

RESEARCH LETTER

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Key Points:

- PM_{2.5} exposure is causing 570,000 premature mortalities
- Economic cost of estimated premature mortalities is about 640 billion USD
- PM_{2.5} should be the primary target for reducing pollution impacts on health

Supporting Information:

- Supporting Information S1

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Premature mortality in India due to PM_{2.5} and ozone exposure

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Abstract This bottom-up modeling study, supported by new population census 2011 data, simulates ozone (O₃) and fine particulate matter (PM_{2.5}) exposure on local to regional scales. It quantifies, present-day premature mortalities associated with the exposure to near-surface PM_{2.5} and O₃ concentrations in India using a regional chemistry model. We estimate that PM_{2.5} exposure leads to about 570,000 (CI95: 320,000–730,000) premature mortalities in 2011. On a national scale, our estimate of mortality by chronic obstructive pulmonary disease (COPD) due to O₃ exposure is about 12,000 people. The Indo-Gangetic region accounts for a large part (~42%) of the estimated mortalities. The associated lost life expectancy is calculated as 3.4 ± 1.1 years for all of India with highest values found for Delhi (6.3 ± 2.2 years). The economic cost of estimated premature mortalities associated with PM_{2.5} and O₃ exposure is about 640 (350–800) billion USD in 2011, which is a factor of 10 higher than total expenditure on health by public and private expenditure.

1. Introduction

Surface ozone (O₃) and fine particulate matter (PM_{2.5}) are major atmospheric pollutants directly affecting human health by causing cardiovascular and respiratory diseases. The environmental risk caused by exposure to these pollutants has increased in many parts of the world as a result of human activity [Burnett *et al.*, 2014; Lelieveld *et al.*, 2015; Apte *et al.*, 2015]. Poor air quality is an important societal issue specifically for developing countries like India [Ghude *et al.*, 2014] where rapid expansion of industrial, urban, and traffic emissions have significantly increased the air pollution especially over the last two decades [Ghude *et al.*, 2013]. Climate change can further influence air quality, as studies have shown, causing O₃ and PM_{2.5} to increase in many developing regions of the world [Horowitz, 2006; Fang *et al.*, 2013].

Measurement data from National Ambient Air Quality Monitoring networks [Central Pollution Control Board (CPCB), 2014], satellite observations, estimates from emission inventories, and regional model calculations [Ghude *et al.*, 2013; Jena *et al.*, 2015a] unambiguously show elevated levels of air pollutants in many regions of India. Therefore, the potential risk to health is higher for populations in these regions. As indicated by the 2011 census of India's statistics, around 32% (~0.4 billion people) of India's 1.2 billion population lives in urban areas. About 78% of the total 141 cities in the country exceed the PM_{2.5} standard, 90 cities have critical levels, and 26 have the most critical levels, exceeding the PM standard by over 3 times [CPCB, 2014]. The Global Burden of Disease estimates ranks pollution as the fifth largest killer in India [World Health Organization (WHO), 2014]. Estimates of the respiratory mortality show India to rank second among the countries globally affected by PM_{2.5} [Silva *et al.*, 2013] and O₃ pollution [Lelieveld *et al.*, 2013]. It is anticipated that recent upward trends in transportation, industrial and energy sectors, urbanizations, population growth in India along with climate change will raise the levels of O₃ and PM_{2.5} in the future, which could worsen the vulnerability of a growing population.

Over the past decades, numerous studies have estimated premature mortality associated with O₃ and PM_{2.5} pollution on global to regional scales using high-resolution global chemistry transport models [Silva *et al.*, 2013; Lelieveld *et al.*, 2013; Fang *et al.*, 2013] or surface-based measurements [Cohen *et al.*, 2004] and using satellite measurements [Van Donkelaar *et al.*, 2010; Global Burden of Diseases (GBD), 2010; Brauer *et al.*, 2012; Apte *et al.*, 2015]. Most of these studies were focused on the United States and Europe and relatively little work has been devoted to India. Studies that relate air quality and human health have been performed only focusing on a few urban areas of India [Gupta, 2011; Guttikunda and Goel, 2013; Chate *et al.*, 2013; Nagpure *et al.*, 2014] using aggregated data of local air quality. This study assesses the impact of only outdoor

air quality on health for all of India on district scale. To our knowledge, the present study is the first high-resolution district-scale modeling study for India to investigate and quantify premature mortality due to O_3 and $PM_{2.5}$ exposure. Here we estimate the burden of present-day anthropogenic air pollution on premature human mortality using a high-resolution regional chemistry transport model with anthropogenic emissions from Hemispheric Transport of Air Pollution (HTAP)-v2, combined with the latest district-wise population count data sets [India Office of the Registrar General and Census Commissioner, 2011] for India. It should be noted that the model is unable to simulate microenvironments (such as near roadways) and our analysis does not consider indoor exposure from cooking and heating.

