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Daily nonaccidental mortality associated with short-term $PM_{2.5}$ exposures in Delhi, India

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Background: Ambient particulate matter of aerodynamic diameter less than 2.5 microns $PM_{2.5}$) levels in Delhi routinely exceed World Health Organization (WHO) guidelines and Indian National Ambient Air Quality Standards (NAAQS) for acceptable levels of daily exposure. Only a handful of studies have examined the short-term mortality effects of PM in India, with none from Delhi examining the contribution of $PM_{2.5}$.

Objectives: We aimed to analyze the association between short-term $PM_{2.5}$ exposures and daily nonaccidental mortality in Delhi, India.

Methods: Using generalized additive Poisson regression models, we examined the association between daily $PM_{2.5}$ exposures and nonaccidental mortality between June 2010 and December 2016. Daily exposures to $PM_{2.5}$ were estimated using an ensemble averaging technique developed by our research group, and mortality data were obtained from the Municipal Corporations of Delhi and the New Delhi Municipal Council.

Results: Median exposures to $PM_{2.5}$ were 91.1 μg/m^3 (interquartile range = 68.9, 126.2), with minimum and maximum exposures of 21.4 μg/m^3 and 276.7 μg/m^3, respectively. Total nonaccidental deaths recorded in Delhi during the study period were 700,512. Each 25 μg/m^3 increment in exposure was associated with a 0.8% (95% confidence intervals [CI] = 0.3, 1.3%) increase in daily nonaccidental mortality in the study population and a 1.5% (95% CI = 0.8, 2.2%) increase in mortality among those with 60 years of age or over. The exposure-response relationship was nonlinear in nature, with relative risk rising rapidly before tapering off above 125 μg/m^3. Meeting WHO guidelines for acceptable levels of exposure over the study period would have likely averted 17,526 (95% CI = 6,837, 25,589) premature deaths, with older and male populations disproportionately affected.

Discussion: This study provides robust evidence of the impact of short-term exposure to $PM_{2.5}$ on nonaccidental mortality with important considerations for various stakeholders including policymakers and physicians. Most importantly, we find that reducing exposures significantly below current levels would substantially decrease the mortality burden associated with $PM_{2.5}$.

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Introduction

Global evidence indicates that ambient air pollution is a major public health hazard, with harmful associations being reported with respect to all-cause mortality,[1–3] and cause-specific mortality from cardiovascular and respiratory diseases.[4–7]

Air pollution exposure is a major and growing risk factor for ill health and premature death in India, with the most recent subnational burden of disease estimates for India estimating that over 980,000 deaths annually are associated with exposure to ambient particulate matter of aerodynamic diameter less than 2.5 microns ($PM_{2.5}$).[8] These estimates are based on concentration-response curves estimated elsewhere and have been treated with scepticism by the Indian administrators. With a rapidly growing transport fleet, vast availability of highly polluting coal, under-regulated construction activity, and increasing migration

What this study adds

Delhi is one of the most polluted cities on the planet, with ambient $PM_{2.5}$ levels routinely exceeding acceptable levels and contributing to a significant burden of disease. This is the first study to provide evidence of the association between short-term $PM_{2.5}$ exposures and mortality in Delhi. We found that $PM_{2.5}$ exposures were significantly associated with increased daily nonaccidental mortality, with adults over 60 years of age disproportionately affected. Given the nature of the E-R relationship, meeting WHO and National standards for air quality could significantly reduce the mortality burden from $PM_{2.5}$ exposures.

to major urban centers, India is home to 21 of the 30 most polluted cities globally.[9]

Although large parts of India experience air pollution beyond levels considered acceptable for daily and annual exposure by the WHO, Delhi has received particular media and policy attention over the years.[9] Annual average exposures to $PM_{2.5}$ in Delhi exceeded 130 μg/m^3 in 2016, having grown steadily over the 7 years between 2010 and 2016, with large spatial and temporal variations.[10] Seasonal factors, high emissions related to fireworks combustion during religious events and periodic crop-stubble burning also contribute to extremes of $PM_{2.5}$ in the winter which can sometimes breach 1,000 μg/m^3 in the very short term.[11]

High levels of PM exposures in the short and long term have been shown to have health effects in India as well, with ambient PM exposure a key risk factor for the burden of disease in the country.[8,11–17] However, there is limited evidence on the short-term effects of $PM_{2.5}$ on mortality and morbidity in India, with only one time-series study conducted till date anywhere in the country documenting the effect on daily mortality.[18,19] Cited regularly by policymakers as an area for remedial action, the generation of in-country studies of this nature would bolster the case for action by building a local evidence base on the short-term effects of ambient $PM_{2.5}$ exposure.

