waste sites (i.e. dumps), and both in developing and developed countries, though waste combustion is more

widespread in developing countries (Wiedinmyer *et al* 2014). While combustion of waste is ubiquitous in rural areas, there are also significant emissions in urban areas, potentially leading to pollution exposure of dense populations (Wiedinmyer *et al* 2014).

Until recently, inventories of pollutant emissions from combustion processes either did not include emissions from uncontrolled domestic-waste combustion (e.g. Bond *et al* 2007, Janssens-Maenhout *et al* 2010), or the domestic-waste-combustion emissions were assumed to be low relative to other sources in all locations (Bond *et al* 2004). However, Wiedinmyer *et al* (2014) recently created an emissions inventory focused specifically on the emissions from the uncontrolled open combustion of domestic-waste products. The total estimated $PM_{2.5}$ emissions were 10 Tg yr^{-1}, globally, an order of magnitude higher than a previous estimate in Bond *et al* (2004), and similar to the emissions from biofuel combustion in Bond *et al* (2004). The more-recent estimates were higher due to newer assumptions in waste production, fraction burned, and emission factors. The largest driver of the difference in emissions was the estimated amount of annual waste burned: Bond *et al* (2004) estimated a total of 33 Tg, while Wiedinmyer *et al* (2014) predict 970 Tg. This discrepancy is in part due to the assumption in Bond *et al* (2004) that no waste combustion occurs in rural developing countries. Thus, ambient air quality particularly in developing countries may be substantially impacted by domestic-waste combustion.

PM air pollution is a major contributor to the Global Burden Of Disease (GBD), and the most important environmental contributor to morbidity and mortality in the world (Lim *et al* 2012). Thus, exposure to ambient pollution from uncontrolled domestic-waste combustion may have serious health implications, yet these health effects have not been quantified. A number of past studies have linked exposure to ambient PM with aerodynamic diameters less than 2.5 μm ($PM_{2.5}$) to increased risk of mortality for adults from ischaemic heart disease (IHD), cerebrovascular disease (CeVD, or stroke), chronic obstructive pulmonary disease (COPD), and lung cancer (LC) (Pope *et al* 2002, Laden *et al* 2006, Krewski *et al* 2009, Burnett *et al* 2014). Using a combination of satellite and ground observations, and chemical-transport models to estimate $PM_{2.5}$ exposure from all sources, the GBD Study 2010 estimated 3.1 million premature mortalities, globally, in 2010 (Lim *et al* 2012).

For global/regional scale studies, chemical-transport models have been used to estimate surface-level ambient $PM_{2.5}$ exposure for use in health-impact assessments (e.g. Anenberg *et al* 2010, Lelieveld *et al* 2013, 2015, Butt *et al* 2016). Despite uncertainties in emissions and atmospheric processes, models have an advantage over ground-based measurements in that they provide exposure estimates in regions where few surface measurements are available. In addition, models can provide source-specific $PM_{2.5}$ estimates, thereby allowing for policy-relevant mortality estimates from specific emission sectors (e.g. Corbett *et al* 2007, Butt *et al* 2016). Recently, Lelieveld *et al* (2015) used the ECHAM model to estimate source-specific mortalities, finding dominant sources in Asia are from residential solid-fuel combustion and biomass burning; however, this study does not include emissions from open waste combustion.

One potential drawback of using chemical-transport models on a global scale is that computational efficiency often limits model spatial resolution. Lower-resolution global models average $PM_{2.5}$ concentrations over larger areas, limiting the ability to predict very-polluted regions and sharp gradients outside these regions. As emissions are often co-located with population, this may lead to a dependence of mortality estimates on model resolution. Punger and West (2013) estimated ambient $PM_{2.5}$ mortalities in the United States using different model resolutions, and find substantially (~30%) lower estimates when scaling $PM_{2.5}$ to spatial scales representative of global models (>250 km) relative to 12 km. One method to correct for sub-grid scale gradients involves using satellite aerosol optical depth (AOD) at resolutions higher than the model (e.g. ~10 km versus ~200 km) to interpolate $PM_{2.5}$ exposure fields at the resolution of the satellite AOD. However, this method relies on the ratio of modeled AOD to surface $PM_{2.5}$ (van Donkelaar *et al* 2015). Ford and Heald (2016) estimated 20% uncertainty in mortality due to methodology in estimating surface $PM_{2.5}$ concentrations from satellite AOD. Additionally, while this method was used in several studies to estimate all-source $PM_{2.5}$ mortality (Lim *et al* 2012, Evans *et al* 2013), satellite observations cannot be used to isolate individual sources of $PM_{2.5}$; one would need to assume that spatial distribution of AOD from each individual source follows the spatial distribution of the total AOD.

