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Historical Trends in $PM_{2.5}$-Related Premature Mortality during 1990-2010 across the Northern Hemisphere

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Short running title: Historical trends in $PM_{2.5}$ related mortality

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Abstract

Background: Air quality across the northern hemisphere over the past two decades has witnessed dramatic changes, with continuous improvement in developed countries in North America and Europe, but a contrasting sharp deterioration in developing regions of Asia.

Objective: This study investigates the historical trend in the long-term exposure to $PM_{2.5}$ and $PM_{2.5}$-related premature mortality ($PM_{2.5}$-mortality) and its response to changes in emission that occurred during 1990-2010 across the northern hemisphere. Implications for future trends in human exposure to air pollution in both developed and developing regions of the world are discussed.

Methods: We employed the integrated exposure-response model developed by Health Effects Institute to estimate the $PM_{2.5}$-mortality. The 1990-2010 annual-average $PM_{2.5}$ concentrations were obtained from the simulations using WRF-CMAQ model. Emission mitigation efficiencies of SO_2, NO_x, NH_3 and primary PM are estimated from the $PM_{2.5}$-mortality responses to the emission variations.

Results: Estimated $PM_{2.5}$-mortalities in East Asia and South Asia increased by 21% and 85% respectively, from 866,000 and 578,000 in 1990, to 1,048,000 and 1,068,000 in 2010. $PM_{2.5}$-mortalities in developed regions, i.e., Europe and high-income North America decreased substantially by 67% and 58% respectively.

Conclusions: Over the past two decades, correlations between population and $PM_{2.5}$ have become weaker in Europe and North America due to air pollution controls but stronger in East Asia due to deteriorating air quality. Mitigation of primary PM appears to be the most efficient way for increasing health benefits, i.e., providing the largest mortality reduction per unit emissions. However, reductions in emissions of NH_3 are needed to maximize the effectiveness of NO_x emission controls.

Introduction

Fine particles, i.e., $PM_{2.5}$ defined as aerosols with aerodynamic diameter of 2.5 μm or less in the atmosphere, have been well known for their potential negative impacts on human health that can affect the respiratory, cardiovascular and cerebrovascular system. The robust association between ambient $PM_{2.5}$ mass concentration and public health has been demonstrated by epidemiologic studies across the world (Pope et al. 1995; Katsouyanni et al. 2001; Pope and Dockery 2006; Chen et al. 2011), but most previous studies focused on a limited range of ambient annual average concentrations, typically from approximately 5 $\mu g/m^3$ to 30 $\mu g/m^3$, in developed countries (Dockery et al., 1993; Pope et al., 1995; Pope and Dockery, 2006). Recent studies (e.g., Pope et al., 2009; Pope et al., 2011; Burnett et al., 2014) enabled the extension of the traditional calculation method for global scale applications by using increments of cigarette smoking as equivalent concentration of $PM_{2.5}$. As one example, the integrated exposure-response model (IEM) reported in Global Burden of Disease 2010 was successfully applied in an estimation of health impacts of long-term exposures to $PM_{2.5}$ at the global scale, and the result suggests the number of deaths attributable to ambient $PM_{2.5}$ was 3.2 million worldwide in 2010 (Lim et al., 2012).

Assessment of the avoided human health impacts associated with air quality improvements is critical for the design and implementation of air pollution control policy. Some studies have estimated air quality changes and health benefits from emission mitigations by using sensitivity analysis with a chemical transport model. For example, Fann et al. (2009) quantified the health benefits from controls on different locations, sources, and emission types by employing the Community Multiscale Air Quality (CMAQ) response surface model. They further characterized the $PM_{2.5}$ air quality impacts and human health benefits from 17 emission sectors across the

Continental U.S. by using the Comprehensive Air Quality Model with Extensions (CAMx) source apportionment air quality modeling techniques (Fann et al, 2012). Lee et al. (2015) calculated the sensitivities of global $PM_{2.5}$ related premature mortality (here after referred to as $PM_{2.5}$-mortality) to emissions by using the adjoint of the GEOS-Chem chemical transport model. Lelieveld et al. (2015) used the zero-out method in a global atmospheric chemistry model to investigate the link between premature mortality and seven emission source categories for conditions representative of 2010. To date, however, little effort has been devoted towards assessing long-term exposure to $PM_{2.5}$ and health impacts associated with recent historical trends in air pollution. Compared to the sensitivity analysis which relies highly on the baseline situation (in regards of the nonlinearly responding functions), a historical trend analysis is better constrained, since it is based on actual documented changes. However, a challenge with the approach is the quantification of a reliable historical trend in $PM_{2.5}$ concentrations with adequate spatial resolution.