Methods

Environmental data

Monitored $PM_{2.5}$ data were not widely available for large periods especially in the early part of our study period (2010–2016), with spatial and temporal resolution of monitored $PM_{2.5}$ increasing gradually toward the end of said period. To ensure adequate temporal coverage of $PM_{2.5}$ data, we utilized predicted $PM_{2.5}$ from a model developed by members of this study group as part of the India GEOHealth Hub.[20] The model described in detail by our colleagues utilizes an ensemble averaging approach to predict $PM_{2.5}$ at high temporal and spatial resolution (daily at 1 × 1 km grids over Delhi).[10] In a multistage approach, the model utilized satellite-derived AOD, ground-monitored $PM_{2.5}$, land-use variables, meteorological variables, and chemical transport models to predict 24-hour average $PM_{2.5}$ exposures at 1 × 1 km grids across Delhi with a cross-validated prediction accuracy of 80% over the study period.

From this model, we took the gridded 24-hour $PM_{2.5}$ predictions and averaged them at a city level to derive a single daily $PM_{2.5}$ exposure metric. Daily lagged exposure metrics were generated as well. Meteorological variables such as temperature and relative humidity were obtained from global climate reanalysis data of the European Centre for Medium-Range Weather Forecasts (ECMWF), which are downscaled from 25 × 25 km grids. Daily lagged variables were generated for temperature and relative humidity as in the case of $PM_{2.5}$.

Mortality data

The registry of births and deaths in the National Capital Territory of Delhi are maintained by the five municipal corporations, namely the North, South, and East Delhi Municipal Corporations (which fall under the Municipal Corporation of Delhi), the New Delhi Municipal Council (NDMC), and the Delhi Cantonment Board. We obtained deidentified mortality records for the period 2010–2016 from the Municipal Corporation of Delhi (North, South, and East) and the New Delhi Municipal Council. Deaths registered in these regions together accounted for 97%–99% of all deaths that occurred in Delhi during the study period. Data were not available for the Delhi Cantonment Board area of the city because this area falls under the purview of the Indian Army.[21] These data were then cleaned and aggregated to generate a count for daily deaths to be used in our analyses. Although the availability of data on age for all, and sex for three of the four municipal corporations (not available for NDMC) enabled us to conduct further stratified analyses, discrepancies in coding the cause of death prevented us from conducting analyses of cause-specific mortality.

Statistical analyses

We studied the association between daily count of nonaccidental deaths and daily $PM_{2.5}$ exposure. The analysis period covered six and a half years (June 2010 to December 2016), and we examined the effect of temperature both as a variable in the model and as an effect modifier in the relationship between $PM_{2.5}$ and daily nonaccidental mortality. We used generalized additive models (GAMs) in a time-series design to examine the association, and the relationship was modeled using quasi-Poisson regression to account for overdispersion.

In our GAM framework, we used a linear term for $PM_{2.5}$ to generate point estimates, and a penalized spline to explore non-linearity in the exposure-response relationship. We also examined the effect of different lagged exposures to $PM_{2.5}$ using the distributed lag non-linear model (DLNM) framework before choosing the lag-0 $PM_{2.5}$ average as our exposure of interest.[22,23] Additionally, we also examined the effect of different lagged effects of temperature and included in our final model a penalized spline for 5-day average temperature before the day of the event. Also included in the model were a penalized spline for 5-day relative humidity and a natural spline for time in days with 1.5 degrees of freedom per season per year to account for long-term trends and year-specific seasonality. In line with previous published work, we also recognized in our model that Delhi has five distinct seasons.[24] A dummy variable for day of the week was also included. The final model used was as follows:

$$\log\left(E\left[Y_i\right]\right) = \alpha + \beta_1 . PM_{2.5} + \beta_2 . s(Temp) + \beta_3 . s(RH) + \beta_4 . ns\left(Time_i, df = 49\right) + \beta_5 . DOW_i$$

where $E\left[Y_i\right]$ is the expected count of nonaccidental deaths on day i; α is the intercept; β_{1-5} are regression coefficients; $PM_{2.5}$ is the 2-day average $PM_{2.5}$ exposure; *Temp* is 5-day average temperature exposure; *RH* is 5-day relative humidity; *Time* is time in days from June 1, 2010; *DOW* is day of the week; *s* is a penalized spline function, and *ns* is a natural spline.