In this study, we provide the first estimates of mortality due to chronic exposure to $PM_{2.5}$ from domestic-waste combustion. We include the recent Wiedinmyer *et al* (2014) waste-combustion inventory into the global/regional chemical-transport model, GEOS-Chem. In section 2, we discuss the model setup and methodology for estimating mortality. In section 3.1, we present model results for waste-combustion $PM_{2.5}$ and in section 3.2 we present the global and country-level attributed mortality. In section 3.3, we estimate the deaths averted due to complete removal of emissions. In section 3.4, we test the sensitivity of mortality rates to uncertainties in emission mass and model resolution. We share our conclusions in section 4. In a separate study, Kodros *et al* (2016), we estimate the aerosol radiative impacts from domestic-waste combustion.

2. Methods

2.1. Chemical-transport model overview

To estimate $PM_{2.5}$ concentrations, we use the Goddard Earth Observing System chemical-transport model (GEOS-Chem) version 10.01. GEOS-Chem is driven by GEOS-5 assimilated meteorology fields (http://gmao.gsfc.nasa.gov), and includes PM tracers for black carbon, organic aerosol, dust, sea salt, sulfate, nitrate, and ammonium in addition to 52 gas-phase species. Globally, we use the EDGAR emissions inventory with regional overwrites as described in the supplemental material. Waste-combustion emissions are not included in these base GEOS-Chem emissions inventories. We incorporate to GEOS-Chem the waste-combustion inventory of Wiedinmyer *et al* (2014), which estimates the mass of waste burned in urban and rural areas for developing countries, and rural areas for developed countries. The inventory primarily uses emission factors compiled in Akagi *et al* (2011). Estimated waste-combustion emissions include 0.6 Tg yr^{-1} of black carbon and 5.1 Tg yr^{-1} of primary organic carbon, as well as more-minor contributions from gas-phase species including sulphur dioxide, ammonia, and nitrous oxides, which contribute to additional $PM_{2.5}$ (<10% of the total addition) through chemical reactions. Evaluation of the modeled aerosol burden with and without waste-combustion emissions is included in the supplemental material.

We perform GEOS-Chem simulations for year 2010 with a pair of simulations: one with emissions from all sources (including waste burning; 'WASTE_ON'), and another, otherwise-identical simulation but without waste-combustion emissions ('WASTE_OFF'). Comparison of these pairs of simulations allows us to isolate the impacts of waste combustion on $PM_{2.5}$ concentrations and mortality. In order to test the sensitivity of mortality rates to uncertainties in waste-combustion emission mass, we assume waste-combustion $PM_{2.5}$ scales linearly with PM emission mass and simply double ('HIGHMASS') and half ('LOWMASS') modeled waste-combustion $PM_{2.5}$ concentrations. The factor-of-2 uncertainty in emission mass is similar to uncertainties in waste-combustion emission factors reported in Akagi *et al* (2011). In order to test the sensitivity of mortality rates to model resolution we repeat the WASTE_ON and WASTE_OFF simulations at three resolutions: 2° × 2.5° (~200 km) and 4° × 5° (~400 km) resolution globally, and a 0.5° × 0.666° (~50 km) resolution over Asia.

2.2. Calculation of premature mortality

Our methodology to estimate mortalities from waste-combustion $PM_{2.5}$ is based on the methods used in the GBD 2010 to estimate ambient $PM_{2.5}$ mortalities. We discuss a number of uncertainties and limitations of this method in section 4. We use year 2010 gridded population (from the NASA Socioeconomic Data and Application Center (SEDAC, http://sedac.ciesin.columbia.edu/) and baseline mortality rates compiled for the GBD 2013 for IHD, CeVD, COPD, and LC for adults (ages greater than 25) (Naghavi *et al* 2015). Baseline mortalities are reported at the country level, and we aggregate them to the model resolution assuming no sub-national gradients. Data on baseline mortality variability at a sub-national scale were not available.

The fraction of all premature mortalities due only to $PM_{2.5}$ is based on concentration-response functions (CRFs) that relate exposure to ambient $PM_{2.5}$ to increased risk of premature mortality from specific diseases. We calculate the relative risk (RR) from all $PM_{2.5}$ sources (RR_{total}) using the integrated CRFs of Burnett *et al* (2014). We choose the Burnett *et al* (2014) study as it uses smoking and household air pollution to constrain the risk at high $PM_{2.5}$ concentrations (such as ambient $PM_{2.5}$ found in India and China). The Burnett *et al* (2014) study makes several assumptions relevant to this study: the toxicity of $PM_{2.5}$ is not dependent on composition, increased mortality risk is a result of long-term exposure, CRFs apply globally, there exists a minimum $PM_{2.5}$ threshold (modeled as a uniform distribution ranging from 5.8 to 8.8 $\mu g\, m^{-3}$) below which no further negative health impacts are assumed to occur, and the relationship between relative risk of mortality and $PM_{2.5}$ exposure is nonlinear. We note that CRFs are an active area of research, and our results are likely sensitive to our choice of CRF. Burnett *et al* (2014) fits coefficients using a Monte Carlo approach, reporting 1000 sets of coefficients for each cause (and each age group for CeVD and IHD). We use these sets of coefficients to determine median, 5th, and 95th percentiles of mortality. The attributable fraction of premature mortalities by $PM_{2.5}$ is then calculated from the RR by: $(RR_{total} - 1)/RR_{total}$.

