Maji et al., *Cogent Environmental Science* (2016), 2: 1193110
http://dx.doi.org/10.1080/23311843.2016.1193110

cogent environmental science

ENVIRONMENTAL HEALTH | RESEARCH ARTICLE

Human health risk assessment due to air pollution in 10 urban cities in Maharashtra, India

Kamal Jyoti Maji[1]*, Anil Kumar Dikshit[1,2] and Ashok Deshpande[3]

Received: 15 March 2016
Accepted: 19 May 2016
First Published: 29 May 2016

*Corresponding author: Kamal Jyoti Maji, Center for Environmental Science and Engineering (CESE), Indian Institute of Technology Bombay, Mumbai 400076, Maharashtra, India
E-mails: kamaljm@iitb.ac.in, kjmaji@gmail.com

Reviewing editor:
Peter Christiaan Speldewinde, The University of Western Australia, Australia

Additional information is available at the end of the article

Abstract: This study assesses human health risk in 10 cities in Maharashtra, India, in terms of mortality and morbidity due to three critical pollutants (i.e. PM_{10}, SO_2, and NO_2). Risk of mortality/morbidity due to air pollution (Ri-MAP) model adopted in air quality health impact assessment (AirQ) software is used to evaluate the direct health impacts of various critical air pollutants in various cities in Maharashtra during the period 2004–2013. The result shows that excess number of mortality and morbidity in Nagpur, Thane, Aurangabad, Kolhapur, and Chandrapur is in increasing trend, while cities like Mumbai and Solapur are in decreasing trend, and other cities as Pune, Nashik, and Navi-Mumbai are in a steady-state condition. Cities having highest annual average excess number of total mortality, cardiovascular mortality, and respiratory motility in one million population are Mumbai (1,192, 724, and 121) (high population city), Chandrapur (944, 533, and 98) (low population city), Navi-Mumbai (797, 492, and 84), and Pune (733, 449, and 78) in decreasing order. Cities having highest annual average of hospital admission due to respiratory disease and cardiovascular disease among one million population are in decreasing order: Mumbai (1,519 and 582), Chandrapur (1,173 and 451), Navi-Mumbai (986 and 378), Pune (901 and 348), and Solapur (797 and 320).

Subjects: Environment & Health; Environment & the City; Environmental Change & Pollution

Keywords: air pollution; human health risk; relative risk; mortality; morbidity

1. Introduction

In epidemiology, "risk" is defined as a measure of the statistical likelihood of having severity of adverse events (e.g. illness or death) due to exposure to some factors (e.g. toxic chemical) (Lowrance, 1976). Health risk is the probability or chance of exposure to a hazardous substance, which makes humans sick and it is equal to multiplication of hazard and exposure. The human health risk (HHR)

ABOUT THE AUTHOR

The author joined the Indian Institute of Technology Bombay in December 2011 as a research scholar. His doctoral work is focused on "Air quality monitoring networking in an urban city and human health risk assessment due to air pollution". His research interests include the use of satellite remote sensing data for health risk assessment, atmospheric chemistry, low-cost indoor air pollution removal technology, and heavy metal contamination in deep sea fish.

PUBLIC INTEREST STATEMENT

The present research is very useful to the pollution control authorities and public, as air pollution level is increasing year by year and pollution is highly related to human death and illness. Metropolitan cities in Maharashtra like Mumbai, Chandrapur, Navi-Mumbai, and Pune have the highest impact of air pollution. Huge number of vehicles, high energy demand, open burning of solid waste, open coal mining, thermal power plant, road dust, and coal burning are the major sources of air pollution. There is a need to generate public awareness about air pollution in these cities.

assessment involves four major steps as follows (NRC, 1983): (1) Hazard identification—elements with known toxicity (like PM_{10}, SO_2, and NO_2 are responsible for different health effects like cardiovascular mortality (CM), respiratory mortality (RM), COPD, etc.). The knowledge about how hazardous a substance (pollutants) is comes from animal experiments or long time human studies. (2) Exposure assessment involves estimation of amount of hazardous pollutants inhaled by a certain population. Exposure information comes in two ways: (a) monitor substance concentration at different places in human community or (b) from dispersion models that account exposure based on released amount of hazardous substance from different sources. (3) Concentration–response assessment: it reflects the probability of health effects based on the dose of inhaled air pollutants. Finally, (4) HHR assessment is calculated by a mathematical model which based on the exposure and dose–response assessments.

Proper knowledge of exposure, baseline incidence of mortality or morbidity for every pollutant as well as concentration–response functions from epidemiological studies help account trends in perilous human health effects associated with alternative scenarios (EOHSP, 2007).

In India, about 0.62 million premature excess number of death cases occur due to outdoor air pollution and became the fifth leading cause of death after high blood pressure, indoor air pollution, tobacco smoking, and poor nutrition in 2012 (NYT, 2014). The economic cost of health impacts due to air pollution is about USD 80 billion in 2010, equivalent to 5.7% of gross domestic product (GDP) in India. Serious health consequences due to PM_{10} coming from fossil fuels burning amount to about 3% (1.7% by outdoor air pollution and 1.3% by indoor air pollution) of India's GDP. The huge amount of health cost for outdoor/indoor is pollution due to fine particulate matter are primarily driven by an elevated exposure of skillful young population in urban area that results a substantial cardiopulmonary and RM and mobility load among adults (WB, 2013).

Due to high population, high energy demand, large number of industries, and huge number of vehicles, cities are facing most environmental challenges in air pollution. Thus, cities tend to be high risk areas and their human community vulnerable to air pollution-induced unfavorable health impacts like CM, RM, chronic obstructive pulmonary disease (COPD), etc. (Butler et al., 2008; Gurjar, Butler, Lawrence, & Lelieveld, 2008; Madronich, 2006; Molina & Molina, 2004). Such risk needs to be calculated to help the pollution control authorities to ameliorate the sustainability of city life. Modeling of human exposure by air pollutants is most important for the evaluation of HHR (Vostal, 1994).

The case study in this paper focuses on quantitative assessment of HHR, like total mortality (TM), CM, RM, COPD, hospital admissions respiratory disease (HARD), and hospital admissions cardiovascular disease (HACD), due to three critical pollutants (PM_{10}, SO_2, and NO_2) in 10 urban areas (cities) (Mumbai, Pune, Nagpur, Thane, Nashik, Aurangabad, Solapur, Navi-Mumbai, Kolhapur, and Chandrapur) in Maharashtra in India from 2004 to 2013.

Here, the risk of mortality/morbidity due to air pollution (Ri-MAP) model adopted in air quality health impact assessment (AirQ) software has been used to assess direct HHR due to three criteria air pollutants present in urban areas (cities).

2. Methodology

The relative risk (R_r) is the probability an exposed group will develop disease relative to the probability of an unexposed group developing the same disease due to air pollutants (Rothman, Greenland, & Lash, 2008). The approach adopted in the present research is HHR assessment, using the AirQ 2.2.3 software (WHO, 2004) developed by the WHO European Centre for Environment Health, Bilthoven Division. This software adopted Ri-MAP model which is used in the present study to estimate the potential impact of exposure to particular air pollutants on the health of people living in an urban area during a certain time period.

The HHR assessment is based on the population attributable risk (PAR) (also called "population attributable risk proportion") concept, defined as the fraction of the excess rate of disease in a given population distinguishable to exposure to a particular atmospheric pollutant, assuming a proven causal relation between exposure and excess rate of disease with no major confounding effects in that association (Gefeller, 1992; Northridge, 1995). The PAR can be easily calculated by the following general equation:

$$\mathrm{PAR} = \sum [\{R_r(c) - 1\}] \times p(c) / \sum [\{R_r(c) - 1\} \times p(c) + 1] \quad (1)$$

where $R_r(c)$ is the changed relative risk for the health outcome in category "c" of exposure and $R_r(c) = 1 + (C_a - C_w) \times (R_r - 1)/10$. C_a is the ambient air pollutant concentration, C_w is the WHO recommended threshold level of that pollutant, and R_r is the relative risk of exposure–disease relation (the ratio of the conditional disease probabilities among exposed and non-exposed). $p(c)$ is the proportion of the population in category "c" of exposure.

If the baseline frequency (at WHO recommended threshold concentration value) of selected health outcomes (i.e. I_w) in the population under investigation is known, then the excess number of cases (ENCs) per unit population (rate) attributed to the exposure in population (i.e. I_E) is calculated as (WHO, 1999):

$$I_E = I_w \times \mathrm{PAR} \quad (2)$$

Consequently, the frequency of the outcome to the non-exposed population (i.e. I_{NE}) can be calculated as follows:

$$I_{NE} = I_w - I_E = I_w \times (1 - \mathrm{PAR}) \quad (3)$$

Finally, at a certain category of exposure (c) with known R_r and the estimated incidence in non-exposed population having population size under investigation (P), the ENCs ($\Delta N(c)$) can be calculated:

$$\Delta N(c) = [R_r(c) - 1] \times p(c) \times I_{NE} \times P \quad (4)$$

Equation (4) is used to estimate ENCs of mortality or morbidity in the exposed population. In practice, however, the range of the estimated health outcome (i.e. uncertainty of the impact) is greater due to measurement errors in exposure assessment because pollutant concentration changes time to time and it depends on the area (e.g. industrial or residential) and non-statistical uncertainty of the concentration–response function (WHO 1999, 2003).

