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Association of aerosols, trace gases and black carbon with mortality in an urban pollution hotspot over central Indo-Gangetic Plain

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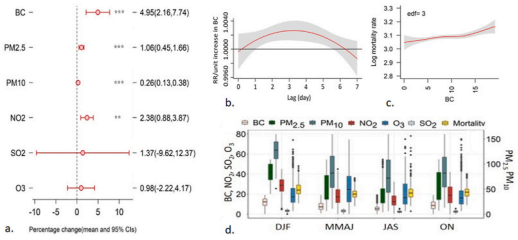
HIGHLIGHTS

- Mortality estimate of black carbon aerosols and multiple trace gases are reported.
- Mortality estimate was particularly high for BC followed by NO_2 levels.
- All the pollutants showed significant lag effect between lag 0–1 and lag 0–6 days.
- Synergistic effect was noted when BC, Res.$PM_{2.5}$ and NO_2 were combined.
- Mortality estimates of BC aerosols, $PM_{2.5}$ and NO_2 were higher during hazy days.

GRAPHICAL ABSTRACT



Exposure-response relations of air pollution and pre-mature mortality in Varanasi.

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ABSTRACT

The short term effect of multiple air pollutants e.g. aerosols (black carbon, BC; $PM_{2.5}$ and PM_{10}) and trace gases (NO_2, SO_2, and O_3) on all-cause mortality was systematically investigated in a typical urban pollution hotspot over central Indo-Gangetic Plain (IGP). To our knowledge, this would be the first report of mortality estimates for exposure to BC aerosols and multiple trace gases over South Asia. Daily all-cause mortality and ambient air quality were analyzed from 2009 to 2016 following a semiparametric quasi-Poisson regression model adjusting mean temperature (Tmean), relative humidity (RH), and long term time trend (Time) as potential confounders. Single pollutant model clearly established the significant impact of BC aerosols (against 10-unit increase in pollutant; 4.95%, 95% CI: 2.16–7.74), NO_2 (2.38%, 95% CI: 0.88–3.87%) and $PM_{2.5}$ exposure (1.06%, 95% CI: 0.45–1.66%) on mortality. The inclusion of co-pollutants in the multi-pollutant model increased the individual mortality risks for BC aerosols (7.3%). Mortality estimates were further stratified considering different effect modifiers viz. sex, age, place of death, and season. Almost in all the cases statistically insignificant differences in effect modification were noted for all the pollutants except PM_{10}. We also explored a distributed lag nonlinear model to estimate the lag effect and all the pollutants showed significant lag up to 3 days while BC showed lag effect up to 5 days. The exposure-response curves for individual air pollutants were mostly linear, while a considerable increase in mortality was noted for an exposure >15 μg m^{-3} for BC aerosols and >60 μg m^{-3} for $PM_{2.5}$. The effect estimates of air pollutants during haze and no-haze days were also defined. During haze days,

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mortality rose to 6.11% and 3.06% for each 10-unit increase in BC and NO_2 exposure, respectively. Significant effect of BC aerosol exposure on human mortality was established which reaffirms its inclusion as a potential health regulator for epidemiological studies.

1. Introduction

Exposure to air pollution has been linked with many negative health impacts including cardiovascular and pulmonary diseases, neonatal conditions, bronchitis, asthma, and lung cancer (Cohen et al., 2005; Lelieveld et al., 2015). Both acute and chronic exposures to air pollutants has been reported to have negative implications on human health (Balakrishnan et al., 2019; Huang et al., 2018). However, most of the pollutions based epidemiological studies essentially relate exposure to particulate mass concentration (PM_{10} and/-or $PM_{2.5}$) that invariably generalize all particulates with equal toxicity without distinguishing individual by its source and composition, which genuinely have different health consequences (Tuomisto et al., 2008; Cao et al., 2012; Lelieveld et al., 2015; Singh et al., 2020). Only a few researchers *viz*. Janssen et al. (2011), Cao et al. (2012), Geng et al. (2013), Wang et al. (2013), and Lelieveld et al. (2015) have explored health implications caused by the exposure of multiple air pollutants and individual particulate type (like desert dust and smoke).

Among many air pollutants, airborne fine particulates ($PM_{2.5}$) are most frequently reported to be associated with negative health impacts especially in terms of mortality and morbidity; both globally (Smith et al., 2009; Janssen et al., 2011; Cao et al., 2012) and over India (Chowdhury et al., 2018; Balakrishnan et al., 2019; Saini and Sharma 2020). Particulates emitted exclusively from the combustion processes *viz*. combustion of wood, residential oil, coal, and petroleum are mostly toxic and are frequently associated with adverse health effects (Krzyżanowski et al., 2005; Cao et al., 2012; Thurston et al., 2013), compared to the particulates from non-combustion sources (like crustal emissions, sea salt). This has been specifically emphasized in the context of black carbon aerosols (BC) or in some cases as elemental carbon (EC) which are short-lived climate-forcer with primary emissions from residential combustion and automobile sector. The sources of BC aerosols varies spatially as residential combustion of biomass/biofuels like dried animal manure, coal, wood, and agricultural waste are primarily responsible for BC emissions over India (Venkataraman et al., 2005) compared to the automobile exhaust emissions over Europe and North America (Bond et al., 2004; Klimont et al., 2017). Large number of epidemiological studies provide extensive evidences of significant positive association of BC exposure with cardiopulmonary morbidity, respiratory mortality and other adverse health impacts (Hennebergeret al., 2005; Ostro et al., 2007; Smith et al., 2009; WHO, 2012; Geng et al., 2013; Wang et al., 2013; Janssen et al., 2011; Luben et al., 2017; and references therein). Exposure to BC aerosols typically causes inflammation in pulmonary tissues inducing a range of mediators altering cardiac functions, or irritant receptor-mediated stimulation of parasympathetic pathways (Smith et al., 2009). It also influences myocardial repolarization leading to the risk of sudden cardiac death (Hennebergeret al., 2005), depression of ST-segment (Gold et al., 2005), and inflammation of the airway through high nitric oxide exhalation (Mar et al., 2005). Black carbon shows higher association with cardiovascular mortality compared to other aerosol components and serves as a better health indicator against total particulate mass (Roemer and Van Wijnen, 2001; Lipfert et al., 2006; Janssen et al., 2011; WHO, 2012). However, health specific impacts of BC are not spatially consistent and vary significantly in different locations and for communities. Likewise, for multi-pollutant health studies, using county-level data over the USA Lipfert et al. (2006) reported a highest impact of elemental carbon (EC) on all-cause mortality, followed by nitrate; Cao et al. (2012) found nitrate to demonstrate the strongest association with all-cause and cardiovascular mortality in Xi'an while Smith et al. (2009), considering 18-years nationwide data over the USA, showed a stronger effect of EC on mortality in combination with sulfate and ozone exposure.