We estimate total premature adult mortality from $PM_{2.5}$ as the product of population, cause and age specific baseline mortality rates, and attributable fraction from all $PM_{2.5}$ sources in each model gridcell. We estimate 2.7 million premature adult mortalities from ambient $PM_{2.5}$ in the 2° × 2.5°-resolution simulation (figure S1). This number is lower than estimates of 3.1 million from Lelieveld *et al* (2015) and Lim *et al* (2012); however, it is within the reported uncertainty ranges (2.5 to 3.7 million for Lim *et al* 2012 and 1.5 to 4.6 million for Lelieveld *et al* 2015). The coarser resolution of our global model likely contributes to the lower estimate.

To attribute the *number of mortalities owing to waste-combustion emissions*, we scale total mortalities from all $PM_{2.5}$ by the fraction of $PM_{2.5}$ from waste combustion (attribution method). This is represented by equation (1):

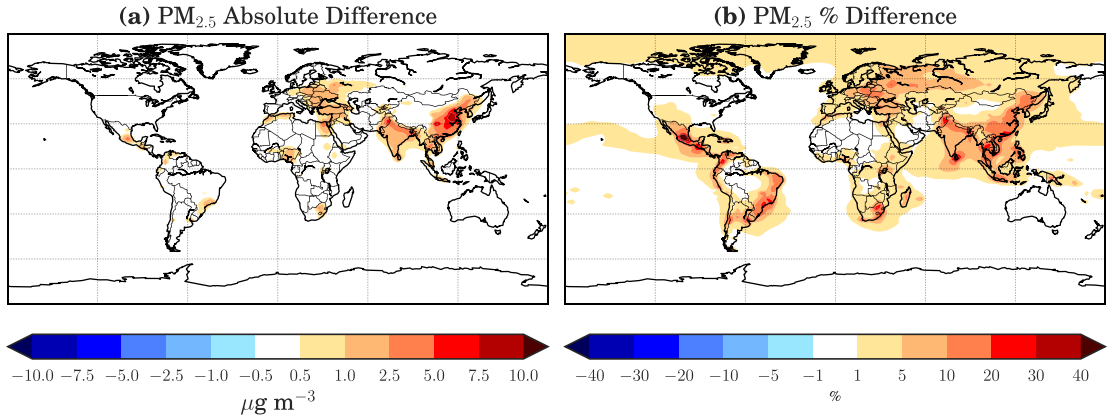

Figure 1. The (a) absolute difference and (b) percent difference in modeled boundary layer $PM_{2.5}$ from including emissions from combustion of domestic waste in our simulations at 2° × 2.5° resolution.

$$M_{\text{waste}} = \frac{PM_{2.5,\text{WASTE\_ON}} - PM_{2.5,\text{WASTE\_OFF}}}{PM_{2.5,\text{WASTE\_ON}}} \times M_{\text{all (WASTE\_ON)}}, \quad (1)$$

where M_{waste} is the premature mortalities due to waste combustion, $PM_{2.5,\text{WASTE\_ON}}$ and $PM_{2.5,\text{WASTE\_OFF}}$ are the modeled $PM_{2.5}$ concentrations in the WASTE_ON and WASTE_OFF simulations, respectively, and $M_{\text{all (WASTE\_ON)}}$ is the premature mortalities due to all sources in the WASTE_ON simulation. Additionally, we estimate the *number of mortalities averted due to removal of waste-combustion emissions* by subtracting the total mortalities in the WASTE_OFF simulation from the total mortalities in the WASTE_ON simulation (subtraction method). This is represented by equation (2):

$$M_{\text{waste}} = M_{\text{all (WASTE\_ON)}} - M_{\text{all (WASTE\_OFF)}}, \quad (2)$$

where $M_{\text{all (WASTE\_OFF)}}$ is the premature mortalities from all sources in the WASTE_OFF simulation. The subtraction method yields different results than the attribution method as the CRF has strong nonlinearities (discussed below).