The AirQ software uses WHO specified input of R_r values (per 10 μg/m^3 increase of concentration for hazardous substances) and corresponding baseline incidences (per 10^5 population) for different air pollutants (particulate matter having aerodynamic diameter ≤ 10 (PM_{10}), sulfur dioxide (SO_2), and nitrogen dioxide (NO_2), etc.) as well as types of diseases (e.g. cardiovascular, respiratory, COPD, and HARD) associated with those values (Table 1), based on various previous studies (Burnett, Dales, Brook, Raizenne, & Krewski, 1997; Poloniecki, Atkinson, de Leon, & Anderson, 1997; Sunyer et al., 1997; Touloumi & Katsouyanni, 1997).

The simple method using all air quality monitoring station data in the city and taking average of the concentrations for each time unit is followed to evaluate human exposure. This average value (daily) is the indicator of exposure of the entire city population. For example, for Chandrapur, daily data of six monitoring stations located throughout the city are taken and the average of three critical pollutants (i.e. PM_{10}, SO_2, and NO_2) is used for each year.

Table 1. WHO specified default values of relative risk (RR) (per 10 μg/m³ increase of daily averages for PM_{10}, SO_2, and NO_2) with 95% confidence intervals (CI)

Pollutants	Mortality/Morbidity	Relative risk (RR)[b] (95% CI) per 10 μg/m³	Baseline incidence per 100,000[c]
PM_{10}	Total mortality[x]	1.0074 (1.0062–1.0086)	1,013
	Cardiovascular mortality[y]	1.008 (1.005–1.018)	497
	Respiratory mortality[z]	1.012 (1.008–1.037)	66
	Hospital admission respiratory disease	1.008 (1.0048–1.0112)	1,260
	Hospital admission cardiovascular disease	1.009 (1.006–1.013)	436
SO_2	Total mortality[x]	1.004 (1.003–1.0048)	1,013
	Cardiovascular mortality[y]	1.008 (1.002–1.012)	497
	Respiratory mortality[z]	1.01 (1.006–1.014)	66
	COPD[a] morbidity (hospital admission)	1.0044 (1–1.011)	101.4
NO_2	Cardiovascular mortality[y]	1.002 (1–1.004)	497
	COPD[a] morbidity (hospital admission)	1.0038 (1.004–1.0094)	101.4

[a]COPD: Chronic Obstructive Pulmonary Disease.

[b]Lower and upper limits (range) of RR values.

[c]Baseline Incidence per 100,000 is based on threshold limit given in WHO guideline.

[x]International Classification of Diseases (ICD) code number: ICD-9-CM < 800.

[y]ICD-9-CM 390–459.

[z]ICD-9-CM 460–519.

3. Study area

Maharashtra is a state in the western region and is the second-most populous region in India with a population of 112 million (9.28% of India's population) and its capital Mumbai is the highest populated city in India and its population is approximately 12.48 million (CI, 2011). Maharashtra is one of the most developed states in India, contributing 25% of the country's industrial output and 23.2% of its GDP (2010–2011) (DDCH, 2014). The state's economy mainly depends on agriculture and industries. Major industries are included in chemical products, electrical and non-electrical machinery, pharmaceuticals, textiles, petroleum, and allied products. The huge amount of population makes Maharashtra the largest energy user but conservation mandates, mild weather, and strong environmental movements make its per capita energy use lowest of any Indian state (EGI, 2014). The high electricity demand of the state constitutes about 13% of the total electricity generated in India, which mainly comes from fossil fuels such as coal and natural gas-based power plants (IndianPowerSector.com, 2015).

The case study in this paper focuses on quantitative assessment of HHR based on Ri-MAP model in 10 cities in Maharashtra from 2004 to 2013.

The Ri-MAP model depends mainly upon ambient air pollution concentrations and population data. The daily average ambient air pollution concentrations (μg/m³) for criteria pollutants, namely; PM_{10}, SO_2, and NO_2 from all monitoring stations in 10 cities from 2004 to 2013, used in this study were monitored and estimated by Maharashtra Pollution Control Board (MPCB) (MPCB, 2014). Population data of all cities in 2001 and 2011 are taken from Census of India (CI, 2001) to calculate the exponential growth factor for all cities. Population growth is estimated by $P = P_0 \exp(kt)$, where P, P_0, t, and k denote final population, initial population, time (year), and exponential growth factor,

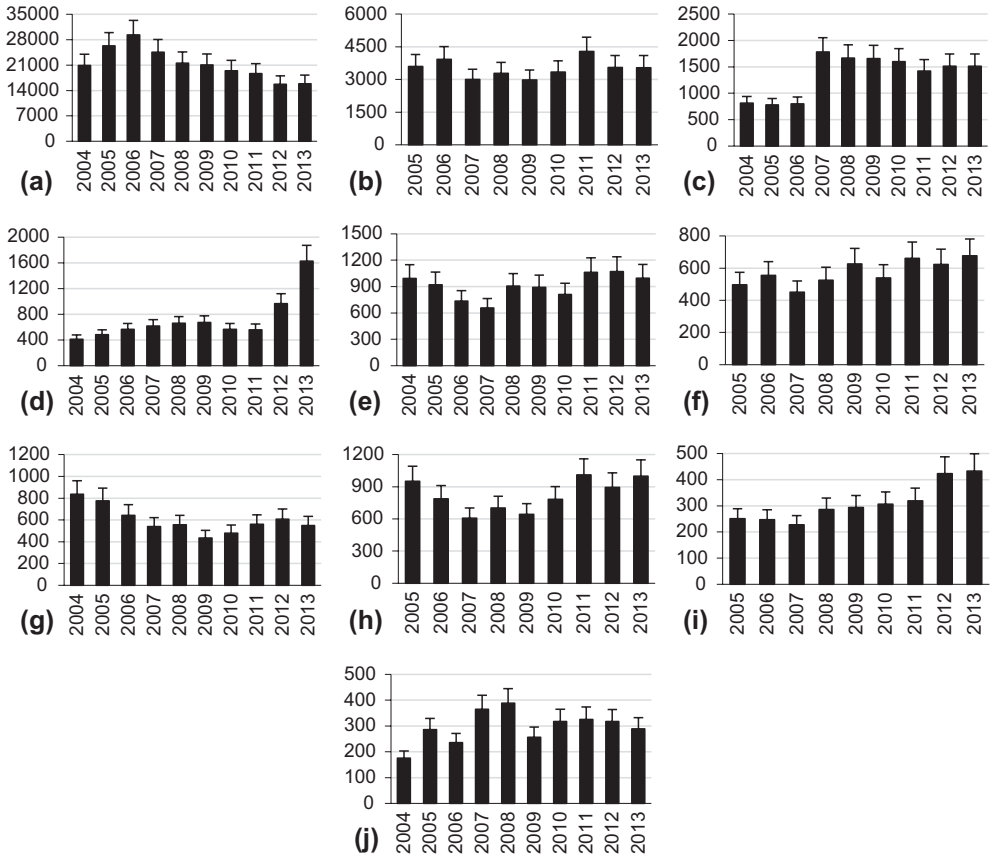

Figure 1. ENCs of TM in Maharashtra (a) Mumbai, (b) Pune, (c) Nagpur, (d) Thane, (e) Nashik, (f) Aurangabad, (g) Solapur, (h) Navi-Mumbai, (i) Kolhapur, and (j) Chandrapur.

respectively. The highest populated city is Mumbai followed by Pune, Nagpur, Thane, Nashik, Aurangabad, Solapur, Navi-Mumbai, Kolhapur, and Chandrapur in decreasing order.

4. Results

The model used the WHO default value for relative risk for three critical pollutants (i.e. PM_{10}, SO_2, and NO_2) and the results obtained from the case study, using Ri-MAP model incorporating exposure response Equations (1)–(4). City-wise ENCs of mortality/morbidity of TM, CM, RM, COPD, HARD, and HACD have been illustrated from Figures 1–6.

4.1. Mumbai

Figures 1(a), 2(a), 3(a), 4(a), 5(a), and 6(a) represent the trend of the ENCs of TM, CM, RM, COPD (hospital admission), HARD, and HACD in the megacity Mumbai. In 2004, ENC of TM was 20,955 (17,689–24,028 at 95% CI) and then 15,872 (13,424–18,235 at 95% CI) in 2013. In the case of CM, ENC in 2004 was 13,139 (7,023–24,894 at 95% CI) and 9,962 (5,355–19,881 at 95% CI) in 2013. RM in Mumbai city in 2004 was 2,187 (1,514–4,724 at 95% CI), then it became 1,628 (1,125–3,901 at 95% CI) in 2013. Excess number of COPD, HARD, and HACD was 699 (53–1,677 at 95% CI), 25,430 (16,018–33,988 at 95% CI), and 9,755 (6,801–13,312 at 95% CI) in 2004 and 580 (57–1,384 at 95% CI), 20,527 (12,757–27,777 at 95% CI), and 7,905 (5,445–10,950 at 95% CI) in 2013, respectively. ENCs in all types of mortality and morbidity were maximum in 2006 but after that there was a decreasing trend. The observed ENCs were highest in the year 2006 for all mortality and morbidity because of higher PM_{10} concentration. PM_{10} was mostly responsible for the ENCs of TM (91.1–97.4%), CM (76.3–86%), RM (86.3–95.9%), and 100% responsible for HARD and HACD. Evidence of ENCs due to NO_2 of CM (7.8–13.7%) and hospital admission of COPD (70.5–90.4%) had been observed from years 2004–2013 in Mumbai city. There was minimum evidence observed for TM (2.6–8.9%), CM (4.2–13.8%), RM (4.1–13.7%), and COPD (9.6–29.5) due to SO_2 pollution.

