Environmental Research Letters

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Environmental Research Letters

LETTER

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Keywords: emissions, air quality, India, health, rural, urban

Supplementary material for this article is available online

OPEN ACCESS

RECEIVED
8 March 2018

REVISED
30 April 2018

ACCEPTED FOR PUBLICATION
3 May 2018

PUBLISHED
25 May 2018

Abstract

Ambient air pollution in India contributes to negative health impacts and early death. Ground-based monitors often used to quantify health impacts are located in urban regions, yet approximately 70% of India's population lives in rural communities. We simulate high-resolution concentrations of fine particulate matter (PM) and ozone from the regional Community Multi-scale Air Quality model over northern India, including updated estimates of anthropogenic emissions for transportation, residential combustion and location-based industrial and electrical generating emissions in a new anthropogenic emissions inventory. These simulations inform seasonal air quality and health impacts due to anthropogenic emissions, contrasting urban versus rural regions. For our northern India domain, we estimate 463 200 (95% confidence interval: 444 600–482 600) adults die prematurely each year from $PM_{2.5}$ and that 37 800 (28 500–48 100) adults die prematurely each year from O_3. This translates to 5.8 deaths per 10 000 attributable to air pollution out of an annual rate of 72 deaths per 10 000 (8.1% of deaths) using 2010 estimates. We estimate that the majority of premature deaths resulting from $PM_{2.5}$ and O_3 are in rural (383 600) as opposed to urban (117 200) regions, where we define urban as cities and towns with populations of at least 100 000 people. These findings indicate the need for rural monitoring and appropriate health studies to understand and mitigate the effects of ambient air pollution on this population in addition to supporting model evaluation.

1. Introduction

Worldwide, more than 7 million adult premature deaths each year are attributable to ambient air pollution (Lim *et al* 2012, Forouzanfar *et al* 2015, 2016). An additional estimated 600 000 children are annually subject to premature mortality resulting from long-term ambient air pollution exposure (UNICEF 2016). Human health impacts from air pollution disproportionately affect populations in countries undergoing rapid industrialization, population growth, urbanization, and motorization. In India, annual premature mortality estimates due to ambient air pollution are estimated between 580 000 (Ghude *et al* 2016) to more than one million people (Health Effects Institute 2017). Premature deaths due to air pollution can bring significant monetary losses to the Indian economy (Ghude *et al* 2016). However, many health analyses fail to reflect differences across urban versus rural populations, important considering India's population is largely rural.

The Global Burden of Disease (GBD) studies seek to quantify health effects (mortality and morbidity) due to risk factors, including environmental risks such

as ambient outdoor air pollution (Brauer *et al* 2012, Lim *et al* 2012, Lelieveld *et al* 2015, Forouzanfar *et al* 2016). Estimates to ambient air pollution risk-attributable mortality and morbidity are calculated using baseline mortalities for specific diseases and dose-response relationships derived from cohort studies. However, evaluating ambient air pollution impacts requires accurate quantitative estimates of concentrations at the surface, where people are subject to harmful exposure effects.

Pollutant exposures are estimated using a combination of atmospheric chemistry models, satellite observations, and *in situ* measurements. Traditionally, surface measurements reflect the best recording of ambient concentrations to determine exposure-response relationships. Yet, in India, surface monitoring is largely limited to urban regions as opposed to rural villages, and available observations supported by the Central Pollution Control Board (CPCB) in India do not have an extensive history (Central Pollution Control Board 2008, ENVIS Centre on Control of Pollution 2016). We can fill in some of these gaps using long-term satellite tropospheric column averages, which show spatial variations in increased pollution over time e.g. Duncan *et al* (2015) that may have slowed in recent years (Hilboll *et al* 2017). Previous health impact assessments in India have used satellite observations to infer surface concentrations and health impacts of $PM_{2.5}$ at the district level (Chowdhury and Dey 2016). Advanced chemical transport models are increasingly used to focus on pollution across India regionally, from evaluating and developing NO_x emissions inventories (Ghude *et al* 2013, Jena *et al* 2015) to testing modeled O_3 sensitivities in the region (Surendran *et al* 2015, Sharma *et al* 2016), in addition to estimating premature mortality due to air pollution (Ghude *et al* 2016). However, some models may be too coarse to resolve large topographic gradients that affect modeled airflow and thus pollutant distributions, something particularly important in northern India due to the Himalayas. Yet the current capacity of atmospheric chemistry models to produce surface concentrations remains largely limited by our understanding of emissions inventories.

Emissions remain challenging to quantify as a result of ongoing industrialization, motorization, and urbanization in India. Many different emissions inventories for use in atmospheric chemistry models have been used for understanding air quality impacts in India (Zhang *et al* 2009, Amann *et al* 2011, EC-JRC/PBL 2011, Kurokawa *et al* 2013, Guttikunda and Jawahar 2014, Pandey *et al* 2014, Sadavarte and Venkataraman 2014), however emissions estimates remain highly uncertain in magnitude—NO_x and SO_2 range between 5–6.75 Tg and 7–9 Tg per year respectively—and location (Saikawa *et al* 2017). In addition estimating rural emissions by extrapolation from urban data can be problematic. Karambelas *et al* (2018) found large low biases across modeled concentrations evaluated with satellite and surface observations, demonstrating the importance of having representative inventories and high-resolution chemical transport models. Emissions inventories are invaluable to air quality modeling and for estimating human health impacts due to pollution exposure, playing a vital role in advancing air quality and health understanding in India.