3. Results

3.1. Model increases in $PM_{2.5}$ due to waste-combustion emissions

Figure 1 shows the absolute increase (panel a) and percent increase (panel b) in $PM_{2.5}$ from waste-combustion emissions at 2° × 2.5° model resolution. Waste-combustion emissions lead to over 10% increases in ambient $PM_{2.5}$ concentrations in South and South-East Asia, Eastern Europe, Central America and Coastal South America. Notably, $PM_{2.5}$ increases by more than 40% in Sri Lanka and central Mexico (e.g. Mexico City). The absolute increases in $PM_{2.5}$ are greatest in eastern China and northern India, but there are anomalously large fractional increases in grid cells corresponding to large urban areas such as Johannesburg, Cairo, Moscow, and Mexico City.

3.2. Global and country-level mortality rates due to waste-combustion $PM_{2.5}$

The mortality rates per area associated with waste-combustion $PM_{2.5}$ calculated through the attribution method are shown in figure 2(a), and global annual mortalities listed by cause of death are in table 1. We attribute waste-combustion $PM_{2.5}$ to 270 000 (5th–95th:213 000–328 000) adult mortalities per year (calculated as the sum of IHD, CeVD, COPD, and LC). The majority of these mortalities are caused by IHD (120 000, 5th–95th:106 000–136 000) and CeVD (108 000, 5th–95th:86 000–130 000). Spatially, the highest concentration of deaths occurs in eastern China and northern India where mortality exceeds 900 deaths per 10^4 km^2 (a 100 × 100 km box) per year. There is also a substantial number of mortalities in Eastern Europe. Significant increases in $PM_{2.5}$ co-located with dense populations lead to greater than 300 deaths per 10^4 km^2 in densely populated cities. Figure 2(c) shows the waste combustion mortality rate per 10^6 people (in each grid cell). A combination of large relative $PM_{2.5}$ increases and high baseline mortality rates in Eastern Europe and Russia lead to a similar mortality rate as in Asia. Figure 2(c) also shows more widespread health impacts in Africa and the Middle East, where lower population densities limit the total number of annual mortalities.

At the country level, the estimated premature mortality rates due to waste-burning $PM_{2.5}$ (normalized either by population or area) varies by several orders of magnitude between regions and economic level (see supplemental table 1). This mortality range is due to (1) differing amounts of waste generation (and hence emissions) and pollution transport leading to differing $PM_{2.5}$ concentrations, (2) large variations in total number and density of the exposed population, and (3) differences in baseline mortality rates. In addition, non-waste-combustion $PM_{2.5}$ plays an important role. In both WASTE_ON and WASTE_OFF, some countries are uniformly below the minimum concentration threshold set as the counterfactual in