Anthropogenic emissions of primary aerosol and gaseous precursors of $PM_{2.5}$ have witnessed dramatic changes over the past two decades across the northern hemisphere. During the period 1990-2010, SO_2 and NO_x emissions across the U.S. decreased by about 66% and 50%, respectively (e.g., Xing et al., 2013), while emissions have increased dramatically in many developing regions. These changing emissions have resulted in contrasting trends in the regional aerosol burden across the northern hemisphere. Understanding the historical trend in long-term exposure to $PM_{2.5}$, as well as the health benefits from the air pollution controls in the developed countries and the health risks arising from the deterioration of air quality in developing countries, is crucial for policy design in the future.

This study aims to provide an estimation of historical trends in long-term exposure to $PM_{2.5}$

and its impact on $PM_{2.5}$-mortality during 1990-2010 across the northern hemisphere. The method used to calculate $PM_{2.5}$-mortality and emission mitigation efficiency (EME) is described in section 2. The estimated long-term exposure to $PM_{2.5}$ and mortality and their response to changes in emissions is provided in section 3. Uncertainties and implications for future trends in air pollution exposure are discussed in section 4.

Method

Historical emission and $PM_{2.5}$ concentration trend

Historical emission trends of gaseous pollutants and primary particles used in this study are derived from the combination of the year-specific EDGAR emission inventory (v4.2) for the period 1990-2008 and an extrapolation for the years of 2009 and 2010 based on recent estimates of activity change (Xing et al, 2015a). The SO_2, NO_x, VOC and primary PM emissions in Europe and high-income North America have seen continuous reductions by 5.4/5.4%, 1.5/1.8%, 3.3/3.3%, and 4.8/4.6% per year, respectively during 1990–2010. In contrast, SO_2, NO_x, and VOC emissions in East Asia have increased continuously by 3.2%, 4.2%, and 2.3% per year, respectively. Stricter controls on primary PM emissions have been implemented in China since 2003 (Wang and Hao, 2012), thus its +0.3% per year increase rate is much smaller compared to gaseous pollutants. NH_3 emissions exhibit an increasing trend both in China (+2.6% per year) and the United States (+1.6% per year) but exhibit a declining trend in Europe (-1.0% per year) due to stricter controls. Additional details on the emission processing for the model simulations are provided in Xing et al. (2015a).

The 1990-2010 gridded annual-average $PM_{2.5}$ concentrations with 108km×108km resolution over the northern hemisphere (see domain coverage presented in Figure 1) are obtained from simulations with a hemispheric version of the Weather Research and Forecast

(WRF) model coupled with the CMAQ model developed by U.S. Environmental Protection Agency (Wong et al. 2012; Wang et al. 2014). CMAQ is a sophisticated modeling system which has multiple capabilities of simulating concentrations of fine particulate and other air pollutants in the atmosphere involving complex pollutant interactions on urban (e.g., Xing et al. 2011), regional (e.g., Appel et al. 2008) and hemispheric (Mathur et al. 2012; 2014) scales. An extensive examination of the model's ability to capture trends in gaseous precursors, $PM_{2.5}$ chemical composition and aerosol burden has been conducted through comparison with multi-decadal trends in observations. First, the ability of WRF-CMAQ in reproducing the historical trend in AOD over the northern hemisphere has been quantitatively evaluated through a comparison of simulated values over the 21-years with six satellite-retrieved AOD products including AVHRR, TOMS, SeaWiFS, MISR, MODIS-Terra and MODIS-Aqua as well as long-term historical records from 11 AERONET sites during the 1990-2010 period (Xing et al., 2015b). Second, the model performance for simulation of gaseous species and $PM_{2.5}$ composition was future evaluated through comparison with measurements at several ground observation networks mostly over Europe and North America over the past two decades. Results suggested that model simulated ambient $PM_{2.5}$ trends over the past two decades largely agree with those derived from observations (Xing et al. 2015a).