In this study, we present a high-resolution anthropogenic emissions inventory to identify pollution impacts on urban and rural populations. We update anthropogenic emissions from the Greenhouse Gas-Air Pollution Interactions and Synergies (GAINS) model (0.5° by 0.5°) using urban and rural population densities and emissions activity information for predominant anthropogenic sectors—domestic combustion and transportation. Applying this new updated emissions inventory at a high horizontal resolution (12 km by 12 km), the US Environmental Protection Agency (EPA) Community Multi-scale Air Quality (CMAQ) model, yields premature mortality estimates that are higher in rural regions compared to urban areas with populations greater than 100 000 people. Results demonstrate the sensitivity of health impact calculations to emissions inventories, and the value of expanding surface monitoring in rural India to improve air quality and accuracy of health impact assessments of the region.

2. Methods

2.1. Modeling

Air quality modeling was performed using the regional CMAQ model version 5.1. Previously, CMAQ has been used in India to assess O_3 regimes (Sharma *et al* 2016, Sharma and Khare 2017) and evaluate urban and rural emissions uncertainties (Karambelas *et al* 2018). We completed simulations for four seasonally representative months using 2010 meteorology and emissions: January, April, July, and October for winter, pre-monsoon spring, monsoon, and post-monsoon fall respectively. Model processes include surface- and upper-level emissions, photolysis, gas- and particle-phase chemistry, deposition, and dispersion Byun and Schere (2006). Simulations include the Carbon Bond 05 (CB05) chemical mechanism (Yarwood *et al* 2005) and AERO 6 aerosol mechanism, the inclusion of windblown dust (Dong *et al* 2015) with enhancements following Karambelas *et al* (2018), and in-line lightning NO_x production (Allen *et al* 2012). Boundary and initial conditions are from a larger, 36 km by 36 km domain (Karambelas *et al* 2018). Meteorology is simulated using the Weather Research and Forecasting (WRF) model v3.2 and Preprocessing System with European Center for Medium-Range Weather Forecasting ERA-Interim globally gridded reanalysis data (Dee *et al* 2011). WRF is used to interpolate weather data from an 80 km resolution over 60 vertical layers at 6 hour increments

to our model domain over 36 vertical sigma layers from the surface to approximately 150 hPa using the Grell cumulus parameterization Grell and Devenyi (2002). For use in CMAQ, WRF output was ultimately preprocessed with the Meteorology-Chemistry Interface Processor (MCIP). Planetary boundary height, temperature, and precipitation from MCIP and evaluated with TRMM show MCIP captures seasonality in meteorology for the region, though underestimates precipitation (supplemental figure 1 available at stacks.iop.org/ERL/13/064010/mmedia).

2.2. Emissions

We use biogenic, biomass burning, and anthropogenic emissions for year 2010 gridded to 12 by 12 km and vertically distributed (supplemental table 1). Monthly biogenic emissions are from the Community Land Model with the Model of Emissions and Gases from Nature (Guenther *et al* 2006) (downloaded from http://lar.wsu.edu/megan/guides.html). Monthly biomass burning emissions are from the Global Fire Emissions Database including small fires (GFED v4.1s) (van Der Werf *et al* 2010) and were vertically distributed following methods used in Karambelas *et al* (2018). Annual total anthropogenic emissions for 2010 are from the GAINS Model gridded globally following the ECLIPSE (Evaluating the Climate and Air Quality Impacts of Short-Lived Pollutants) project version 5a (Stohl *et al* 2015, Klimont *et al* 2017). In addition to regridding anthropogenic emissions from the global 0.5 degree by 0.5 degree to 12 km by 12 km over northern India, we distribute anthropogenic emissions vertically according to Simpson *et al* (2012). Updates according to urban and rural activities and population distributions where cities and towns greater than 100 000 people are designated as urban were included for the domestic combustion and transportation sectors to provide enhanced detail at the higher domain resolution. Emissions from electricity generation were also updated based on power plant location information. More details on population data and energy sector emissions updates can be found in the supplemental information. Our urban designation is different than used for population distribution in India, that states an urban population must have (1) at least a population of 5000; (2) 75% of the male working population is employed in non-agricultural work; and (3) the density is at least 400 people per square kilometer. Our urban-rural designations result in a distribution of 79% rural and 21% urban, and is approximately 2% more rural (less urban) than the 2001 population distribution off of which our data is based.

Solid fuel based cooking is one of the main sources of primary PM emissions in India, particularly among rural communities where biomass is freely available and access to clean fuels like liquid petroleum gas (LPG) or cookstoves that use them may be limited in availability and cost. Recent studies found residential combustion emissions are attributable for 20%–25% of premature deaths associated with ambient $PM_{2.5}$ air pollution (Conibear *et al* 2018, GBD MAPS Working Group 2018). We reallocate emissions from the domestic cooking sector using state totals and gridded population data following greater LPG consumption in urban regions and more traditional biomass burned in rural regions. Information on distinctions between urban and rural domestic cooking fuel use were from Pachauri *et al* (2013), and updated emissions were gridded according to urban and rural population activity fractions across each state. We did not include other socio-demographic factors such as proximity to forest edge (Winijkul *et al* 2016a, 2016b), yet we recognize this may be a relevant factor in emissions allocation.