We conducted sensitivity analyses using same-day temperature as a linear term with thin plate spline of temperature and $PM_{2.5}$, or as a categorical term (Cold [<15°C]; Normal [15–31°C], and Hot [≥32°C]) in interaction with the exposure of interest. We also examined effect modification by season, sex, and different age strata through the introduction of an interaction term with the primary exposure. We excluded outliers of exposure (1st and 99th percentiles) in our analyses. All statistical analyses were conducted using R statistical software version 4.0.1 (R Foundation for Statistical Computing, Vienna, Austria) using the *mgcv* package version 1.8-31 and *dlnm* package version 2.4.2.

Results

Summary statistics

Between June 2010 and December 2016, there were 700,512 nonaccidental deaths across the four municipal corporations for which we have data. The median nonaccidental deaths per day were 288 (interquartile range [IQR] = 258, 320), median $PM_{2.5}$ exposure was 91.1 μg/m^3 (IQR = 70.1, 126.2), and the median age at death was 60 (IQR = 40, 72). There was high variability in $PM_{2.5}$ exposure across seasons, with the minimum recorded exposure being 27 μg/m^3 (higher than WHO guidelines for daily

exposure), and the maximum being over 10 times levels considered acceptable. Summary statistics of other meteorological variables and breakdown of key variables by strata are provided in Table 1 below.

Regression results

Table 2 presents the increases in relative risk of mortality for every 25 μg/m^3 increase in $PM_{2.5}$ exposure overall across the entire dataset as well as among the various strata we examined. Overall, we observed a 0.8% (95% confidence intervals [CI] = 0.3, 1.3%) increase in nonaccidental mortality with every 25 μg/m^3 increase in exposure. There was significant heterogeneity across age groups with every 25 μg/m^3 increase in exposure contributing to a 1.5% (95% CI = 0.8, 2.2%) increase in mortality among those with 60 years of age or over, and a 0.3% (95% CI = 0.1, 0.5%) increase in those between 40 and 59 years of age. A significant effect was also observed in both males and females, with each 25 μg/m^3 exposure increment contributing to 0.9% and 0.7% increase in mortality, respectively. There was a large negative effect observed in those under the age of 40, and this population subgroup was therefore excluded from the final analyses presented below, with detailed information on effect estimates and exposure-response for this group presented in eTable 2; http://links.lww.com/EE/A150. Detailed results are provided below in Table 2. A plot with effect estimates and 95% CIs are also presented in Figure 1.

Exposure response

The overall exposure-response (E-R) relationship showed a steep increase in risk below 75 μg/m^3, reduction in slope between 75 and 125 μg/m^3 and tapering off thereafter as shown in Figure 2. This exposure-response relationship was mirrored in the over 60 and 40–59 age strata (Figure 3). Both males and females showed a rapid increase in risk below 125 μg/m^3 albeit at different scales of risk, with E-R curves differing beyond that level of exposure. There was significant heterogeneity in exposure-response across seasons with winter and spring showing increasing risk throughout the exposure range (eFigure 1b; http://links.lww.com/EE/A150).

Table 1.

Descriptive statistics

Variables	Median	Min	25th %ile	75th %ile	Max
$PM_{2.5}$ exposure					
Daily exposure (μg/m^3)	91.1	21.4	68.9	126.2	276.7
Weather conditions					
5-day average temperature (°C)	25.7	8.4	17.8	28.5	36.9
5-day average relative humidity (%)	69.3	9.2	54.6	80.1	98.3
Daily deaths (count)					
Overall	279	88	252	308	485
Age Strata (years)					
≥60	136	37	118	159	274
40–59	74	18	64	83	144
<40	68	29	60	76	132
Sex[a]					
Male	148	32	132	167	277
Female	94	21	83	107	169
Age at death					
Overall	60	0	40	72	95

[a]Analyses restricted to East, North and South Delhi Municipal Corporations

Table 2.