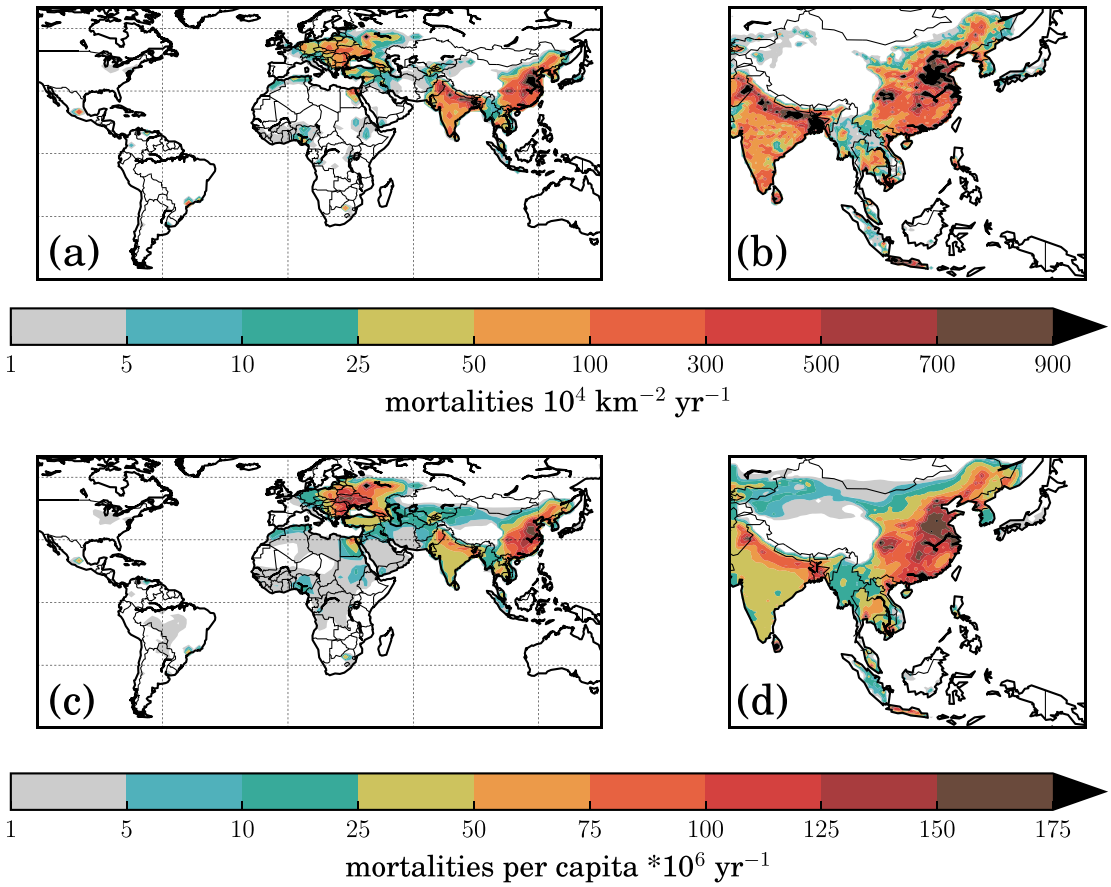

Figure 2. Annual mortalities from chronic exposure to ambient $PM_{2.5}$ from waste combustion per 10^4 km^2 in the (a) 2° × 2.5° global domain and (b) in the 0.5° × 0.666° Asian domain as well as per 10^6 people in the (c) 2° × 2.5° global domain and (d) in the 0.5° × 0.666° Asian domain.

Table 1. Annual mortalities (thousands) by cause and model resolution for the global and Asian domains due to exposure to $PM_{2.5}$ from combustion of domestic waste.

	Resolution	IHD	CeVD	COPD	LC	Sum
Global Domain	4° × 5°	103[a] (91–116)	89 (71–107)	23 (12–34)	12 (6–34)	227 (180–275)
	2° × 2.5°	120 (106–136)	108 (86–130)	27 (14–40)	15 (7–21)	270 (213–328)
	2° × 2.5°-HIGHMASS	229 (203–258)	208 (165–249)	52 (28–77)	30 (14–41)	518 (180–275)
	2° × 2.5°-LOWMASS	62 (55–70)	55 (44–66)	13 (7–20)	7 (3–11)	138 (109–167)
	2° × 2.5°-SUBTRACT	70 (63–80)	84 (67–98)	23 (14–30)	13 (8–17)	191 (151–224)
Asia Domain	4° × 5°	76 (68–84)	76 (61–91)	21 (11–31)	11 (6–15)	184 (146–222)
	2° × 2.5°	90 (81–101)	93 (74–110)	24 (13–36)	13 (6–19)	221 (174–266)
	0.5° × 0.666°	113 (102–126)	120 (96–143)	29 (15–44)	16 (8–23)	279 (221–336)

Abbreviations: IHD, ischaemic heart disease; CeVD, cerebrovascular disease; COPD, chronic obstructive pulmonary disease; LC, lung cancer.

[a] The first number is the median estimate, and the 5th and 95th percentile are in parentheses.

Burnett *et al* (2014) for $PM_{2.5}$ health effects (though, we note that the actual minimum concentration threshold has not been clearly identified in the literature). By aggregating mortalities to the country level, we find the countries with the largest total mortalities due to waste combustion are China, India, Pakistan, and Russia. These four countries amount to 78% of the global mortality. The countries with the highest mortalities per capita are Montenegro, Bulgaria, Moldova, and Ukraine. Normalizing by mass of waste generated, we find that the countries with the highest mortalities per mass of waste generated are Nepal, Montenegro, Uruguay, and Bulgaria (supplemental table 1). Supplemental figure S4 shows box-and-whisker plots of country-level generated waste per capita per year (a) (from Wiedinmyer *et al*, 2014), mortality rates per capita (b), and mortality per mass of waste generated (c) split between 4 economic strata: high income, upper-middle income, lower-middle income, and low income. While on average high-income

countries generate two times more waste, only 10 people die for every Tg of waste generated compared to 82 deaths for every Tg of waste generated in low- to upper-middle-income countries. This difference is driven by a higher fraction of waste generated being burned in low- to upper-middle income countries compared to high-income countries.

3.3. Deaths averted due to removal of waste-combustion emissions

We estimate 191 000 (5th–95th:51 000–224 000) premature mortalities per year may be saved by eliminating waste combustion (subtraction method; see explanation in section 2.2) (table 1 and figure S6). Thus, removing waste combustion would reduce premature mortality rates by a smaller number than the mortality rate attributed to waste combustion. This is due to the nonlinear CRFs where the mortality response to $PM_{2.5}$ saturates with increasing $PM_{2.5}$ concentrations. This saturation effect is strongest in the heavily polluted regions of India and China (which lie on the sub-linear portion of the CRF). This saturation effect is partly balanced out by a larger number of mortalities avoided in cleaner regions where waste-combustion emissions elevates $PM_{2.5}$ concentrations from below to above minimum-$PM_{2.5}$-threshold values.

