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Nonlinear relationships between air pollutant emissions and $PM_{2.5}$-related mortality in the Beijing-Tianjin-Hebei region

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Abstract

A direct and quantitative linkage of air pollution-related health effects to emissions from different sources is critically important for decision-making since protecting public health is a final goal of air pollution controls. While many studies have attributed the $PM_{2.5}$-related health effects to emission sources, they have seldom examined the complicated nonlinear relations between the two. Here we investigate the nonlinear relationships between $PM_{2.5}$-related premature mortality in the Beijing-Tianjin-Hebei (BTH) region, one of the most polluted regions in the world, and emissions of multiple pollutants from multiple regions and sectors, through a combination of a chemical transport model, an extended response surface model, and concentration-response functions. In the BTH region, 129.2 thousand and 18.7 thousand people die annually due to long-term and short-term exposures to $PM_{2.5}$, respectively. Among all pollutants, both long-term and short-term mortalities are most sensitive to emissions of primary inorganic $PM_{2.5}$ (defined as all chemical components of primary $PM_{2.5}$ other than organics), followed by NH_3, nonmethane volatile organic compounds and intermediate volatility organic compounds (NMVOC+IVOC), and primary organic aerosol (POA). The sensitivities of long-term mortality to emissions of primary inorganic $PM_{2.5}$, NH_3 and NO_x increase significantly with the increase of reduction ratio, while

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the sensitivities to POA, NMVOC+IVOC and SO_2 roughly remain constant at various reduction ratios. The emissions of primary inorganic $PM_{2.5}$, especially those from residential and commercial sources, contribute a larger fraction of mortality in winter than in other seasons. When emissions of multiple pollutants or those from both local and nonlocal emissions are controlled simultaneously, the sensitivity of long-term mortality substantially increases with the increase of reduction ratio, and this sensitivity is much larger than the arithmetic sum of the sensitivities to emissions of individual pollutants or from individual regions. In order to achieve large marginal health benefits, we suggest that stringent control measures on primary $PM_{2.5}$ emissions should be enforced (especially in winter), that NO_x emissions should be jointly controlled over a larger region beyond BTH, and that multi-pollutant and regional collaboration control strategies should be implemented.

Keywords

$PM_{2.5}$; health effect; CMAQ/2D-VBS; Beijing-Tianjin-Hebei region; Extended Response Surface Model (ERSM)

1 Introduction

Atmospheric fine particle ($PM_{2.5}$) pollution has adverse effects on human health (Shang et al., 2013; Lelieveld et al., 2015). The effects are especially important in the Beijing-Hebei-Tianjin (BTH) urban agglomeration region, one of the most heavily polluted regions in China (Wang et al., 2017; Cai et al., 2017). It's estimated that monetize health losses caused by $PM_{2.5}$ pollution in the BTH region can reach 134.3 billion RMB, which accounts for 2.16% of the GDP in this region (Lv and Li, 2016). Protecting public health is the ultimate goal of air pollution control. Therefore, it is crucially important for environmental decision making to quantitatively link the $PM_{2.5}$-related health impacts to air pollutant emissions from different sources.

$PM_{2.5}$ pollution is formed through complex physical and chemical processes. As a result, the relationships between $PM_{2.5}$ concentrations and emissions of precursors are nonlinear, and the nonlinearity is especially significant in urban agglomerations (Zhao et al., 2015; Zhao et al., 2017; Xing et al., 2017). In addition, many studies (Martenies et al., 2015; Burnett et al., 2014; Burnett et al., 2018) pointed out that the relationships between $PM_{2.5}$ concentrations and the resulting health effects are also nonlinear. When these two relationships are overlaid, we can expect highly nonlinear relationships between the $PM_{2.5}$-associated health effects and the emissions of precursors. Quite a few studies (Fann et al., 2012; Wang et al., 2015; Heo et al., 2016; Andersson et al., 2009) have attributed the $PM_{2.5}$-associated health effects to emission sources, but to be best of knowledge, none of them have explicitly considered the aforementioned nonlinear relationships, which brings inaccuracy to the source attribution results. Therefore, it's a meaningful and also challenging scientific issue to quantify the complex nonlinear responses of $PM_{2.5}$-related health impacts in urban agglomerations to emissions of key individual precursors from major emission sources.

In this study, we quantitatively evaluate the nonlinear relationships between premature mortality caused by long-term/short-term exposures to $PM_{2.5}$ in the BTH region and

emissions of multiple air pollutants from multiple regions and sectors, by combining a three-dimensional air quality model, concentration-response functions, and a response surface modeling technique. Based on these assessment results, we provide policy suggestions that maximize the marginal benefits on public health.

