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Premature Mortality Attributable to Particulate Matter in China: Source Contributions and Responses to Reductions

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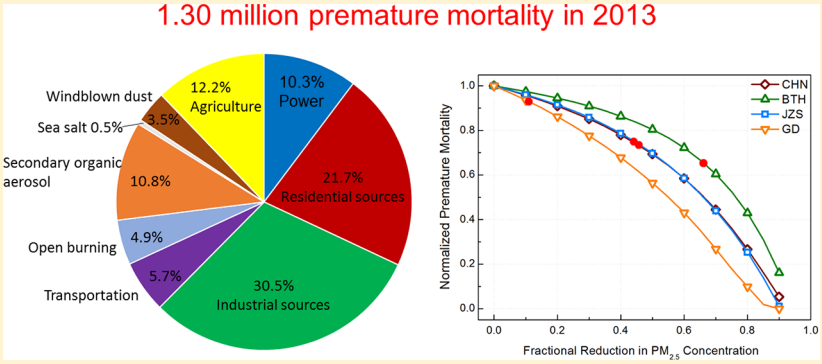
Supporting Information



ABSTRACT: Excess mortality (ΔMort) in China due to exposure to ambient fine particulate matter with aerodynamic diameter ≤2.5 μm ($PM_{2.5}$) was determined using an ensemble prediction of annual average $PM_{2.5}$ in 2013 by the community multiscale air quality (CMAQ) model with four emission inventories and observation data fusing. Estimated ΔMort values due to adult ischemic heart disease, cerebrovascular disease, chronic obstructive pulmonary disease, and lung cancer are 0.30, 0.73, 0.14, and 0.13 million in 2013, respectively, leading to a total ΔMort of 1.3 million. Source-oriented CMAQ modeling determined that industrial and residential sources were the two leading sources of ΔMort, contributing to 0.40 (30.5%) and 0.28 (21.7%) million deaths, respectively. Additionally, secondary ammonium ion from agriculture, secondary organic aerosol, and aerosols from power generation were responsible for 0.16, 0.14, and 0.13 million deaths, respectively. A 30% ΔMort reduction in China requires an average of 50% reduction of $PM_{2.5}$ throughout the country and a reduction by 62%, 50%, and 38% for the Beijing−Tianjin−Hebei, Jiangsu−Zhejiang−Shanghai, and Pearl River Delta regions, respectively. Reducing $PM_{2.5}$ to the CAAQS grade II standard of 35 μg m^{-3} would only lead to a small reduction in mortality, and a more stringent standard of <15 μg m^{-3} would be needed for more remarkable reduction of ΔMort.

1. INTRODUCTION

Outdoor air pollution is linked to various health effects.[1−7] Globally it is estimated to have caused 3.3 million premature deaths in 2010.[8] The health risk occurs predominantly in developing countries, particularly in Asia,[8,9] due to the combination of a fast increase of population, industrialization, urbanization, and associated energy consumption and a lack of sufficient emission control measures. China has been suffering serious air pollution in recent decades. Observational studies revealed that annual concentrations of ambient fine particulate matter with aerodynamic diameter ≤2.5 μm ($PM_{2.5}$) are more than 5 times higher than the World Health Organization (WHO) guideline value in many populous cities.[10−13] Sustained exposure to high $PM_{2.5}$ concentrations greatly threatens public health in the country.[14−16]

Recognizing the severity of the air pollution, the Chinese government had set a target in 2013 to reduce the $PM_{2.5}$ level

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by up to 25% in major metropolitan areas by 2017.[17] Many studies are undergoing to investigate the pathways to achieve the target. However, it is still unclear how public health will be affected by $PM_{2.5}$ reduction. To design effective control plans and set priorities for air pollution controls to better protect public health, the need to assess premature mortality caused by air pollution, to identify its source contributions, and to evaluate its responses to air quality improvement is urgent.

Premature mortality caused by outdoor air pollution in China has been estimated in a few global and regional studies. Lelieveld et al.[8] estimated that 1.36 million premature deaths in China were attributed to outdoor air pollution in 2010. This study applied the improved exposure response functions described by Burnett et al.[18] to account for health effects at high $PM_{2.5}$ concentrations, such as those that occur frequently in China. However, this estimation is still quite uncertain due to the coarse grid resolution (approximately 110×110 km^2) used in the chemical transport model (CTM) and the uncertainties associated with emission inventory. A few studies have examined the effects of grid resolution on air quality predictions in North America and Europe.[19−23] In general, the increase of grid resolution from several tens of kilometers to a few kilometers improves the agreement between the predictions and observations. The improvement is more significant for the primary PM components than for the secondary components and is more significant in winter than in summer.[19] Observation data sets were also used to improve model predictions. Brauer et al.[24] generated a fused global $PM_{2.5}$ estimation at high resolution ($0.1° \times 0.1°$) utilizing the average $PM_{2.5}$ estimated by the satellite retrieved aerosol optical depth at $0.1° \times 0.1°$ and a global CTM prediction of $PM_{2.5}$ at $1° \times 1°$ suballocated to $0.1° \times 0.1°$ based on population density. The fused global estimation was further adjusted using a global adjustment function derived from the correlation analysis of the observed surface $PM_{2.5}$ at monitoring sites and the corresponding fused concentrations. More recently, Liu et al.[25] estimated the premature deaths due to $PM_{2.5}$ exposure in China using a regional CTM with 45×45 km^2 resolution assimilated with hourly observations from 506 monitoring sites.

Identifying source contributions to premature mortality can help design effective plans and set priorities for air pollution controls. Large differences were observed in the air pollution characteristics in different regions of China due to significant differences in emission source sectors and climate conditions. Studies to quantify the source contributions to air pollution and premature mortality at the province level are needed. Global models are limited in providing such detailed information at a subregional scale. Source-oriented regional CTMs were developed to provide detailed source information for health studies[26] and were used to study the sources of $PM_{2.5}$ in China,[27−29] which makes detailed analysis of source contributions to air pollution related health impact possible.