Transportation emissions were adjusted according to vehicle type: mopeds, motor-cycles, light duty cars, light duty trucks, buses, and heavy duty trucks. Based on best available information from Indian transportation research on vehicle population composition in the Mumbai metropolitan region (Shirgaonkar 2012) and personal communications (with Dr. Jens Borken, AIR, IIASA in July 2015), emissions for urban and rural regions per vehicle type are distributed according to ratios in supplemental table 2. Equal distribution is given to urban and rural mopeds due to their accessibility for both low-income and middle-class households. Urban populations operate more motorcycles, light duty cars and trucks, while buses and heavy-duty trucks are operated in greater frequency in rural areas. Assumptions regarding vehicle fleet age are not included in the redistribution. Urban and rural emissions were gridded based on respective population distributions.

The final emissions inventory included the updated sectors merged with non-updated sectors including agriculture, non-road, industry, and other sectors.

2.3. Estimating premature mortality

We use parameters from integrated exposure response functions from the GBD 2013 study (Cohen *et al* 2016, Forouzanfar *et al* 2015, World Health Organization 2016) to calculate the relative risk associated with diseases attributable to an excess concentration of ambient $PM_{2.5}$. The attributable fraction of cause-specific mortality related to excess ambient air pollution is multiplied by our population dataset to estimate the number of premature deaths per grid cell.

We estimate the effects of long-term exposure to $PM_{2.5}$ on deaths due to chronic obstructive pulmonary disease (COPD), ischemic heart disease (IHD), lung cancer, and stroke. Baseline mortality rate estimates for these outcomes in India are from the World Health Organization (WHO) (World Health Organization 2017) and have been previously used for ambient air pollution health assessments in India (Chowdhury and Dey 2016, Ghude *et al* 2016). Our work follows methods similar to Ghude *et al* (2016), Burnett *et al* (2014) and Apte *et al* (2015). We estimate annual premature

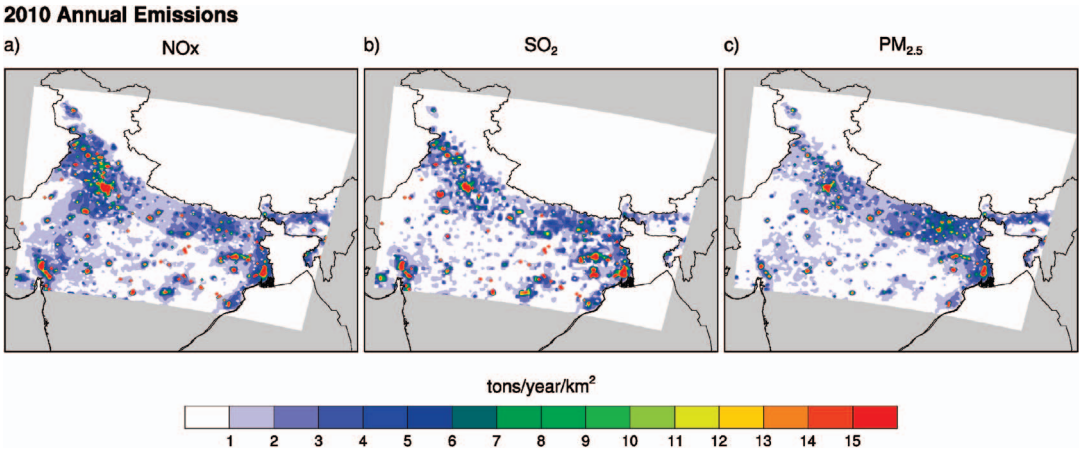

Figure 1. Anthropogenic emissions for India (ECLIPSE v5a; www.iiasa.ac.at/web/home/research/researchPrograms/air/Global_emissions.html) with updated population and activity information for (*a*) annual total NO_x, (*b*) SO_2, and (*c*) primary $PM_{2.5}$. Emissions are shown for India only, yet emissions exist elsewhere in the domain including in Pakistan, Bangladesh, and Nepal. All non-model domain regions are colored in gray.

mortality (ΔM_j) attributable to estimated annual average $PM_{2.5}$ concentrations as a result of disease *j*, where instances of IHD and stroke are age-dependent, following:

$$\Delta M_j = Y_j \times \frac{RR_{j-1}}{RR_j} \times P \quad (1)$$

where Y_j is the baseline mortality rate for a particular disease and P is total population. We apply the same baseline mortality rates across India, but we recognize that there are limitations to this method. We use relative risk tables parameters from GBD (2013) in calculating relative risk for disease *j* using an integrated exposure response (IER) function following Burnett *et al* (2014):

$$RR_j = 1 + a_j \times [1 - \exp(-\gamma_j(\Delta PM_{2.5})^{\delta_j})] \quad (2)$$

where α, γ, and δ are disease-dependent parameters (Burnett *et al* 2014, Apte *et al* 2015). $\Delta PM_{2.5}$ is the excess $PM_{2.5}$ beyond an annual average counterfactual minimum risk concentration commonly placed between 5.8–8.0 $\mu g\,m^{-3}$ (Chowdhury and Dey 2016, Ghude *et al* 2016). Other recent estimates have used nonlinear power law functions derived from cohort studies to estimate relative risk attributable to $PM_{2.5}$ in India which result in lower premature mortality estimates compared to using IER methodology (Chowdhury and Dey 2016) and references therein, and we include analysis using this method in the supplemental information. We calculate the 95% confidence interval of disease-specific relative risk at each grid cell to define a range of mortality estimates attributable to $PM_{2.5}$ exposure.