2 Methods

2.1 Health impact assessment methodology

In this study, we estimate the health effects due to both short-term and long-term exposures to ambient $PM_{2.5}$. For long-term health impact assessment, Burnett et al. (2014) proposed integrated expose-response (IER) function, which were constructed by combining risk estimates from studies of ambient and household air pollution, and active/second-hand smoking that cover a full range of $PM_{2.5}$ exposure up to about 30000 μg/m^3 (Cohen et al., 2017; Burnett et al., 2014). The IER function is given by Eq. 1, which has been proved to fit best to the actual risk estimates among a variety of equation forms (Cohen et al., 2017; Burnett et al., 2014).

$$\Delta Y_i = y_{i,0} P \times \frac{RR_i - 1}{RR_i}$$

$$RR_i = \begin{cases} 1, & C < C_0 \\ 1 + \alpha_i \left[1 - e^{-\gamma_i (C - C_0)^{\delta_i}} \right], & C \geq C_0 \end{cases} \quad (1)$$

where ΔY_i refers to $PM_{2.5}$-induced mortality from endpoint i; $y_{0,i}$ refers to the actual mortality rate of endpoint i at the current $PM_{2.5}$ concentration (C); P refers to exposed population; C_0 refers to threshold $PM_{2.5}$ concentration below which no health impact is expected. α_i, γ_i, and δ_i are regression parameters for endpoint i. According to the IER function, the change in mortality due to a unit concentration change decreases significantly with the deterioration of $PM_{2.5}$ pollution. Recently, Burnett et al. (2018) developed new concentration-response functions for long-term $PM_{2.5}$ exposures based on only cohort studies of ambient air pollution, which resulted in larger mortality estimates. However, this would not change our major conclusions about the nonlinear emission-mortality relationships, since the general shape of the functions retained. In this study, we adopt the same health endpoints and IER parameters (α_i, γ_i, and δ_i) as Burnett et al. (2014). These endpoints include ischemic heart disease (IHD), chronic obstructive pulmonary disease (COPD), lung cancer, and stroke. The disease specific baseline mortality rates by age and gender are obtained from the from Institute of Health Metrics and Evaluation (Global Burden of Disease Collaborative Network, 2017).

For health impact assessment of short-term exposure to $PM_{2.5}$, the concentration-response relationships are derived in epidemiological studies based on time series analysis of $PM_{2.5}$ and health. In most studies (Shang et al., 2013; Wang et al., 2015; Dominici et al., 2002; Kan and Chen, 2004), the incidence of mortality caused by air pollution is considered to be subject to Poisson distribution. Subsequently, the relationship between mortality and $PM_{2.5}$

concentrations can be regressed in the following form by using Poisson regression (log-linear regression) or similar methods (Kan et al., 2008; Dominici et al., 2002; Shang et al., 2013; Kan and Chen, 2004).

$$\Delta Y_i = y_{0,i} P\left[1 - e^{\beta_i (C - C_0)}\right] \quad (2)$$

where ΔY_i, $y_{0,i}$, P, C, and C_0 have the same meaning as defined above. β_i is a regression coefficient derived from epidemiological studies, which refers to excess risk of mortality per each increase in 1 μg/m^3 of $PM_{2.5}$. Reasonable regression results have been obtained using Eq. 2 in over 25 epidemiological studies conducted in China (Shang et al., 2013), where the $PM_{2.5}$ concentrations range from very small to very large (mean concentration > 170 μg/m^3), indicating the applicability of Eq. 2 to the Chinese environments. Based on Eq. 2, the relationship between ΔY_i and C presents a slightly convex function form, indicating subtly less marginal effect at larger concentration increase. The health endpoints considered in the short-term assessment include all-cause mortality, respiratory mortality, and cardiovascular mortality. The baseline mortality rates are derived from China Health and Family Planning Statistics Yearbook 2015 (National Health and Family Planning Commission of China, 2015) and Huang and Zhang (2013), and the parameter estimates are taken from Chen et al. (2011). It should be noted that, in addition to mortality, the short-term $PM_{2.5}$ exposure also leads to various types of morbidity (e.g., cardiovascular and respiratory hospital admissions or outpatients). We focus on mortality in the present study since it accounts about 80% of the total monetized health losses (Kan and Chen, 2004; Wu, 2016). It's also worth noting that the estimation methods of both long-term and short-term mortality assume that the health effects depend only on the inhaled amount of $PM_{2.5}$ and are independent of the chemical composition, which appears reasonable in view of the available quantitative epidemiological studies. However, some studies have reported that some aerosol species, such as the carbonaceous aerosols, could be significantly more toxic than others (Tuomisto et al., 2008; Lelieveld et al., 2015). This relative toxicity of different aerosol species may affect the relative contributions of different emission sources to $PM_{2.5}$-related mortality, which warrants further in-depth study.

The age-specific population data at city level in 2014 are acquired from the statistical bureaus of Beijing, Tianjin and Hebei, and the sub-city distribution of population is based on the LandScan dataset at 30"×30" (approximately 1 km×1 km) resolution (Oak ridge national laboratory, 2016). The spatial distribution of population is shown in Fig. S1. To give the uncertainty of the health effects, we calculate 95% confidence intervals (CIs) using 95% CIs of the parameters of the concentration-response functions for both short-term and long-term assessments.

We use sensitivity analysis method for health effect source apportionment. The sensitivity of health effects to emissions can be calculated with the following formula:

$$S_{i,j} = \frac{\left(H_{i,j} - H_{i,0}\right)/H_{i,0}}{R_j - 1}, \left(0 \le R_j \le 1\right) \quad (3)$$

Where $S_{i,j}$ refers to the sensitivity of health endpoint i to emission source j; R_j refers to the ratio of emissions from source j to the base-case emissions; $H_{i,j}$ is the health effect of endpoint i when the emission ratio of source j is R_j; $H_{i,0}$ refers to the health effect in the base case. For each emission source, we calculate $S_{i,j}$ for reduction ratios ($1-R_j$) of 10%, 30%, 50%, 65%, 80% to assess the nonlinear relationships between health effects and emission reductions.