Estimating responses of premature mortality to air quality improvement can help set reasonable air pollution control targets. In a recent study, Lee et al.[30] estimated the sensitivity of global $PM_{2.5}$-related mortality due to the increase in precursor emissions using an adjoint version of the GEOS-Chem model. However, the estimations were given at either a uniform increase of 1 kg km^{-2} yr^{-1} or a 10% increase, and the study did not apply China-specific emission and the most recent baseline mortality data from the Chinese government. Even though a decrease in excess mortality can be expected, responses of premature mortality to targeted $PM_{2.5}$ reduction goals remain unknown due to the nonlinear relationship between excess mortality and $PM_{2.5}$ concentrations.[31]

The objectives of the present study are to provide an improved estimation of the premature mortality attributable to $PM_{2.5}$ at the province level in China by using more China-specific data and higher spatial resolution, to quantify contributions to premature mortality from major sources, and to determine its responses to different $PM_{2.5}$ reduction targets.

2. METHODS

2.1. Calculation of Premature Mortality. The relative risk, or risk ratio (RR), is the ratio of the probability of an event occurring (for example, developing a disease) in an exposed group to the probability of the event occurring in a comparison, nonexposed group. In this study, the RRs of several causes of premature mortality [chronic obstructive pulmonary disease (COPD), lung cancer (LC), adult ischemic heart disease (IHD), and cerebrovascular disease (CEV)] from long-term outdoor $PM_{2.5}$ exposure are calculated using the integrated exposure response function (eq 1), as suggested by Burnett et al.[18]

$$\mathrm{RR}(C) = 1 + \alpha\{1 - \exp[-\beta(C - C_{\mathrm{cf}})^{\delta}]\} \quad (1)$$

where α, β, and δ are parameters fitted for different causes based on data from exposure of cigarette smoke at high concentrations and of ambient air pollution at low concentrations. C is the annual average $PM_{2.5}$ exposure in units of μg m^{-3} and C_{cf} is the counter-factual concentration below which it is assumed that there is no additional risk. Once the RR is calculated, the premature (excess) mortality can be calculated using eq 2

$$\Delta\mathrm{Mort} = y_0 P[(\mathrm{RR} - 1)/\mathrm{RR}] = y_0 P \times \mathrm{AF} \quad (2)$$

where y_0 is the baseline mortality rate due to a particular disease category and P is the population. $(\mathrm{RR} - 1)/\mathrm{RR}$ represents the attributable fraction (AF), which is the proportion of the disease in the exposed group that can be attributed to exposure.

The baseline mortalities of male and female population for each 5-year age group in the urban and rural areas in 2013 were obtained from the China Public Health and Family Planning Statistical Yearbook 2014 (CPHFPSY 2014). In addition, another set of baseline mortality data was obtained from the World Health Organization (WHO) database. The sensitivity of the predicted excess mortality using the WHO data is discussed in the Discussion section. Tables S1 and S2 of the Supporting Information (SI) summarize the mortality data used in this study. Gridded population data were obtained from the Oak Ridge Nation Laboratory's LandScan database at 1 km for 2008[32] and was scaled up to 2013 using the reported provincial population data from the National Bureau of Statistics of China, assuming that the population spatial distribution within a province does not change. The 1-km resolution data were reprojected into the same resolution of the CTM domain. The age distribution in 2013 (see Table S3, SI) was obtained from the Population Division of the Department of Economic and Social Affairs, United Nations, and applied to all regions.

2.2. Estimation of $PM_{2.5}$ Exposure and Source Contributions. The Community Multiscale Air Quality (CMAQ) model[33,34] developed by the United States Environmental Protection Agency at a horizontal spatial resolution of 36×36 km^2 with 18 vertical layers up to 20 km above the surface was used to estimate $PM_{2.5}$ concentrations in China

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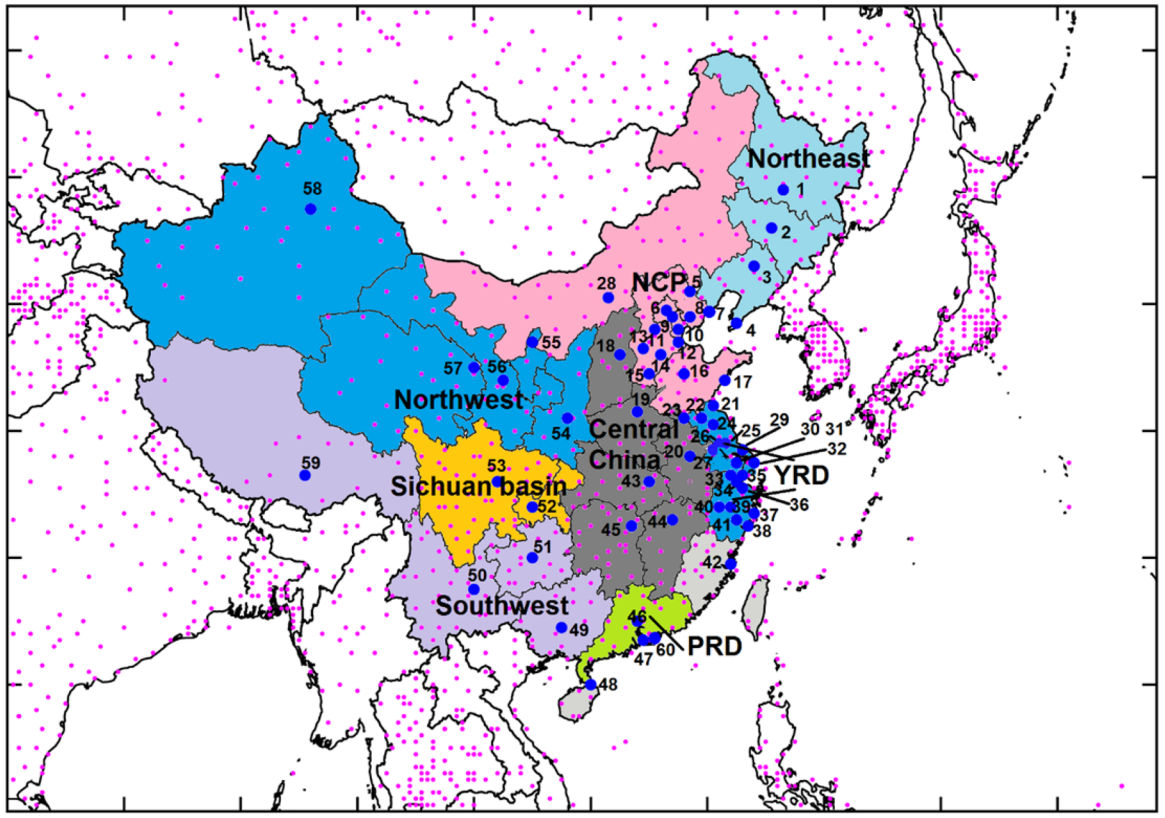