The health effects in terms of mortality due to BC aerosol exposure have never been evaluated in India except studies on cross-sectional associations between BC with blood pressure and hypertension (Curto et al., 2019). Availability of city-specific health statistics like cause-specific mortality and morbidity is limited over the Indian cities, so is the BC mass concentration which is measured only over a few urban environments across India. In contrast, systematic monitoring of other air quality parameters ($PM_{2.5}$, PM_{10}, NO_2, SO_2, and O_3) is now functional over most of the Indian cities under the National Air Quality Monitoring Programme. This motivated us to evaluate the individual as well as the cumulative impact of BC aerosol, fine ($PM_{2.5}$), and coarse (PM_{10}) particulates, and trace gases (SO_2, NO_2, O_3) on premature mortality in an urban pollution hotspot in Northern India. Our analysis was novel as only few studies over India have evaluated the effect of multiple air pollutants on mortality (Jayaraman and Nidhi, 2008; Maji et al., 2017) while none considering the individual particulate types (like BC aerosols).

The manuscript reports the short-term effect of BC aerosols along with other criteria air pollutants on mortality using time series data from a typical urban pollution hotspot over Indo-Gangetic Plain (IGP), South Asia. Besides, we also investigated the effects of air pollution on the short-term mortality risk during haze and no-haze days. Haze is a typical environmental condition under which the exposure to air pollutants (particularly fine particulates) increases excessively. There are many inferences regarding negative health impacts of haze on human health, as reported in Hong Kong (by Chak Ho et al., 2018), Guangzhou (by Zhang et al., 2014), and in Beijing (by Zhang et al., 2015 and Liang et al., 2017). However, over South Asia, there is no report on the possible impact of haze on human mortality. Considering such limitations, we tried to establish the impact of haze on human mortality, constrained by the impact of individual air pollutants. To the best of our knowledge, mortality estimates of BC aerosol were reported for the first time over South Asia which may have greater implications in prioritizing early warning systems, and in developing mitigation/- adaptation policies over the region.

2. Material and methods

2.1. Study area

Entire South Asia has been documented to have an excessive burden of air pollutants with many associated sources like biomass and waste burning, automobile emissions and, soil and desert dust (Singh et al., 2017, and references therein). However, the northern part of this geographical region, the IGP, often remains the center of investigation focusing on the air pollution-agriculture-health-sustainability nexus. It is one of the most fertile regions of the world, accounting for a major fraction of India's food production and sustains 60% of India's population that is primarily sensitive to changing climate (Mall et al., 2018; Sonkar et al., 2019) and pollution-related health impacts (Chowdhury et al., 2018).

The study was conducted in Varanasi (25°16′N, 82°59′E; 82 m MSL), a typical urban pollution hotspot in central IGP (Fig. S1) which experiences very high aerosol loading (decadal mean aerosol optical depth, AOD±SD: 0.67 ± 0.28; annual mean $PM_{2.5}$: 82 ± 66 μg m^{-3}) and trace gas concentrations throughout the year (Murari et al., 2017; Shukla et al., 2017; Kumar et al., 2018). Decadal increasing trends both in AOD (0.017 $year^{-1}$; Kumar et al., 2018) and BC aerosols (0.9 μg m^{-3} yr^{-1};

Manoj et al., 2019; Srivastava et al., 2019) are concurrent with the population growth rate of Varanasi (17%). The city supports a population of 12 million (MHA, 2011), with a very high population density (14, 598 Km^{-2}). Varanasi itself has numerous kinds of air pollution sources but emissions from biomass and backyard incineration, automobiles, household emissions, soil, and road dust resuspensions are the dominating ones (Murari et al., 2020; Singh et al., 2021). Besides, there is evidence of the prevalence of a subsidence zone over the central IGP by prevailing westerly which also facilitates the gradual accumulation of air pollutants over the region (Di Girolamo et al., 2004; Kumar et al., 2018). The city represents a humid subtropical climate with four distinct seasons: winter (DJF), summer (pre-monsoon, MAMJ), monsoon (JAS), and post-monsoon (ON); summer is relatively hot and dry, winter is cold and humid while monsoon accounts for the major proportion of annual precipitation.

2.2. Data

Daily mortality data (excluding accidental mortality) between January 1, 2009, and December 31, 2016, for Varanasi city, was collected from the Municipal Corporation of Varanasi. Mortality data were classified following the International Classification of Diseases 10th revision (ICD10) into all-cause mortality (A00–R99), mortality due to cardiovascular diseases (I00–I99), and respiratory diseases (J00-J98). The mortality data were also classified according to age (≤4, 5–44, 45–64, ≥65 years), sex (male/female), and place of death (institutional and non-institutional deaths) (Singh et al., 2019). Meteorological data, including mean temperature (Tmean), relative humidity (RH), and precipitation were obtained from the India Meteorological Department, New Delhi. The daily visibility data was assessed from ASOS-AWOS-METAR dataset maintained by Iowa Environmental Mesonet (IEM).

Ambient air quality data for $PM_{2.5}$, PM_{10}, NO_2, SO_2, and O_3 (2009–2016, all-inclusive) was acquired from Real-time Air Quality Data inventory of the Central Pollution Control Board (CPCB), available at https://app.cpcbccr.com/ccr. The CPCB air quality monitoring station is close to the city center and represents emission sources of the city. Initially, the hourly concentration of each pollutant was checked for quality and was averaged for 24 h. Pollutant concentrations above 97.5% CI and below 2.5% CI of the 8-year annual average concentration was excluded from the analysis (as outliers) to avoid unexpected coefficient of association and unspecified events. Real-time black carbon (BC) mass concentration at 880 nm was measured using Aethalometer (AE42, Magee Scientific, USA) within the Banaras Hindu University (BHU) campus. The BC monitoring station mainly encompasses emissions from residential and commercial activities, while both the stations (BHU and CPCB) also receive contributions from biomass burning, road dust, and other emissions sources. A detailed discussion on BC measurement protocol and related uncertainties are discussed in Kumar et al. (2017) and Singh et al. (2018).