2.2 Determination of exposed concentrations

The exposed concentrations of $PM_{2.5}$ is one of the most important input variables in health effect assessment. Three-dimensional chemical transport models (CTMs) such as the Community Multi-scale Air Quality (CMAQ) model are frequently used to obtain exposed pollutant concentrations under specific emission scenarios. To improve the simulation of secondary organic aerosol (SOA), Zhao et al. (2016) incorporated the two-dimensional volatility basis set (2D-VBS) framework in CMAQ and developed the CMAQ/2D-VBS model. In this study, we use CMAQ/2D-VBS to obtain exposed concentrations of $PM_{2.5}$ to evaluate the health effects in the base case.

The simulation period is January, March, July and October in 2014, representing four seasons. The Weather Research and Forecasting (WRF) model version 3.7 is used to simulate the meteorological field to provide input data for the CMAQ/2D-VBS model. The configurations of WRF and CMAQ/2D-VBS and the emission inventory used in this paper are same as Zhao et al. (2017). We apply double-nesting simulation domains, with grid resolutions of 36 km×36 km and 12 km×12 km. The first domain covers China and its surrounding areas, and the second domain covers the BTH region, as shown in Fig. 1. The simulated results from WRFv3.7 and CMAQ/2D-VBS model generally agree well with ground observations (Zhao et al., 2017).

The 3-D CTM modeling is time-consuming and expensive when being used in source apportionment, so it’s not efficient to support decision making. The Extended Response Surface Modeling (ERSM) method established by Zhao et al. (2015) uses hundreds of simulation scenarios from a CTM and advanced statistical techniques to enable quick prediction of $PM_{2.5}$ concentrations in any given emission scenario, and it shows good performances in several recent studies (Zhao et al., 2015; Zhao et al., 2017; Xing et al., 2017). In this study, we use the ERSM technique to acquire exposed concentration in multiple emission scenarios to comprehensively assess the complex nonlinear relationships between various emission sources and $PM_{2.5}$-related mortality.

The establishment and validation of ERSM in the BTH region have been described in Zhao et al. (2017), so we only summarize several key points. First, we defined 5 target regions in the BTH region, i.e., Beijing, Tianjin, Northern Hebei, Eastern Hebei, and Southern Hebei (see Fig. 1). Next, we used 1121 scenarios simulated by CMAQ/2D-VBS to establish the ERSM prediction system, which maps atmospheric $PM_{2.5}$ concentrations versus emissions of 55 combinations of regions, sectors, and pollutants. We assessed the performance of the ERSM prediction system using the “out-of-sample” and 2D-isopleths validation methods, and showed satisfying accuracy and stability of the prediction system.

3 Results and Discussion

3.1 Health effects in the base case

The long-term $PM_{2.5}$-associated mortality from COPD, IHD, lung cancer, and stroke in the BTH region are shown in Fig. 2.

According to our assessment, 17.42 (95% CI, 9.45–24.40) thousand, 36.29 (27.24–48.48) thousand, 13.53 (5.19–18.19) thousand, and 61.91 (27.71–79.93) thousand people die of COPD, IHD, lung cancer, and stroke due to long-term exposure to $PM_{2.5}$ in the BTH region annually. Stroke is the most important health endpoint, accounting for 48% of the total premature deaths due to long-term exposure. Beijing, Tianjin, and cities in Southern Hebei are areas where people most severely suffer from $PM_{2.5}$-related mortality, and Zhangjiakou, Chengde, Qinhuangdao are cities with the least mortality.

For the long-term exposure to $PM_{2.5}$, the air pollution seems like a background that chronically affect people who live in the region. The short-term exposure to $PM_{2.5}$, however, is quite different, because it raises more concerns when heavy pollution episode occurs. As Fig. 2 shows, 18.72 (11.20–25.65) thousand people die due to short-term exposure to $PM_{2.5}$ in the BTH region annually, among which 59% and 16% die of cardiovascular and respiratory diseases, respectively. Similar to long-term effects, the majority of short-term health effects come from Beijing, Tianjin, and cities in Southern Hebei.

We also assess the spatial distribution of health effects due to $PM_{2.5}$ pollution, and the long-term mortality from stroke is shown in Fig. 1. The spatial distribution characteristics of short-term mortality is highly similar (Fig. S2). Comparing the spatial distribution of the health effects with population and $PM_{2.5}$ concentrations (Fig. S1), we can conclude that the health effects are strongly correlated with population. For this reason, high mortality appears in urban areas.

3.2 Nonlinear relationships between air pollutant emissions and health effects

We assess the nonlinearity between pollutant emissions control and health effects using the ERSM technique and the sensitivity analysis method described in Sections 2.1 and 2.2. For long-term exposure, to simplify the analysis, we sum up the mortality from COPD, IHD, lung cancer, and stroke to approximately represent the total $PM_{2.5}$-related mortality. As shown in Fig. 3, among all pollutants, the annual mortality due to long-term exposure to $PM_{2.5}$ is most sensitive to emissions of primary inorganic $PM_{2.5}$, which is defined as all chemical components of primary $PM_{2.5}$ other than organics, including black carbon, metals, crustal elements, etc. Primary organic aerosol (POA) is treated separately because it undergoes chemical reactions and produces SOA in the CMAQ/2D-VBS model, whereas primary inorganic $PM_{2.5}$ is assumed to be chemically inert. Among all sources of primary inorganic $PM_{2.5}$, the industry sector makes the largest contribution, followed by the residential and commercial sectors, while the contribution from power plants is negligible. The sensitivity of mortality to primary inorganic $PM_{2.5}$ emissions increases gradually by ~20% when the emission reduction ratio increases from 10% to 80%. In contrast, the sensitivity of $PM_{2.5}$ concentrations remains constant regardless of reduction ratio according to our previous study (Zhao et al., 2017). The difference is explained by the fact that the

mortality reduction due to a unit drop of $PM_{2.5}$ concentration is larger at lower $PM_{2.5}$ concentration range, according to the curvilinear shape of the IER function. For this reason, emission of primary inorganic $PM_{2.5}$ are expected to be controlled as stringent as possible to efficiently reduce the long-term mortality.