Relative risk change for every 25 μg/m^3 increase in $PM_{2.5}$ exposure

	For 25 μg/m^3 increase in $PM_{2.5}$ exposure
Group	**Relative risk (95% CI)**
Overall	**1.008 (1.003, 1.013)**
Age Strata	
≥60	**1.015 (1.008, 1.022)**
40–59	**1.003 (1.001, 1.005)**
Sex[a]	
Male	**1.009 (1.002, 1.015)**
Female	**1.007 (1.005, 1.010)**

[a]Analyses restricted to East, North and South Delhi Municipal Corporations

Effect modification

We analyzed effect modification by temperature using interaction terms. When introducing either a linear temperature term (as a thin plate spline in interaction with the exposure), or as a categorical term in interaction with the exposure, we did not observe any effect modification by temperature. We also tested effect modification by season of death using a categorical variable for season in interaction with the exposure of interest. We observed differing E-R curves across different seasons with Winter and Spring showed an upward slope across the entire exposure spectrum, although summer showed an upward slope for exposures below 100 μg/m^3 (eFigure 1c; http://links.lww.com/EE/A150). We introduced different degrees of freedom for the natural spline of time to control for long-term trends and seasonality but observed no significant changes in E-R.

Potential mortality displacement

To understand the negative slopes we observed for the under-40 age category, we conducted analyses to examine whether there was a harvesting effect at play. Harvesting or mortality displacement occurs when high-risk groups (such as the elderly or those with preexisting conditions) in a population experience an increase in mortality in the short-term relative to healthy subjects in a population due to an external factor such as air pollution exposure.[25,26] In our case, we hypothesized that there would be heightened risk at shorter lags of exposure with risk declining thereon. We examined the $PM_{2.5}$ exposure-mortality association over an increasing lag time up to 30 days using the DLNM approach outlined by Bhaskaran et al., (2013). This showed heightened risk at shorter lags with risk declining there-on, indicative of 'harvesting' or short-term mortality displacement for the whole population, but not specifically for under-40s.[22] Our second hypothesis was that high-risk subjects were likely to die at lower ambient concentrations of $PM_{2.5}$, with healthier subjects remaining largely unaffected, causing a sharp increase in risk at lower exposures, with either a tapering off or even an inverse slope thereafter. If misread, these inverse slopes could be construed as conferring a protective effect with rising levels of air pollution, which we know it does not. We therefore explored the potential for a harvesting effect by examining only days under the median exposure of 91.1 μg/m^3. These analyses did not reveal any significant change in E-R for those under-40 (eFigure 1a; http://links.lww.com/EE/A150).

Mortality averted by reducing exposures

To calculate the potential mortality averted by improving ambient air quality by 25 μg/m^3, we utilized the approach followed by Dominici et al., (2006) where M or attributable mortality is defined as M = (exp (β * Δx) -1)*N, with β being the RR estimate for a 1 μg/m^3 increase in 2-day average $PM_{2.5}$ exposures, Δx being 25 μg/m^3, and N being the total number of deaths in a defined time period (or for the specific subcategory).[27] Table 3 below lists the annual reduction in deaths with a 25 μg/m^3 reduction in exposure across different strata for the year 2016. Our estimates showed the highest risk population being those

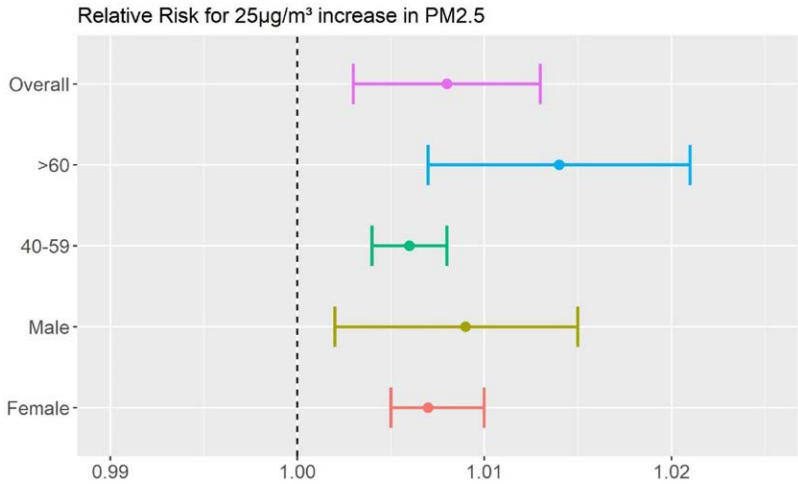

Figure 1. Relative risk change for every 25 µg/m^3 increase in $PM_{2.5}$ exposures.

over the age of 60 years, and this is reflected in the potential mortality reduction, with a 25 µg/m^3 reduction in exposure over the entire study period potentially averting 7,697 (95% CI = 6,788, 8,607) deaths among that age group.