Both BC and $PM_{2.5}$ have missing observations due to instrumental error, power issue, and instrument calibration which was later modelled to fill the gaps. Two models were constituted to impute the missing $PM_{2.5}$ (24%) and BC (32%) concentrations. Modelled data were validated using the 10-fold cross-validation (CV) method to avoid potential over-fitting. The training dataset was randomly split into 10 subsets each containing approximately 10% of the data. In each round of CV, nine subsets were used for model fitting and the rest for validation. The missing $PM_{2.5}$ was imputed by fitting a generalized additive model using PM_{10} as a main predictive variable, adjusted to the time-varying nonlinear meteorological variables (temperature and relative humidity). Fig. S2 shows the correlation between the observed and predicted $PM_{2.5}$. The fitted model showed a good agreement with the daily mean $PM_{2.5}$ (R^2:0.89; RMSE 28.57). It should be noted that the predicted values were only used to fill the missing $PM_{2.5}$ when PM_{10} concentrations were available.

The missing BC data were imputed using a Random Forest machine learning algorithm considering BC mass concentration as a function of measured $PM_{2.5}$ and carbon monoxide; as both represent emissions primarily from the combustion sources. To take into account the diurnal variation of BC concentration, hourly concentration was initially modelled, before averaging it to 24 h. Fig. S3 (a, b) shows the scatter plot of the 10-fold cross-validation and the test unseen results for BC. The performance of the fitted random forest model shows good agreement for both the 10-fold cross-validation method and the unseen test dataset. The CV and test results indicate that the random forest model well estimated the hourly BC concentration (R^2: 0.77; RMSE: 4). The daily predicted BC values (Fig. S4) showed lower RMSE (2.38) and higher R^2 (0.87) compared to hourly observation. The modelled BC observation was only used to fill the missing value when observed $PM_{2.5}$ was available.

2.3. Statistical methods

Time-series analysis with a semiparametric quasi-Poisson regression model was used to assess the effect of single and multiple air pollutants on daily mortality. In a single-pollutant model, we introduced one pollutant at a time as a linear term. The model was controlled for seasonality, long-term trend (Peng et al., 2009), and potential non-linear confounding effects of Tmean and RH (Chen et al., 2012) using penalized cubic smoothing spline. The purpose of adjusting temporal effects was to eliminate time trends and seasonality from the mortality count and to estimate the effects of short-term change in exposure. The confounding effects of a day of the week and the public holiday didn't contribute significantly to the model and thus excluded (Table S3).

$$\mathrm{Log}\{\mathrm{E}[(Mortality_t)]\} = \alpha + \beta P_{(t)} + f_1(RH_t) + f_2(Tmean_t) + f_3(Time_t) \quad (1)$$

where, β is the regression coefficients corresponding to the air pollutant "P" (BC, PM_{10}, $PM_{2.5}$, SO_2, NO_2, and O_3) over time't'; f_1, f_2, f_3 are the smoothed function (penalized cubic smoothing spline) of nonlinear confounding factors such as time, RH and Tmean. For each of the nonlinear components in the above and rest of the analysis, a penalized cubic smoothing technique was used and degrees of freedom (df) were estimated by the algorithm based on Generalized Cross Validation (GCV) score (Hastie and Tibshirani 1990). Hence, we allowed the algorithm to choose suitable df based on cross validation referred to as estimated degrees of freedom (edf).

In the multipollutant model, we considered residual $PM_{2.5}$ (Res. $PM_{2.5}$), BC, and NO_2 together as linear terms in the core model.

$$\mathrm{Log}\{\mathrm{E}[(Mortality_t)]\} = \alpha + \beta_1 ResPM_{2.5\,(t)} + \beta_2 BC_t + \beta_3 NO_{2\,(t)} + f_1(RH_t) + f_2(Tmean_t) + f_3(Time_t) \quad (2)$$

where β_1 to β_3 are the regression coefficients corresponding to the air pollutants Res.$PM_{2.5}$, BC, and NO_2 over time 't'; rest remained identical as in equation (1). To obtain the Res.$PM_{2.5}$, we regressed the $PM_{2.5}$ against BC, SO_2, and NO_2 where all three were added together in the core model as a linear term. The predicted $PM_{2.5}$ was subtracted from the observed $PM_{2.5}$ to get the residual $PM_{2.5}$. Thus, the $PM_{2.5}$ concentration contributed due to other sources than BC, NO_2, and SO_2 were

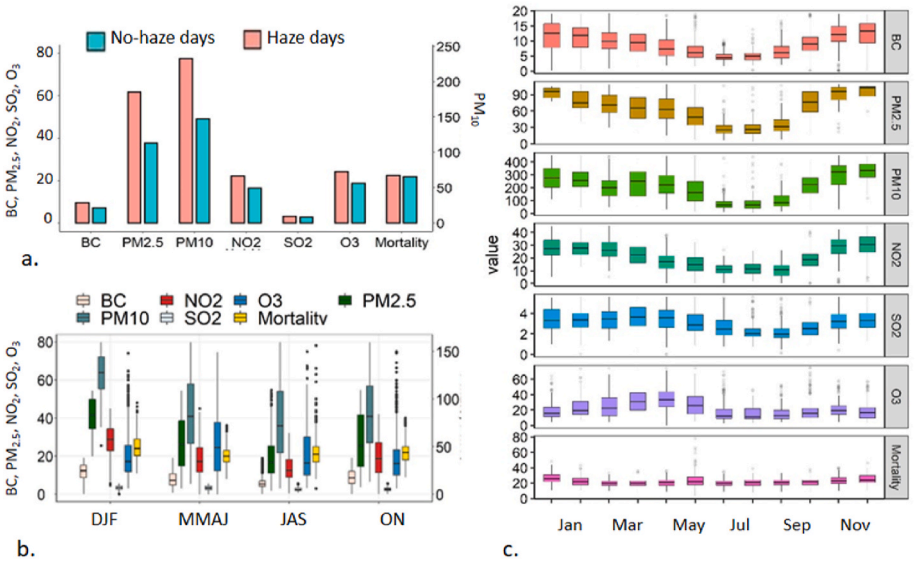

Fig. 1. Time-series graph for air pollutants and all-cause mortality in Varanasi.

termed as 'Res.$PM_{2.5}$. Since the average SO_2 level was much lower than the national standard (Fig. 1c) and as both SO_2 and O_3 did not show any significant association with all-cause mortality, we excluded them further. We have included NO_2 along with BC to predict $PM_{2.5}$ to account for the gas to particle conversions of NO_2, both via homogeneous and heterogeneous reactions, which frequently constitute a greater fraction of particulate mass (Hodan and Barnard, 2004). Here, partial determination of NO_2, SO_2, and BC with $PM_{2.5}$ were 0.38, 0.09, and 0.37 respectively (Table S4).