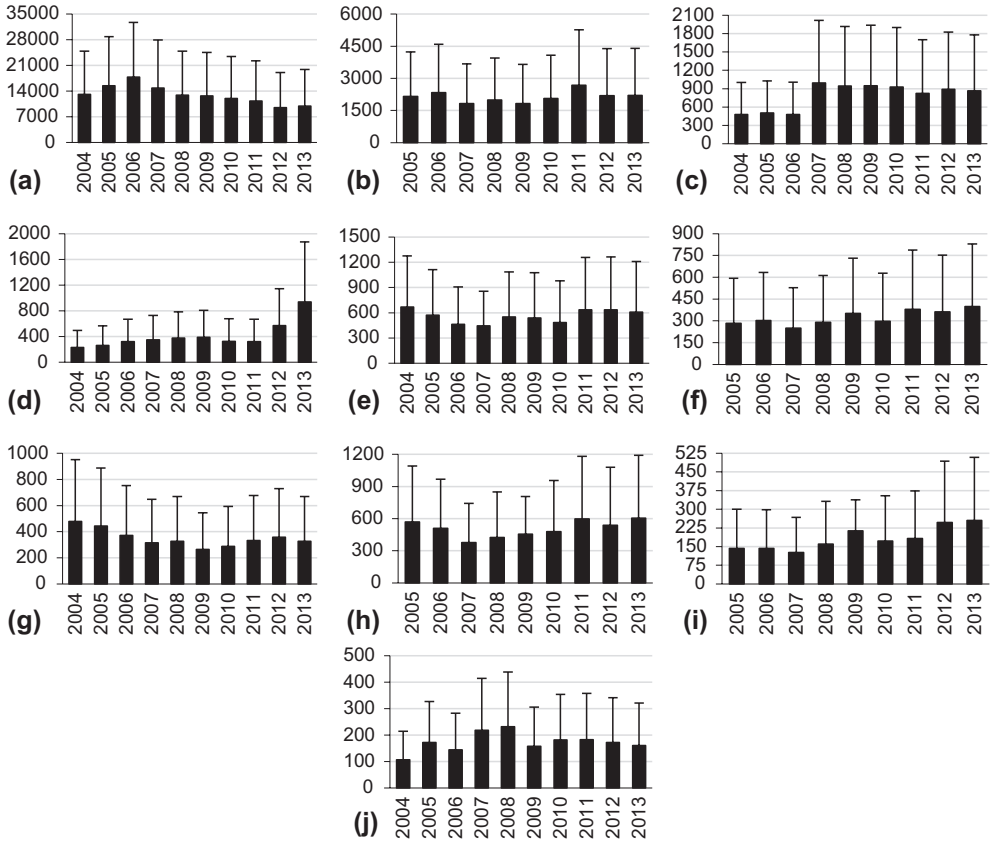

Figure 2. ENCs of CM in Maharashtra (a) Mumbai, (b) Pune, (c) Nagpur, (d) Thane, (e) Nashik, (f) Aurangabad, (g) Solapur, (h) Navi-Mumbai, (i) Kolhapur, and (j) Chandrapur.

4.2. Pune

Trend of ENCs of mortality and morbidity in Pune city from 2005 to 2013 is shown in Figures 1(b) (TM), 2(b) (CM), 3(b) (RM), 4(b) (COPD), 5(b) (HARD), and 6(b) (HACD). There was no specific change of trend on mortality and morbidity observed except some dips. Annual average ENCs of TM, CM, RM, COPD, HARD, and HACD were 3,502 (2,939–4,040 at 95% CI), 2,143 (1,161–4,250 at 95% CI), 372 (253–890 at 95% CI), 97 (7–235 at 95% CI), 4,303 (2,659–5,853 at 95% CL), and 1,660 (1,138–2,314 at 95% CI), respectively, during the study period. Responsibility of PM_{10} pollution for ENCs of TM, CM, and RM was 89.7–93.5, 76.5–82.5, and 84.5–90%, respectively, in Pune city. NO_2 was responsible for 6–10 and 60–73% of ENCs of CM and COPD. Contribution of SO_2 was observed for ENCs of TM (6.5–10.3%), CM (10.5–16.3%), RM (10–15.5%), and hospital admission of COPD (27–40%).

4.3. Nagpur

Except COPD, the ENCs of mortality and morbidity had not significant change from 2004 to 2006, but in 2007, it increased suddenly and then a small decreasing trend was found in Nagpur city (Figures 1(c), 2(c), 3(c), 4(c), 5(c), and 6(c)). About 108–127% growth was observed from 2006 to 2007 in ENCs of TM (Figure 1(c)), CM (Figure 2(c)), RM (Figure 3(c)), HARD (Figure 5(c)), and HACD (Figure 6(c)) due to increase in annual average concentration of PM_{10} from 58 to 112 μg/m^3. In 2004 the ENCs of TM were 812 (681–939 at 95% CI), 1,785 (1,512–2,051 at 95% CI) in 2007 and became 1,514 (1,276–1,745 at 95% CI) in 2013. In case of CM, in 2004, the ENCs were 478 (269–1,003 at 95% CI) followed by 996 (603–2,017 at 95% CI) in 2007, and 864 (508–1,779 at 95% CI) in 2013. In RM, the ENC in 2004 was 86 (58–231 at 95% CI), then in 2013, it became 157 (107–401 at 95% CI) with annual average 141 (96–357 at 95% CI). Annual average ENC of hospital admission COPD was 24 (2–60 at 95% CI). Excess number of HARD and HACD was 1,048 (638–1,446 at 95% CI) and 406 (275–576 at 95% CI) in 2004, then they became 1,974 (1,214–2,698 at 95% CI) and 763 (520–1,096 at 95% CI) in 2013, respectively. The contribution of PM_{10} for the ENCs of TM, CM, and RM was 90.2–98, 73.7–92.6, and

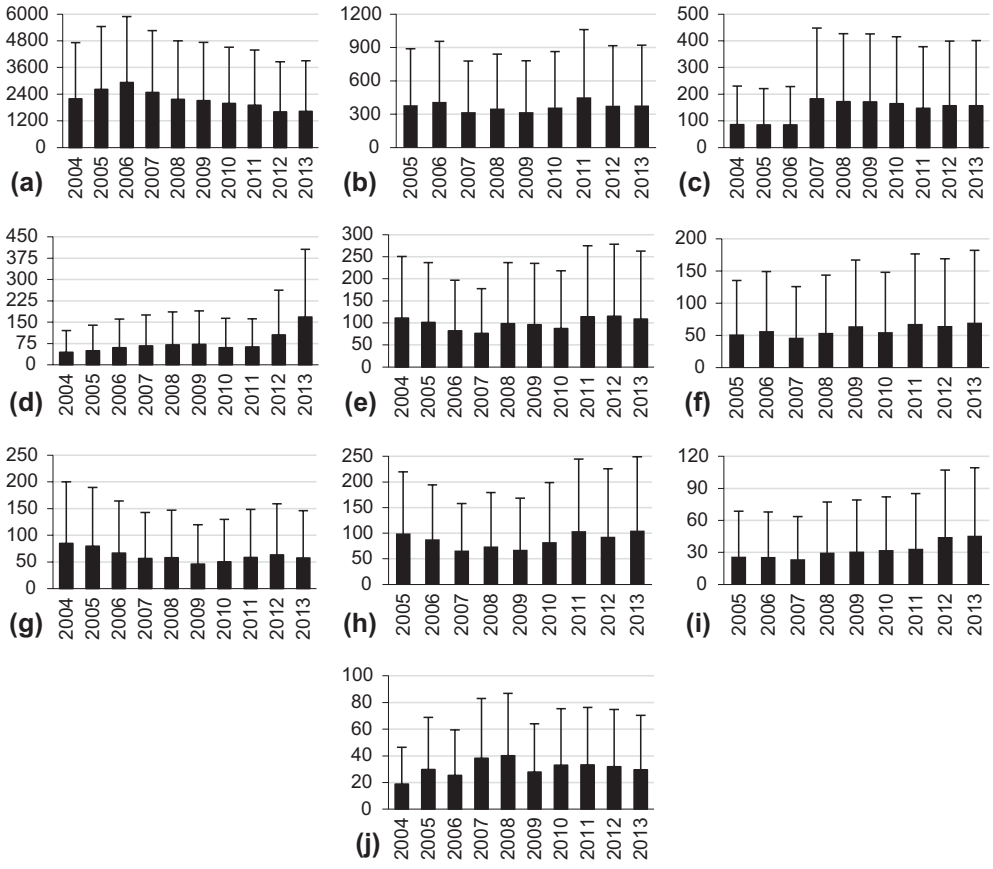

Figure 3. ENCs of RM in Maharashtra (a) Mumbai, (b) Pune, (c) Nagpur, (d) Thane, (e) Nashik, (f) Aurangabad, (g) Solapur, (h) Navi-Mumbai, (i) Kolhapur, and (j) Chandrapur.