We also estimated the potential mortality averted if exposure levels were reduced from the median of 91.1 µg/m^3 to reference levels such as the WHO guidelines for acceptable exposure and the National Ambient Air Quality Standards for acceptable exposure. These results are presented below in table 4.

Reducing the exposure from the median of 91.1 µg/m^3 to the WHO guideline level of 10 µg/m^3 would have potentially averted 17,526 (95% CI = 6,837, 25,589) deaths (approx. 7 deaths a day) among the entire study population, with the vast majority of averted mortality observed among the elderly population, and largely among men. Similarly, reducing exposure from the median to the NAAQS for 24-hour exposure of 40 µg/m^3 would have potentially averted 10,983 (95% CI = 4,297, 17,501) deaths during the course of the study period (approx. 4.5 deaths per day).

From the exposure-response relationships generated as part of this study, it is evident that the risks posed are nonlinear in nature requiring interventions that aim to achieve reductions in ambient $PM_{2.5}$ that are far below the current median exposure in our study of 91.1 µg/m^3 and even below those of the NAAQS. Additionally, it is also clear that the effect is heterogenous across age strata, and the increased risk to sensitive groups must be taken into effect in planning appropriate interventions. Consequently, exposures must be reduced significantly from current levels to ensure appropriate reductions in risk especially to susceptible populations.

Discussion

This study represents one of the first to examine the effect of $PM_{2.5}$ exposures on daily mortality in an Indian setting.[18,19] We found in our analyses a strong association between changes in $PM_{2.5}$ exposures and increased mortality in Delhi, with the effect being far stronger in older adults compared with the younger age groups. There were also heterogeneities in effects across sex. Exposure-response for the overall study population indicated a rapid increase in risk at lower exposures with a tapering off at

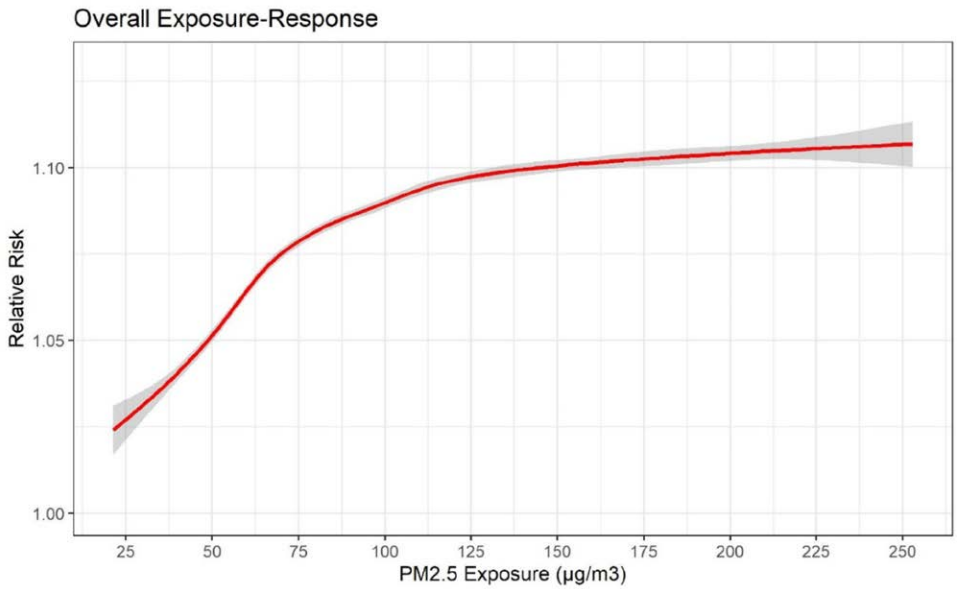

Figure 2. Exposure-response relationship among the entire study population.