Among the precursors, the long-term mortality is primarily sensitive to the emissions of NH_3, nonmethane volatile organic compounds and intermediate volatility organic compounds (NMVOC+IVOC), and POA, and their relative importance differs according to reduction ratio and region (Fig. 3). The sensitivities of mortality to emissions of POA, NMVOC+IVOC and SO_2 roughly remain constant at various reduction ratios. In contrast, the sensitivities to NH_3 emissions increase substantially with the increase of reduction ratio due to a transition from NH_3-rich to NH_3-poor conditions. The sensitivities of mortality to NO_x emissions can be either negative or positive, depending on region and reduction ratio — they tend to be more negative at small reduction ratio and more positive at large reductions, since a small reduction in NO_x emissions is likely to produce more oxidants due to a NMVOC-limited photochemical regime, thus leading to formation of more secondary inorganic and organic aerosols. However, the results would be quite different if the NO_x emissions outside the BTH region are jointly controlled. As shown in Fig. S3, when NO_x emissions in the whole China are uniformly reduced, the sensitivities of mortality in BTH are positive even at a small reduction ratio. The reason is that NO_x emission reductions in upwind regions are more likely to result in a net $PM_{2.5}$ decrease compared with local emission reductions, since the photochemistry typically changes from a NMVOC-limited regime in local urban areas at surface to a NO_x-limited regime in downwind areas or at upper levels (Xing et al., 2011). Therefore, it is critically important to enforce stringent NO_x emission controls over a large spatial area beyond the BTH region.

Regarding emission sectors, the contributions of SO_2 and NO_x emissions are dominated by non-point sources (Fig. 3b). When all pollutants are controlled together, the sensitivity of mortality dramatically increases with reduction ratio, and this sensitivity is remarkably larger than the sum of the sensitivities to emissions of individual pollutants, indicating extra health benefits will be achieved if a multi-pollutant control strategy is implemented.

As for short-term exposure, we assess the sensitivities of all-cause mortality to emissions of various pollutants from various sectors (Fig. 4). The characteristics of the results are largely similar to those of long-term exposure. What is found to be different is that, when the emission reduction ratio increases from 10% to 80%, the sensitivity of mortality to primary inorganic $PM_{2.5}$ emissions increases so slightly that it seems to be constant. This can be explained by the fact that the nonlinearity of the concentration-response function for short-term assessment (Eq. 2) is much weaker than that of the IER function (Eq. 1). When all pollutants are controlled together, the sensitivity of mortality is similar to or less than the sum of the sensitivities to emissions of individual pollutants, and the sensitivity only increases slightly with reduction ratio.

We further analyze the seasonal feature of sensitivity of short-term mortality to emissions of various pollutants and sectors. We choose Beijing and Northern Hebei as examples, as shown in Fig. 4b. The sensitivities show significant discrepancy in different months. In

winter, primary inorganic $PM_{2.5}$ contributes a much larger fraction of mortality than other seasons, as a result of weaker vertical mixing and slower reactions of gaseous precursors. Among different sources of primary inorganic $PM_{2.5}$, the residential and commercial sectors make relatively larger contributions in winter due to household heating. The sensitivities of mortality to gaseous precursors are generally larger in summer than in winter because of accelerated chemical reactions due to stronger radiation and higher temperature. The sensitivities to NO_x are more complicated — they are mostly negative in winter and positive in summer, and change from negative to positive with the increase of reduction ratio in spring and autumn, because the photochemistry is prone to be NMVOC-limited in winter and NO_x-limited in summer (Zhao et al., 2013; Zhao et al., 2015; Xing et al., 2017).

3.3 Nonlinear relations between region-specific emission control and health effects

We examine the nonlinear relationships between region-specific emission control and health effects by using the same method applied in Section 3.2. Here we select long-term mortality in Beijing as an example, as shown in Fig. 5. For any pollutant, the mortality in Beijing is most sensitive to local emissions among the five regions, but the relative contributions of local and non-local sources differ significantly according to pollutants. For primary inorganic $PM_{2.5}$, POA, and SO_2, local emissions account for more than 60% of the total contributions from all regions. With respect to NH_3 and NMVOC+IVOC, local emissions account for about 50% and one third, respectively. The sensitivity of mortality to local primary inorganic $PM_{2.5}$ emissions increases by ~20% when the reduction ratio increases from 10% to 80%, while the sensitivity to local NH_3 emissions nearly doubles in response to such increase in the reduction ratio. For SO_2, POA, and NMVOC+IVOC, no obvious change in sensitivities with the reduction ratio is noted. For NO_x, the sensitivity to local emissions is negative, whereas the sensitivities to emissions from other regions, especially Southern Hebei from which a large amount of air pollutants are transported to Beijing (Chang et al., 2018), could change from negative to positive when the reduction ratio increases from 10% to 80%.