Our estimates of $PM_{2.5}$ -caused premature mortalities are based on GBD [2010], applying integrated exposure response functions developed by Burnett *et al.* [2014], which accounts for health effect at even higher exposure concentrations. We have estimated premature mortalities due to ischemic heart disease (IHD), cerebrovascular disease (CEV, stroke), chronic obstructive pulmonary disease (COPD), and lung cancer (LC) for adult population, and acute lower respiratory illness (ALRI) for infants (<5 years old) linked to $PM_{2.5}$ exposure. For estimating COPD linked to O_3 exposure we used the exposure response function by Ostro [2004]. Additionally, this study calculates the economic loss and lost life expectancy due to $PM_{2.5}$ exposure. Surface O_3 and $PM_{2.5}$ concentrations over India vary significantly among the Indian states, largely due to differences in emission patterns and regional meteorology (Figure S1 in the supporting information). Therefore, we also address the state-wise impacts of air quality degradation on premature mortality, economic cost, and lost life expectancy.

2. Method

2.1. Regional Chemistry Modeling

We used version 3.6.1 of the regional Weather Research and Forecasting model coupled with chemistry (WRF-Chem) to simulate hourly surface O_3 and $PM_{2.5}$ distributions for the entire year of 2011 at 36 km horizontal resolution in order to resolve urban and rural regions. The model was driven by National Centers for Environmental Prediction Final (GFS/FNL) meteorological reanalysis fields. The model uses MOZART-4 gas-phase chemistry linked to the GOCART aerosol scheme. The GOCART aerosol model simulates five major types of aerosols, namely, sulfate, black carbon, organic carbon, dust, and sea salt. Anthropogenic emissions of CO, NO_x , SO_2 , NMVOC, PM10, $PM_{2.5}$, and BC/OC are taken from the HTAP-v2 inventory [http://edgar.jrc.ec.europa.eu/htap_v2/]. Fire emissions were provided to the model using the Fire INventory from NCAR (FINNV1). More details on the modeling setup are discussed by Jena *et al.* [2015b].

Kumar *et al.* [2012] discuss in detail the validation of meteorological fields over India and shows that the index of agreement for important meteorological parameters is greater than 0.6, indicating that WRF-Chem is capable of simulating the variations around the observed mean. Detailed descriptions of the chemistry simulations, including a discussion of the anthropogenic, biogenic and fire emissions, chemical boundary conditions, and meteorological inputs are given by Ghude *et al.* [2013] and Jena *et al.* [2015a]. We evaluate the model performance by comparing modeled aerosol optical depth (AOD_{550}) with MODIS AOD_{550} (Figure S2), and simulated monthly mean $PM_{2.5}$ and O_3 with observations from different ground sites in India (Figures S3 and S4). The model reproduces the observed distribution of AOD_{550} very well (mean bias = -0.08 ± 0.1) but shows lower values over the Indo-Gangetic Plain (IGP) region. The model also reproduces the seasonality of surface $PM_{2.5}$ of the sites in India. Both observations and model results show the largest $PM_{2.5}$ concentrations for Delhi and Agra, but the model tends to overestimate summertime and underestimate wintertime values (Figure S3). For ozone, the model shows a similar seasonality as the observations, but for most sites the model is biased high throughout the year. While the $PM_{2.5}$ model bias is relatively low, the model bias is large for surface O_3 (about 10–30 ppb) at some of the sites in India. Most of these observational sites are situated in urban/suburban locations which generally represent very local conditions and may not be indicative of the overall model performance. High NO_x emissions [Jena *et al.*, 2015b], coarser model grid spacing [Kumar *et al.*, 2012], and underestimation of dry deposition [Martin *et al.*, 2014] could also be contributing factors that may lead to an overestimation of ozone production and should be investigated in further detail. As will be discussed later, we try to account for the high model bias in O_3 when estimating premature mortality.