Estimates of health impacts due to long-term exposure to $PM_{2.5}$

The $PM_{2.5}$-mortality estimated in this study includes five causes of premature mortality: ischemic heart disease (IHD), cerebrovascular disease (stroke), chronic obstructive pulmonary disease (COPD), lung cancer (LC) for adults over 25, and acute respiratory lung infection (ALRI) for children under 5. The method is based on the algorithm in the Environmental Benefits Mapping and Analysis Program - Community Edition (BenMAP CE) (e.g., Wang et al. 2015),

released by the United States Environmental Protection Agency, and the Integrated exposure–response model from global burden of disease (GBD) (Lim et al. 2012; Burnett et al. 2014).

The concentration-response functions is summarized as below:

$$Mortality_{PM2.5} = \sum_{i=IHD,stroke,COPD,LC,ALRI} incidence_{0,i} \times PAF_i \times Population \quad [1]$$

in which, $incidence_{0,i}$ is the baseline incidence rate of the cause-specific premature mortality of i. The value of $incidence_{0,i}$ is based on Naghavi et al. (2015). PAF_i is population attributable fraction (PAF) of the cause-specific premature mortality of i. The value of PAF is defined by the following equation:

$$PAF_i = (RR_i - 1)/RR_i \quad [2]$$

where RR_i is the relative risk for the cause-specific premature mortality of i,

$$\begin{cases} \text{for } C < C_0,\ RR_i(C) = 1 \\ \text{for } C \geq C_0,\ RR_i(C) = 1 + \alpha \times \{1 - exp[-\gamma \times (C - C_0)^{\delta}]\} \end{cases} \quad [3]$$

where, C is the ambient $PM_{2.5}$ concentration and C_0 is the threshold value of $PM_{2.5}$ concentration below which there is no additional risk assumed in this study. The estimates of relative risks (i.e. RR_i) were obtained from a lookup table developed by Apte et al. (2015).

The gridded population data are interpolated from the 1990, 1995, 2000, 2005 and 2010 Gridded Population of the World, Version 3 (GPWv3) (CIESIN et al. 2005). The population age structures were obtained from Ahmad et al. (2001). The original population data on a 0.25 degree × 0.25 degree grid was apportioned to individual grid pixels matching the model grid (108km ×108km). The population-weighted average $PM_{2.5}$ is estimated from the annual-average $PM_{2.5}$ concentrations weighted by the population. To quantify the correlations between population and $PM_{2.5}$, we define the ratio of population-weighted average $PM_{2.5}$ to regional average $PM_{2.5}$ as the "population scale factor" (PSF). Thus large PSF (>1) indicate positive correlation between population and $PM_{2.5}$, while small PSF (<1) indicate negative correlation between population and

$PM_{2.5}$.

Calculation of EME

EME, an index to represent the health benefits from unit emission reduction in different source categories, has previously been estimated using sensitivity analysis methods (Fann et al., 2009, 2012; Lee et al., 2015; Lelieveld et al., 2015). In this study, we utilized the changes in air pollution-attributed health effects and emissions over the past two decades to estimate the EME. The simulation of year 1990 is defined as the baseline scenario. Each of the other years simulated from 1991-2010 then represent 20 scenarios with different emission and meteorological conditions relative to 1990. A principle similar to that in source apportionment methods was adopted; changes in concentrations of inorganic aerosols (i.e., sulfate, nitrate and ammonium) are attributed to changes in emissions of their corresponding precursors (i.e., SO_2, NO_x and NH_3, respectively), though it is important to note that this is only a first-order approximation due to the nonlinear coupling of the atmospheric SO_x-NO_x-NH_x system. Changes in concentrations of other inorganic particles (excluding sulfate, nitrate and ammonium) as well as primary organic aerosols are attributed to the change in their emissions (noted as primary PM). Since $PM_{2.5}$ mass is still considered the most robust indicator of mortality impacts in epidemiologic cohort studies of long-term exposure (Chen et al., 2008), the assumption here is that the health effects of exposure to $PM_{2.5}$ are independent of source and composition (Lee et al., 2015). Thus, the impact of various pollutants on mortality are estimated to be proportional to their contribution to PM mass. However, considering the non-linearity of the risk-curve, these estimates could be considered to be on the higher side for cases where one component changes but the others are constant. The year specific EME for each year in the period 1991-2010 relative to the base year of 1990, for an aerosol species, p, is then estimated as follow:

$$Mortality_{p,y} = \frac{Mortality_y}{Concentration_{PM_{2.5},y}} \times Concentration_{PM_p,y} \qquad (y = 1990 \ldots 2010) \quad [4]$$

$$EME_{p,y'} = \frac{Mortality_{p,y'} - Mortality_{p,1990}}{Emission_{p,y'} - Emission_{p,1990}} \qquad (y' = 1991 \ldots 2010) \quad [5]$$

$$p = \{SO_2,\ NO_x, NH_3, primary\ PM\}$$

$$PM_p = \{SO_4^{2-}, NO_3^-, NH_4^+, other\ inorganic\ particles\ and\ primary\ organic\ aerosols\}$$

To limit the influence of year-to-year variations in meteorological condition, the 21-year average EME for a particular species was estimated from a linear regression of the changes in $PM_{2.5}$-mortality and emissions of 1991-2010 relative to 1990.

Results

Population-weighted $PM_{2.5}$ and population scaled factor (PSF)

We focus our analysis on 6 selected regions within the northern hemisphere simulation domain displayed in Figure 1, including two high population and also polluted regions (East Asia and South Asia), two well-developed regions (Europe and high-income North America), and two regions within or downwind of deserts (North Africa and the Middle East, Eastern and Western Sub-Saharan Africa). The distribution of surface $PM_{2.5}$ concentrations across grid cells grouped by population in 6 regions is presented in Figure 2. In East Asia and South Asia, most of the population is concentrated in the areas with high annual-average $PM_{2.5}$ concentrations exceeding 10 μg m^{-3} which is the WHO air quality guideline for $PM_{2.5}$. Additionally, compared to 1990, in 2010 a larger population (i.e., area under the curve) is exposed to higher $PM_{2.5}$ levels, suggesting that greater health risks due to $PM_{2.5}$ likely occurred in recent years in these two regions. In the well-developed regions, i.e., Europe and high-income North America, population exposure to high $PM_{2.5}$ levels (above10 μg m^{-3}) was much greater in 1990 compared to 2010, indicating the health benefits from air quality improvement resulting from emission reductions. In the two

desert regions, i.e., North Africa and the Middle East, Eastern and Western sub-Saharan Africa, the range of $PM_{2.5}$ levels to which populations are exposed are similar in 1990 and 2010, but due to population growth a much greater number of people are exposed to $PM_{2.5}$ pollution now compared to 1990. Also, the increased populations are mainly concentrated in the areas with high $PM_{2.5}$ levels (> 10μg m^{-3}), suggesting greater health risks due to $PM_{2.5}$.

To further investigate the correlation between $PM_{2.5}$ and population in 6 typical regions, Figure 3 presents comparisons of the historical trend of population-weighted and regional average $PM_{2.5}$ concentrations from 1990 to 2010. Not surprisingly, contrasting trends in $PM_{2.5}$ concentrations are found between developing and developed regions. These trends are comparable with trends in exposure estimates recently suggested in van Donkelaar et al (2015). Developing regions of East Asia and South Asia exhibit continually increasing trends in regional average $PM_{2.5}$ concentrations of +0.66 and +0.43 μg m^{-3} yr^{-1}, respectively, while developed regions including Europe and high-income North America have experienced decreasing trends in regional average $PM_{2.5}$ concentrations of -0.37, and -0.14 μg m^{-3} yr^{-1}, respectively. The population-weighted $PM_{2.5}$ in East Asia and South Asia increased from 24.6 and 15.6 μg m^{-3} in 1990, to 36.1 and 23.0 μg m^{-3} in 2010, while the population-weighted $PM_{2.5}$ in Europe and high-income North America decreased from 16.6 and 14.5 μg m^{-3} in 1990 to 9.6 and 9.1 μg m^{-3} in 2010.