We estimate premature deaths from COPD due to long-term exposure of estimated annual average concentrations of maximum daily 8 hour average (MDA8) O_3 following Ghude *et al* (2016) and Ostro (2004). We use the relative risk calculation:

$$RR = \left[\frac{X+1}{X_0+1}\right]^{\delta} \quad (3)$$

where the minimum-risk concentration, X_0, is 37.6 ppb (range: 33.3–41.9 ppb) and the factor δ is 0.1521, both derived from the GBD assessment (Lim *et al* 2012). This minimum-risk concentration range was used to estimate minimum and maximum mortality impacts. To estimate ΔM attributable to annual average MDA8 O_3, we substitute RR from equation (3) into equation (1). Premature mortality for both $PM_{2.5}$ and O_3 is estimated for adults 25 years and older, meaning our estimates are likely conservative considering they do not take into account infant, child, and young adult mortality. Finally, we apply grid-cell specific population distributions to assess differences across urban and rural regions, with urban defined as towns and cities with greater than 100 000 people in total respectively.

3. Results

3.1. Updated annual emissions for northern India

Domain-wide 2010 annual emissions of anthropogenic NO_x, SO_x, and primary $PM_{2.5}$ (elemental carbon [EC], organic carbon [OC]) indicate spatial variability across the Indo-Gangetic Plain. Emissions vary across the region, with many instances of localized high values indicative of regions of relatively higher population density. Emissions of NO_x (figure 1(*a*)) and SO_2 (figure 1(*b*)) exhibit similar distributions, with annual totals in the north India region (model domain) of 2.3 Tg N (2.6 Tg N) and 6.3 Tg (6.9 Tg) respectively, where there are many concentrated regions of emissions and fewer emissions from area sources, however this is much more pronounced among the SO_2 emissions due to power plants being a main source. Emissions of NO_x are slightly greater than reported in a recent

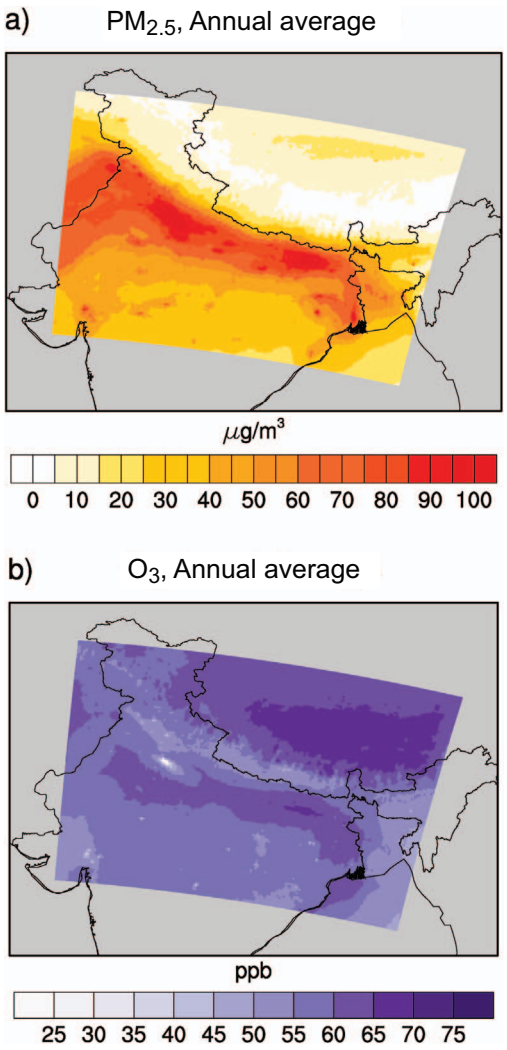

Figure 2. Four-month estimated annual average concentrations of (*a*) $PM_{2.5}$ and (*b*) maximum daily 8 hour O_3 using January, April, July, and October monthly average concentrations for use in relative risk calculations. $PM_{2.5}$ concentrations are greatest along the Indo-Gangetic Plain where population density is greatest. O_3 concentrations reflect regions of slightly lower O_3 due to inhibited O_3 formation as well as NO_x titration along the Indo-Gangetic Plain with seasonal variations.

total India 2005 inventory of 1.9 TgN derived using satellite tropospheric columns (Ghude *et al* 2013), but emissions of both NO_x and SO_2 are less than the range (5–6.5 TgN and 7–9.5 Tg SO_2 respectively for 2010) of a recent comparative analysis (Saikawa *et al* 2017), as expected considering our simulations cover the northernmost half of India.