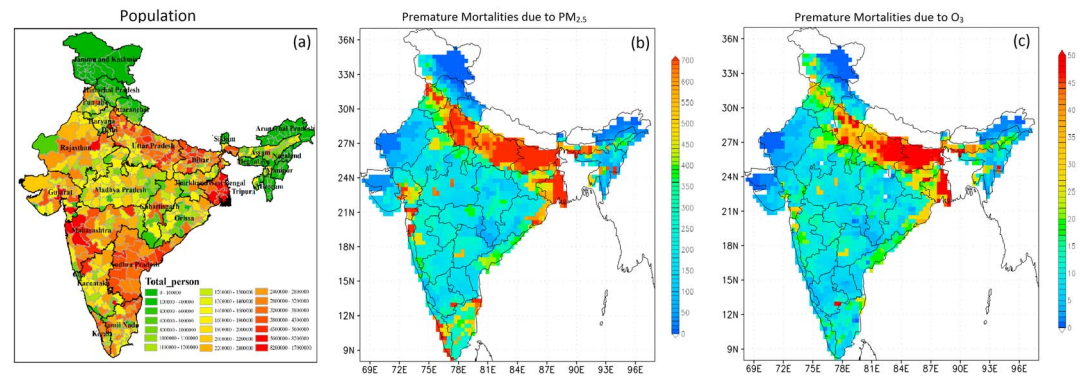


Figure 1. Distribution of (a) district-wise India's population and total premature mortalities due to (b) $PM_{2.5}$ and (c) O_3 exposure in 2011. (Unit: premature mortalities per grid box) (AP (Andhra Pradesh), AR (Arunachal Pradesh), AS (Assam), BR (Bihar), CG (Chhattisgarh), DL (Delhi), GA (Goa), GJ (Gujarat), HP (Himachal Pradesh), HR (Haryana), JH (Jharkhand), JK (Jammu and Kashmir), KA (Karnataka), KL (Kerala), MH (Maharashtra), ML (Meghalaya), MN (Manipur), MP (Madhya Pradesh), MZ (Mizoram), NL (Nagaland), OR (Orissa), PB (Punjab), RJ (Rajasthan), SK (Sikkim), TN (Tamil Nadu), TR (Tripura), UK (Uttarakhand), UP (Uttar Pradesh), WB (West Bengal)).

2.2. Health Impact

The latest district-wise population count and age structure data set used in this work is taken from the Registrar General and Census Commissioner, Government of India for the year 2011 (Figure 1a). These district-wise population data are mapped to the 36 km model grid using GIS-based statistical methodology. According to the Census 2011, around 31% (37 million) of the residents are 0–14 years old and 5% (~6.6 million) of the residents are above 65 years old. Approximately, 32% of this population (in all age groups) resides in urban areas.

The premature mortality attributable to anthropogenic O_3 and $PM_{2.5}$ has been estimated employing the human health impact function given in *Burnett et al.* [2014], *Lelieveld et al.* [2015], and *Apte et al.* [2015] for present-day concentrations. To estimate the premature mortalities we combined the results with epidemiological exposure response functions by using the following relationship:

$$\Delta M = \delta c[(RR - 1)]/RR]P \quad (1)$$

In equation (1), ΔM is a function of the baseline mortality rate of a particular disease category δc for countries estimated by the *World Health Organization (WHO)* [2012]. P is the population count for the specific age category. In this work the baseline mortality rate for a specific disease is obtained from the WHO health statistics and health information system. RR is relative risk and $(RR - 1)/RR$ is the attributable fraction. *Burnett et al.* [2014] developed integrated exposure response functions that constrain the shape of the C-R relationship using mortality data for even higher $PM_{2.5}$ exposure concentration, which may represent the countries where $PM_{2.5}$ levels can be much higher such as East and South Asia. The value of RR attributable to $PM_{2.5}$ exposure is calculated for different disease categories for adults (IHD, CEV (stroke), COPD, and LC, and acute lower respiratory illness (ALRI) for infant population). Here we have used RR derived by *Burnett et al.* [2014] that parameterizes the dependence of RR on concentration (X) based on the metaanalysis of observed data:

$$RR = 1 + \alpha\{1 - \exp[-b(X - X_0)^p]\} \quad (2)$$

For each disease categories, X_0 represents the theoretical minimum-risk concentrations (range: 5.8–8.0 $\mu g m^{-3}$). We also adopted the bounds representing the 95% confidence interval (CI) which was derived by *Burnett et al.* [2014] from 1000 sets of coefficients and exposure response functions based on Monte Carlo simulations. For estimating COPD linked to O_3 exposure we applied the exposure response function by *Ostro* [2004]:

$$RR = [(X + 1)/(X_0 + 1)]^\delta \quad (3)$$

where $X_0 = 37.6$ [Lim et al., 2012] and is the average of the range of 33.3–41.9 ppbv O_3 , and δ is 0.1521 [Lim et al., 2012]. More discussion on uncertainties and sensitivity calculations that address the shape of exposure response function is given in detail in *Burnett et al.* [2014] and *Lim et al.* [2012].