Figure 1. Model domain (spatial resolution: 36 × 36 km^2). Blue circles: cities with annual $PM_{2.5}$ concentration data. Purple dots: meteorological stations. NCP: North China Plain provinces. YRD: Yangtze River Delta provinces and cities. PRD: Pearl River Delta provinces and cities (including Hong Kong and Macau Special Administrative Regions). Names of the cities and their provinces (italic text denotes cities that are provincial capitals): 1, *Harbin* (Heilongjiang province); 2, *Changchun* (Jilin); 3, *Shenyang* (Liaoning); 4, Dalian (Liaoning); 5, Chengde (Hebei); 6, *Beijing* (Beijing); 7, Qinhuangdao (Hebei), 8, Tangshan (Hebei); 9, Langfang (Hebei); 10, *Tianjin* (Tianjin); 11, Baoding (Hebei); 12, Cangzhou (Hebei); 13, *Shijiazhuang* (Hebei); 14, Hengshui (Hebei); 15, Handan (Hebei); 16, *Jinan* (Shandong), 17, Qingdao (Shandong); 18, *Taiyuan* (Shanxi); 19, *Zhengzhou* (Henan); 20, *Hefei* (Anhui); 21, Lianyungang (Jiangsu); 22, Suqian (Jiangsu); 23, Xuzhou (Jiangsu); 24, Huai'an (Jiangsu); 25, Taizhou (Jiangsu); 26, Yangzhou (Jiangsu); 27, *Nanjing* (Jiangsu); 28, *Huhehaote* (Neimeng); 29, Nantong (Jiangsu); 30, Suzhou (Jiangsu); 31, Wuxi (Jiangsu); 32, *Shanghai* (Shanghai); 33, Huzhou (Zhejiang); 34, *Hangzhou* (Zhejiang); 35, Jiaxing (Zhejiang); 36, Shaoxing (Zhejiang); 37, Zhoushan (Zhejiang); 38, Wenzhou (Zhejiang); 39, Jinhua (Zhejiang); 40, Quzhou (Zhejiang); 41, Lishui (Zhejiang); 42, *Fuzhou* (Fujian); 43, *Wuhan* (Hubei); 44, *Nanchang* (Jiangxi); 45, *Changsha* (Hunan); 46, *Guangzhou* (Guangdong); 47, Zhuhai (Guangdong); 48, *Haikou* (Hainan); 49, *Nanning* (Guangxi); 50, *Kunming* (Yunnan); 51, *Guiyang* (Guizhou); 52, *Chongqing* (Chongqing); 53, *Chengdu* (Sichuan); 54, *Xi'an* (Shaanxi); 55, *Yinchuan* (Ningxia); 56, *Lanzhou* (Gansu); 57, *Xining* (Qinghai); 58, *Wulumuqi* (Xinjiang); 59, *Lasa* (Xizang); 60, Shenzhen (Guangdong).

(Figure 1). While it is desirable to use even higher spatial resolution, higher-resolution emission inventories are not available for all of China at this point. An ensemble of four simulations using four different sets of anthropogenic emission inventories was used to provide a combined estimation of $PM_{2.5}$ concentrations. The inventories used in this study include (1) the Multiresolution Emission Inventory for China (MEIC) developed by Tsinghua University (http://www.meicmodel.org), (2) the Emission Inventory in Asia, version 2 (REAS2),[35] (3) the emission prepared by the School of Environment of Tsinghua University (SOE),[36] and (4) the Emissions Database for Global Atmospheric Research (EDGAR),[37] version 4.2. As the study focuses on air pollution in China, anthropogenic emissions in other countries within the model domain were generated using the REAS2 in all four simulations. Emissions from other sources were also identical in the four simulations. Biogenic emissions were generated using the Model for Emissions of Gases and Aerosols from Nature (MEGAN), version 2.1. Open burning emissions were generated from a satellite-based fire inventory from NCAR.[38] Dust[39] and sea salt emissions[40] were generated inline during simulations. The gas-phase photochemistry was simulated using SAPRC-11[41] with modifications to provide a more detailed treatment of isoprene oxidation products, including epoxydiols.[42] The sixth-generation aerosol module in CMAQ was also modified to include additional heterogeneous reactions for the formation of secondary organic aerosol (SOA),[43,44] as well as secondary nitrate and sulfate. The meteorological inputs were generated using the Weather Research and Forecasting (WRF) Model, version 3.61. Model performance with regard to O_3 and $PM_{2.5}$, elemental and organic carbon, and SOA using MEIC has been documented previously.[45,46] A comparison of the four emission inventories and four sets of simulations, as well as an ensemble of the surface $PM_{2.5}$ predictions using the four inventories, was described by Hu et al.[47] In summary, the ensemble concentrations show improved agreement with available observations at 60 cities in 2013. The mean fractional bias and mean fractional error are −0.11 and 0.24, respectively, which are better than the mean fraction bias (−0.25 to −0.16) and mean fractional error (0.26−0.31) of any individual simulation. Figure S1 (SI) shows the observed annual concentrations and mean fractional bias of each individual