Particulate emissions are largely dominated by the domestic combustion sector, emissions from which are strongly dependent on population density and availability of combustible materials. Total $PM_{2.5}$ emissions for the north India domain (2.7 Tg) are about one-third of those used in recent studies covering all of India (9–9.1 Tg) (Venkataraman *et al* 2017, GBD MAPS Working Group 2018). Emissions indicate a high number of localized peaks in $PM_{2.5}$ emissions, and, unlike NO_x and SO_2 emissions, broad regions of lower levels of emissions (figure 1(*c*)). Variations in emissions magnitude indicate relatively densely populated villages among low-populated rural areas and the effect these different population distributions have on total emissions. Adjusting emissions according to urban-rural population and activity distribution information resolves detailed urban and rural differences for more precise air quality modeling.

3.2. Estimated annual concentrations of surface $PM_{2.5}$ and O_3

Estimated annual average concentrations of $PM_{2.5}$ and MDA8 O_3 using four seasonally representative months are shown in figure 2; monthly average concentrations are shown in supplemental figure 2. Modeled average concentrations for $PM_{2.5}$ and MDA8 O_3 are 25.5 $\mu g\,m^{-3}$ and 41.7 ppb in northern India respectively (figures 2(*a*) and (*b*) respectively). Concentrations of $PM_{2.5}$ in the New Delhi National Capital Region are much higher (167.4 $\mu g\,m^{-3}$). Concentrations of both pollutants are relatively higher along the Indo-Gangetic Plain (IGP), corresponding with population density. South of the IGP, annual mean concentrations of $PM_{2.5}$ are comparatively low, between 30 and 40 $\mu g\,m^{-3}$ with some localized instances of concentrations greater than 50 $\mu g\,m^{-3}$ including near Ahmedabad to the west and Kolkata to the east. Localized instances of higher concentrations are predominantly from primary $PM_{2.5}$ species such as elemental and organic carbon (supplemental figure 3). Ozone concentrations exhibit local minima in highly populated urban areas, for instance in Ahmedabad and most noticeably Delhi, where concentrations average 34.3 ppb as a result of NO_x titration due to modeled high NO_2 concentrations in excess of 20 ppb.

We evaluate model performance for 2010 with available observations from India's CPCB. Modeled O_3 exhibits a minimal high bias (NMB = +3.5%) across nine monitor locations. However at Delhi monitor locations model biases are actually low on average in comparison due to an overestimation of modeled O_3 depletion from NO_x titration. Average high O_3 biases exhibited across northern India is exhibited in other CMAQ (Sharma *et al* 2016 and WRF-Chem Ghude *et al* 2016) analyses. Compared to two sites in Delhi, modeled daily $PM_{2.5}$ concentrations exhibit a slight high bias of +6.0%. Despite low model biases, normalized mean errors are quite high (O_3: 83.4%; $PM_{2.5}$ 45.7%) and spatial correlations are poor (O_3: −0.29; $PM_{2.5}$: +0.41). Evaluation with CPCB measurements is limited due to challenges such as unavailable data and the influence of very local sources on measured concentrations. Finally, to determine if the model can represent annual average values of $PM_{2.5}$ and O_3 from the four month simulations, we compare our modeled values with long-term average concentrations at available monitors from 1 January 2010– 31 December 2014 (detailed in the supplemental information). The four-month