3.4. Sensitivity of mortality rates to emission mass and model resolution

Wiedinmyer *et al* (2014) acknowledges large uncertainties in $PM_{2.5}$ emission mass. To test the sensitivity of mortality rates to uncertainties in emission mass, we introduce a factor-of-2 uncertainty in waste-combustion $PM_{2.5}$ mass. Halving waste-combustion $PM_{2.5}$ (LOWMASS) results in 138 000 (5th–95th:109 000–167 000) mortalities per year and doubling $PM_{2.5}$ (HIGHMASS) results in 518 000 (5th–95th:410 000–626 000) through the attribution method (table 1 and figure S5). The relationship between waste-combustion $PM_{2.5}$ and mortality is sub-linear (additional $PM_{2.5}$ impacts health less when $PM_{2.5}$ concentrations are already high) because many of the waste-combustion source regions occur in already heavily polluted areas; thus, the waste-combustion emissions in these regions lead to fewer mortalities than if $PM_{2.5}$ concentrations were lower.

In order to explore the dependence on estimated attributed mortality rates to model resolution, we use a coarser $4^\circ \times 5^\circ$ and finer $2^\circ \times 2.5^\circ$ global simulation and a $0.5^\circ \times 0.666^\circ$ simulation over Asia. The total number of annual mortalities in the global $4^\circ \times 5^\circ$ resolution simulation is 16% lower than the $2^\circ \times 2.5^\circ$ resolution simulation (table 1 and figure S7). The decrease in mortality rates with coarser resolution is caused by two main factors. First, as grid-box area increases, ambient $PM_{2.5}$ is averaged over a larger area and may reduce concentrations below the minimum threshold for mortality (between 5.8 and 8.8 $\mu g\,m^{-3}$) in some locations. Second, higher-resolution simulations are better able to co-locate dense urban populations with $PM_{2.5}$ increases from waste combustion. Figure 2(b) shows the annual mortality per $10^4\,km^2$ and figure 2(d) shows annual mortality per 10^6 people over Asia at $0.5^\circ \times 0.666^\circ$ resolution. In this domain, total mortalities increase from 184 000 (5th–95th:146 000–222 000) in the $4^\circ \times 5^\circ$, to 221 000 (5th–95th:174 000–266 000) in the $2^\circ \times 2.5^\circ$, to 279 000 (5th–95th:221 000–336 000) in the $0.5^\circ \times 0.666^\circ$ resolution simulation. The higher-resolution simulation predicts more mortalities just in the Asia domain than the $2^\circ \times 2.5^\circ$ simulation predicts globally, which highlights the importance of model resolution when using simulated $PM_{2.5}$ concentrations to estimate mortality rates.

Figure 3 shows the fractions of the population impacted by waste-combustion $PM_{2.5}$ and mortality risk. Figure 3(a) shows the complementary cumulative distribution functions (CCDF) of the percent of the global population exposed to different levels of $PM_{2.5}$ from domestic-waste combustion. In the global $2^\circ \times 2.5^\circ$ domain, 50% of the total population is exposed to a greater than 1.3 $\mu g\,m^{-3}$ increase in $PM_{2.5}$ from waste combustion, while 10% is exposed to a greater than 5.3 $\mu g\,m^{-3}$ increase. Increases in exposure due to waste-combustion $PM_{2.5}$ are generally smaller in the coarser $4^\circ \times 5^\circ$ domain due to dilution of emissions into larger grid cells. Figure 3(b) shows the corresponding CCDFs for the relative increase in $PM_{2.5}$-mortality risk in the WASTE_ON compared to the WASTE_OFF simulation. The all-cause relative risk is the mean value of the relative risk of the four causes weighted by the proportion of each cause to the total baseline mortality. For the two global resolutions, more than 50% of the population has greater than a 0.5% increased risk of mortality by any cause due to waste-combustion emissions, while 10% of the population has a greater than 1.5% increased risk of mortality. While in figure 3(a) 91% of the population is exposed to at least a 0.1 $\mu g\,m^{-3}$ increase in $PM_{2.5}$, only 75% of the population has a greater than 0.02% increased risk of mortality in figure 3(b) (due to minimum $PM_{2.5}$ thresholds in the CRF). Figures 3(c) and (d) show increased $PM_{2.5}$ and increased risk of mortality in the Asian domain for the three model resolutions. The majority of global mortalities occur in Asia (table 1), where a larger fraction of the population is exposed to waste-combustion $PM_{2.5}$ and thus increased risk of mortality. All three resolutions show that nearly 99% of the population in Asia is exposed to at least a 0.1 $\mu g\,m^{-3}$ increase in $PM_{2.5}$ and nearly 83% of the population have an greater than 0.02% increased risk of mortality from waste combustion. The $0.5^\circ \times 0.666^\circ$ resolution simulation predicts that a higher fraction of the population is exposed to larger increases in $PM_{2.5}$ and mortality risk than the coarser resolutions due to the reasons discussed above.