Additionally, for all regions except North Africa and the Middle East, the population-weighted $PM_{2.5}$ concentrations are always higher than the regional averages. This can be explained by the positive correlation in spatial distributions between population and $PM_{2.5}$ concentrations. In regions where ambient $PM_{2.5}$ is mainly caused by anthropogenic sources, populated areas are usually also polluted, leading to a positive correlation between population

and $PM_{2.5}$. However, in regions where natural sources (wind-blown dust) are dominant, e.g., North Africa and the Middle East, since dust areas are not amenable for living, a negative spatial correlation between population and $PM_{2.5}$ concentrations is expected, resulting in lower population-weighted $PM_{2.5}$ compared to the regional averages.

The PSF value can be used to measure the extent of correlation between population and $PM_{2.5}$. Large PSF (>1) indicate positive correlation between population and $PM_{2.5}$. Figure 3g summarizes the PSF trends in the 6 regions. Large 21-year average PSF (around 2.0) are found in East Asia, Europe and high-income North America. Small PSF are found in North Africa and Middle East (around 0.79), indicating a negative correlation between the population and $PM_{2.5}$ concentration. Additionally, contrasting PSF trends during 1990-2010 are evident in the 6 regions. The PSF trends are statistically significant at the 95 % confidence level ($p = 0.05$) in all regions except East Asia and Eastern and Western Sub-Saharan Africa. In East Asia and South Asia, the simultaneous growth of population and $PM_{2.5}$ concentrations during past two decades leads to an increasing trend in PSF (from 1.99/1.22 in 1990 to 2.11/1.27 in 2010), suggesting more serious health impacts from exposure to air pollution. Conversely, Europe and high-income North America exhibit decreasing trends in PSF, suggesting increasing health benefits from air pollution controls.

Estimated $PM_{2.5}$-mortality

The estimated annual-average $PM_{2.5}$-mortality for 1990 and 2010 and its change from 1990 to 2010 in the 6 regions are summarized in Table 1. Among the 6 regions, estimated $PM_{2.5}$-mortality in 2010 is highest in two populated regions with poor air quality, i.e., East and South Asia, where the $PM_{2.5}$-mortality increased by 21% and 85% respectively, from 866,000 and 578,000 in 1990, to 1,048,000 and 1,068,000 in 2010. North Africa and the Middle East, Eastern

and Western Sub-Saharan Africa also experienced a substantial growth of mortality risk due to $PM_{2.5}$ over the past two decades, with increase in $PM_{2.5}$-mortality increased of 38% and 53%, respectively. However, in developed regions including high-income North America and Europe, the $PM_{2.5}$-mortality decreased by 58% and 67% respectively in 2010 compared to 1990. The $PM_{2.5}$-mortality estimates in this study are comparable in magnitude with results in a previous study (Lim et al., 2012) as shown in Table 1. The differences in estimates compared to Lim et al (2012) arise primarily due to differences in the spatial resolution employed in this analysis, since the non-linearity of the risk function enlarges the uncertainties of $PM_{2.5}$-mortality estimates related to the exposure allocation.

The $PM_{2.5}$-mortality is estimated from the product of three factors (see Equation 1), i.e., population, PAF (which depends on $PM_{2.5}$ concentrations), and baseline incidence rate of the cause-specific mortality (influenced by living conditions, access to medical care, etc.). Thus, the trend in $PM_{2.5}$-mortality can be driven by changes in these three factors. To estimate the relative contributions to $PM_{2.5}$-mortality changes from these three factors, analysis was conducted by designing three control scenarios. Impacts due to the change of PAF were estimated from the difference between the base case and a control case in which population and baseline incidence rate of the cause-specific mortality are the same as the base case but the $PM_{2.5}$ concentration in 2010 is set to be that in 1990. Similarly, impacts due to change in population (or due to change of baseline incidence rate of the cause-specific mortality) were estimated from the difference between the base case and the control case in which the population (or baseline incidence rate of the cause-specific mortality) in 2010 is set to be that in 1990, but the other two factors were kept the same as in base case.