To investigate the implication of pollutant concentrations measured during haze and/- no-haze days on daily mortality, we followed the single pollutant model with subset haze days and no haze days. A "haze day" was defined as a calendar day when the visibility is < 5 km for at least 1 h, relative humidity <95%, with no fog, no mist, and no precipitation (Ho et al., 2018). We also explored the interaction between haze and no haze day and pollutant concentration but no significant effect on all-cause mortality was evident (not included in the text, Table S5).

To account for the effect modification by individual characteristics in the air pollutant-mortality association, we fitted equation (1) separately for each factor such as age, gender, place of death, and season as a dependent variable keeping the rest unchanged (as in equation (1)). Regression coefficients for age/-gender/-place of death/-season were compared within the group by Wald χ2-test to measure for equality assuming independence across the group (Diggle et al., 1994). To test the robustness of the results, we also performed different sensitivity analyses. The time lag effects of individual pollutants were established by exploring a restricted distributed lag model for 7 days' lag with a polynomial of degree two for Eq. (1) (Schwartz, 2000). Besides, we explored the sensitivity of the RR estimate to the time lag effect of temperature (Tmean) and RH up to 5 days lag using the single and cumulative lag model and 5 days (Lag 1 to Lag 5) mean value to control for the confounding effects of previous day's temperature and RH in the core model (Eq. (1)). To do so, the Eq. (1) was adjusted for different temperature lag one at a time (Lag 1 to Lag 5) for the single lag model and by adding the next lag each time in the cumulative lag model (lag 0 + lag 1, lag 0 + lag1 + lag 2 …. .) keeping RH at the current day. After comparing the temperature lag models the one with the highest deviance explained and lowest coefficient of variation was selected as the best fit, and the same process was repeated for RH fixing Tmean at lag 1 for BC and $PM_{2.5}$, Lag 3 for PM_{10} and lag 0 for NO_2.

The choice of degrees of freedom (3–6 degrees of freedom) of temporal effect on mortality were analyzed too. We have also explored the association of air pollutants with cause-specific mortalities, but due to under-reporting and/-or absence of robust cause-specific mortality data, results remained inconclusive, therefore, not reported. We have also checked different lag days in the core model, and find that the effect estimates on the same day are much robust than considering lag days based on deviance explained, standard error, GCV score as well as the width of the confidence interval. Hence we considered same day exposure in the core model for the entire analysis.

The dose-response curve for different air pollutants was generated to explore the possibility of a nonlinear relationship (as a potential violation of linearity assumption between mortality and pollutant in the core model) to observe the changes in mortality caused by the different levels of pollutant exposure. We refitted our core model (equation (1)) as follows:

$$\mathrm{Log}\{\mathrm{E}[(Mortality_t)]\} = \alpha + f(P_{(t)}) + f_1(RH_t) + f_2(Tmean_t) + f_3(Time_t) \quad (3)$$

where f ($P_{(t)}$) is the smooth function of the average pollutant concentration on day t; rest remain unchanged. The degrees of freedom were selected based on the GCV score.

All the results from the above analysis were presented as per unit change in pollutant concentration and/-or percent change in mortality per ten-unit change in pollutant concentration with 95% confidence interval (CI). All data were analyzed by statistical software R version 3.5.1 (R Core Team, 2018); using the R -package "mgcv" (version 1.8–18.) (Wood, 2006), random forest, and "dlnm" (version 2.3.2.) (Gasparrini, 2011). The statistical tests were two-tailed and result with p-value < 0.05 were considered statistically significant.

3. Results and discussion

Summary statistics for mortality data, air pollutants, and weather variables are included in Table S1. In between the eight years (2009–2016), a total of 64,712 non-accidental deaths were considered for the analysis that showed a daily mean (±SD) mortality of 22 (±6) with more male decedents (60%). Overall, cardiorespiratory mortality (RD and CVD) accounted for 3.4% of total non-accidental mortality, 65% of which were male.

A very high concentration of almost all the pollutants was noted throughout, particularly during ON and DJF (Fig. 1). The annual mean (±SD) concentration of $PM_{2.5}$ and PM_{10} was 104 (±86) and 219 (±135) μg m^{-3} respectively; well above the national ($PM_{2.5}$, 40 μg m^{-3}; PM_{10},

60 μg m^{-3}; NAAQS 2016) and WHO ($PM_{2.5}$, 10 μg m^{-3}; PM_{10}, 20 μg m^{-3}; WHO 2005) permissible limits. Other air pollutants (BC, NO_2, and SO_2) that have direct emissions especially from the combustion sources, have also recorded a very high mixing ratio with considerable seasonal variations. The annual mean (±SD) BC aerosols based on 24 h average concentration measured between 2009 and 2016 was 9.7 (±6.3) μg m^{-3}, while the daily mean range in between 0.02 and 19.2 μg m^{-3}. The correlation coefficients for the air pollutants ranged from 0.50 to 0.86 and the detailed description is provided in Table S2. Daily variations in BC concentration were more closely associated with $PM_{2.5}$ and NO_2 ($r > 0.5$), indicating their similarity in origin, predominately being emitted from vehicle exhaust and biomass burning. A total of 1958 days were classified as haze days (67% of total), with a slightly declining trend for the year 2016 (214 days) compared to 2009 (238 days). Overall, haze days accounted for a total of 43,753 all-cause mortality, 3.4% of which were diagnosed for cardiorespiratory diseases (RD and CVD) compared to 0.7% for no-haze days. Interestingly, haze days were frequent both during summer (554) and winter months (472) compared to the rest of the year. Characteristically, the concentration of PM_{10}, $PM_{2.5}$, and BC was particularly high during haze days with 58%, 65%, and 36% increase respectively in their mean concentration compared to the no-haze days (Fig. 1).