For any individual pollutant, when emissions from all regions except Beijing are controlled together, the overall sensitivity of mortality nearly equals the arithmetic sum of the sensitivities to each single region, indicating quasi-linear effects of emission control among region outside Beijing. Nevertheless, when emissions from all regions including Beijing are controlled together, the overall sensitivity of mortality exceeds the arithmetic sum of the sensitivities to emissions from each single region, and the differences are more pronounced when the reduction ratio is large and/or multiple pollutants are controlled simultaneously (see the rightmost columns in Fig. 5). This implies that additional health benefits will be achieved when multi-pollutant and regional collaboration control strategies are implemented. Although we used Beijing as an example in the preceding analysis, similar increment in sensitivity due to multi-region and multi-pollutant controls is also found for other regions in BTH.

4 Conclusion and implications

In this study, we systemically assessed the nonlinear relationships between $PM_{2.5}$-related mortality and air pollutant emissions in the BTH region by integrating the CMAQ/2D-VBS chemical transport model, the ERSM technique, and state-of-the-art concentration-response functions.

About 17.42 thousand, 36.29 thousand, 13.53 thousand and 61.91 thousand people die of COPD, IHD, lung cancer, and stroke respectively due to long-term exposure to $PM_{2.5}$ in the BTH region annually. The annual mortality due to short-term exposure to $PM_{2.5}$ is about 18.72 thousand. The $PM_{2.5}$-related health effects are most severe in Beijing, Tianjin, and cities of Southern Hebei.

Among all pollutants, premature mortality due to both long-term and short-term exposures to $PM_{2.5}$ is most sensitive to emissions of primary inorganic $PM_{2.5}$. The sensitivity of long-term mortality to primary inorganic $PM_{2.5}$ increases significantly with reduction ratio, as a result of the curvilinear IER function. For precursors, the $PM_{2.5}$-related mortality is primarily sensitive to emissions of NH_3, NMVOC+IVOC, and POA, and their relative importance differs according to reduction ratio and region. The sensitivities of mortality to NH_3 and NO_x increase significantly with the increase of reduction ratio, while the sensitivities to POA, NMVOC+IVOC and SO_2 roughly remain constant at various reduction ratios. For long-term exposure, when all pollutants are controlled together, the sensitivity of mortality significantly increases with reduction ratio, and it's much larger than the sum of the sensitivities to emissions of individual pollutants. For short-term exposure, however, the sensitivity of mortality to joint control of all pollutants is similar to the sum of sensitivities to individual pollutants.

The sensitivities of mortality to emission controls differ with seasons. In winter, emissions of primary inorganic $PM_{2.5}$, especially those from residential and commercial sources, contribute more to mortality than in other seasons. In contrast, the sensitivities of mortality to gaseous precursors are generally larger in summer than in winter.

Over 60% of the contributions from emissions of primary inorganic $PM_{2.5}$, POA, and SO_2 to long-term mortality in Beijing are attributed to local emissions. For NH_3 and NMVOC +IVOC, local emissions account for a smaller fraction, i.e., 50% and one third, respectively. When emissions from local and nonlocal emissions are controlled simultaneously, the overall sensitivity of mortality exceeds the arithmetic sum of the sensitivities to emissions from each single region, and the differences are more pronounced for larger reduction ratio and/or for joint control of multiple pollutants.

The findings from this study are important for the development and optimization of health-oriented air pollution control policies in BTH and its surrounding areas. First, a sharp reduction in emissions of primary $PM_{2.5}$ is suggested considering that $PM_{2.5}$-related mortality is most sensitive to it among all pollutants and that the sensitivity increases with reduction ratio. A particular focus shall be placed on primary $PM_{2.5}$ emissions from residential and commercial sectors in winter, which have been largely neglected in China's control policies until recently. Second, stringent control policies should be implemented for

NO_x and NH_3 emissions in the BTH region in view of their increasing sensitivities with reduction ratio. For NO_x, joint controls over a larger region beyond BTH are necessary in order to avoid possible side effects. Finally, multi-pollutant, multi-sector and regional collaboration control strategies are recommended wherever possible, because the sensitivity of long-term mortality to the joint emission controls are substantially larger than the sum of sensitivities to controls of individual pollutants or regions.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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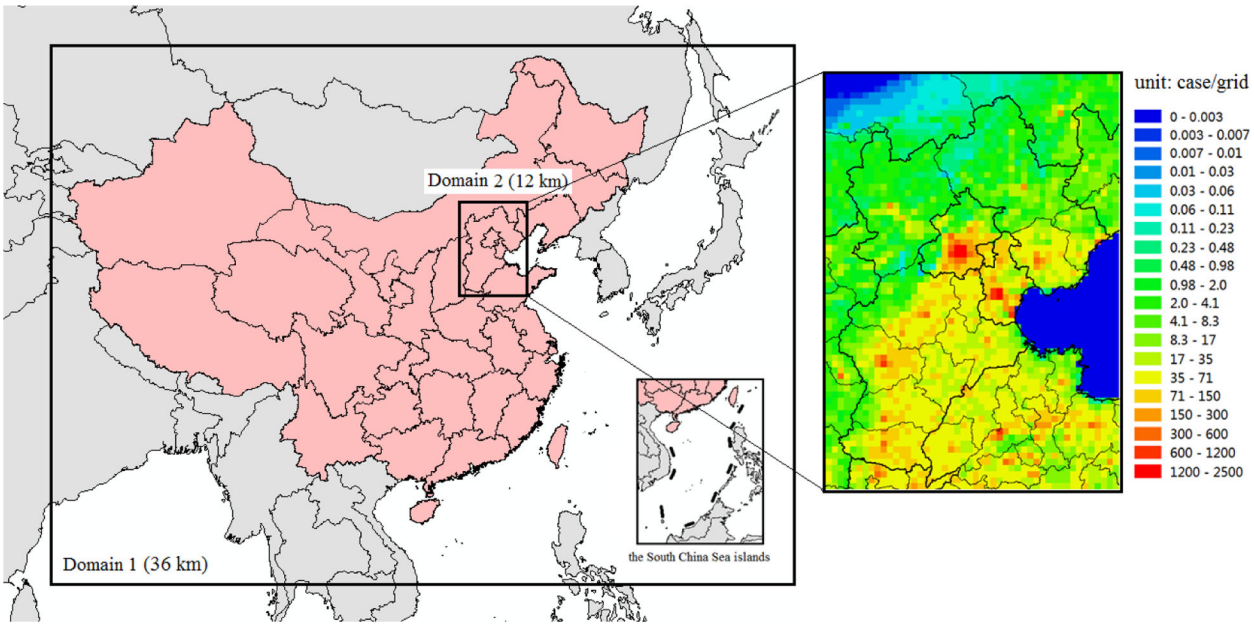