85.4–98.8%, respectively. Moreover, the accountability of NO_2 was 3.9–11.5 and 72.7–87.5% for ENCs of CM and hospital admission of COPD. ENCs of TM, CM, RM, and COPD due to SO_2 were 2–9.8, 3.5–14.8, 3.2–14.6, and 12.5–27.3%, respectively, in Nagpur city during the study period.

4.4. Thane

Trend of ENCs of mortality and morbidity in Thane increased from 2004 to 2013 except a few dips (Figures 1(d), 2(d), 3(d), 4(d), 5(d), and 6(d)). In 2004, the ENC of TM was 413 (347–478 at 95% CL) and became 1,627 (1,372–1,872 at 95% CL) in 2013 (Figure 1(d)). Accountability of CM was 231 (138–493 at 95% CL) in 2004, but in 2013, it became 938 (546–1,874 at 95% CL). In RM, ENC in 2004 was 44 (29–121 at 95% CL), then in 2013, it was 169 (116–406 at 95% CL). Observed ENCs of COPD, HARD, and HACD were 4 (0–10 at 95% CL), 540 (328–748 at 95% CL), and 210 (141–299 at 95% CL) in 2004 and 26 (2–64 at 95% CL), 2,073 (1,285–2,811 at 95% CL), and 799 (549–1,110 at 95% CL) in 2013, respectively. Accountability of PM_{10} pollution for ENCs of TM, CM, and RM was 86.7–100, 79–97, and 83–100%, respectively, in Thane from 2004 to 2013. Responsibility of NO_2 was 1.8–4.9% for ENCs of CM. The contribution of SO_2 was observed for ENCs of TM (0–13.3%), CM (0–19.3%), and RM (0–17%).

4.5. Nashik

Figures 1(e), 2(e), 3(e), 4(e), 5(e), and 6(e) illustrate the trend of the ENCs of TM, CM, RM, COPD, HARD, and HACD in Nashik from 2004 to 2013. There was no significant change of trend except some dips in 2006, 2007, and 2010. Annual average ENCs of TM, CM, RM, COPD, HARD, and HACD were 904 (754–1,047 at 95% CI), 559 (295–1,102 at 95% CI), 99 (66–237 at 95% CI), 25 (1–61 at 95% CI), 1,063 (651–1,622 at 95% CL), and 409 (279–575 at 95% CI), respectively, during the study period. PM_{10} was responsible for 77.4–91.2, 60.3–81.1, and 68.9–86.8% of ENCs of TM, CM, and RM, respectively. For

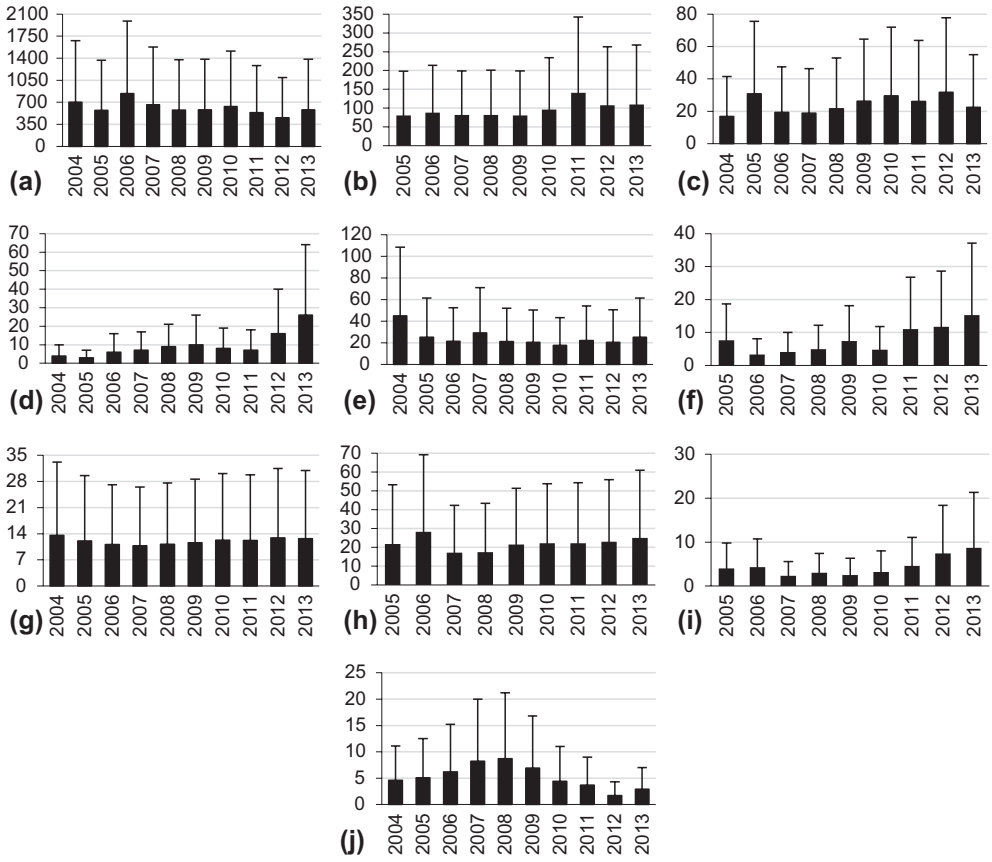

Figure 4. ENCs of COPD (hospital admissions) in Maharashtra (a) Mumbai, (b) Pune, (c) Nagpur, (d) Thane, (e) Nashik, (f) Aurangabad, (g) Solapur, (h) Navi-Mumbai, (i) Kolhapur, and (j) Chandrapur.

SO_2, the figures were 8.8–22.6, 14.4–32.2, and 13.2–31.1% for ENCs of TM, CM, and RM, respectively, and very less evidence had been obtained for NO_2.

4.6. Aurangabad

From 2005 to 2013, the ENCs of mortality and morbidity were increasing in trend with some dips in Aurangabad (Figures 1(f), 2(f), 3(f), 4(f), 5(f), and 6(f)). In 2005, the ENC of TM was 497 (419–574 at 95% CI) and it became 677 (571–781 at 95% CI) in 2013 (Figure 1(f)). In case of CM, ENC in 2005 was 284 (167–593 at 95% CI) and in 2013, it was 399 (227–829 at 95% CI) (Figure 2(f)). About 52 (35–135 at 95% CI) number of cases of RM were observed in 2005, but in 2013, it was 70 (48–182 at 95% CI) (Figure 3(f)). During the study period, ENCs of COPD were very low: 8 (1–19 at 95% CI) in 2005 and 15 (2–37 at 95% CI) in 2013 (Figure 4(f)). HARD and HACD were 654 (401–898 at 95% CI) and 253 (172–356 at 95% CI) in 2005, but they became 893 (548–1,223 at 95% CI) and 345 (235–485 at 95% CI) in 2013 (Figures 5(f) and 6(f)). PM_{10} was responsible for most of the ENCs of TM, CM, and RM and it contributed 98.3–100, 88.2–97.2, and 97.3–100%, respectively. NO_2 contributed almost 2.8–9.1% in CM but the contribution of SO_2 in ENCs of mortality and morbidity was very low; but after 2010, that increased about 3%.

4.7. Solapur

Figures 1(g), 2(g), 3(g), 4(g), 5(g), and 6(g) illustrate the decreasing trend of ENCs of TM, CM, RM, COPD, HARD, and HACD in Solapur city from 2004 to 2013. In 2004, the ENC of TM was 837 (710–960 at 95% CI) and it became 549 (462–634 at 95% CI) in 2013. Accountability of CM was 479 (284–952 at 95% CI) in 2004, then in 2013, it was 327 (183–669 at 95% CI). About 85 (59–200 at 95% CI) number of cases of RM were found in 2004, then in 2013, it was 58 (39–146 at 95% CI). The ENC of COPD was very low and a steady-state trend followed throughout the study period and annual average was 12 (1–29 at 95% CI). In case of HARD and HACD, ENCs were 1,093 (682–1,473 at 95% CI) and 420