Using the model-simulated annual mean $\text{PM}_{2.5}$ and O_3 fields, the premature mortalities for five different diseases are estimated for population in each model grid. The state and national level mortalities are estimated for the year 2011 by summing all grid within the state and national boundaries (Figure 1a). We find higher O_3 and $\text{PM}_{2.5}$ concentrations over most of the densely populated regions but with a strong seasonal variability (Figure S1).

We also evaluate state and nationwide economic cost of the health impact associated with the $\text{PM}_{2.5}$ and O_3 exposure during 2011. We used the value of the statistical life (VSL) to evaluate the cost of premature mortalities. VSL is widely used in the USA and Europe in cost-benefit analysis to assess the benefits of saving lives [OECD, 2014]. In India, estimates of VSL on original country-specific studies are scarce. Therefore, the benefits transfer approach has been applied, with estimates of VSL from developed countries being transferred after adjusting for differences in income or national per capita output as suggested in OECD [2014] and Chen *et al.* [2015]. To establish the India-specific VSL we used the following equation:

$$\text{VSL}_{\text{IC}} = \text{VSL}_{\text{Db}} \times (G_{\text{IC}}/G_{\text{DC}})^{\beta} \times (1 + \% \Delta P + \% \Delta Y)^{\beta} \quad (4)$$

VSL_{IC} is estimated VSL for India for current year, VSL_{Db} is adopted VSL for developed countries (e.g., USA or Europe) for the base year, G_{IC} and G_{DC} are the Gross Domestic Product per capita at the Purchasing Power (PPP) in India and developed countries for the current year, $\% \Delta P$ and $\% \Delta Y$ are the percentage increase in consumer price and real GDP per capita growth in India from base year to current year, respectively. β is income elasticity of VSL. Using this approach, we estimate a VSL of USD 1.1 million for India in 2011 (see Supporting Information S1).

For calculating lost life expectancy due to $\text{PM}_{2.5}$ exposure we followed the estimate in Pope *et al.* [2009]. As per their estimate, an increase of $1 \mu\text{g}/\text{m}^3$ in $\text{PM}_{2.5}$ exposure decreases mean life expectancy by about 0.061 ± 0.02 years. This estimate is derived for higher-income countries, where average $\text{PM}_{2.5}$ concentrations are lower than those found for India and may introduce some additional uncertainties in our estimates.

3. Results

3.1. Health Impact Assessment

Figure 1a shows the population distribution for India in 2011, while Figures 1b and 1c show estimates of $\text{PM}_{2.5}$ -related premature mortalities (per grid box) linked to CEV, COPD, IHD, LC, and ALRI, and O_3 -related mortalities by COPD in 2011, respectively. The estimated premature mortalities are a function of both the population and air quality in each grid cell resulting in high premature mortalities in heavily polluted and populated areas. As seen in Figure 1, premature mortality is widespread in India. The Indo-Gangetic Plain (IGP) shows highest estimated premature mortalities due to both ground level $\text{PM}_{2.5}$ (300–800 excess cases/grid box) and O_3 (25–60 excess cases/grid box), followed by Southern India (Tamil Nadu and Kerala), and Mumbai-Gujarat Industrial corridor. Among the different polluted areas, the largest share of premature mortalities is in the IGP region with about 42% for $\text{PM}_{2.5}$ exposure and 45% for O_3 exposure. This is a point of concern because one eighth of the world population resides in this region and most models and future projections based on Representative Concentration Pathways (RCPs) emission scenarios predict significant increase in O_3 and $\text{PM}_{2.5}$ concentrations from this region due to climate change [Horowitz, 2006; Fang *et al.*, 2013]. The number of cases of premature mortalities due to $\text{PM}_{2.5}$ exposure from IGP region is estimated to be $\sim 240,000$. (95%CI: 140,000–300,000). This is equivalent to the estimate of 240,000 (in 2005) deaths globally attributed to $\text{PM}_{2.5}$ contribution from surface transportation [Chambliss *et al.*, 2014]. Substantial premature mortalities due to $\text{PM}_{2.5}$ (300–600 excess cases/grid boxes) and O_3 (10–40 excess cases/grid box) exposure are also seen in the northeastern India, although this region is moderately populated. This region shows less influence from anthropogenic activities [Ghude *et al.*, 2013] but elevated levels of $\text{PM}_{2.5}$ and surface O_3 during premonsoon time as a result of biomass burning [Jena *et al.*, 2015a] (Figure S1). Figures 1b and 1c also distinguish metropolitan areas and big cities such as Delhi, Mumbai, Kolkata, and Bangalore as locations where air pollution causes a significant number of premature mortalities.