3.1. Mortality risk of air pollutants

3.1.1. Single pollutant model

The results from the generalized additive model described in eq. (1) for a single pollutant model with estimated degrees of freedoms approximately 9, 7, and 3 for time, temperature, and relative humidity respectively are shown in Fig. 2. The result compares the individual effect of air pollutants on mortality. For every 10-unit increase in ambient concentration of individual pollutant, mortality was enhanced by 4.95% for BC aerosols (95% CI: 2.16–7.74%), 2.38% for NO_2 (95% CI: 0.88–3.87%) and 1.06% for $PM_{2.5}$ (95% CI: 0.45–1.66%). In comparison, the effect estimates for PM_{10} on all-cause mortality was relatively low (0.26%, 95% CI: 0.13–0.38%). For the rest of the pollutants (SO_2 and O_3), the effect on mortality was statistically insignificant and had a minimum individual impact (<1.4%), therefore SO_2 and O_3 were not included in stratification analyses. Evidence from this study showed that the current level of black carbon concentration in Varanasi is significantly associated with all-cause mortality which vowed it to consider as a potent health indicator of air pollution exposure compared to other matrices. However, when BC and $PM_{2.5}$ were compared for change in effect per interquartile range, the effect of $PM_{2.5}$ was higher than BC as BC is a constituent of $PM_{2.5}$ and thus $PM_{2.5}$ shows the cumulative effect due to particles that have a diameter less than 2.5 μm including BC. Our estimate for Varanasi was lower compared to the estimates reported by Janssen et al. (2011) for each 10 μg m^{-3} increase in $PM_{2.5}$ (1.9%) and EC (14.5%) using pooled analysis for all-cause mortality (Klemm et al., 2004; Ostro et al., 2007; Cakmak et al., 2009). Also, Smith et al. (2009) reported a 1.006 and 1.06 relative risk of all-cause mortality for each 1 μg m^{-3} increase in long-term exposure to $PM_{2.5}$ and EC respectively over the USA. In contrast, when compared against the individual composition of $PM_{2.5}$, Ostro et al. (2007) found no statistical evidence of the impact of EC on all-cause mortality in California but had the highest impact on cardiovascular mortality, followed by nitrate aerosols. Cao et al. (2012) in Xian, China also reported the highest individual impact by nitrate aerosols on all-cause and cardiovascular mortality followed by EC.

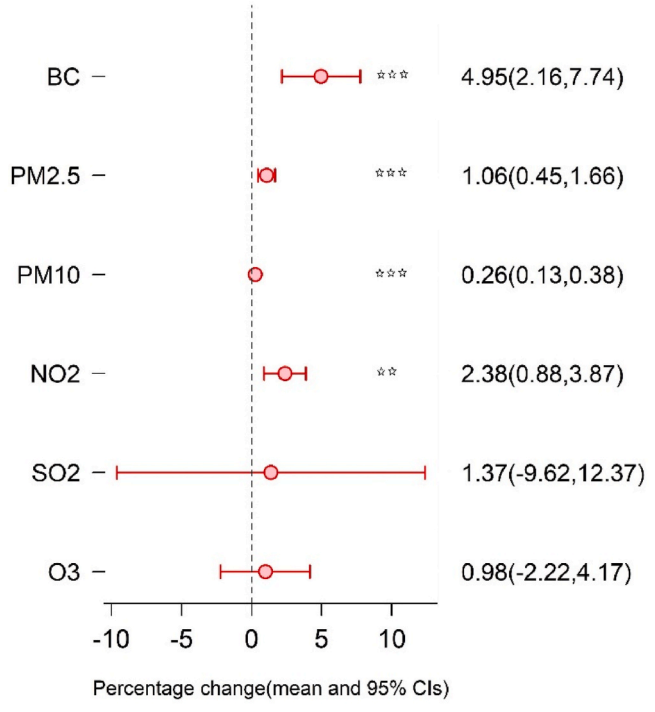

Fig. 2. Percent change in mortality associated with 10-unit increase in air pollution exposure.

After BC, NO_2 was another important pollutant to influence mortality in Varanasi. Both cross-sectional and longitudinal studies indicate NO_2 as a robust indicator of traffic pollution which induces impairment of lung function, exacerbation of asthma and non-asthma respiratory symptoms, and other cardiovascular complications. However, it should be noted that although we find a statistically significant association between mortality and NO_2 exposure, a considerable level of uncertainty still exists for a time-series analysis as often impacts of NO_2 are complicated by the co-existence of other pollutants, especially by particulate matter and ozone.

3.1.2. Multiple pollutant model

We also explored the change in mortality by concurrent exposure to multiple air pollutants, because the individual impact of pollutants is exceedingly rare in the real-world, and pollutants possibly induce additive, synergistic or antagonistic impact when they co-exist in the environment. The variation in $PM_{2.5}$ was partly defined by the individual contribution of BC (37.8%), SO_2 (9%), and NO_2 (38.5%) while, the combined contribution of all three was 55.5% (Table S4). Therefore, to improve the power and stability in the multi-pollutant model, we did not introduce $PM_{2.5}$ in the multi-pollutant model; instead, we used the residual of $PM_{2.5}$ (Res.$PM_{2.5}$), that excludes the variation in $PM_{2.5}$ attributed to BC, SO_2, and NO_2 but is contributed by some unknown sources. The result from the multi-pollutant model showed an increase in individual mortality risk of BC aerosols and Res.$PM_{2.5}$ for all the concerned cases (Table 1). The effect estimates of BC increased the most (7.30%) suggesting it as an important health regulator in combination with Res.$PM_{2.5}$ and NO_2, followed by Res.$PM_{2.5}$ (1.51%, 95% CI: 0.71–2.31%), while being highly statistically significant. The effect estimates of NO_2 reduced and became non-significant when adjusted for co-pollutants (0.77%, 95% CI: 1.88, 3.42%) which possibly indicated the more strong impact of BC and fine particulates on mortality risk

Table 1
Percent change in mortality with 10 μg m^{-3} increase in exposure of BC, Res$PM_{2.5}$ and NO_2.

Variables	Temp	% Change in mortality (95% CI)
All cause mortality	Res$PM_{2.5}$	**1.51 (0.71, 2.31)**
	BC	**7.30 (2.33, 12.27)**
	NO_2	0.77 (−1.88, 3.42)

Note. Effects of different pollutants on mortality with 95% CI in terms of percentage change in mortality per 10- μg m^{-3} change of pollutant. Values in bold are significant at $p < 0.05$.

compared to NO_2. However, as discussed in section 3.1.1, the health impacts of NO_2 often get complicated by the presence of aerosols, and there is evidence of both synergistic (Luben et al., 2018) and antagonistic effects (Yu et al., 2013) of NO_2-aerosols association, particularly for complications related to cardiovascular diseases.