Figure 4 displays the $PM_{2.5}$-mortality changes due to three factors in 6 regions. In East Asia

and South Asia, the increase in $PM_{2.5}$ concentration causes an increase in mortalities of 247,000 and 219,000, respectively. The benefits for the $PM_{2.5}$-mortality reduction from the improvement in living conditions and the quality of medical care has been offset by the increased health risk from the deterioration of air quality. Compared to East Asia, the total increase of $PM_{2.5}$-mortality in South Asia is larger because of a larger contribution from population growth and a smaller reduction from the improvement of living conditions and the quality of medical care. In Europe and high-income North America, the decrease in $PM_{2.5}$ concentrations is the major reason for the $PM_{2.5}$-mortality reduction, overwhelming the impact from the other two factors. In the two desert areas, i.e., North Africa and the Middle East, Eastern and Western Sub-Saharan Africa, changes in $PM_{2.5}$-mortality are dominated by population growth. A small fraction of the growth of $PM_{2.5}$-mortality during 1990-2010 is caused by the deterioration in local air quality.

Response of $PM_{2.5}$-mortality to changes in local emissions

Analysis of $PM_{2.5}$-mortality changes to emission variations can be used to estimate the relative contributions of SO_2, NO_x, NH_3 and primary PM emissions to mortalities. Figure 5 displays the relative change in $PM_{2.5}$-mortality associated with the emission variation of these four pollutants in the period of 1991-2010 relative to 1990. The relative contribution of emissions to $PM_{2.5}$-mortality varies substantially in the three regions where anthropogenic emissions are the dominant sources for $PM_{2.5}$. In East Asia, about 89% of the growth in $PM_{2.5}$-mortality over the past two decades is associated with the increase of SO_2, NO_x and NH_3 emissions which have not been as well controlled as primary PM emission, suggesting that the health risk arising in East Asia is mostly due to the increase in secondary inorganic aerosols. The importance of secondary inorganic sources in contributing to $PM_{2.5}$-mortality in East Asia was also noted in Lee et al. (2015). In high-income North America, the $PM_{2.5}$-mortality reduction is

mainly from the primary PM and SO_2 controls which reduced $PM_{2.5}$-mortality by 20,000 and 19,000 respectively from 1990 to 2010. However, such health benefits from primary PM and SO_2 controls are partially (about 11% of $PM_{2.5}$-mortality reduction from primary PM and SO_2 controls during 1990-2010) counteracted by the increased health risk from the growth of ammonia emissions which results in increase of aerosol nitrate and ammonium concentrations. Since the sensitivity shown in Figure 5 is only a first-order approximation, the small negative NH_3 sensitivity noted in late 2000s is associated with the substantial reduction of NO_x emissions which leads to the reduction of particulate ammonium. In Europe, simultaneous controls of all pollutants maximize the health benefit from the emission reductions. The $PM_{2.5}$-mortality was reduced by 87,000, 91,000, 60,000 and 51,000 from 1990 to 2010 due to the emission mitigation of primary PM, SO_2, NO_x and NH_3, respectively.

The EME is a useful indicator to quantify the emission mitigation efficiency in reducing the health risk. Figure 6 presents the relationship between the change in $PM_{2.5}$-mortalities ($\Delta PM_{2.5}$-mortality) and emissions (ΔEmission) for four pollutants in three regions during 1991-2010 relative to 1990. The EME is estimated from the linear regression of $\Delta PM_{2.5}$-mortality / ΔEmission (i.e., the slope of the scatter plots in Figure 6). In East Asia, highly linear correlations between $\Delta PM_{2.5}$-mortality and ΔEmission are noted for SO_2 and NH_3, and the EMEs are 9.8 and 10.8 per k-ton emissions of SO_2 and NH_3 respectively. NO_x has the highest EME, i.e., 17.9 k-ton^{-1} among all pollutants. This is because the simultaneous increase of NH_3 emissions facilitates the formation of aerosol nitrate thus enlarging the $PM_{2.5}$-mortality sensitivity to NO_x emissions. Compared to other pollutants, the relationship between primary PM emission controls and $PM_{2.5}$-mortality response during 1991-2010 displays no clear trend, indicating that the variation of primary PM emissions does not dominate this relationship in East Asia. As illustrated in Figure