To illustrate the state-wise pattern of premature mortalities, we present a bar chart (Figures 2a and 2b) showing the excess number of cases due to $\text{PM}_{2.5}$ and O_3 exposure for various health outcomes (Adults: IHD, CEV (stroke), COPD, and LC; infants: ALRI). The state with the highest premature mortalities due to $\text{PM}_{2.5}$ exposure is Uttar

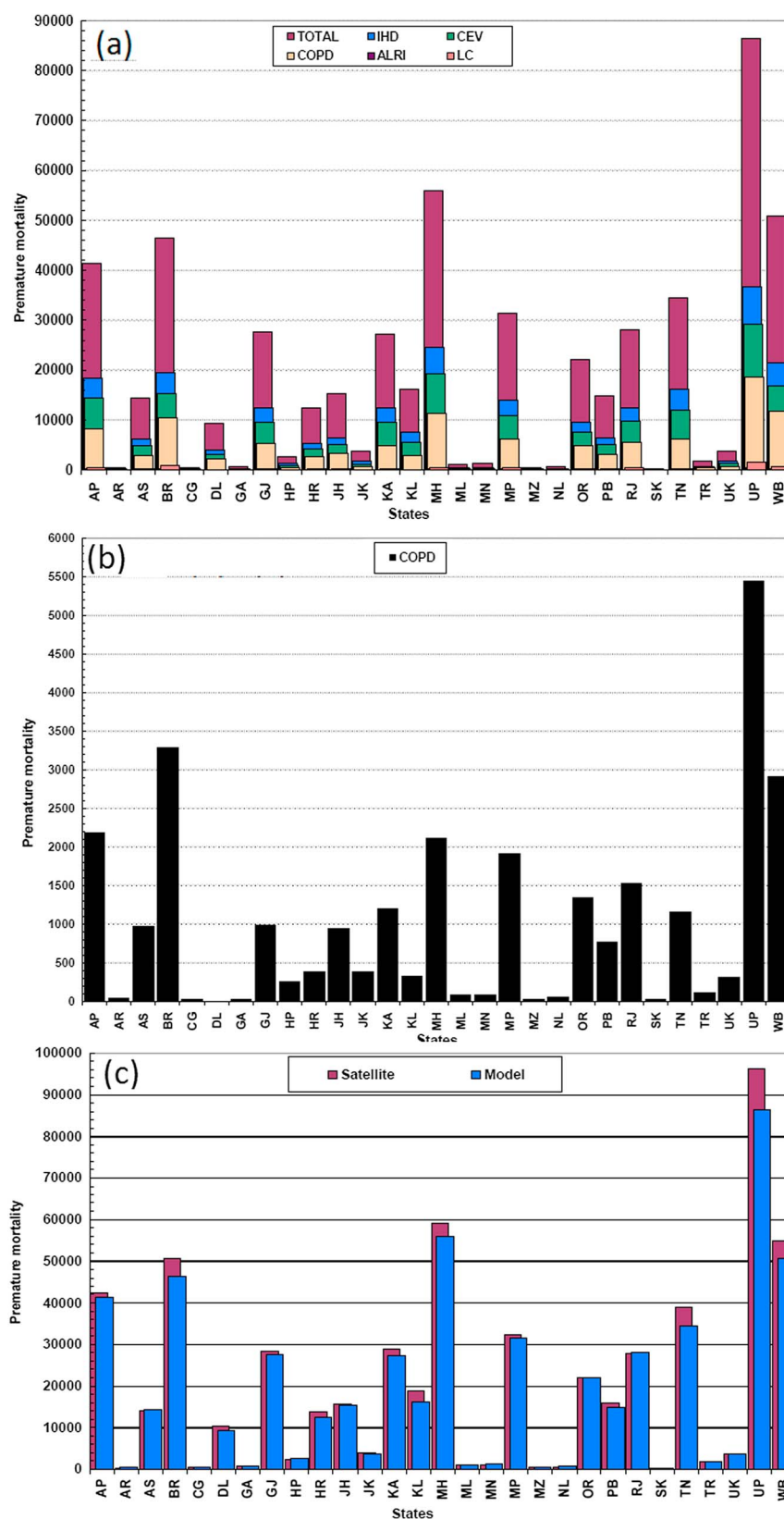


Figure 2. Estimates of the state-wise premature mortalities due to (a) $PM_{2.5}$ and (b) O_3 exposure for various health outcomes (TOMR, CARD, RESP, and COPD) in 2011. (c) State-wise premature mortalities estimated from the satellite-derived and simulated $PM_{2.5}$ concentrations.