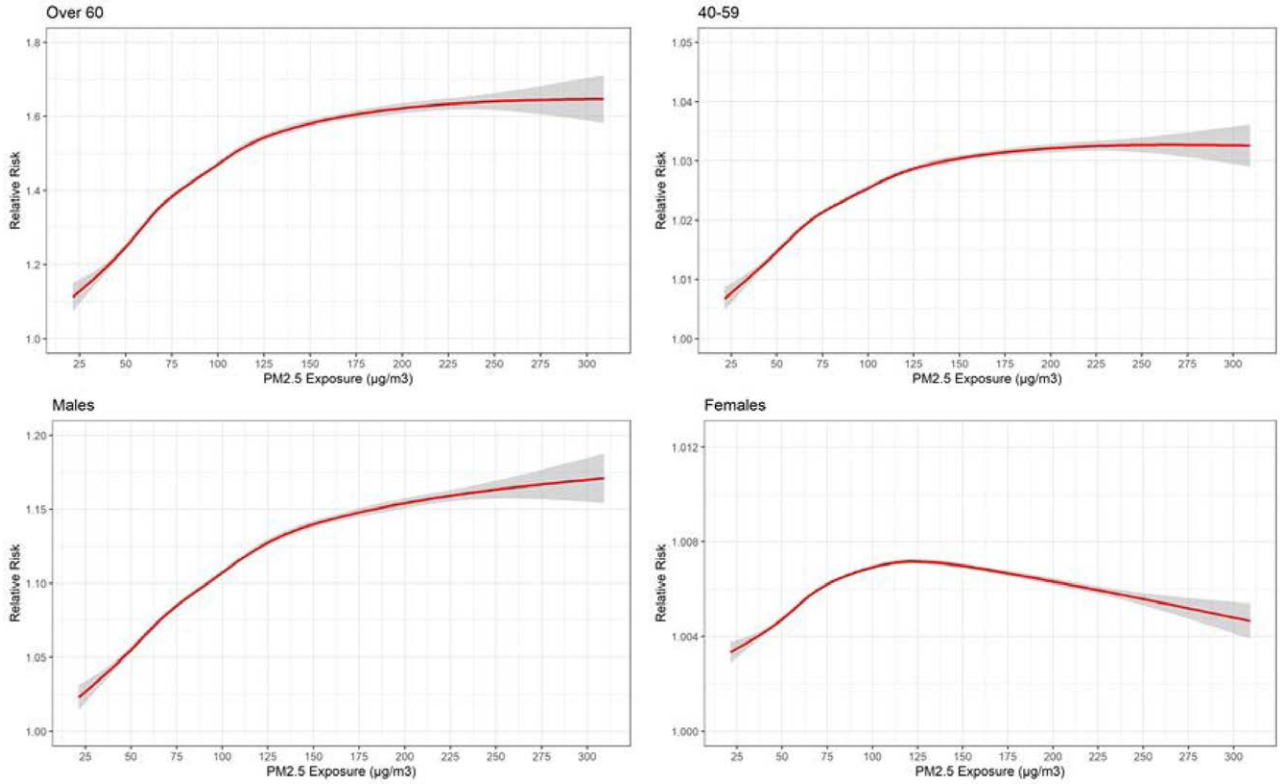

Figure 3. Exposure-response relationship among different strata.

levels above 125 µg/m^3. Similar heterogeneities were observed across other strata of interest.

Overall, while the effects on mortality in our study population were significant, they were smaller in magnitude than those reported in previous studies conducted in western settings.[2,3,28,29] For a 10 µg/m^3 increase in $PM_{2.5}$ exposure, we estimated a 0.31% increase in nonaccidental mortality which was comparable to results from a multi-city study conducted in China in 2016 that reported a 0.22% increase in total mortality for each 10 µg/m^3 increment in exposure.[30] Our estimates were lower, however, than those of a multi-city study conducted in the United States which estimated 1.18% increase in total mortality with each 10 µg/m^3 increase in 2-day average $PM_{2.5}$ exposure.[3]

The weaker effects of $PM_{2.5}$ on mortality observed in our study compared with those conducted in Europe or the United States may be attributable to several factors. First, various source apportionment studies conducted in Delhi have shown that the composition of $PM_{2.5}$ in the city comprises a large proportion of crustal material from road and construction dust, wind-blown mineral dust, and other sources.[31–33] These proportions are comparable to those observed in several Chinese cities, but much higher than in European or American cities.[30] Although for the purposes of this study, we maintain the generally accepted principle of $PM_{2.5}$ equitoxicity, it is likely that the relative lower toxicity of crustal material may have contributed to our lower estimates.[34,35]

Second, the E-R observed in our study indicated a plateauing of risk above 125 µg/m^3 of $PM_{2.5}$. A similar saturation effect was observed in Chinese studies as well.[30] analyzing the E-R across different strata also showed significant heterogeneity with those below the age of 40 years showing no effect on mortality. When we examined whether this was a by-product of mortality displacement or harvesting, we did not observe any evidence of this, with the E-R for the under-40 age group remaining negative in slope (eFigure 1a; http://links.lww.com/EE/A150). We also observed in our study differences in effect magnitude between males and females, which may be attributable to varying levels of outdoor activity, profession, and therefore differing exposure levels to ambient $PM_{2.5}$.