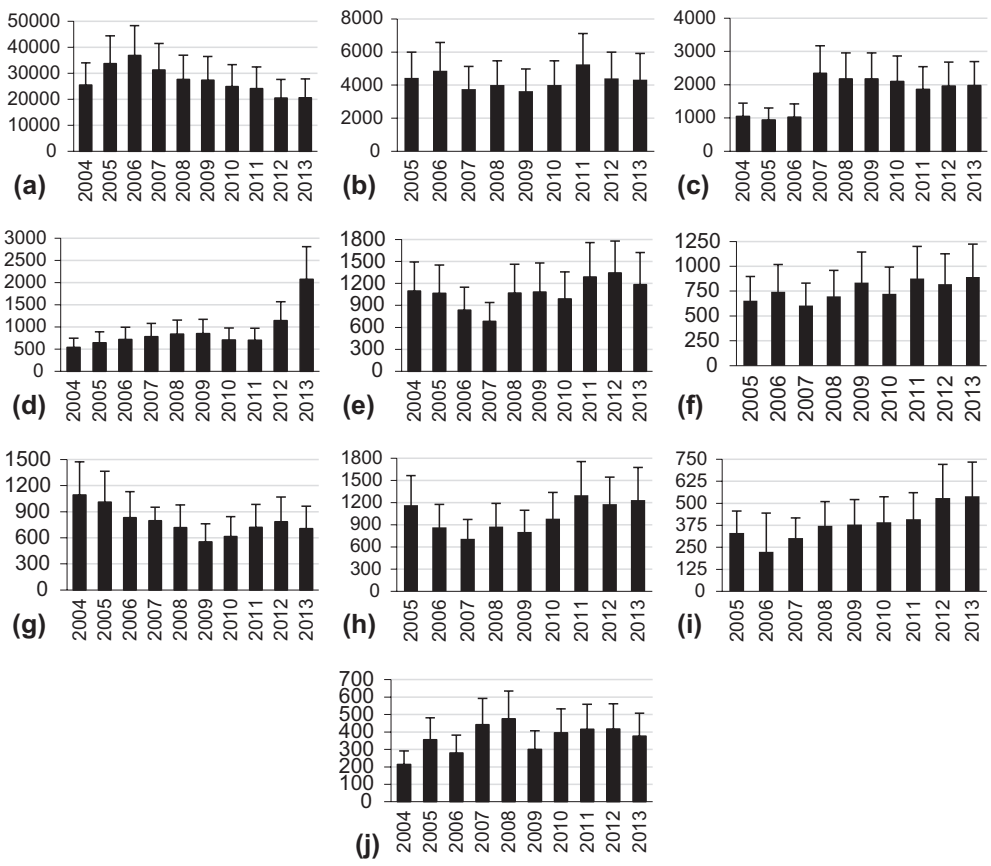

Figure 5. ENCs of HARD in Maharashtra (a) Mumbai, (b) Pune, (c) Nagpur, (d) Thane, (e) Nashik, (f) Aurangabad, (g) Solapur, (h) Navi-Mumbai, (i) Kolhapur, and (j) Chandrapur.

(291–580 at 95% CI) in 2005, then they became 705 (433–965 at 95% CI) and 272 (186–383 at 95% CI) in 2013. PM_{10} was majorly responsible for most of ENCs of TM, CM, and RM and it contributed 94.9–97.8, 82.8–90, and 92.2–96.6%, although NO_2 was responsible for 5.7–9.2 and 78.4–85.3% of CM and COPD. There was contribution of SO_2 for ENCs of TM (2.2–5.1%), CM (3.7–8.2%), RM (3.4–7.8%), and COPD (14.7–21.6).

4.8. Navi-Mumbai

Trend of ENCs of mortality and morbidity in Navi Mumbai decreased from 2005 to 2009; after that it increased further (Figures 1(h), 2(h), 3(h), 4(h), 5(h), and 6(h)). In 2005, ENC of TM was 952 (803–1,093 at 95% CI) followed by 642 (539–741 at 95% CI) in 2009, then it became 999 (843–1,151 at 95% CI) in 2013 (Figure 1(h)). In case of CM, in 2005, the ENC was 570 (319–1,092 at 95% CI) followed by 454 (213–807 at 95% CI) in 2009 and 605 (332–1,192 at 95% CI) in 2013 (Figure 2(h)). In RM, the ENC in 2005 was 99 (69–220 at 95% CI), then in 2013, it became 105 (72–249 at 95% CI) with annual average 87 (59–204 at 95% CI) during the study period (Figure 3(h)). Annual average ENC of hospital admission COPD was very low: 22 (2–54 at 95% CI) (Figure 4(h)). In the case of HARD, ENC was 1,168 (733–1,566 at 95% CI) in 2005, 804 (495–1,098 at 95% CI) in 2009, and 1,236 (766–1,677 at 95% CI) in 2013 (Figure 5(h)). ENC of HACD in 2005 was 448 (312–615 at 95% CI), in 2009, it was 311 (212–435 at 95% CI) followed by 476 (327–662 at 95% CI) in 2013 (Figure 6(h)). Accountability of PM_{10} pollution for ENCs of TM, CM, and RM was 82.4–96.3, 67–86, and 75–94%, respectively, in Navi-Mumbai from 2005 to 2013. Accountability of NO_2 was 6.1–9 and 46–81% for ENCs of CM and COPD. There was contribution of SO_2 for ENCs of TM (3.7–17.6%), CM (6.2–26%), and RM (6–25%).

4.9. Kolhapur

Figures 1(i), 2(i), 3(i), 4(i), 5(i), and 6(i) represent the smooth increasing trend of the ENCs of TM, CM, RM, COPD, HARD, and HACD in Kolhapur city from 2004 to 2013. In the case of TM, in 2005, the ENC

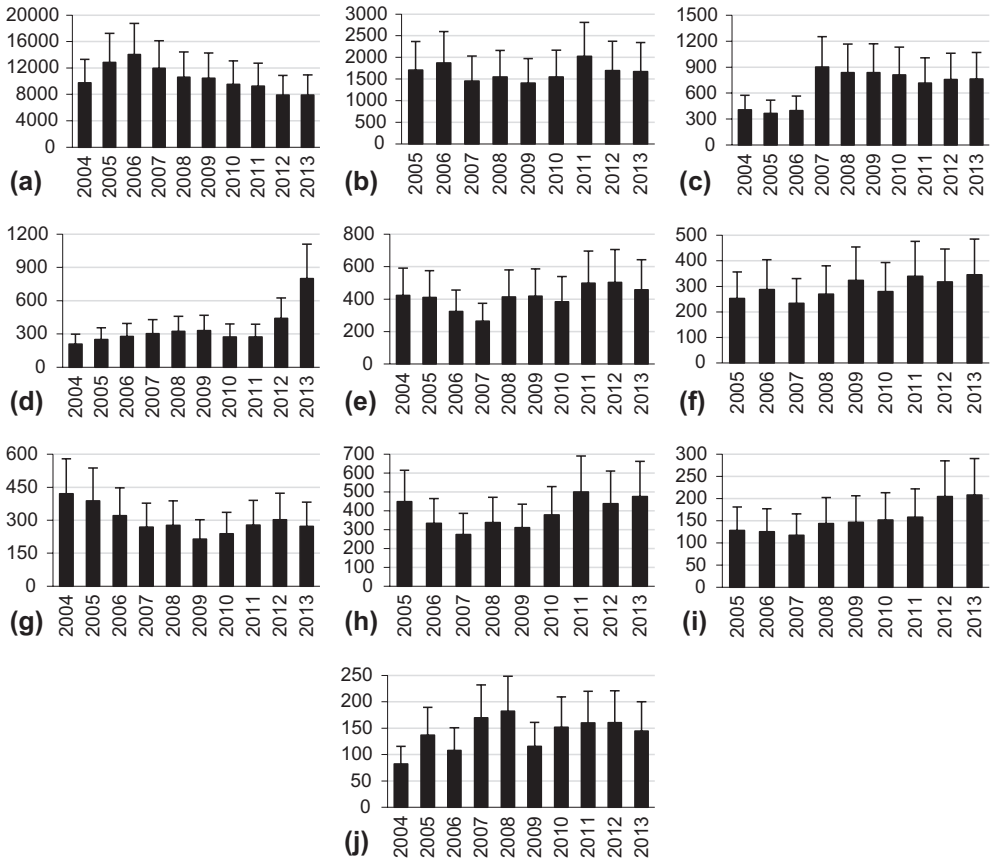

Figure 6. ENCs of HACD in Maharashtra (a) Mumbai, (b) Pune, (c) Nagpur, (d) Thane, (e) Nashik, (f) Aurangabad, (g) Solapur, (h) Navi-Mumbai, (i) Kolhapur, and (j) Chandrapur.

was 251 (211–289 at 95% CI) and it became 433 (364–499 at 95% CI) in 2013. In 2005, ENC of CM was 143 (84–301 at 95% CI), then in 2013, it became 244 (144–509 at 95% CI). In the case of RM and COPD, ENC was very low, 26 (18–69 at 95% CI) and 4 (0–10 at 95% CI), in 2005, then it became 46 (31–109 at 95% CI) and 9 (1–21 at 95% CI) in 2013. In HARD, ENC in 2005 was 332 (203–456 at 95% CI) then in 2003 it was 540 (334–734 at 95% CI). ENC of HACD in 2005 was 129 (87–181 at 95% CI) and it became 208 (143–290 at 95% CI) in 2013. The contribution of PM_{10} for the ENCs of TM, CM, and RM was 93.3–99.2, 83.4–94.4, and 89.7–98.7%, respectively. Moreover, the accountability of NO_2 was 1.7–6.8 and 56–92.5% for ENCs of CM and hospital admission of COPD. ENCs of TM, CM, RM, and COPD due to SO_2 were 0.8–6.7, 1.4–11, 1.3–10.3, and 7.5–44%, respectively, during the study period.