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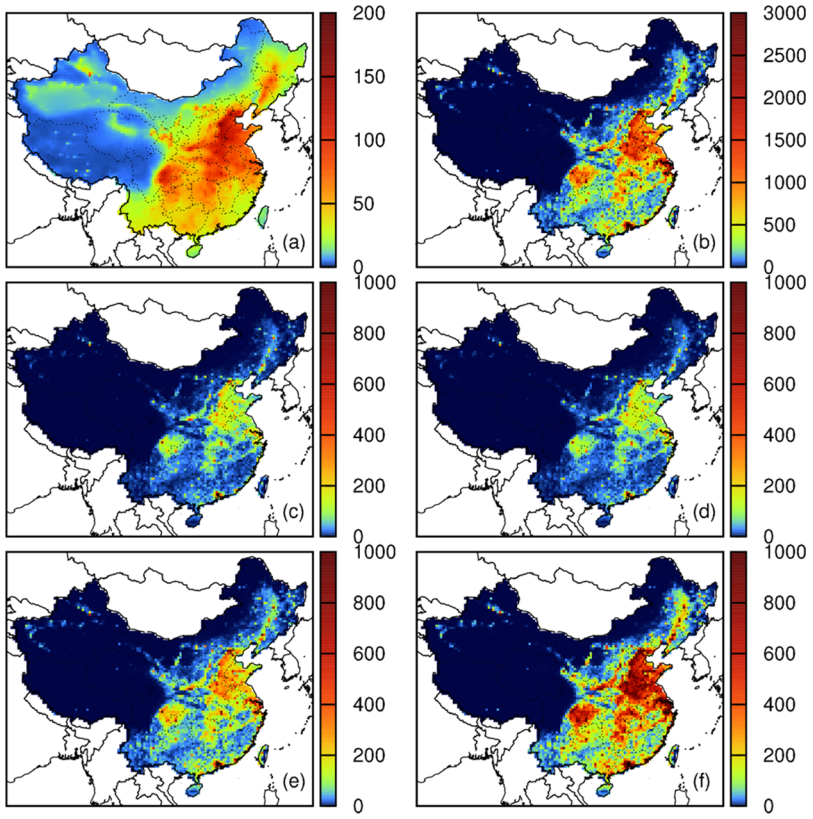

Figure 2. (a) Observation-fused annual $PM_{2.5}$ concentrations ($\mu g\ m^{-3}$), (b) total premature mortality (death per area of 36 × 36 km^2), and premature mortality due to (c) chronic obstructive pulmonary disease (COPD), (d) lung cancer (LC), (e) ischemic heart disease (IHD), and (f) cerebrovascular disease (CEV) in China.

simulation as well as the merged concentration at the 60 cities. More details of the model configuration and performance analyses can be found in the papers mentioned and the references therein.

To further reduce the biases in exposure estimation, an observation data fusing technique[48] was used to adjust the merged concentration using the spatially interpolated difference between predicted and observed annual $PM_{2.5}$ concentrations at the 60 cities. In this study, the differences were interpolated to the grid cells without observation using inverse distance weighting. Adjustment to the predicted concentrations was only made to grid cells with a population density more than 50 persons per km^2. Each city was considered to have an influence radius of 72 km, beyond which its influence was considered negligible. The impacts of ensemble and observation data fusing on predicted population weighted concentrations and excess mortality at the provincial level are explored in Table S8 (SI).

Source-oriented versions of the CMAQ model[27,28] were used to quantify the contributions to primary PM and secondary inorganic PM (ammonium, nitrate, and sulfate) from gas-phase precursors. Source contributions were determined by expanding the gas and aerosol mechanisms to use source-specific species to independently track emissions of primary PM and secondary inorganic PM precursors and the formation of secondary aerosol. The sources tracked in this study were power generation, residential use, industries, transportation, open burning, agriculture, sea salt, and windblown dust. Source contributions to SOA were not determined, and therefore, SOA was listed as a separate source category. Fractional source contributions to annual $PM_{2.5}$ concentrations were determined using the MEIC inventory alone and were used to apportion the excess mortality determined from the observation-fused ensemble concentrations.

MEIC is developed on the basis of more local data, as the developers have access to resources and information that might not be available to REAS2 and EDGAR developers. It was assumed that access to local data will lead to better estimation of emissions and thus more reasonable source contribution calculations, even though MEIC does not yield consistently better predictions of $PM_{2.5}$ concentrations at all locations and times (which could be affected by other factors, such as emissions from natural sources and meteorological inputs). More details of the source apportionment using MEIC can be found in the work by Shi et al.[49] A comparison of sectorial emissions for the four inventories and potential uncertainty in source apportionment of excess mortality is included in Figure S5 (SI).

2.3. Estimation of Uncertainties. One thousand sets of α, β, C_{cf}, and δ estimations, as used by Burnett et al.[18] in developing the exposure-response function (eq 1), were used to estimate the uncertainties in the predicted RR and AF for each cause of death due to uncertainties in the exposure−response function. The data were obtained from http://ghdx.healthdata.org/sites/default/files/record-attached-files/IHME_CRCurve_parameters.csv (last accessed on June 13, 2017). The same exposure−response functions were used to calculate RR for all ages greater than 30. Since age-specific parameters for relative risk of IHD and CEV are available, the effect of using these age-specific RR calculations was also explored. For each grid cell within China, 1000 RR values were generated using the 1000 sets of α, β, C_{cf}, and δ and the observation-fused ensemble annual $PM_{2.5}$ concentration. The RR values were then used to

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Environ. Sci. Technol. XXXX, XXX, XXX−XXX

Table 1. Relative Source Contributions[a] (%) to Premature Mortality from COPD, LC, IHD, and CEV in Adults ≥30 Years Old Due to Long Term Exposure of Ambient $PM_{2.5}$ from Power Generation (P), Residential Use (R), Industries (I), Transportation (T), Open Burning (OB), Secondary Organic Aerosol (SOA), Sea Salt (SS), Windblown Dust (WD), and Agricultural NH_3 (A)