Both single and multi-pollutant models established clear evidence of effect estimates of BC aerosols on human mortality. This is in line with the findings of other concurrent epidemiological researchers both over Asian (Cao et al., 2012; Geng et al., 2013; Wang et al., 2013) and global cities (Roemer and Van Wijnen, 2001; Henneberger et al., 2005; Smith et al., 2009; Janssen et al., 2011). Wang et al. (2013) concluded higher effect estimates for BC aerosols against $PM_{2.5}$ and $PM_{2.5\text{-}10}$ using a two-pollutant model in Shanghai, China. For the same city, the effect estimates for BC and $PM_{2.5}$ are reported to increase from their individual estimates to 2.3%–4.9% and from −5% to 2.4% respectively, when adjusted for one another (Geng et al., 2013). However, Hoek et al. (2000) reported a decrease in effect estimates for $PM_{2.5}$ and BC in the Netherlands when compared against their individual impact.

3.1.3. *Mortality stratified by effect modifiers*

The population response in terms of change in mortality (in %) against 10-unit change in individual pollutant concentration stratified by means of age, sex, place of death, and season are included in Fig. 3. Both $PM_{2.5}$ and PM_{10} induced greater mortality risk in male while the female population was affected more by NO_2 exposure, and there was no variation in mortality risk by sex for BC aerosols. A similar nonsignificant estimate for sex of the targeted population was also reported by Bravo et al. (2015), Maji et al. (2017), and Geng et al. (2013) for other Asian cities. We noted a higher rate of change in mortality for elderly people (>65 years) particularly for BC (6.7%, 95% CI: 2.87, 10.43%) and NO_2 exposure (4.1%, 95% CI: 2.12, 6.12%), while population within the age 5–44 years were more influenced by $PM_{2.5}$ and PM_{10} exposure in addition to BC aerosols. However, within the group, differences were insignificant. In absence of socio-economic profile of the targeted population, we used place of death as a proxy (Zhang et al., 2017), considering non-institutional deaths as an indicator of low socioeconomic condition, lack of health insurance and other health care facilities leaving them vulnerable to high pollutant exposure (Bravo et al., 2015; Singh et al., 2018). The difference between effect estimates for institutional and non-institutional deaths was significant for all the pollutants except for BC. Non-institutional deaths were particularly high for $PM_{2.5}$ and NO_2 exposure. The risk of mortality was further classified by seasons. In winter we note a significant association of mortality with pollutants. BC was associated with a 6.80 (95% CI: 2.37, 11.23%) increase

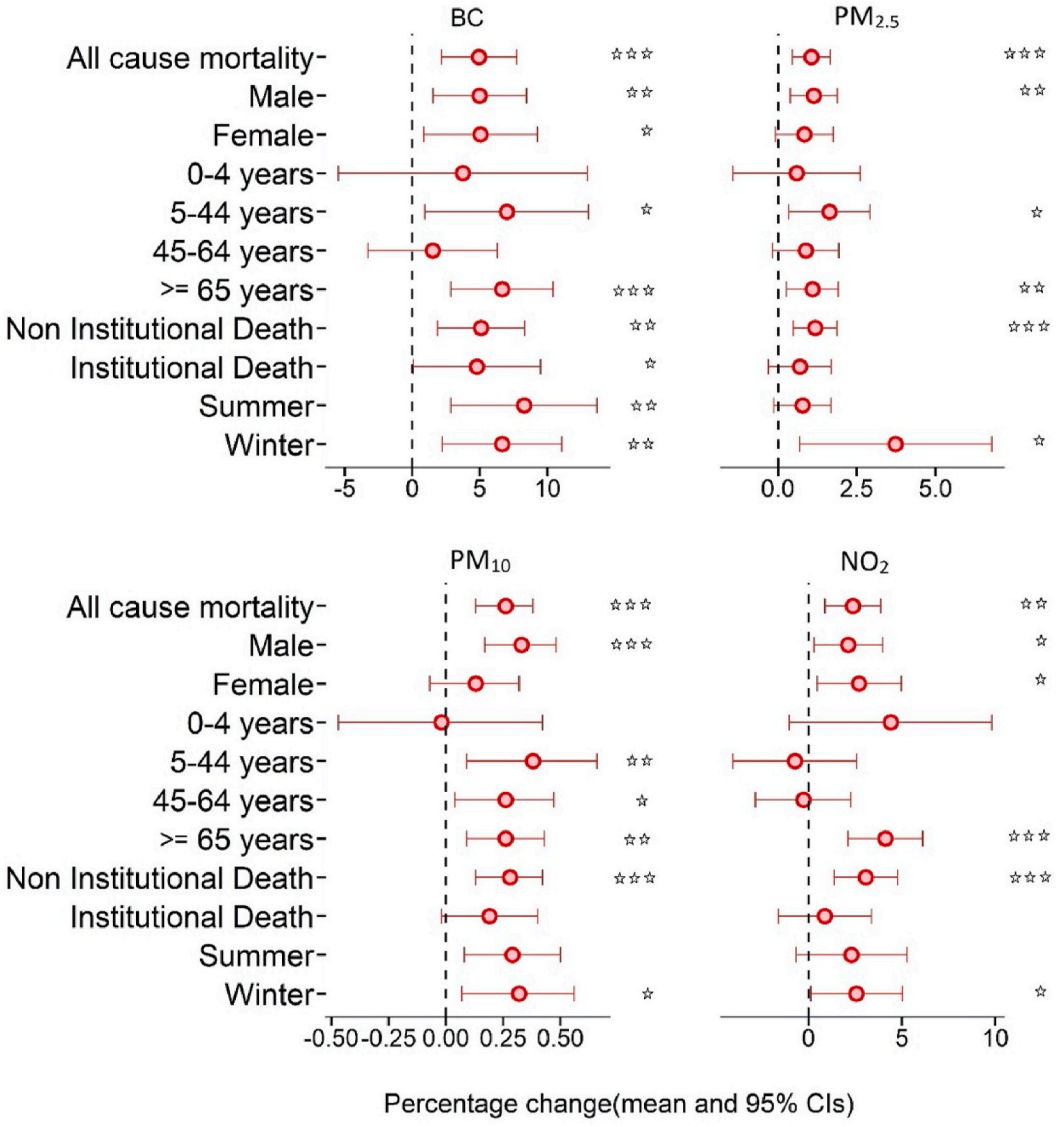

Fig. 3. Air pollution exposure and mortality stratified by sex, age and regional climate.

in mortality during winter, while the association with $PM_{2.5}$ was 3.73% (95% CI: 0.68, 6.79%), and with NO_2 was 2.57% (95% CI: 0.13, 5.01%). However, there was an insignificant difference between regression coefficients in summer and winter. Also, the impact was low and insignificant for the rest of the seasons (Fig. 3). Previous studies, in general, have shown that the effect of BC, particulate matter (PM_{10}, $PM_{2.5}$) and gaseous pollutant (SO_2, NO_2, and O_3) were high during winter (Geng et al., 2013; Zhang et al., 2017). However, some studies have also concluded a higher effect of PM_{10} during summer (Guo et al., 2014).