Our results hold several important findings of public health significance. First, our finding that vulnerability to short-term $PM_{2.5}$ exposures is much higher in individuals above the age of 60 is consistent with other studies conducted around the world and indicates the need to deliver tailored messaging to protect this vulnerable subgroup.[3,36,37]

Table 3.

Mortality potentially averted for every 25 µg/m^3 reduction in exposure

Group	Total deaths	Reduction in deaths over study period for 25 µg/m^3 reduction in exposure (95% CI)
Overall	700,512	5,474 (2120, 8812)
≥60 years	349,473	5,113 (2856, 7357)
40–59 years	183,196	598 (198, 997)
Male	373,983	3,166 (753, 5564)
Female	234,362	1,723 (1201, 2243)

Table 4.

Potential deaths averted over study period by reducing exposure from the median exposure (91.1 µg/m^3) to reference levels

Group	WHO—10 µg/m^3 (95% CI)	NAAQS—40 µg/m^3 (95% CI)
Overall	17,526 (6837, 25,589)	10,983 (4297, 17,501)
≥ 60 years	16,202 (9138, 23,081)	10,111 (5725, 14,346)
40–59 years	1,928 (641, 3204)	1,212 (403, 2011)
Male	10,127 (2,433, 17,622)	6,344 (1,530, 10,996)
Female	5,519 (7,161, 3,862)	3,460 (2,424, 4,483)

Second, the heterogeneity in effect across different age groups and sexes indicates the need for targeted interventions that reduce exposures based on preexisting risk profiles, modes of exposure (e.g. profession), and time spent outdoors.

Third, the E-R curves show us that risk rises most rapidly at lower concentrations, tapering off at levels above 125 μg/m^3. From a policy perspective, it is vital therefore that a roadmap to improve ambient air quality be not only focused on the extreme exposures of winter, but also on those closer to the median exposure of 91.1 μg/m^3 experienced throughout most of the year. Additionally, the targets for improvement in air quality must also be commensurate to the added risk, which in this case would mean achieving WHO guidelines for acceptable exposure.

Fourth, the absence of an effect on mortality for those under the age of 40 does not indicate that air pollution is not harmful to this population subgroup. Indeed, the wealth of evidence from India and elsewhere showcases the harmful effects of exposure on morbidity for children and young persons,[8] and the inference we draw from these results are that young persons are not dying from short-term changes in exposure and that they may be more affected by longer term exposures.

As one of the first studies examining the acute effects of $PM_{2.5}$ on mortality in India, our study would hopefully not be the last. Differing $PM_{2.5}$ exposures and compositions in different parts of the country necessitate the generation of more local evidence, and as such, the findings of this study are not generalizable to the rest of the country.[31,38,39] Our study does however have additional strengths including a large pool of data which allowed the study to have more than adequate power to estimate the effects observed.

Our study did also have some limitations. Given the use of a city average $PM_{2.5}$ metric as our exposure, there is likely to be some nondifferential exposure misclassification which may lead to an underestimate of the association. Second, the quality of cause of death coding made it impossible for us to undertake analyses by cause of death. There is also a lack of clarity on how these raw cause of death codes are cleaned and analyzed before being published as part of annual cause of death statistics. Third, our model did not generate predictions for other pollutants, and we were therefore unable to include additional variables for Nitrogen or Sulphur Oxides in our models. Fourth, we did not have information on the sex of the individual for one of the four municipalities, which meant the stratification of our analyses by sex was limited to 3 of 4 municipalities. Finally, since this study only captured deaths in Delhi, the results are not generalizable to other parts of the country.

Conclusion

As one of the largest epidemiologic studies conducted in India on the harmful effects of air pollution, this study provides robust evidence of the impact of short-term exposure to $PM_{2.5}$ on nonaccidental mortality. Although the magnitude of the effects were lower than those observed in Western Europe or the United States, the far higher levels of ambient $PM_{2.5}$ observed, the nature of the E-R, and the significant impact on vulnerable subgroups represents the need for substantial and sustained year-round action to reduce all sources of air pollution in Delhi and the rest of India.

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