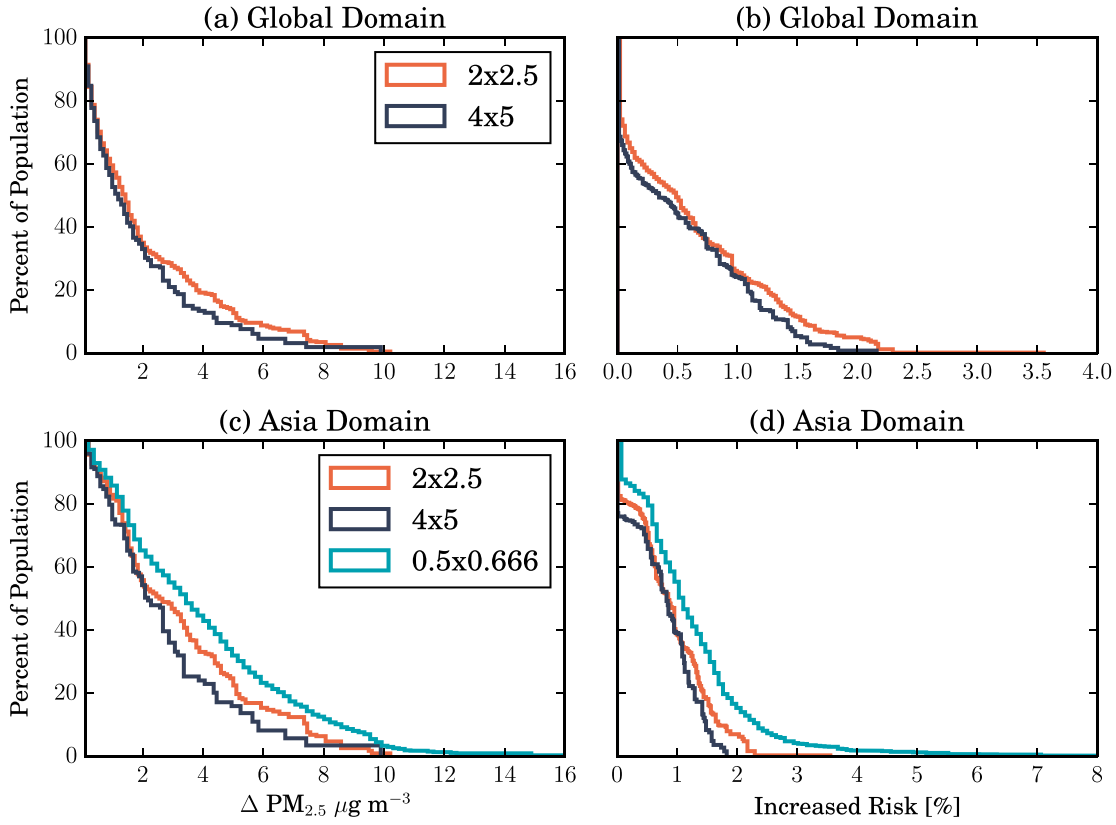

Figure 3. Complementary cumulative distribution functions weighted by percent of the population for (a) the change in $PM_{2.5}$ due to combustion of domestic waste for the global domain, (b) the increased risk for mortality due to combustion of domestic waste for the global domain, (c) the change in $PM_{2.5}$ for the Asia domain, and (d) the increased risk of mortality for the Asia domain.

4. Discussion and conclusions

As stated in the results, we estimate 270 000 (5th–95th:213 000–328 000) annual adult mortalities from waste combustion. Our estimate of mortality attributed to waste combustion is similar to recent estimates of mortalities due to $PM_{2.5}$ from solid-fuel cookstoves (300 000–400 000 mortalities per year), another emission source associated with the developing world (Chafe *et al* 2014, Butt *et al* 2016). Waste-combustion emissions are approximately 10% of our total ambient $PM_{2.5}$ mortalities and 9% of the total ambient $PM_{2.5}$ mortalities estimated in the GBD 2010. We note that our estimate of global mortalities from waste-combustion emissions are of a similar magnitude to the Lelieveld *et al* (2015) estimate of mortalities from $PM_{2.5}$ from biomass burning (~165 000), land traffic (~165 000), and industrial emissions (~231 000); however, we note that our total mortality estimate is 10% lower than Lelieveld *et al* (2015).