Table 1. Premature Mortalities Due To PM_{2.5} and O₃ Exposure in India During 2011

Health Endpoints		Premature Mortalities (95% CI) (Simulated PM _{2.5})	Premature Mortalities (Satellite PM _{2.5})
1	Ischemic heart disease (IHD) ^a	250,000 (190,000–310,000)	260,000
2	Cerebrovascular disease (CEV, stroke) ^a	190,000 (70,000–240,000)	200,000
3	Chronic obstructive pulmonary disease (COPD) ^a	117,000 (61,000–165,000)	135,000
4	Lung cancer (LC) ^a	2,700 (1,000–3,600)	3,100
5	Acute lower respiratory illness (ALRI) ^b	7,300 (4,900–9,000)	8,200
6	COPD due to O ₃ ^a	31,000 (23,000–39,000)	

^aFor adult population (>25 years old).^bFor infants (<5 years old).

Pradesh (UP), which accounts for about 15% (about 86,000 excess cases) of all premature mortalities in India during 2011, followed by Maharashtra (MH, 10%), and West Bengal (WB, 9%) and Bihar (BR, 8%). Other states with high premature mortalities due to PM_{2.5} are Andhra Pradesh (AP), Tamil Nadu (TN), Gujarat (GJ), Karnataka (KA), Madhya Pradesh (MP), Orissa (OR), and Rajasthan (RJ), which collectively make up for 32% of the countrywide premature mortalities. For O₃-related mortalities by COPD, the greatest premature mortalities are found in UP, with about 5500 excess cases (about 18%), followed by BR (11%), WB (9.5%), and MH and AP (7%). It can be seen that, premature mortalities due to O₃ exposure for other states show a similar pattern to that of PM_{2.5} premature mortalities.

The premature mortalities for different health outcomes are summarized in Table 1. Our estimate of nationwide premature mortalities related to PM_{2.5} exposure in 2011 is about 570,000 people (47 people per 10⁵ population) with the 95% confidence interval, 320,000–730,000. For adults (age >25 years) premature mortalities linked to IHD, CEV (stroke), COPD, and LC are estimated to be about 250,000 (95%CI: 190,000–310,000), 190,000 (95%CI: 70,000–240,000), 120,000 (95%CI: 61,000–160,000), and 2700 (95%CI: 1000–3600) people, respectively. Premature mortalities (ALRI) for children <5 years old are estimated at about 7300 (95%CI: 4900–9000). Our nationally aggregated estimate of O₃-related premature total mortality by COPD is about 31,000 (95%CI: 23,000–39,000) people, which agrees very much with the estimate by *Lelieveld et al.* [2015].

In addition to simulated PM_{2.5} concentration, we determined the annual estimate of ground level PM_{2.5} using column aerosol optical depth (AOD) [Van Donkelaar et al., 2010] from the MODIS satellite instruments and coincident simulated vertical aerosol profiles from simulations described in section 2.1 (see Supporting Information S1). We further estimated premature mortalities from the satellite-derived surface PM_{2.5} for 2011 similar to estimates made in recent studies [GBD, 2010; Brauer et al., 2012; Apte et al., 2015]. Figure 2c shows the comparison between premature mortalities estimated from the satellite and simulated PM_{2.5} concentrations for different states of India, and Table 1 shows the comparison for the different health outcome. It can be seen that satellite-based total mortalities are a little bit higher than the simulated PM_{2.5} exposure. Our nationwide estimated premature mortalities from satellite-derived PM_{2.5} exposure is about 601,000 people, which is about 6% higher than the estimate derived from the simulated PM_{2.5} exposure. The total number of mortalities estimated (both from simulated and satellite-derived PM_{2.5} concentration) in this study compares closely to the WHO [2014] estimate of 0.62 million, Apte et al. [2015] estimate of 0.57 million, and Lelieveld et al. [2015] estimate of 0.62 million (South Asia) deaths due to air pollution in India. It is double in magnitude to that of 300,000 deaths globally caused by human impact of global warming and climate change [GBD, 2010]. It should be noted that premature mortalities estimated in this study is in addition to near-source exposure such as indoor cooking and heating [Lim et al., 2013].