	P	R	I	T	OB	SOA	SS	WD	A
Beijing (BJ)	10.0	26.2	31.7	7.8	2.3	7.4	0.0	2.9	11.7
Tianjin (TJ)	10.5	25.1	33.8	7.3	1.8	6.8	0.0	2.3	12.3
Hebei (HE)	11.2	22.8	34.6	6.9	1.7	7.1	0.1	2.6	12.9
Shanxi (SX)	12.4	20.1	34.6	4.8	1.9	8.7	0.0	5.6	11.8
Neimeng (NM)	13.2	25.3	28.9	3.7	1.6	6.8	0.0	9.5	11.1
Liaoning (LN)	11.4	22.8	35.0	5.9	1.5	7.0	0.4	2.5	13.5
Jilin (JL)	11.7	25.6	31.8	5.2	1.5	7.1	0.3	2.8	13.9
Heilongjiang (HL)	10.0	32.3	26.5	5.0	2.4	7.6	0.3	3.7	12.3
Shanghai (SH)	12.3	12.7	40.9	6.5	3.6	7.8	1.0	3.2	12.0
Jiangsu (JS)	12.0	19.2	33.2	7.3	3.3	9.2	0.5	2.1	13.2
Zhejiang (ZJ)	12.3	12.0	33.7	6.4	6.9	11.1	1.2	3.3	13.2
Anhui (AH)	10.8	25.1	28.5	7.0	3.6	10.5	0.2	1.9	12.2
Fujian (FJ)	10.3	10.9	32.5	4.6	10.0	12.8	1.8	5.2	11.9
Jiangxi (JX)	9.2	17.2	27.7	5.3	12.5	14.1	0.5	2.6	11.0
Shandong (SD)	11.6	22.7	32.8	7.5	1.7	7.6	0.3	2.0	13.7
Henan (HA)	11.1	22.9	31.3	7.4	2.1	8.8	0.1	2.3	13.9
Hubei (HB)	9.5	24.5	30.9	5.7	3.9	10.4	0.1	2.2	12.7
Hunan (HN)	8.7	21.1	29.0	5.2	9.7	11.9	0.2	2.3	11.8
Guangdong (GD)[b]	9.5	15.2	30.1	4.8	8.8	16.0	1.6	4.0	10.1
Guangxi (GX)	7.8	17.5	26.7	4.0	12.0	17.9	0.8	3.0	10.3
Hainan (HI)	7.3	18.2	21.8	3.6	9.1	23.6	3.6	3.6	9.1
Chongqing (CQ)	9.0	26.1	29.8	4.2	3.7	11.2	0.3	2.8	12.9
Sichuan (SC)	7.7	29.8	26.9	4.4	3.9	12.8	0.1	3.0	11.5
Guangzhou (GZ)	10.1	24.5	26.3	3.3	7.1	12.9	0.3	3.5	12.1
Yunnan (YN)	6.2	14.5	20.2	1.9	21.6	23.3	0.2	4.3	7.8
Xizang (XZ)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100.0	0.0
Shaanxi (SN)	10.2	28.1	26.9	5.0	1.8	11.2	0.2	5.0	11.8
Gansu (GS)	9.4	26.2	23.4	4.3	2.0	10.2	0.0	14.5	10.2
Qinghai (QH)	9.3	24.1	24.1	3.7	1.9	7.4	0.0	20.4	9.3
Ningxia (NX)	12.3	20.0	26.2	3.1	1.5	9.2	0.0	18.5	9.2
Xinjiang (XJ)	13.8	10.1	33.9	2.8	0.9	3.7	0.0	24.8	10.1
Taiwan (TW)	10.2	10.2	23.7	3.4	11.9	18.6	5.1	6.8	10.2
CN[c]	10.3	21.7	30.5	5.7	4.9	10.8	0.5	3.5	12.2

[a]Source contributions are based on fractional contributions of each source to the predicted $PM_{2.5}$ mass concentrations. [b]Including Hong Kong Special Administrative Region (SAR) and Macau SAR [c]Including Hong Kong SAR, Macau SAR, and Taiwan Province

generate 1000 estimations of premature mortality using eq 2 with the gridded population data. The mean of the 1000 premature mortality values was used as the central estimation, and the 2.5 and 97.5 percentile values among the 1000 premature mortality values were considered as the lower and upper estimations of the 95% confidence intervals (CI95). Premature mortalities (central and lower and upper bound estimations) for each 5-year sex/age group were estimated and added to get the overall premature mortality and the associated uncertainties for the grid cell. For provincial premature mortality estimation, the central estimations at each grid cell within a provincial boundary were added and considered as the central estimation for the province. A similar approach was used to generate the upper and lower bound estimations for the province. For grid cells that belong to multiple provinces, the fractional areas that the grid cell falls into in each province were used to split the estimated premature mortality.

2.4. Evaluating the Effectiveness of $PM_{2.5}$ Reduction on Reducing Premature Mortality. The China Council for International Cooperation on Environment and Development proposed to reduce $PM_{2.5}$ concentrations in the Beijing–Tianjin–Hebei (BTH) region in the North China Plain (NCP) by over 35% relative to the concentration levels in 2013 and in the Jiangsu–Zhejiang–Shanghai (JZS) region in the Yangtze River Delta (YRD) by 30%. The Pearl River Delta (PRD) region should meet grade II of the Chinese Ambient Air Quality Standard (CAAQS, 35 $\mu g\ m^{-3}$).[50] Coordinated emission reduction plans have been proposed for these regions to achieve the targets. To evaluate the effectiveness of the proposed PM reduction goals on reducing premature mortality, calculations were performed for regional and provincial mortality from 5% to 95% $PM_{2.5}$ concentration reductions.

3. RESULTS

3.1. Estimated Regional and Provincial Premature Mortality. Figure 2a shows the observation-fused annual $PM_{2.5}$ concentrations in China for 2013, with a maximum of >200 $\mu g\ m^{-3}$ in the southern part of the NCP and in the Sichuan Basin. The population-weighted annual $PM_{2.5}$ concentration is 62.6 $\mu g\ m^{-3}$, which is higher than that estimated by Brauer et al.[24] (54.8 $\mu g\ m^{-3}$) and used in the Global Burden of Disease (GBD) 2013[51] but lower than that used in the GBD 2010 (72.6 μg

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Environ. Sci. Technol. XXXX, XXX, XXX−XXX

m^{-3}).[52] The population-weighted annual $PM_{2.5}$ concentration based on the original CMAQ simulation without observation fusing is 59.5 $\mu g\ m^{-3}$. Since the original CMAQ results are lower than observations at a number of urban areas with high population density, the higher estimation based on the observation-fused data likely better reflects the exposure level.