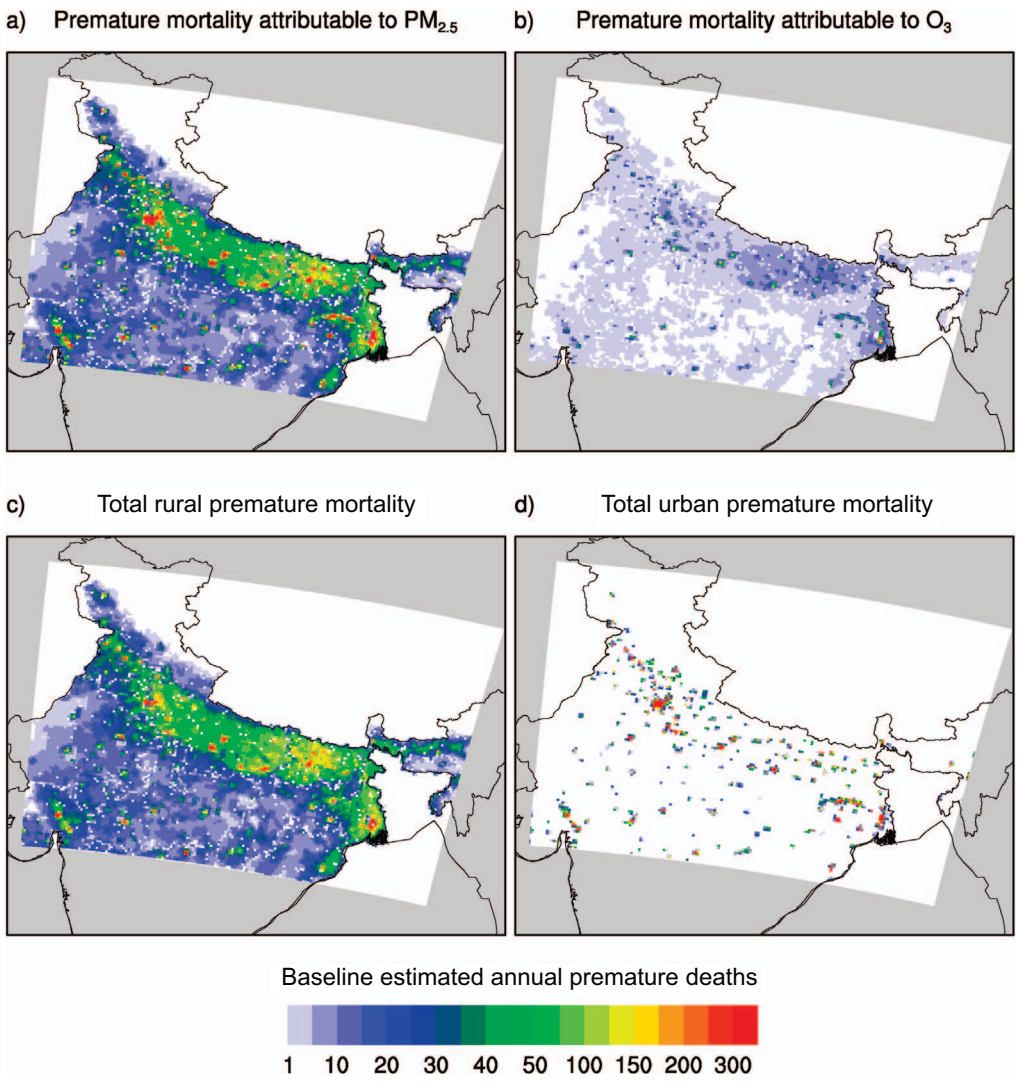

Figure 3. Annual estimates of premature mortality in deaths per 12 km by 12 km grid cell for adults greater than 25 years due to excess exposure to (*a*) $PM_{2.5}$, (*b*) MDA8 O_3. Average deaths per grid cell due to $PM_{2.5}$ (O_3) in India are 19 (2) adults, and maximum deaths per grid cell are 5580 (320) adults. Using ratios of grid-total urban and rural distributions, we also estimate (*c*) rural and (*d*) urban premature mortality distributions and quantify air quality impacts on these populations where the maximum deaths per grid cell due to $PM_{2.5}$ (O_3) are 4450 (200) in urban regions and 3020 (170) in rural regions.

average concentrations compare well with observations at nine monitor locations in Delhi for $PM_{2.5}$—163 $\mu g\,m^{-3}$ modeled and 160 $\mu g\,m^{-3}$ observed—and at 23 monitor locations in the IGP for annual average O_3—22 ppb modeled and 21 ppb observed. Evaluation metrics for NO_2, SO_2, and PM_{10} for 2010 are shown in supplemental table 3.

3.3. Adult premature mortality in northern India

Using representative annual average concentrations of $PM_{2.5}$ and O_3, we calculate premature mortality attributable to these pollutants within northern India.

Estimated population-weighted annual average concentrations of $PM_{2.5}$ are 72.1 $\mu g\,m^{-3}$ in northern India. We estimate 463 200 (95% CI: 444 600–482 600) annual premature adults deaths resulting from excess $PM_{2.5}$ pollution in northern India. Concentrations and adult premature deaths from COPD, IHD, stroke, and LC are greatest along IGP (figure 3(*a*)), where most grid cells reflect annual premature deaths between 40 and 75 adults and several localized hotspots estimate more than 300 adults deaths attributable to $PM_{2.5}$ (i.e. in New Delhi). In far northwestern India where the population and emission density is low, there are some cells where zero deaths are attributable to $PM_{2.5}$. Estimated annual average modeled concentrations in Delhi and Kolkata are 152.7 $\mu g\,m^{-3}$ and 144.4 $\mu g\,m^{-2}$, respectively, well above the Indian National Ambient Air Quality Standard (NAAQS) of 40 ug m^{-3} (annual average) and the WHO guideline of 10 ug m^{-3}. The populations of Delhi and Kolkata are 16.8 and 4.5 million people according to the 2011 Census, respectively. Using the average concentration and total populations of Delhi (National Capital Territory) and Kolkata, we find that approximately 12 200 (11 700–12 800) and 3300 (3600–3900) premature deaths occur each year in Delhi and Kolkata, respectively, as a result of excess $PM_{2.5}$. Our estimate of premature mortality