Waste combustion has high mortality rates due to proximity of emissions to population. In developing countries, substantial amounts of waste combustion can occur in urban areas. We find high mortality densities (>300 deaths per 10^4 km^2 yr^{-1}) in cities such as Mexico City, Moscow, Johannesburg, and Rio de Janeiro. Four countries (China, India, Pakistan, and Russia) account for slightly more than 2/3 of the global mortality burden due to waste combustion. Globally, more than half of the world's population is exposed to an additional 1.3 μg m^{-3} of $PM_{2.5}$ due to waste combustion leading to a 0.5% increased risk of mortality each year just from waste-combustion emissions.

Due to a factor-of-2 uncertainty in $PM_{2.5}$ emission mass, we estimate a range of mortalities per year of 138 000 to 518 000. We note that even on the low end of this range, waste combustion contributes to a substantial number (greater than 100 000) of mortalities per year. Additionally, we estimate that a complete removal of waste-combustion would avoid 191 000 mortalities per year (see explanation in section 2.2). In these sensitivity simulations, the calculated decrease of premature mortality is less than the proportional decrease in $PM_{2.5}$. The sub-linear nature of these sensitivity tests demonstrates that most of the mortalities from waste combustion occur in heavily polluted areas.

Our estimates of premature mortality from waste-combustion $PM_{2.5}$ are likely a lower bound due to the coarse resolution of the global model. Combustion of domestic waste often occurs near where people live and work, sometimes in streets or in front of homes, and none of our model simulations resolve this near-field pollution exposure. The 2° × 2.5° resolution is unable to capture high $PM_{2.5}$ gradients, which are

often co-located with dense populations. We include an additional simulation at $4^\circ \times 5^\circ$ and calculate 227 000 mortalities (16% lower than the estimate at $2^\circ \times 2.5^\circ$). This suggests increasing mortalities at progressively finer model resolutions. Due to the high mortality rates in Asia, we include a higher resolution $0.5^\circ \times 0.666^\circ$ simulation in this domain. We estimate 279 000 mortalities in Asia at this model resolution. The sensitivity of health-response estimates to model resolution has been explored in past studies (e.g. Thompson and Selin 2012, Punger and West 2013).

The methods and data sources used here are similar to the GBD 2010 and several recent health-impact studies (e.g. Lim *et al* 2012, Apte *et al* 2015, Lelieveld *et al* 2015); however, there are substantial uncertainties and limitations inherent in these methods. First, the Burnett *et al* (2014) CRF is largely based on epidemiologic studies that occur in the United States, and while there are age modification factors for CeVD and IHD, there are no modifications for other socio-demographc factors. We assume here that this function applies globally; however, as demographics and pollution sources vary regionally, this may not be a valid assumption. Second, we consider all species of $PM_{2.5}$ to be equally toxic; however, some studies have suggested that combustion particles may be more toxic (Krzyzanowski *et al* 2005). Third, due to lack of data, baseline mortality rates used here are reported at the national or regional level; however, demographics often vary sub-nationally, and national and regional level mortality rates may introduce uncertainty. Chowdhury and Dey (2016) estimate 15% fewer premature mortalities from ambient $PM_{2.5}$ exposure in India when assuming a uniform country-level baseline mortality rate as opposed to varying baseline mortality at the state level using gross domestic product as a proxy. The uncertainties listed here are in addition to uncertainties in emissions (Akagi *et al* 2011, Wiedinmyer *et al* 2014) and model processes.

In this study, we focus on mortality from chronic exposure to ambient $PM_{2.5}$, but we note that waste combustion also emits a number of gas- and particulate-phase toxins that may have additional significant health implications. While generally accepted that these toxins have negative health impacts, the CRFs are less well-developed making it difficult to estimate a global health impact. Thus, because $PM_{2.5}$ response functions exist, it is common to use $PM_{2.5}$ estimates as a proxy for exposure to a broad array of toxic species. Therefore, the net health impacts from waste combustion could be greater than estimates for only $PM_{2.5}$.

Uncontrolled domestic-waste-combustion emissions are a potentially significant emission source that has been largely overlooked in past studies. Waste combustion creates health-effect disparities between high-income countries and lower-income countries, where high-income countries generate more waste but combust less, resulting in a factor of 8 fewer deaths per mass waste of generated than lower-income countries (figure S4). Due to the coarse model resolution and exclusion of gaseous toxic species, our estimate of mortalities from waste-combustion emissions is likely a lower bound. Despite this, we find a substantial burden of disease (9% of mortalities from all $PM_{2.5}$) that are on the same order of magnitude as modeling estimates of industry, land traffic, and biomass burning. Waste combustion also affects aerosol number concentrations and radiative impacts, and we explore this in a separate study (Kodros *et al* 2016).

Acknowledgments

This research has been supported by a grant from the US Environmental Protection Agency's Science to Achieve Results (STAR) program through grant no. 83543801. Although the research described in the article has been funded by the US Environmental Protection Agency's STAR program, it has not been subjected to any EPA review and therefore does not necessarily reflect the views of the Agency, and no official endorsement should be inferred.

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