4.10. Chandrapur

From 2004 to 2008, the ENCs of mortality and morbidity were increasing in trend, but after that, there was decrease in trend in Chandrapur (Figures 1(j), 2(j), 3(j), 4(j), 5(j), and 6(j)). In 2004, the ENC of TM was 175 (147–203 at 95% CI), 388 (328–444 at 95% CI) in 2008, and became 289 (245–332 at 95% CI) in 2013 (Figure 1(j)). In the case of CM, in 2004, the ENC was 107 (58–214 at 95% CI), followed by 232 (130–439 at 95% CI) in 2008 and 161 (98–321 at 95% CI) in 2013 (Figure 2(j)). In RM, the ENC in 2004 was 19 (13–47 at 95% CI), then in 2013, it became 30 (21–70 at 95% CI) with an annual average 31 (21–70 at 95% CI) (Figure 3(j)). Annual average ENCs of hospital admission COPD were very low: 3 (0–13 at 95% CI) (Figure 4(j)). Excess number of HARD and HACD was 213 (131–292 at 95% CI) and 82 (56–116 at 95% CI) in 2004, but they became 376 (234–508 at 95% CI) and 145 (100–200 at 95% CI) in 2013, respectively. The highest ENCs were observed for all types of mortality and morbidity in the year 2008 because of higher concentration of PM_{10}. Responsibility of PM_{10} pollution for ENCs of TM, CM, and RM was 87.7–98.6, 75–95.6, and 81.7–97.8%, respectively, in Chandrapur. NO_2 was responsible for 1.9–6.6 and 47.7–70.6% of ENCs of CM and COPD. SO_2 contributed for ENCs of TM (1.4–12.3%), CM (2.5–19.4%), RM (2.2–18.3%), and hospital admission of COPD (29.4–52.3%).

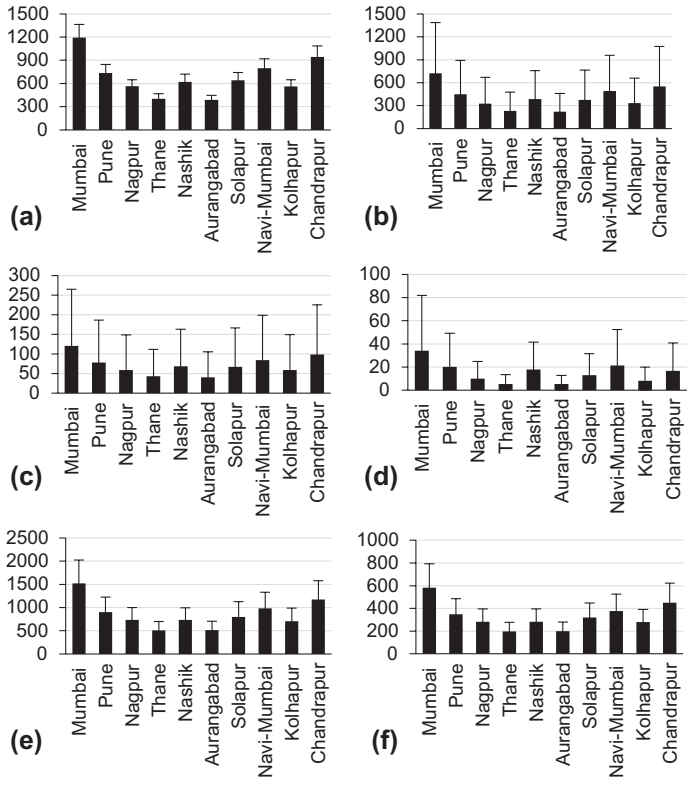

Figure 7. Annual average ENCs in one million of population of (a) TM, (b) CM, (c) RM, (d) COPD (hospital admission), (e) HARD, and (f) HACD in different urban cities in Maharashtra.

5. Discussion

In the above results, the annual average ENCs of mortality and morbidity are almost proportionate to the total population in the city; but in this case study, any area-specific ambient air pollution concentration is not considered to calculate HHR. To avoid biases generated due to different population sizes, different population densities and different areas (i.e. residential or industrial) in different cities, and ENCs in one million population (ENCsOMP) have been estimated (Figure 7). All figures show the range of the annual average ENCsOMP at 95% CI.

5.1. Excess number of mortality/morbidity among one million population

5.1.1. Total mortality

The excess number of mortality (death) has been estimated taking into account the sum total of effects caused by PM_{10} and SO_2. City-wise different trends are observed for the excess number of mortality cases considered among one million population in all cities (Figure 7(a)). Annual average ENCsOMP of TM is highest in Mumbai (1192/yr) while Chandrapur (944/year) is in the second position. On the other hand, Navi-Mumbai (797/year), Pune (733/year), Solapur (643/year), Nashik (622/year) followed by Nagpur (564/year), Kolhapur (562/year), Thane (404/year), and Aurangabad (388/year) are in decreasing order.

5.1.2. Cardiovascular mortality

Annual average ENCsOMP of CM follow the same trend too (Figure 7(b)); it is highest in Mumbai (724/year) followed by Chandrapur (533/year), Navi-Mumbai (492/year), and Pune (449/year). On the other hand, Nashik (385/year), Solapur (376/year), Kolhapur (332/year), Nagpur (327/year), Thane (231/year), and Aurangabad (219/year) are in decreasing order. The excess number of CM is owing to the total effect of PM_{10}, SO_2, and NO_2.

5.1.3. Respiratory mortality

Annual average excess number of RM in one million population is relatively low (Figure 7(c)). Figure shows that annual average ENCsOMP of RM was highest in Mumbai (121/year). Subsequent to Mumbai, top five cities in this list are Chandrapur (98/year), Navi-Mumbai (84/year), Pune (78/year), Nashik (68/year), and Solapur (67/year). The excess number of RM has been calculated taking into account the sum total of PM_{10}, SO_2, and NO_2.

5.1.4. Chronic obstructive pulmonary disease

Trend of annual average ENCsOMP was different from others and was very low because SO_2 and NO_2 were only responsible for COPD (Figure 7(d)). Top five cites with ENCsOMP are Mumbai (34/year), Navi-Mumbai (21/year), Pune (20/year), Nasik (18/year), and Chandrapur (17/year) in decreasing order. Suspended particulate matter (SPM) is primarily responsible for COPD but is not included in this study; so the values are less in all cities.

5.1.5. Hospital admission due to respiratory disease and cardiovascular disease

HARD and HACD are only responsible by PM_{10}. Cities with highest ENCsOMP of HARD (Figure 7(e)) and HACD (Figure 7(f)) are Mumbai (1,519 and 582), Chandrapur (1,173 and 451), Navi-Mumbai (986 and 378), Pune (901 and 348), and Solapur (797 and 320) in decreasing order. ENCsOMP of HARD in other cities are in the range of 510–732 and for HACD in between 197 and 283.

Other case studies were carried out in different cities by different researchers on human health due to atmospheric pollution. AirQ software has been popular to many researchers to assess the human health impact of $PM_{2.5}$ (Boldo et al., 2006) or PM_{10} (Tominz, Mazzoleni, & Daris, 2005). Naddafi et al. (2012) calculated the excess number of mortality under all causes, cardiovascular and respiratory diseases due to PM_{10}, SO_2, NO_2, and O_3 in Tehran, Iran. Study on HHR due to air pollution in two industrial municipalities of Northern Italy was done by Fattore et al. (2011). The study showed that 433, 180, and 72 lives were lost per year for all causes, cardiopulmonary diseases and lung cancer, respectively. An assessment study on other human health impacts was carried out by Gharehchahi et al. (2013) in Shairaz, Iran; Orru, Laukaitienė, and Zurlytė (2012) in Vilnius and Kaunas; Orru et al. (2009) in Tallinn using fine spatial resolution with GIS. Nagpure, Gurjar, and Martel (2014) evaluated HHR in NCT Delhi for the years 1991–2010 and Gurjar et al. (2010) estimated the HHR due to air pollution in different megacities in the world. Results obtained from the studies of health effects due to air pollution in various cities in the world differ substantially, but in all of these as well as our present study, particulate matter has been found to cause the most adverse health effects.

5.2. Cause of air pollution

From the above study, it is observed that excess number of mortality and morbidity is due to particulate matter (PM_{10}) than gaseous pollutants. Mumbai and Chandrapur have the highest rate of ENCs of mortality and morbidity. In Mumbai, different combustion processes are the main contributors for PM, like power plant, open burning, commercial food sector, and road transport, and they contribute 37, 24, 18, and 10%, respectively. A study by National Environmental Engineering Research Institute (NEERI) found that open burning and landfill fires of municipal solid waste (MSW) were a major source of air pollution in Mumbai (CPCB, 2010). The survey results show that about 2% of total generated MSW is burnt on the streets and slum areas, 10% of the total generated MSW is burnt in landfills by management authorities or due to accidental landfill fires, thereby emitting large amounts of CO, PM, carcinogenic HC, and NO_x. In Chandrapur, primary sources of high critical pollutant concentration (i.e. SPM, PM_{10}, SO_2, and NO_2) are open coal mining, lime stone mining, fluoride mining, cement industry, thermal power plant, road dust, natural burning of coal, and domestic coal burning by local people for cooking (MPCB, 2010). In Pune city, highest pollution load of PM_{10} comes from different sources like road dust (61%), vehicular source (18%), industry (1.25%), vegetative burning, and solid fuels burning. For NO_2 emissions, major contributions are from vehicles (95%), industries (2%), and domestic and commercial fuel burning (3%) (ARAI, 2010). Vehicles and industries contribute to high SO_2 emission loads due to fuel burning. Main cause of air pollution in Nashik city is due to plastic

industry, food processing factories, and domestic waste burning. Till December 2013, there are 1.13 million number of registered vehicles in the city, causing a major source of pollution (TI, 2014).