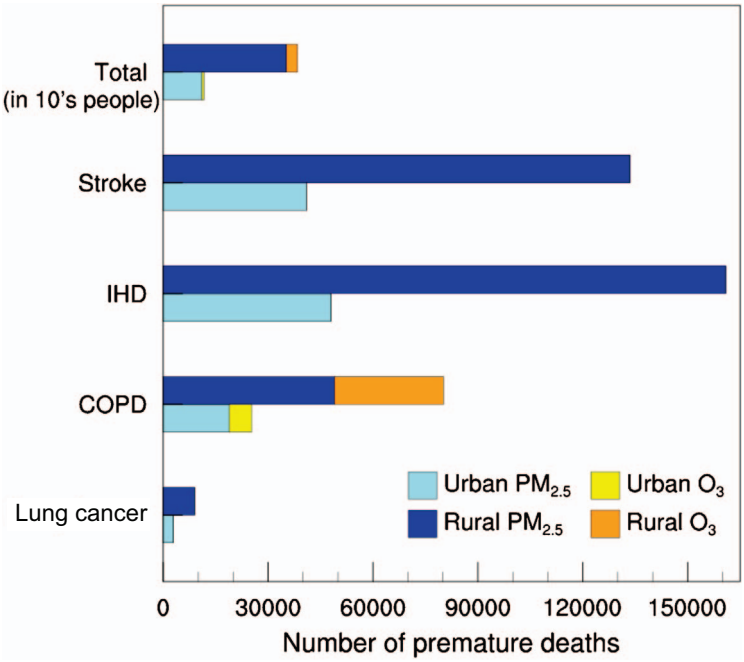

Figure 4. Total annual cause-specific urban (light blue) and rural (dark blue) premature death estimates for $PM_{2.5}$ attributable mortality, and average cause-specific urban (yellow) and rural (orange) premature death estimates for MDA8 O_3 attributable mortality. All premature mortality estimates for MDA8 O_3 are the result of chronic obstructive pulmonary disease (COPD). Total deaths are in tens of people, with the all-cause urban (rural) mortality estimated to be 117 200 (383 600). Stroke and IHD lead to the greatest number of premature deaths, while lung cancer is estimated to cause relatively few premature deaths in both urban and rural regions.

in Delhi is about 50% greater than the 8300 deaths from Amann *et al* (2017), likely due to our average modeled concentration being greater by nearly 30 μg m^{-3} and differences in model resolution, emissions, and population datasets. Values for other cities atop the WHO most polluted cities list are included in the Supplemental Information.

Larger numbers of premature deaths attributable to MDA8 O_3 are evident across the IGP than in central India (figure 3(*b*)). The average population weighted MDA8 O_3 concentration in northern India is 56.5 ppb. Exposure to O_3 contributes to approximately 37 600 (28 500–48 100) adult lives lost annually. In Delhi, the MDA8 O_3 concentration of 34.3 ppb is lower then both the O_3 ambient air quality standards of both India and the WHO (50 ppb) due to NO_x titration, while the average MDA8 O_3 concentration in Kolkata (59.5 ppb) is in excess of these standards. Ozone-attributable COPD deaths in these cities are an estimated 100 and 315, respectively. In contrast to $PM_{2.5}$, there are many grid cells across India where fewer than five premature adult deaths are attributable to O_3, suggesting that $PM_{2.5}$ rather than O_3 plays a larger health risk to the northern India population.

3.3.1. *Urban and rural adult premature deaths*

Using urban and rural population distributions, we find that excess $PM_{2.5}$ and O_3 pollution affects rural populations three to five times larger, with annual premature adult deaths in rural regions of 383 600 adults (figure 3(*c*)) compared to 117 200 deaths in urban regions (figure 3(*d*)). An estimated 352 400 premature deaths in rural regions are attributable to excess $PM_{2.5}$, compared to an estimated 110 800 adults in urban regions. Rural deaths correspond with a greater quantity of primary particulate emissions (2.3 Tg of elemental and organic carbon) versus urban (0.48 Tg). An urban-rural effect is found for O_3 as well, where the number of premature deaths in rural (urban) regions due to O_3 pollution is 31 200 (6400) adults. However, per total urban and rural population and in comparison to the total death rate of 72 people per 10 000, urban (rural) deaths attributable to ambient air pollution are 6.4 (5.6) per 10 000, such that ambient pollution in 2010 contributed to 8.1% of deaths.

Ambient $PM_{2.5}$-attributable premature deaths are greatest in rural regions for all diseases (figure 4). By far the greatest cause of death as a result of $PM_{2.5}$ is from ischemic heart disease (IHD), causing an estimated 160 900 premature deaths of adults annually. Total deaths from $PM_{2.5}$-attributable IHD is lower in urban areas at 48 000 premature deaths. Stroke contributes to the second greatest cause of $PM_{2.5}$ related premature mortality, with an estimated 133 500 (41 000) people in rural (urban) areas. COPD due to both $PM_{2.5}$ and O_3 affects a smaller number of people in both rural and urban regions (80 200 rural and 25 300 urban annually), and lung cancer has the lowest impact according to our estimates (9000 rural and 2900 urban annually). All of these estimates were based only on population exposure with no weight given to economic measures such as access to healthcare, however this may play

a considerable role in survival rate against a disease (Chowdhury and Dey 2016).

3.4. Limitations

This study has several limitations. One limitation is deficiencies in other emissions sectors, for instance, data is particularly lacking for the industrial sector. Further, emissions inventories in India remain highly uncertain. Recently, an emissions inventory comparison noted large uncertainties, on the order of 150%–325%, across several inventories for different pollutants from the domestic combustion sector (Saikawa *et al* 2017). Updates to support reducing uncertainties in the domestic combustion sector have been included in this study, yet still requires considerable research.