Fig. 1.

The simulation domain in this study (left) and the spatial distribution of deaths from stroke due to long-term exposure to $PM_{2.5}$ pollution in the BTH region (right).

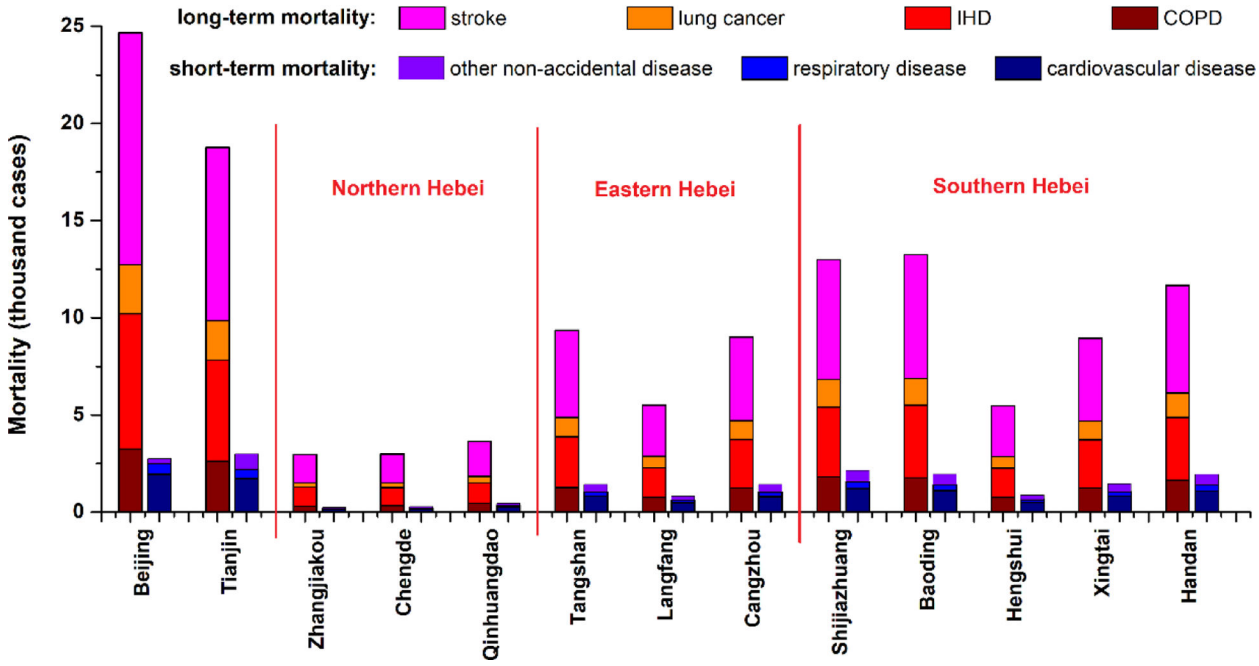

Fig. 2.

Premature mortality due to $PM_{2.5}$ pollution in the BTH region. The two sets of colored bars for each city refer to mortality due to long-term and short-term exposures, respectively. Only median values of the mortality estimates are displayed in this figure.