5.3. Uncertainty analysis

There may be several uncertainties because study areas are not categorized into residential or industrial together with instrumental error and so uncertainty is owing to relative risk measurements. Central health risk with the addition of lower and upper ranges for the 95% CI based on the input parameters (Table 1) for pollutants is estimated using Ri-MAP model. In each of Figures 1-7, solid bars show calculated values of ENCs or ENCsOMP of mortality and morbidity and thin vertical lines show their lower and upper limits. Uncertainties in calculation of ENCs of TM, CM, CM, hospital admission of COPD, HARD, and HACD are presented in Figures 1-6 as error bars. On the other hand, uncertainty estimated in city-wise annual average ENCsOMP is shown in Figure 7 representing 95% CI. Figures show that CM and COPD have the highest uncertainties while RM has the least uncertainty in most of the cities in Maharashtra.

Error in health risk assessment is due to uncertainty involved in relative risk. Relative risk is calculated using generalized additive model (GAM) in cohort study. Other parameters involved in the cohort study are temperature, humidity, due point, and rain fall. These parameters are most pronounced in CM; it has remained practically constant in the case of RM (Dholakia, Bhadra, & Garg, 2014; Jerrett et al., 2008; Ren & Tong, 2008).

5.4. Limitation and assumption made

There are a number of methodological uncertainties and limitations in the approach which need further improvement to make the method robust. In particular:

(a) Relative risk values used in this study are experimentally developed in United States of America, but a lot of uncertainty is involved when these values are used in any other country like India, as the climatic conditions and economic backgrounds differ starkly from United State of America.

(b) Pollutants are generally of mixed kind—outdoor and indoor air pollutants—associated with synergistic effect which is not considered in the study. While calculating the combined exposure to different pollutants, quite often, an assumption is made that the effects of individual pollutants on human health are additive. However, the simple addition of the effects of each single pollutant would not be totally precise because normally there is a positive relation among atmospheric pollutants (Fattore et al., 2011).

(c) Here, the area-specific (i.e. industrial or residential area) mortality and morbidity have not been considered.

(d) The accuracy of the air quality data as available through MPCB is uncertain due to a wide variety of reasons such as frequent power cut, man power availability problem, calibration error, and failures of air quality monitoring instruments that might cause an error.

(e) In this study, only PM_{10}, SO_2, and NO_2 with WHO threshold limits are considered, but fine particulate matter $PM_{2.5}$ and ozone have not been considered which might have more health impacts.

6. Conclusion

HHR has been estimated in terms of ENCs in different urban area (cities) in Maharashtra using Ri-MAP model. Excess number of mortality and morbidity is observed in Nagpur, Thane, Aurangabad, Kolhapur, and Chandrapur in increasing trend and in decreasing trend in Mumbai and Solapur, but in steady-state condition in Pune, Nashik, and Navi-Mumbai. Annual average excess number of TM, CM, and RM of one million of population is highest in Mumbai and Chandrapur, followed by Navi-Mumbai, Pune, and Nashik. The primary responsible pollutant for HHR in all cities is PM_{10}, but NO_2 and SO_2 are also similarly responsible in Chandrapur. Hence, the pollution control authorities in Maharashtra

urgently need proper policies to elevate ambient air quality in terms of PM_{10} level to decrease the economic costs of air pollution-related health impact. COPD in all the cities is shown very low because SPM, the primary responsible pollutant, is not considered in the case study. The estimated ENCs are only with reference to pollutants' concentration in excess of the levels adopted as per WHO guidelines. However, pollutant concentrations lower than the WHO guidelines also have excess morbidity and mortality because of long time exposure. Actual number of mortality/morbidity cases is higher than the calculated one, owing to lack of data for other pollutants. For more extensive study to estimate health impact, one needs all the relevant pollutants such as TSP, $PM_{2.5}$, PM_1, O_3, CO, heavy metals, and polyaromatic hydrocarbons. In developing countries like India, the ratio of $PM_{2.5}$ and PM_{10} is very high, about 0.65 (Satsangi, Kulshrestha, Taneja, & Rao, 2011), which is much higher than USA (Pace, 2005) and Europe (Barmpadimos, Keller, Oderbolz, Hueglin, & Prévôt, 2012). And long-term exposure to $PM_{2.5}$ is strongly related with ischemic heart disease, cerebrovascular disease, COPD, lung cancer, and acute lower respiratory infections. Thus, long-term epidemiological study related to $PM_{2.5}$ should be performed in India in future because of the presence of high outdoor $PM_{2.5}$ concentration in India and its subcontinent countries (Dey et al., 2012). A new wave of pollution control initiatives is needed to stem the current crippling levels of air pollution. It will be appropriate to initiate similar studies in megacities all over the world; however, the governing parameter (i.e. relative risk) in WHO model should be developed for country-specific studies. The current study shows the importance of evaluation and assessment of health impacts of air quality on a local scale to protect the environment. This study was based on the assumption that the entire population of a city was exposed to the average concentration levels of all air quality monitoring stations. Future studies may use Benefits Mapping and Analysis Program (BenMAP) to calculate the number of air pollution-related deaths and illnesses in finer resolution.

Acknowledgment
The author gratefully acknowledge Maharashtra Pollution Control Board (MPCB), India for giving the permission to use air quality parametric data in this research. The first author thanks Council of Scientific & Industrial Research (CSIR) for providing the scholarship to carry out research at CESE, IIT Bombay.

Funding
The authors received no direct funding for this research.

Author details
Kamal Jyoti Maji[1]
E-mails: kamaljm@iitb.ac.in, kjmaji@gmail.com
Anil Kumar Dikshit[1,2]
E-mail: anildikshit@iitb.ac.in
Ashok Deshpande[3]
E-mail: ashok_deshpande@hotmail.com
[1] Center for Environmental Science and Engineering (CESE), Indian Institute of Technology Bombay, Mumbai 400076, Maharashtra, India.
[2] School of Business, Environment and Society, Malardalen University, Vasteras, SE 72220, Sweden.
[3] Berkeley Initiative in Soft Computing (BISC) - Special Interest Group (SIG) - Environment Management Systems (EMS), University of California, Berkeley, CA, USA.

Citation information
Cite this article as: Human health risk assessment due to air pollution in 10 urban cities in Maharashtra, India, Kamal Jyoti Maji, Anil Kumar Dikshit & Ashok Deshpande, *Cogent Environmental Science* (2016), 2: 1193110.

References

ARAI. (2010). *Air quality monitoring and emission source apportionment study for city of Pune* [online]. Pune: The Automotive Research Association of India, [ARAI/IOCL-AQM/R-12/2009-10]. Retrieved March 21, 2015, from http://cpcb.nic.in/Pune.pdf

Barmpadimos, I., Keller, J., Oderbolz, D., Hueglin, C., & Prévôt, A. S. H. (2012). One decade of parallel fine (PM2.5) and coarse (PM10–PM2.5) particulate matter measurements in Europe: Trends and variability. *Atmospheric Chemistry and Physics, 12*, 3189–3203. doi:10.5194/acp-12-3189-2012

Boldo, E., Medina, S., Le Tertre, A., Hurley, F., Mücke, H.-G., Ballester, F. ... Eilstein, D. (2006). Apheis: Health impact assessment of long-term exposure to PM2.5 in 23 European cities. *European Journal of Epidemiology, 21*, 449–458. doi:10.1007/s10654-006-9014-0

Burnett, R. T., Dales, R. E., Brook, J. R., Raizenne, M. E., & Krewski, D. (1997). Association between ambient carbon monoxide levels and hospitalizations for congestive heart failure in the elderly in 10 Canadian cities. *Epidemiology, 8*, 162–167. doi:10.1097/00001648-199703000-00007

Butler, T. M., Lawrence, M. G., Gurjar, B. R., van Aardenne, J., Schultz, M., & Lelieveld, J. (2008). The representation of emissions from megacities in global emission inventories. *Atmospheric Environment, 42*, 703–719. doi:10.1016/j.atmosenv.2007.09.060

CI. (2001). *Maharashtra 2001* [online]. Census of India. Retrieved January 23, 2015, from http://censusindia.gov.in/maps/State_Maps/StateMaps_links/maharashtra01.html

CI. (2011). *Maharashtra Population Census data 2011* [online]. Census of India Retrieved January 23, 2015, from http://www.census2011.co.in/census/state/maharashtra.html

CPCB. (2010). *Air quality assessment, emissions inventory and source apportionment studies: Mumbai* [online]. Central Pollution Control Board. Retrieved November 23, 2014, from http://cpcb.nic.in/Mumbai-report.pdf

DDCH. (2014). *Data-book compiled for use of planning commission* [online]. Data-book of DCH. p. 168. Retrieved January 23, 2015, from http://planningcommission.nic.in/data/datatable/0306/Databook_June2014.pdf

Dey, S., Di Girolamo, L., van Donkelaar, A., Tripathi, S. N., Gupta, T., & Mohan, M. (2012). Variability of outdoor fine particulate (PM2.5) concentration in the Indian Subcontinent: A remote sensing approach. *Remote*