A second limitation is using four model months to represent annual average concentrations. Each month was chosen to represent a unique air quality condition: January for the polluted winter due to inversions, April to represent spring windblown dust, July to simulate air quality conditions during the monsoon, and October to represent the onset of the agricultural biomass burning season in northwestern India. However, we note above that the average concentrations estimated using these four months ($PM_{2.5}$: 163 $\mu g\,m^{-3}$, O_3: 22 ppb) are comparable to annual average long-term observations from the CPCB ($PM_{2.5}$: 160 $\mu g\,m^{-3}$, O_3: 21 ppb).

Finally, there are limitations in using the IER methodology to estimate premature mortality in India. The concentration-response functions for ambient air pollution used here were developed in the context of the GBD 2013 study (Forouzanfar *et al* 2015) based on epidemiological studies in comparatively low-pollution regions including the US and Europe. Applying these functions to estimate health effects in a high pollution region such as India relies upon extrapolation to higher levels based on studies from the passive and active smoking literatures (Burnett *et al* 2014) and references therein. The parameters used in the IERs underlie considerable uncertainty, and recent updates in the context of GBD studies since 2010 led to distinctly different estimates of premature deaths. However, in the absence of long-term air pollution cohort studies at high concentrations, using published and documented exposure-response relationships remains the best approach for estimating effects in India, and yields findings that can promote awareness of health implications and the need for observational studies in India to obtain more accurate representation of underlying health status, relevant time spent outdoors, access to healthcare, and genetic predisposition.

4. Conclusion

An estimated 463 200 (95% CI: 444 600–482 600) and 37 800 (28 500–48 100) premature deaths occurred annually in 2010 as a result of excess $PM_{2.5}$ and O_3 concentrations, respectively, in northern India. Compared to prior studies that estimate annual adult premature mortality due to $PM_{2.5}$ (between 486 100 and 1100 000 (Chowdhury and Dey 2016, Ghude *et al* 2016, GBD MAPS Working Group 2018)) and/or O_3 (12 000 (Ghude *et al* 2016)) across all of India, our estimates from this study over northern India are slightly lower for $PM_{2.5}$ (463 200 premature deaths) as expected due to the smaller domain we use in our study, yet are much higher for O_3 (37 800 premature deaths). The discrepancy between these totals may reflect our use of MDA8 O_3 concentrations to consider effects from peak O_3 exposure used for setting air quality standards, whereas Ghude *et al* (2016) used bias-corrected annual average concentrations to estimate average exposure levels. Finally, we note that our estimates are for adult premature mortality, though it is estimated that up to 6% of childhood premature deaths between the ages of five and fifteen are the result of lower respiratory infections attributable to ambient air pollution exposure (Institute for Health Metrics and Evaluation 2016).

We conduct model simulations to generate estimated annual average ambient concentrations of $PM_{2.5}$ and O_3 from the CMAQ model using a new high-resolution anthropogenic emissions inventory updated according to urban and rural population and activity distribution measures for transportation and domestic combustion. Separating total premature deaths according to respective urban and rural areas, we find that 6.4 (5.6) deaths per 10 000 people occur in urban (rural) areas attributable to ambient $PM_{2.5}$ and O_3 concentrations. However, despite the greater rate of deaths per 10 000 people that occur in urban areas, the total amount of premature deaths attributable to air pollution in rural areas (383 600 deaths) is much larger in magnitude than those that occur in urban populations annually (117 200). Our results (76% rural) are in line with those in a modeling study from the Health Effect Institute, that found 75% of premature deaths attributable to air pollution affect rural populations (GBD MAPS Working Group 2018), with a large fraction of premature mortality in rural populations due to residential combustion. We note this similarity in urban-to-rural ratios despite the present study using (1) a more common global anthropogenic emissions inventory with updates as opposed to an inventory developed from prior studies over India, and (2) a higher resolution regional chemical transport model. Rural populations are burdened by ambient air pollution three to five times greater than urban populations, indicating a need for surface air pollution monitoring in rural regions.

This work supports the growing body of research using modeled ambient concentrations to estimate premature mortality associated with exposure to air pollution in India by using high-resolution detailed updates for urban and rural emissions for

transportation and domestic combustion in addition to location-specific power plant emissions. Ultimately, it is challenging to gain a full understanding of the impacts on adult mortality in northern India without significant measurements available to validate air quality model results. Satellite tropospheric vertical column depths (VCDs) can potentially fill in the spatial gaps (Chowdhury and Dey 2016), yet model data are often necessary to derive surface estimates from the VCDs to use in health impacts assessments (Ghude *et al* 2016). Future efforts would benefit from an increased network of surface observations to assess more rigorously air quality model performance and to build confidence in applying these models to quantify human health impacts in the region. With surface observations available in rural regions, we can also better characterize ambient air pollution and the skill of high-resolution emissions inventories to provide the scientific basis for selecting effective emission control strategies to improve air quality in India.

Acknowledgments

We thank three anonymous reviewers for providing valuable feedback. A K acknowledges J Borken for guidance on transportation emissions, W Schoepp for providing gridded urban and rural population data, and The Earth Institute at Columbia University for supporting this research.

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