The total premature mortality due to COPD, LC, IHD, and CEV for adults ≥30 years old is shown in Figure 2b. In major urban areas with high population density, the total mortality can be much greater than 3000 per grid (i.e., 36 × 36 km^2). High premature mortality occurs in the NCP, the Sichuan Basin, the YRD, and the PRD regions. The estimated national $PM_{2.5}$-related premature mortality for adults ≥30 years old in 2013 is approximately 1.30 million, with a CI95 of 0.69−1.78 million (Table S4, SI). This estimation is very close to the estimation by Liu et al.[53] (1.37 million premature deaths, CI95 0.89−1.74) and the estimation by Lelieveld et al.[8] (1.36 million premature deaths including $PM_{2.5}$ and O_3), even though different $PM_{2.5}$ exposure methods and different mortality database were used in these studies. As shown in Figure 2c−f, CEV is the major cause of death (742 000, CI95 253−886), followed by IHD (306 000, CI95 237−527), COPD (144 000, CI95 74−207), and LC (139 000, CI95 48−189). The relative contributions of these diseases to the total mortality are also similar to those estimated by Liu et al.[53] and Lelieveld et al.,[8] as shown in Table S5 (SI). The population-weighted attributable fractions (PAFs) of the premature mortality due to $PM_{2.5}$ for these four diseases were calculated using the gridded population data and the gridded RR calculated using eq 1. On the national level (Table S6, SI), PAFs for COPD, LC, IHD, and CEV are 20% (CI95 11−29%), 25% (CI95 9−36%), 24% (CI95 19−43%), and 42% (CI95 15−52%), respectively.

The premature mortality for each province was also estimated (Table S4, SI). Shandong, located in the south part of the NCP, has the highest $PM_{2.5}$-related premature mortality of 110 000 (CI95 54−146), as well as the third highest per capita mortality of 11.5 per 10 000 person-years. Hebei and Henan top the per capita mortality, with 12.0 and 11.7 per 10 000 person-years, respectively. In addition, eight other provinces also have per capita mortality greater than 10 per 10 000 person-years, which is approximately twice the global average estimated by Lelieveld et al.[8] Only Xinjiang, Xizang, and Taiwan have per capita mortality lower than the global average. Although Xinjiang shows a low mortality of 3.9 per person-years, its capital city Wulumuqi (Urumqi; population ∼3 million) has an observed annual $PM_{2.5}$ concentration of 85 $\mu g\ m^{-3}$, which leads to a much higher per capita mortality than the provincial average.

Relative contributions of COPD (9.5−12.4%) and LC (8.2−12.6%) to the total mortality do not vary significantly among different provinces. However, relative contributions of IHD (21.7−31.6%, Taiwan 46.6%) and CEV (49.9%−58.0%, Taiwan 33.8%) show larger variations, as the PAFs of these two diseases are more sensitive to the changes in $PM_{2.5}$ concentrations in the current range. PAFs were also calculated for each province (Table S6, SI). As the highest $PM_{2.5}$ concentrations occur in Hebei, PAFs for COPD, LC, IHD, and CEV are 27% (CI95 16−38%), 35% (CI95 14−46%), 28% (CI95 23−50%), and 50% (CI95 18−57%), respectively. In addition to the provinces in the NCP, provinces in eastern and central China, as well as in the Sichuan Basin, also have higher PAFs.

3.2. Source Contributions to Premature Mortality. Contributions of eight source categories and SOA to premature mortality were also determined. Unlike the emission subtraction method used by Lelieveld et al.,[8] the source-oriented simulations provide the source contributions in a mass conservative way so that the predicted $PM_{2.5}$ concentrations from all sources add up to the total. Regional distributions of the predicted fractional contributions to $PM_{2.5}$ concentrations associated with these sources are shown in Figure S2 (SI). Industrial and residential sources are the two most significant contributors to $PM_{2.5}$ and thus excess mortality, while the contributions from power generation and transportation are lower (Figure S3, SI). Relative source contributions to $PM_{2.5}$-related premature mortality in each province are listed in Table 1. In most provinces, industries are the largest contributor, including 10 out of the 12 provinces with per capita premature mortality greater than 10 per person-years. In these 10 provinces, the contributions range from 29.0% in Hunan to 34.6% in Hebei and Shanxi. The industrial province Liaoning has the largest industrial contribution (35%). Residential sources generally rank second and are the top contributor in Sichuan (29.8%) and Shaanxi (28.1%). The contributions are higher in northern and western China (20−25%) than in southern China (10−15%). Heilongjiang, the north-most province, has the highest contribution from residential sources (32.3%).

A significant amount of $PM_{2.5}$ in China exists in the form of ammonium nitrate or ammonium sulfate.[28] Agriculture sources, by emitting a large amount of NH_3, account for a significant fraction of $PM_{2.5}$ based on the mass of ammonium ion (NH_4^+). Our estimation suggests that 10−14% of $PM_{2.5}$, and thus the premature mortality, is related to NH_4^+ from agriculture NH_3. Our estimation of agriculture contributions is lower than that of Lelieveld et al.[8] (29%, Table S5, SI), partly because zeroing-out NH_3 emissions from agriculture sources would significantly reduce the formation of secondary nitrate, which is generally due to NO_x emitted from nonagriculture sources.

The estimated contributions of power generation to excess mortality are approximately 10−12% in most provinces. This is lower than the estimation by Lelieveld et al.[8] (18%, Table S5, SI) partly because of the significant reduction of SO_2 emissions from coal-fired power plants in recent years.[54,55] It should be noted that while the contribution of coal in power plants is low, a significant fraction of coal is still used in industrial and residential sources. The contribution of transportation-related sources is approximately 5.7% in China, with the largest contribution being 7.8% in Beijing. This is in accordance of the 3% estimation by Lelieveld et al.[8] (Table S5, SI). Our calculation suggests that SOA accounts for approximately 10% of population exposure to $PM_{2.5}$ in China. Its contribution ranges from 7 to 10% in northern China, mostly from anthropogenic emissions of aromatic compounds, to 10−20% in southern provinces, mostly from biogenic emissions of isoprene and other terpenes.[46] Open burning contributions are higher (10−20%) in southern provinces (e.g., Fujian, Jiangxi, and Yunnan). Windblown dust accounts for 2−3% of the population exposure to $PM_{2.5}$ in most provinces, but its contributions can be as high as 15−25% in Xinjiang and Neimeng (Inner Mongolia), northwest provinces close to the dust source regions.