Sensing of Environment, 127, 153–161. doi:10.1016/j.rse.2012.08.021

Dholakia, H. H., Bhadra, D., & Garg, A. (2014). Short term association between ambient air pollution and mortality and modification by temperature in five Indian cities. *Atmospheric Environment, 99*, 168–174. doi:10.1016/j.atmosenv.2014.09.071

EGI. (2014). *Initiatives to promote clean energy in Maharashtra, India* [online]. Electricity Governance Initiative. Retrieved January 23, 2015, from http://electricitygovernance.wri.org/files/egi/Maharashtra%20Case%20Study.pdf

EOHSP. (2007). *The effectiveness of health impact assessment, scope and limitations of supporting decision-making in Europe* [online]. European Observatory on Health System and Policies. Retrieved January 23, 2015, from http://www.euro.who.int/__data/assets/pdf_file/0003/98283/E90794.pdf

Fattore, E., Paiano, V., Borgini, A., Tittarelli, A., Bertoldi, M., Crosignani, P., & Fanelli, R. (2011). Human health risk in relation to air quality in two municipalities in an industrialized area of Northern Italy. *Environmental Research, 111*, 1321–1327. doi:10.1016/j.envres.2011.06.012

Gefeller, O. (1992). An annotated bibliography on the attributable risk. *Biometrical Journal, 34*, 1007–1012. doi:10.1002/bimj.4710340815

Gharehchahi, E., Mahvi, A. H., Amini, H., Nabizadeh, R., Akhlaghi, A. A., Shamsipour, M., & Yunesian, M. (2013). Health impact assessment of air pollution in Shiraz, Iran: A two-part study. *Journal of Environmental Health Science and Engineering, 11*, 11. doi:10.1186/2052-336X-11-11

Gurjar, B. R., Butler, T. M., Lawrence, M. G., & Lelieveld, J. (2008). Evaluation of emissions and air quality in megacities. *Atmospheric Environment, 42*, 1593–1606. doi:10.1016/j.atmosenv.2007.10.048

Gurjar, B. R., Jain, A., Sharma, A., Agarwal, A., Gupta, P., Nagpure, A. S., & Lelieveld, J. (2010). Human health risks in megacities due to air pollution. *Atmospheric Environment, 44*, 4606–4613. doi:10.1016/j.atmosenv.2010.08.011

IndianPowerSector.com. (2015). *Overview* [online]. IndianPowerSector.com. Retrieved February 22, 2015, from http://indianpowersector.com/home/about/

Jerrett, M., Shankardass, K., Berhane, K., Gauderman, W. J., Künzli, N., Avol, E., ... McConnell, R. (2008). Traffic-Related air pollution and asthma onset in children: A prospective cohort study with individual exposure measurement. *Environmental Health Perspectives, 116*, 1433–1438. doi:10.1289/ehp.10968

Lowrance, W. W. (1976). *Of acceptable risk: Science and the determination of safety*. Los Altos, CA: Kaufmann, William, Incorporated. ISBN 13: 978-0913232309, 10: 0913232300.

Madronich, S. (2006). Chemical evolution of gaseous air pollutants down-wind of tropical megacities: Mexico City case study. *Atmospheric Environment, 40*, 6012–6018. doi:10.1016/j.atmosenv.2005.08.047

Molina, M. J., & Molina, L. T. (2004). Megacities and atmospheric pollution. *Journal of the Air & Waste Management Association, 54*, 644–680. doi:10.1080/10473289.2004.10470936

MPCB. (2010). *Action plane for industrial cluster: Chandrapur*. Maharashtra Pollution Control Board. Retrieved February 14, 2015, from http://cpcb.nic.in/divisionsofheadoffice/ess/Action%20plan%20CEPI-Chandrapur.pdf

MPCB. (2014). *Focus area air quality* [online]. Maharashtra Pollution Control Board. Retrieved December 22, 2014, from http://mpcb.gov.in/envtdata/envtair.php

Naddafi, K., Hassanvand, M. S., Yunesian, M., Momeniha, F., Nabizadeh, R., Faridi, S., & Gholampour, A. (2012). Health impact assessment of air pollution in megacity of Tehran, Iran. *Iranian Journal of Environmental Health Science & Engineering, 9*, 28. doi:10.1186/1735-2746-9-28

Nagpure, A. S., Gurjar, B. R., & Martel, J. (2014). Human health risks in national capital territory of Delhi due to air pollution. *Atmospheric Pollution Research, 5*, 371-380. doi:10.5094/APR.2014.043

Northridge, M. E. (1995). Public health methods–attributable risk as a link between causality and public health action. *American Journal of Public Health, 85*, 1202–1204. doi:10.2105/AJPH.85.9.1202

NRC. (1983). Risk assessment in the federal government: Managing the process. *Committee on the Institutional Means for Assessment of Risks to Public Health. National Research Council*. Washington, DC: National Academy Press

NYT. (2014). *India's Air Pollution Emergency* [online]. The New York Times. Retrieved January 23, 2015, from http://www.nytimes.com/2014/02/14/opinion/indias-air-pollution-emergency.html?_r=0

Orru, H., Laukaitienė, A., & Zurlytė, I. (2012). Particulate air pollution and its impact on health in Vilnius and Kaunas. *Medicina (Kaunas), 48*, 472–477.

Orru, H., Teinemaa, E., Lai, T., Tamm, T., Kaasik, M., Kimmel, V., ... Forsberg, B. (2009). Health impact assessment of particulate pollution in Tallinn using fine spatial resolution and modeling techniques. *Environmental Health, 8*, 7. doi:10.1186/1476-069X-8-7

Pace, G. T. (2005). Examination of the multiplier used to estimate PM2.5 fugitive dust emissions from PM10. *US Environmental Protection Agency*. Retrieved from http://www3.epa.gov/ttnchie1/conference/ei14/session5/pace.pdf

Poloniecki, J. D., Atkinson, R. W., de Leon, A. P., & Anderson, H. R. (1997). Daily time series for cardiovascular hospital admissions and previous day's air pollution in London, UK. *Occupational and Environmental Medicine, 54*, 535–540. doi:10.1136/oem.54.8.535

Ren, C., & Tong, S. (2008). Health effects of ambient air pollution–recent research development and contemporary methodological challenges. *Environmental Health, 7*, 56. doi:10.1186/1476-069X-7-56

Rothman, K. J., Greenland, S., & Lash, T. L. (2008). *Modern epidemiology*, (3rd ed., pp. 758, 53–77 136, 160, 214). Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins, ISBN 0781755646, 9780781755641.

Satsangi, P. G., Kulshrestha, A., Taneja, A., & Rao, P. S. P. (2011). Measurements of PM 10 and PM 2. 5 aerosols in Agra, a semi-arid region of India. *Indian Journal of Radio Space Physics, 40*, 203–210.

Sunyer, J., Spix, C., Quenel, P., Ponce-de-Leon, A., Ponka, A., Barumandzadeh, T., ... Katsouyanni, K. (1997). Urban air pollution and emergency admissions for asthma in four European cities: The APHEA Project. *Thorax, 52*, 760–765. doi:10.1136/thx.52.9.760

TI. (2014). *Vehicles in Nashik region rise by nearly 10%* [online]. The Times of India. Retrieved January 23, 2015, from http://timesofindia.indiatimes.com/city/nashik/Vehicles-in-Nashik-region-rise-by-nearly-10/articleshow/29924015.cms

Tominz, R., Mazzoleni, B., & Daris, F. (2005). Estimate of potential health benefits of the reduction of air pollution with PM_{10} in Trieste, Italy. *Epidemiologia & Prevenzione, 29*, 149–155.

Touloumi, G., & Katsouyanni, K. (1997). Short-term effects of ambient oxidant exposure on mortality: A combined analysis within the APHEA project. *American Journal of Epidemiology, 146*, 177–185. http://dx.doi.org/10.1093/oxfordjournals.aje.a009249

Vostal, J. J. (1994). Physiologically based assessment of human exposure to urban air pollutants and its significance for public health risk evaluation. *Environmental Health Perspectives, 102*, 101–106. doi:10.2307/3431937

WB. (2013). *Diagnostic assessment of select environmental challenges in India* [online]. The World Bank. Retrieved

January 23, 2015, from http://www.worldbank.org/en/news/press-release/2013/07/17/india-green-growth-necessary-and-affordable-for-india-says-new-world-bank-report

WHO. (1999). *Monitoring ambient air quality for health impact assessment. World Health Organization Regional Office for Europe*, Copenhagen: WHO Regional Publications. ISBN 92 890 1351 6, European series; No. 85. (pp. 216, 43–44), ISSN: 0378-2255. Retrieved January 22, 2014, from http://www.euro.who.int/__data/assets/pdf_file/0010/119674/E67902.pdf

WHO. (2003). Health aspects of air pollution with particulate matter, ozone and nitrogen dioxide. *World Health Organization Working Group Bonn, Germany, 13-15*. Copenhagen: WHO Regional Office for Europe. Retrieved November 23, 2014, from http://www.euro.who.int/document/e79097.pdf

WHO. (2004). *Tools for health impact assessment of air quality: The AirQ 2.2 software* [online]. World Health Organization. Retrieved November 23, 2014, from http://www.euro.who.int/en/health-topics/environment-and-health/air-quality/activities/tools-for-health-impact-assessment-of-air-quality-the-airq-2.2-software/download-and-installation-instructions