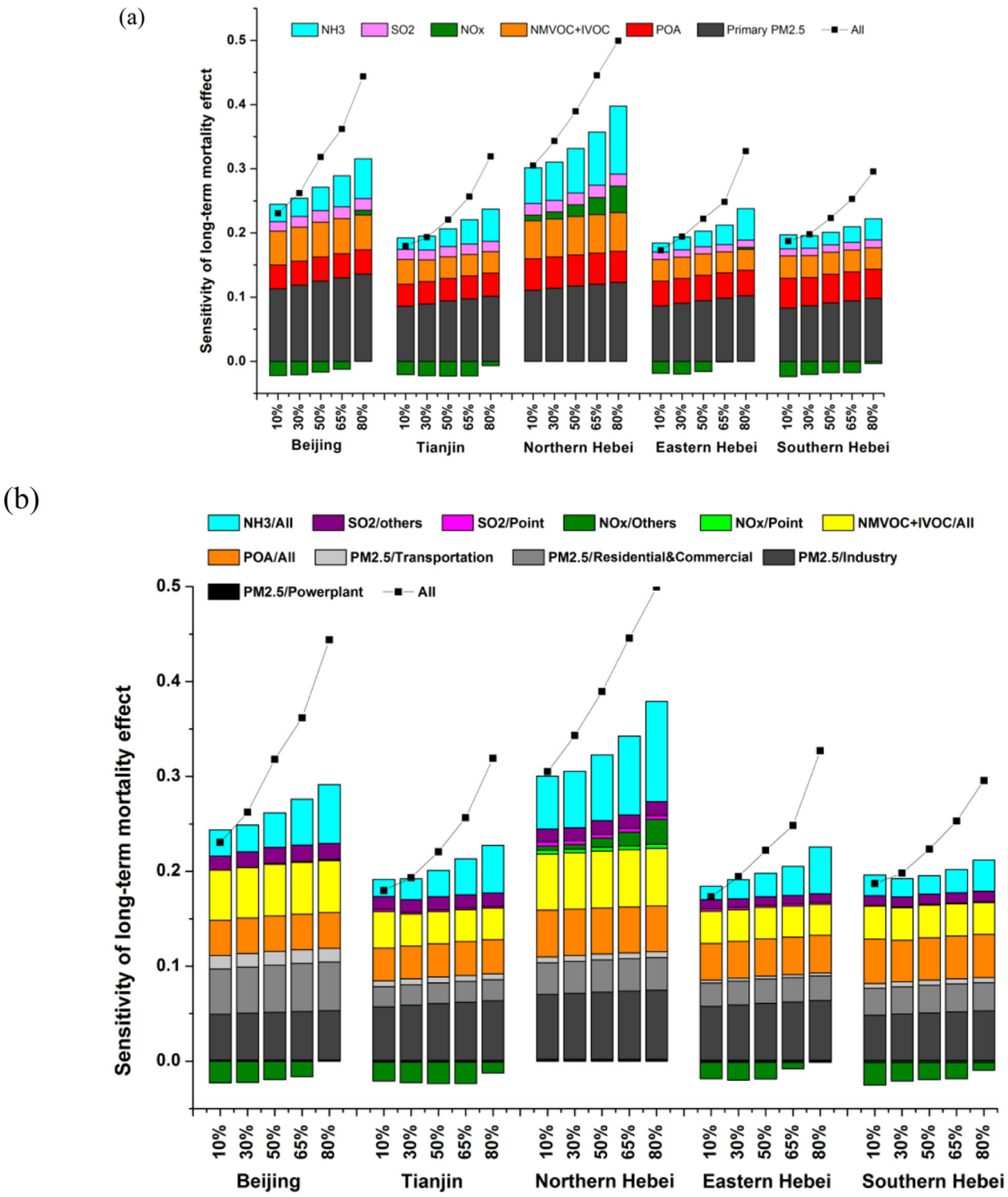

Fig. 3.

Sensitivity of annual mean mortality due to long-term exposure to $PM_{2.5}$ to stepped control of individual air pollutants (a) and individual pollutant-sector combinations (b). The X-axis shows the reduction ratio (= 1 – emission ratio). The Y-axis shows the sensitivity of mortality, as defined by Eq. 3. The colored bars denote the sensitivity of mortality when a

particular emission source is controlled while the others stay the same as the base case; the black dotted line denotes the sensitivity of mortality when all emission sources are controlled simultaneously.