3.3. Effectiveness of $PM_{2.5}$ Reduction on Reducing Premature Mortality. Figure 3a shows that the decrease of excess mortality in the BTH, JZS, and PRD regions is slower in the beginning when $PM_{2.5}$ concentrations are higher, and the marginal benefit of $PM_{2.5}$ reduction to excess mortality

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Environ. Sci. Technol. XXXX, XXX, XXX−XXX

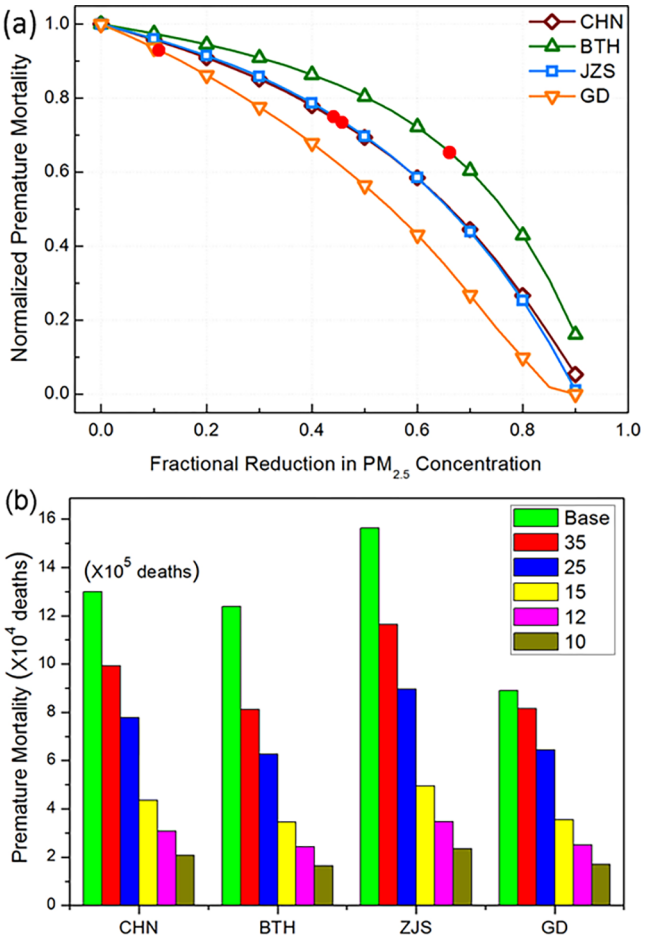

Figure 3. (a) Premature mortality (normalized to 2013 deaths) as a function of fractional reduction in $PM_{2.5}$ concentrations (relative to the 2013 concentrations) for the whole of China (CHN); Beijing, Tianjin, and Hebei (BTH) in the NCP region; Jiangsu, Zhejiang and Shanghai (JZS) in the YRD region; and Guangdong (GD) in the PRD region. The solid dots represent the normalized premature mortalities when $PM_{2.5}$ concentrations are reduced to 35 $\mu g\ m^{-3}$ (CAAQS grade II standard, red dots). (b) Number of premature deaths in CHN, BTH, ZJS, and GD corresponding to the cases shown in panel a. "Base" refers to $PM_{2.5}$ in the year 2013.

increases as PM concentrations decrease. A 35% reduction in $PM_{2.5}$ concentrations in BTH would only lead to an 11% reduction in premature mortality relative to the 2013 level (without accounting for increase of population). A 30% reduction in JZS would lead to a 13% reduction in mortality from the 2013 level. In the PRD region [using Guangdong province (GD) as an example], meeting the CAAQS standard would only result in 8% reduction in mortality. A 40% reduction of $PM_{2.5}$ uniformly in China results in a 22% reduction of premature mortality and even less reduction in the provinces that experience higher mortality (Figure S4, SI). To reduce the premature mortality by 30% from the 2013 levels, a reduction of $PM_{2.5}$ by 50% throughout the country is needed. $PM_{2.5}$ concentrations need to be reduced by 62%, 50%, and 38% for BTH, JZS, and GD, respectively, to achieve a 50% reduction in $PM_{2.5}$-related excess mortality.

Figure 3b evaluates the premature mortality benefit when $PM_{2.5}$ concentrations in the three regions and in China as a whole are reduced to five different levels, i.e., the CAAQS grade II annual standard of 35 $\mu g\ m^{-3}$, the WHO interim target 2 (IT2) of 25 $\mu g\ m^{-3}$, the WHO IT3 of 15 $\mu g\ m^{-3}$, the United States (U.S.) Ambient Air Quality Standards (NAAQS) annual standard of 12 $\mu g\ m^{-3}$, and the WHO guideline level of 10 $\mu g\ m^{-3}$.[56] The premature mortality in China due to $PM_{2.5}$ exposure would be reduced by 24% from 1.30 million (CI95 0.69−1.78) to approximately 1 million (0.99 million, CI95 0.43−1.44) when all China grids meet the current CAAQS grade II standard. On the basis of this study, it appears that China needs to further reduce $PM_{2.5}$ to an even lower level to provide sufficient health benefits. Further reducing the $PM_{2.5}$ concentrations to 25 $\mu g\ m^{-3}$ (WHO IT2), 15 $\mu g\ m^{-3}$ (WHO IT3), 12 $\mu g\ m^{-3}$ (U.S. AAQS), and 10 $\mu g\ m^{-3}$ (WHO guideline), the estimated premature mortalities would be reduced to 779 000 (CI95 358−1225), 436 000 (CI95 229−673), 307 000 (CI95 130−540), and 207 000 (CI95 58−453), respectively. The premature mortality benefit of $PM_{2.5}$ reduction at the province level is shown in Table S7 (SI). In the provinces with high premature deaths due to $PM_{2.5}$ pollution, such as Shandong, Henan, Hebei, Sichuan, and Guangdong, reducing the $PM_{2.5}$ to the WHO IT3 of 15 $\mu g\ m^{-3}$ would results in a significant reduction of excess mortality.

4. DISCUSSION

The estimation of premature mortality and source contributions is affected by $PM_{2.5}$ exposure, the exposure−response coefficients used in RR calculation, and the baseline mortality. In this study, extensive efforts were made to enhance the $PM_{2.5}$ exposure by using higher CTM spatial resolution with more China-specific data, using an ensemble of four sets of CTM simulations with different emission inventories, and fusing CTM predictions with observation data in 60 major cities in China. Our estimation of 1.30 million premature deaths in China due to $PM_{2.5}$ exposure is consistent with the estimation by Liu et al.[53] (1.37 million) and by Lelieveld et al.[8] (1.36 million), despite different $PM_{2.5}$ exposure methods. As shown in Figure 3, predicted premature mortality is not very sensitive to changes in $PM_{2.5}$ concentrations at the current levels in China. Data fusing with a larger radius of influence of 108, 180, and 252 km was examined, and only minor changes were found in the premature mortality estimation for most provinces in China. Therefore, $PM_{2.5}$ exposure estimation leads to small uncertainty in premature mortality estimation for China and countries/regions with $PM_{2.5}$ concentrations similar to that of China.