3.2. Sensitivity analysis

Fig. 4 shows the visual interpretation of the distributed lag nonlinear model (dlnm) for each air pollutant. The lag effect varied by the days of exposure and remained statistically significant between lag 1 to lag 3 days for $PM_{2.5}$, PM_{10} and NO_2 whereas, BC showed an extended lag effect up to 5 days. The cumulative risk of exposure for different air pollutants at different lag is also included in Table S6. We found an increase in cumulative risk with an increase in lag days that reached a maximum at lag 5 for BC (8.95%, 95% CI: 5.27–12.63%) and $PM_{2.5}$ (1.43 95% CI: 0.58, 2.28%), while NO_2 displayed maximum risk at lag 2 (3.00%, 95% CI: 1.35, 4.65) and decreased thereafter. This may typically indicate that the consequent effects of BC aerosols on human mortality extended till the 5th day of exposure, reported similarly (for lag 3) in Shanghai by Geng et al. (2013). In contrast, PM_{10} did not indicate any cumulative lag effect even after 0–7 days of exposure with the lowest attributable mortality varying between 0.28% and 0.34%. A single and cumulative lag model up to a 5-day lag and 5 days mean (Lag 1 to Lag 5) for Tmean and RH was fitted, but no appreciable change was found in the estimates except for BC in temperature lag (Tables S7–S8). Further, the change of degree of freedom (3–6) of temporal effect on mortality did not significantly influence the coefficients (Table S9).

3.3. Exposure-response curve

The exposure-response curves with a 95% confidence interval for individual air pollutants are included in Fig. 5. A dose-response curve with two and three degrees of freedom was found for BC and $PM_{2.5}$, respectively by minimizing the GCV score. However, the curve was found to be linear in the range of 0–7 and 12–15 μg^{-3} for BC and above 50 μg^{-3} for $PM_{2.5}$ (Fig. 5). All the pollutants exhibited an increase in mortality with a corresponding increase in exposure while the trend was not always linear. A considerable increase in the log mortality rate due to BC aerosols was evident for an exposure >15 $\mu g\ m^{-3}$. Similarly for $PM_{2.5}$ exposure, the mortality rate remained almost constant for concentration <60 $\mu g\ m^{-3}$ but increased particularly beyond >60 $\mu g\ m^{-3}$. On contrary, the exposure-response curve for NO_2 and PM_{10} was linear indicating a gradual increase in mortality with a corresponding increase in the level of exposure. For all the cases, the association remained significant and robust due to the narrow confidence band and established the harmful effect of pollutants on human mortality. Although the exposure-response association is subject to the regional features like adaptability/resilience of the population, still we note a similar kind of exposure-response curve as reported in Jinan (Zhang et al., 2017), Shanghai (Wang et al., 2013), and in Delhi (Maji et al., 2017).

3.4. Mortality risk during haze days

In this section, we have established the impact of haze on human mortality, constrained by the impact of individual air pollutants. It should be noted that 68% of monitoring days (2009–2016) were haze days which accounts for both very high $PM_{2.5}$ (mean ± SD: 62 ± 36 $\mu g\ m^{-3}$) and BC exposure (9.5 ± 5.5 $\mu g\ m^{-3}$) thereby, posing a significant threat to the human health. BC aerosols induce a severe impact during haze days with a 6.11% (95% CI; 2.91–9.31%) increase in mortality for every 10-unit increase in BC concentration (Table 2). Similarly, the effect estimates for fine particulates (1.77%, 95% CI: 1.05–2.50%) and NO_2 (3.06%, 95% CI: 1.41–4.71%) during haze days were considerably high and significant. We also note a statistically significant effect of PM_{10} on human mortality during haze days accounting for a 0.38% increase in mortality. In contrast, no significant association of mortality and air pollution was noted during no haze days.

The higher effect of pollutants during haze established the role of BC, fine particulates, and NO_2 exposure on pre-mature mortality in Varanasi, which has also been reported over other Asian cities. Likewise, in Guangzhou, Liu et al. (2014) reported a 3.4–10.4% increase in air pollution-induced mortality during haze days; Goldberg et al. (2001) noted a 1.4% increase in mortality in Montreal due to incremental haze effects and in Hong Kong, Chak Ho et al. (2018) concluded a significant influence of haze events on mortality risk, especially for the population with mental and behavioral disorders. We also tried to isolate the seasonal influence of haze events, as we hypothesized summertime haze to be mostly dominated by dust particles while wintertime haze consisting mainly of a higher amount of smoke particles (Banerjee et al., 2020). However, no statistically significant variation between the seasons was noted in Varanasi (data not shown). Such discrepancies in effect estimates are reported in Guangzhou by Liu et al. (2014) with higher mortality, particularly during cold seasons. Therefore, we also conclude that it is critical to account for the individual effect of aerosol components on mortality during haze days.

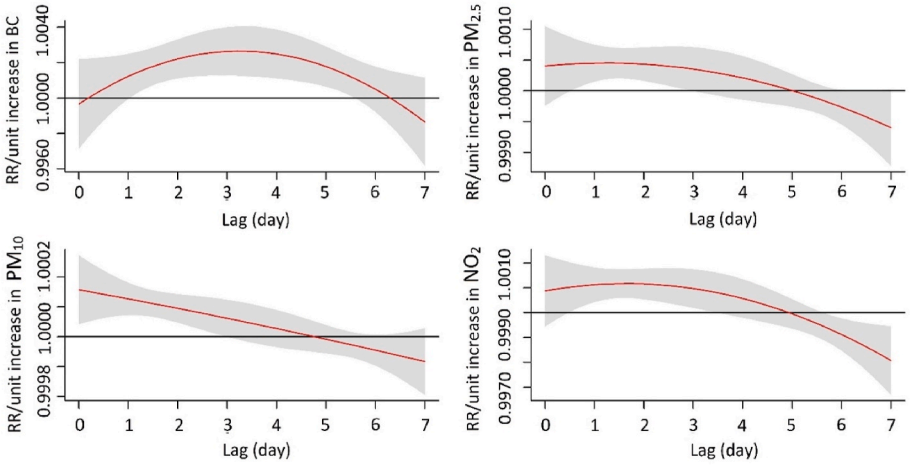

Fig. 4. Lag patterns for air pollution exposure and risk of mortality.