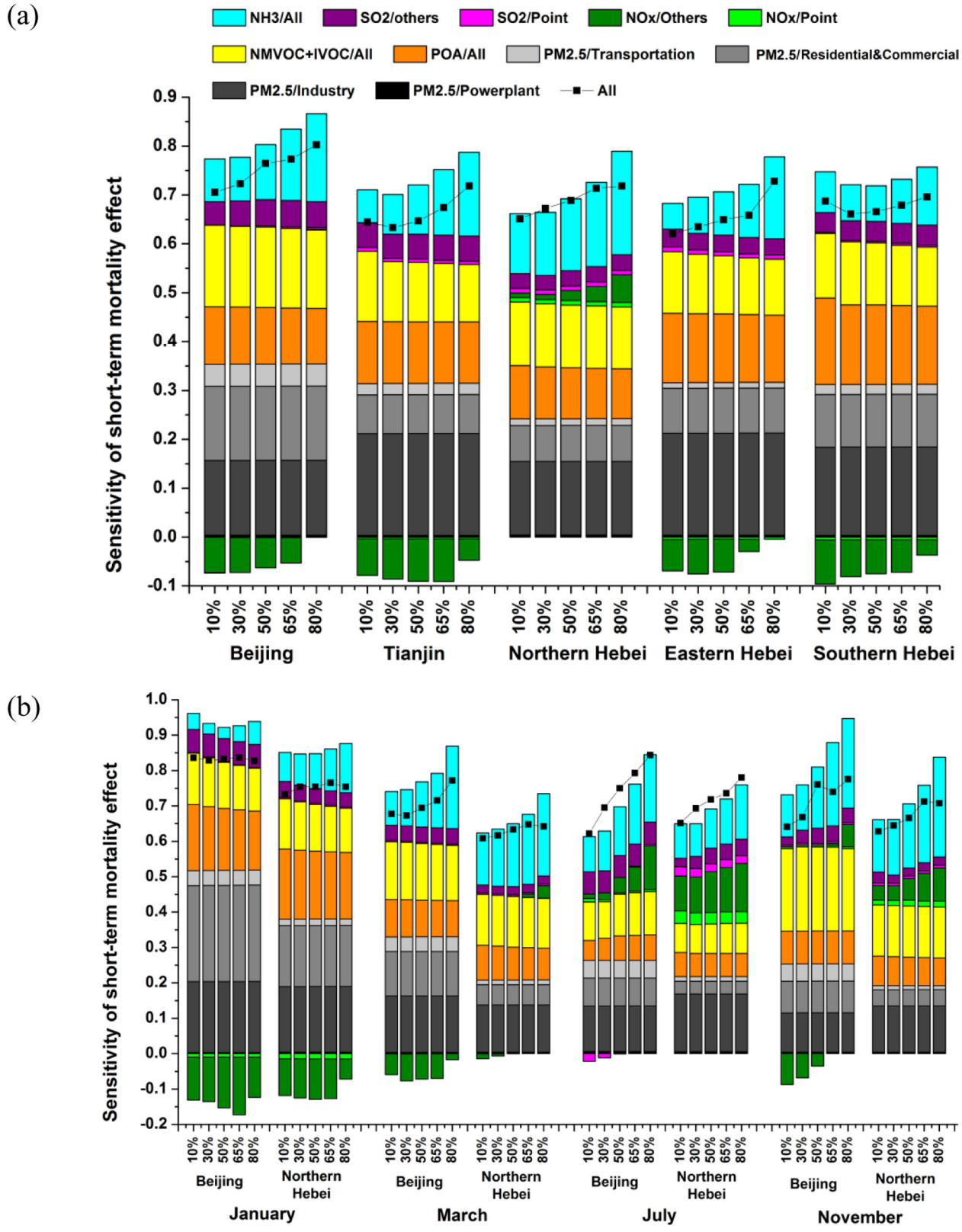

Fig. 4.

Sensitivity of annual mean (a) and monthly mean (b) short-term $PM_{2.5}$-related mortality to stepped control of individual pollutant-sector combinations. The meanings of X-axis, Y-axis, colored bars, and black dotted lines are the same as Fig. 3.

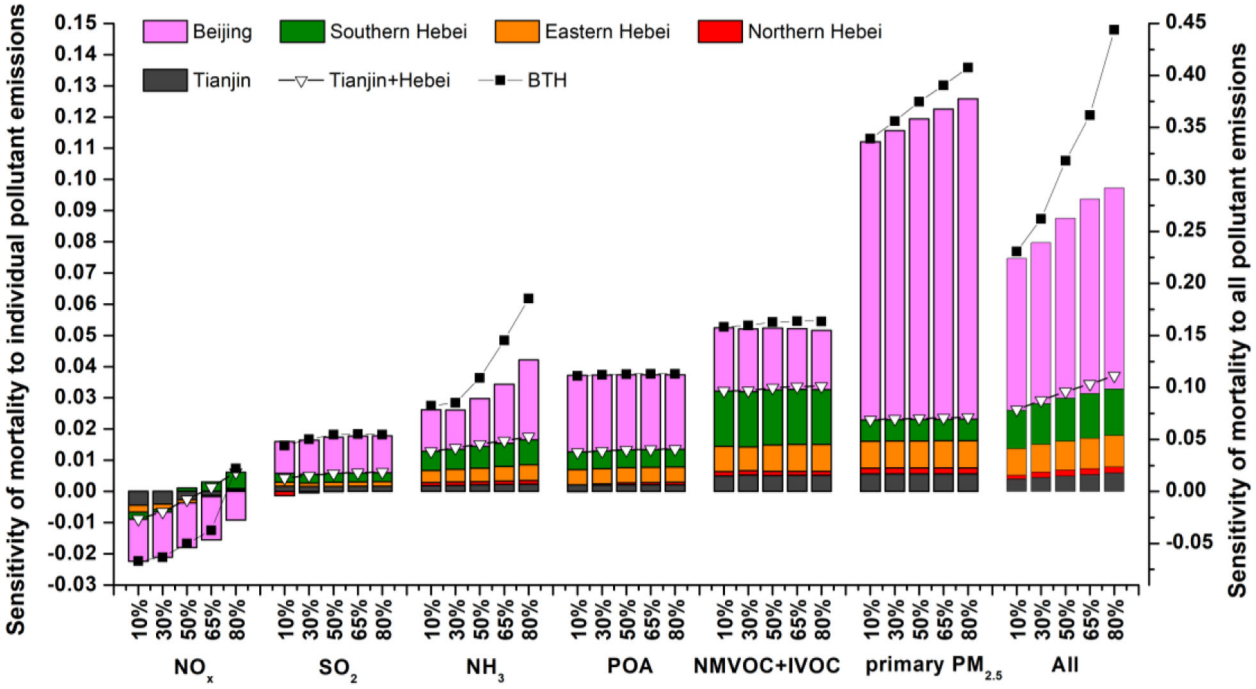

Fig. 5.

Sensitivity of annual mean mortality caused by long-term exposure to $PM_{2.5}$ in Beijing to stepped control of individual/all air pollutants from various regions. The meanings of X-axis, Y-axis, colored bars, and black dotted lines are the same as Fig. 3. Hollow triangle dotted lines denote the sensitivity of mortality when all emission sources from regions except Beijing are controlled simultaneously.