Simulated O₃ concentrations are positively biased by about 10–30 ppb over the India region. Therefore, the estimated premature mortalities (31,000) due to O₃ exposure might be on the higher side. In order to see the impact of bias on estimated premature mortalities due to O₃ exposure we performed additional calculations by reducing O₃ amount by 15 ppb over the entire simulation domain. Assuming that the bias in O₃ (15 ppb) is entirely due to the model performance (and all other factors are correct) and representative for all of India, the bias-corrected mortalities are estimated to be about 12,000 people.

Estimated mortalities are subject to several sources of uncertainty including uncertainties in the model parameterizations (e.g., emissions and physical/chemical parameterizations) and uncertainties in the exposure response functions (e.g., RR values). Taking into account the variability in various emissions for South Asia, the uncertainty in $PM_{2.5}$ emissions could be as much as 30% [Kurokawa *et al.*, 2013]. This adds an uncertainty of about 12% in the estimated premature mortalities due to $PM_{2.5}$ exposure. Additional sources of uncertainties in our estimate lie in the use of a specific chemical mechanism to simulate $PM_{2.5}$ and O_3 concentration. The GOCART aerosol scheme used in the present simulation does not include nitrate and Secondary Organic Aerosol (SOA) production; therefore, nitrate and SOA are missing in the simulated $PM_{2.5}$ concentration. The relative contribution of nitrate and SOA in $PM_{2.5}$ is estimated to be 10% and 15% in India [Behera and Sharma, 2010], which adds uncertainty of about 3% and 5%, respectively, in the estimated premature mortalities due to $PM_{2.5}$ exposure. Variability in different NO_x emission inventories in India leads to an uncertainty of about 15% in daytime-simulated O_3 concentration [Jena *et al.*, 2015b], which adds uncertainty of about 36% in the estimated premature mortalities due to O_3 exposure. Overall uncertainty due to combination of above input parameters translate into about 21% in our premature mortality estimates. RR values used in this study are derived from the integrated exposure response functions that constrain the shape of the C-R relationship using mortality data for higher $PM_{2.5}$ exposure concentration; however, it is based on epidemiological cohort studies conducted in other parts of the globe, which may not necessarily represent conditions in South Asia. This requires future region-specific epidemiological research for constraining the shape of the $PM_{2.5}$ concentration-response relation. Due to the lack of India-specific epidemiological exposure response functions it is hard to quantify related uncertainties in our premature mortality estimates.

We calculated the nationwide and statewide economic cost due to premature mortalities linked to $PM_{2.5}$ and O_3 exposure using equation (4). We computed VSL for India to be about 1.1 million USD in 2011 adjusted by GDP per capita on PPP basis. The state-wise economic cost due to premature mortality of $PM_{2.5}$ and O_3 exposure is largest in Uttar Pradesh (98 billion USD) followed by Maharashtra (62 billion USD), West Bengal (57 billion USD), and Bihar (53 billion USD) (Figure 3a). On a national scale, we estimate the economic cost to be about 640 (350–800) billion USD₂₀₁₁ annually because of total premature mortalities due to $PM_{2.5}$ and O_3 exposure under modeled present-day pollution levels. The total GDP of India was 1840 billion USD and total expenditure on health (public plus private) as percentage of GDP in 2011 for India was 3.9% [OECD, 2013], i. e., approximately 60 billion USD. The economic cost due to $PM_{2.5}$ and O_3 exposure estimated in this study is much larger than the total health expenditure by government in 2011.

We further estimated health burden in terms of life years lost due to $PM_{2.5}$ exposure following the estimates given in Pope *et al.* [2009]. Nationally, $PM_{2.5}$ exposure is estimated to cause a life year lost of about 3.4 ± 1.1 years, on average. However, the regional variability is significant. Figure 3b shows the estimated life years lost due to annual averaged $PM_{2.5}$ exposure by state. The lowest lost life expectancy is noticed in Jammu Kashmir ($\sim 0.6 \pm 0.2$ years) followed by Himachal Pradesh and Sikkim ($\sim 1.2 \pm 0.4$ years). Delhi suffered the greatest lost life expectancy of about 6.3 ± 2.0 years, followed by west Bengal (6.1 ± 1.9 years) and Bihar (5.7 ± 1.9 years), which are also the top three polluted states in India (Figure 3a). This is a point of concern because overall average life expectancy is already low (64 years) in India; ranked 150 worldwide in 2012 and future increase in $PM_{2.5}$ concentration may worsen the situation.