A more important source of uncertainty for premature mortality estimation in China is the exposure−response coefficients. The 95% CIs reported in the previous section indicate relatively large uncertainties with the exposure−response parametrization. With the lack of epidemiologic studies to estimate the association of long-term exposure to high $PM_{2.5}$ levels, coefficients fitted by an integrated exposure−response model by Burnett et al.[18] based on data from exposure of cigarette smoke at high concentrations and of ambient air pollution at low concentrations were used in the study. Even though these coefficients are believed to provide improved estimation, the estimation of premature mortality in China can be further improved with the coefficients derived from long-term epidemiologic studies in China in the future.

Age-specific (but not sex-segregated) parameters for relative risk calculations are only available for IHD and CEV. For consistency, parameters designated as "all ages" were used in previous calculations. A sensitivity analysis was performed using age-specific parameters[18] for IHD and CEV (Table S9, SI). In

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Environ. Sci. Technol. XXXX, XXX, XXX−XXX

general, using age-specific parameters leads to a higher estimation of excess mortality due to IHD by ~12.4% (from 30.2 to 33.9 × 10^4 deaths). However, it also leads to a lower estimation of excess mortality due to CEV by ~14.0% (from 72.6 to 62.5 × 10^4 deaths). Overall, the total excess mortality for the whole country using these age-specific parameters decreases by approximately 4.9% (from 1.30 to 1.25 million deaths).

In this study, it is assumed that $PM_{2.5}$ with different chemical components or source origins poses the same unit health risk. This was also assumed in a number of studies and was supported by the fact that the exposure−response coefficients determined from epidemiological studies show very little spatial variation globally, even though the major and minor chemical compositions of PM show major geographical variations.[57] However, carbonaceous particles from combustion sources are often considered to be more toxic than secondary sulfate and nitrate particles.[58] Lelieveld et al.[8] performed a sensitivity analysis by assuming that carbonaceous components of $PM_{2.5}$ are 5 times more toxic than other components. Diesel particles have been categorized as a toxic air contaminant by the California Air Resources Board due to their strong carcinogenic potential.[59,60] The estimation of premature mortality and its source contributions in China may change when chemical components are considered. However, there are no consistent exposure−response relationships for either $PM_{2.5}$ components or source categories, especially at $PM_{2.5}$ levels similar to those current in highly polluted regions in China, so that a meaningful calculation can be carried out.

Another major source of uncertainty is the baseline mortality. In this study, official country-level mortality data were used. To evaluate the uncertainty from this important input data, the premature mortality calculation was repeated using the baseline mortality data from WHO (Table S2, SI), and the estimated total premature mortality due to the four diseases is 1.66 million (CI95 0.84−2.15). While it is 25% higher than the estimated 1.30 million using the official baseline mortality, it is still well within the 95% CI (0.61−1.83 million).

Another large uncertainty may rise from the spatial distribution of population. Many people from less economically developed regions, such as central and western China, work in more developed regions, mainly the NCP, YRD, and PRD regions. However, as the predicted $PM_{2.5}$ concentrations are high (e.g., >50 $\mu g\ m^{-3}$) in almost all areas with sufficient population density and a generally uniform baseline mortality, using a more accurate population distribution would not significantly change the total premature mortality, although it will change the estimations for the provinces with large net influx or out-flux of populations more significantly.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge on the ACS Publications website at DOI: 10.1021/acs.est.7b03193.

Rural and urban population baseline mortality for COPD, LC, IHD and CEV for different age groups from the China Public Health and Family Planning Statistical Yearbook (Table S1); baseline mortality from the WHO Mortality Database (Table S2); population age distribution data for China, 2013 (Table S3); premature mortality (×10^4 deaths) and 95% CI from chronic obstructive pulmonary disease (COPD), lung cancer (LC), ischemic heart disease (IHD) and cerebrovascular disease (CEV) in adults ≥30 years old due to long-term exposure of ambient $PM_{2.5}$ based on predicted 2013 annual average concentrations (Tables S4); comparison of the cause of excess mortality by disease and sources from this study with those from other studies (Table S5); attributable fractions and their 95% CI for COPD, LC, IHD, and CEV in adults ≥30 years old due to long-term exposure of ambient $PM_{2.5}$ based on predicted 2013 annual average concentrations (Table S6); provincial premature mortality and 95% CI when $PM_{2.5}$ is reduced to 35, 25, 15, 12, and 10 $\mu g\ m^{-3}$ (Table S7); effect of ensemble and observation-fusing on excess mortality (Table S8); excess mortality (×10^4 deaths) calculated using age-specific relative risk parameters for IHD and CEV and the relative changes comparing to the ones without using age-specific parameters (Table S9); grouping of REAS2 and EDGAR emission sectors to match those in MEIC and SOE inventories (Table S10); spatial distribution of (a) observed annual $PM_{2.5}$ concentrations in 60 Chinese cities and fractional bias of the annual concentration for predictions based on EDGAR, MEIC, REAS2, SOE ,and weighted ensemble of the four inventories (Figure S1); relative source contributions to annual average $PM_{2.5}$ based on the MEIC emission inventory in 2013 (Figure S2); premature mortality (death per area of 36 × 36 km^2) in China due to $PM_{2.5}$ from different sources (Figure S3); relative reduction of excess mortality of each province as a function of population-weighted annual average $PM_{2.5}$ concentrations if concentrations were reduced by 40% of their 2013 value throughout the country (Figure S4); comparison of January emissions of NO_x, SO_2, VOC, and $PM_{2.5}$ from MEIC, SOE, REAS2, and EDGAR inventories and relative contributions of power generation, transportation, residential, and industry sectors to total emissions (Figure S5) (PDF)

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Notes

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Environ. Sci. Technol. XXXX, XXX, XXX−XXX

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Environ. Sci. Technol. XXXX, XXX, XXX−XXX