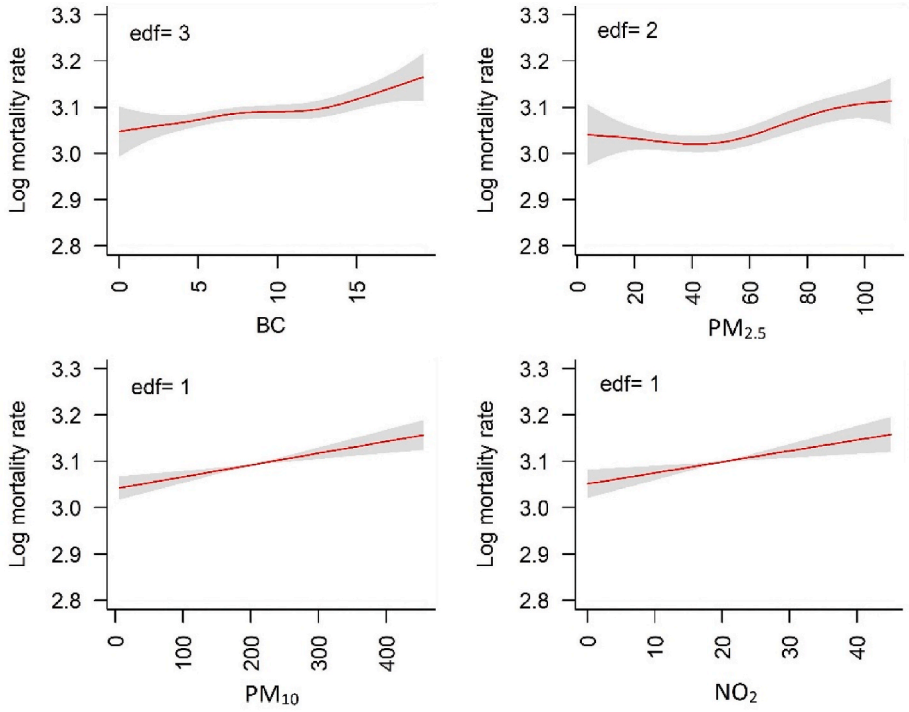

Fig. 5. Exposure-response curves of individual air pollutants.

Table 2
Effect of air pollutants on all-cause mortality during haze and no-haze days.

	Haze Days	No Haze days
	% Change in mortality (95% CI)	% Change in mortality (95% CI)
BC	**6.11 (2.91, 9.31)**	4.15 (−0.61, 8.92)
PM_{10}	**0.38 (0.23, 0.52)**	0.07 (−0.15, 0.30)
$PM_{2.5}$	**1.77 (1.05, 2.50)**	−0.61 (−1.69, 0.46)
NO_2	**3.06 (1.41, 4.71)**	1.01 (−1.73, 3.76)

Note. Effects of different pollutants on mortality with 95% CI in terms of percentage change in mortality per 10 μg m^{-3} change of pollutant. Values in bold are significant at $p < 0.01$. A "haze day" was defined as a calendar day when the visibility is < 5 km for at least 1 h, relative humidity <95%, with no fog, no mist, and no precipitation (Ho et al., 2018).

4. Summary and conclusions

The effect of multiple air pollutants including black carbon aerosols and trace gases (NO_2, SO_2, and O_3) on all-cause mortality is reported for the first time over South Asia. We examined single and multi-pollutant effects on all-cause non-accidental mortality over a typical urban pollution hotspot at central IGP. The individual effect of BC, $PM_{2.5}$, PM_{10}, and NO_2 exposure on total mortality was statistically significant. The combined exposure of BC, NO_2, and $PM_{2.5}$ (residual $PM_{2.5}$) in the multi-pollutant model showed an increase in effect estimates compared to their individual exposure. Among the effect modifiers, both sex and season did not yield any significant differences except the exposure to PM_{10}, while the population within the age group 5–44 years were at greater risk of air pollution-associated mortality. A delayed response of air pollutants on mortality was found that existed up to 0–7 lag days. Further, the dose-response showed an increase in total mortality with the increase in pollutant concentration that was not necessarily linear. The effect estimates of air pollutants based on haze and no-haze days showed higher non-accidental mortality and an increase in the individual effect of air pollutants, particularly for BC and NO_2 exposure during haze events.

The study has two great strengths. Firstly, it considers exposure to black carbon aerosol and establishes its independent effect on all-cause mortality. The detrimental effect of black carbon aerosols remains to be duly acknowledged as limited evidence exists for BC and its effect on public health in India, if not over South Asia. Secondly, the study analyses the multipollutant exposure, which has been reported only over a few urban hotspots thereby, limiting our fundamental understanding of how air pollutants modify individual's effects when they coexist in the environment. Besides, our analysis does include some limitations, especially in considering exposure data from a single monitoring station that could have led to some bias in the estimates. Also, the total mortality could be underreported as not all deaths are registered, but we assumed that the registered deaths satisfactorily displayed the true burden of mortality. There is also a potential for residual confounding, particularly if there were unmeasured time-varying confounders with seasonal patterns that matched pollutants and mortality. Besides, the missingness in included covariates may also lead to bias. A relatively small number of cause-specific mortality data also limits associations of cause-specific mortality with pollution exposure. Also, the lack of information on individual characteristics of the population was unknown.

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CRediT authorship contribution statement

Nidhi Singh: Conceptualization, Methodology, Formal analysis, interpretation, Writing - review & editing, Writing - original draft. **Alaa Mhawish:** Methodology, Formal analysis. **Tirthankar Banerjee:** Conceptualization, Methodology, Writing - review & editing, Writing - original draft, interpretation as well as review and editing of the draft, Funding acquisition. **Santu Ghosh:** Methodology, Writing - review &

editing, Writing - original draft, interpretation as well as review and editing of the draft. **R.S. Singh:** Writing - review & editing, Writing - original draft, interpretation as well as review and editing of the draft. **R. K. Mall:** Methodology, Writing - review & editing, Writing - original draft, interpretation as well as review and editing of the draft, Resources, Funding acquisition.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.atmosenv.2020.118088.

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