4. Conclusion

Our study suggests that the widespread $PM_{2.5}$ and O_3 pollution under present emission levels considerably impact human mortalities and life expectancy in India. The present-day premature mortalities due to $PM_{2.5}$ ($\sim 570,000$) and O_3 (31,000) exposure caused economic cost of approximately 640 billion USD, which is a factor of 10 higher than total expenditure on health by public and private expenditure in India. Simulated O_3 concentrations are positively biased (about 10–30 ppb) over the India region, which might put our estimated premature mortalities (31,000) due to O_3 exposure on the high side. Our bias-corrected total mortalities estimate is less than half of this, about 12,000 people. In April 2015, the Union Environmental Ministry of Government of India launched a national Air Quality Index as a major aggressive initiative for improving air quality in urban areas for air pollution mitigation and to meet clean-air standards for reducing the public health risk. Our estimates on premature mortalities, economic loss, and life lost years provide

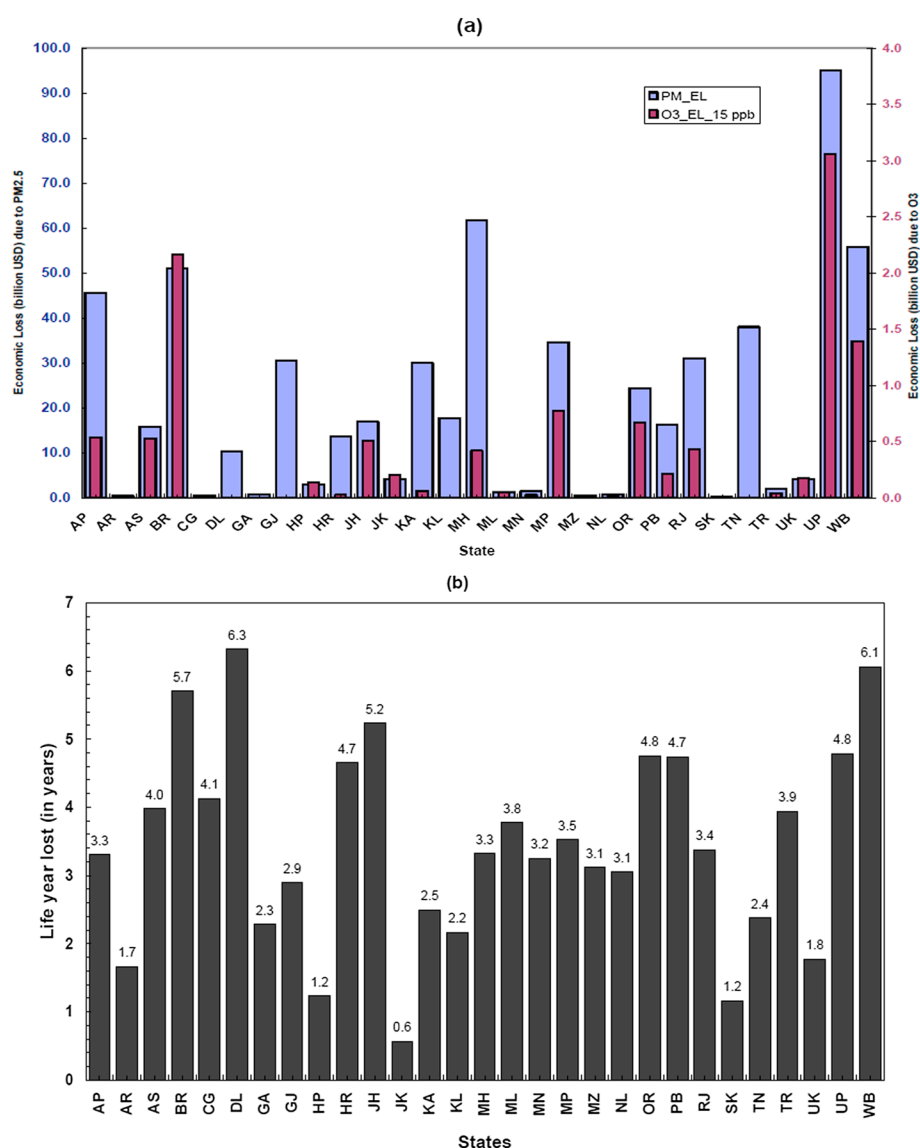


Figure 3. Estimates of the state-wise (a) economic loss due to total mortalities by PM_{2.5} and O₃, and (b) mean lost life expectancy due to PM_{2.5} exposure in India during 2011.

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important information to elective members and policy makers to propose or impose emission controls to benefit reduced public health risk due to exposure to outdoor air pollution. Therefore, these results may have important policy implications considering the projected future increase in PM_{2.5} and O₃ and implementation of revised air quality standard for India.

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