6a, the changes in the magnitude of primary PM emissions in East Asia is much smaller than those of NO_x, SO_2, and NH_3. Further, inter-annual variability in the meteorology may contribute to the variability in the estimated contribution to $PM_{2.5}$ (and its associated mortality). Additionally, uncertainties in estimates of primary PM emissions from household combustion in East Asia could also contribute to the noted weaker relationship. However, a clear positive correlation between the $PM_{2.5}$-mortality response and primary PM emissions is found in high-income North America, where primary PM has the highest EME (12.7 k-ton^{-1}), followed by SO_2 with an EME of 2.3 k-ton^{-1}. Due to the increase of NH_3 emissions over the past two decades, NO_x emission reductions show small or even negative $PM_{2.5}$-related health benefits. This is because even though HNO_3 concentrations decline in response to NO_x emissions, the growth in NH_3 facilitates higher partitioning of nitrate to the aerosol phase in NH_3-limited conditions. These results are consistent with those of Fann et al. (2012) who reported that directly emitted $PM_{2.5}$ had the highest economic value (estimated from mortalities by applying an estimate of the value of statistical life) for a 1 ton emission reduction, while NO_x had the lowest value, and the value for SO_2 was in between these two. In Europe, all pollutants show highly linear correlations with the $PM_{2.5}$-mortality responses. The EMEs for primary PM, SO_2, NO_x and NH_3 are estimated as 28.3, 6.7, 23.7 and 14.8 k-ton^{-1}, respectively. The mitigation of primary PM is most effective for the health benefits, followed by the reduction of NO_x emissions which become more effective when the NH_3 emissions are simultaneously controlled. In contrast, in high-income North America where NO_x emissions have declined but NH_3 emissions have increased, the $PM_{2.5}$-mortality does not show a decline relative to NO_x emissions.

Discussion and conclusion

Historical trends in long-term exposure to $PM_{2.5}$ and its impacts on premature mortality

were investigated for the period of 1990-2010 across the northern hemisphere. The investigation conducted in this study was based on continuous time series of spatially-resolved concentration and population datasets. Contrasting trends in $PM_{2.5}$-mortality over the past two decades are found in developed versus developing regions. Decreasing trends in $PM_{2.5}$-mortality are noted in Europe and high-income North America, and these reductions are mainly associated with the decrease in $PM_{2.5}$ concentrations as well as the improvement in living condition and the quality of medical care. In contrast, in East and South Asia, the increased health risk from the deterioration of air quality in combination with increasing population, offsets the benefits from the improvement in living conditions and the quality of medical care, resulting in an increasing trend in $PM_{2.5}$-mortality over the past two decades.

Strong positive correlations between the spatial distributions of air pollution and population are found in regions where anthropogenic emission sources are dominant. More serious health impacts from air pollution can thus be expected in regions of East Asia where both population and emissions have shown increasing trends (as in the past two decades). On the other hand, additional health benefits from air pollution controls occur when the positive correlation weakens, as witnessed in Europe and high-income North America over past two decades. To maximize the health benefits, one way to weaken such positive correlation could be by encouraging low-density residential developments, or by reducing pollution in high population regions. Such approaches could to be considered in future urban development strategies.

Mitigation of primary PM emissions appears to be the most efficient way for reducing health impacts of exposure to ambient $PM_{2.5}$. Primary PM from combustion of household fuels could be an important contributor to $PM_{2.5}$-mortality in East Asia. The response of $PM_{2.5}$ concentration to NO_x emissions varies significantly under different NH_3 levels (i.e., NH_3-rich or

-poor conditions) (e.g., Wang et al., 2011). Thus, the assessment of NO_x emission mitigation efficiency in reducing the health risk largely depends on associated measures for NH_3 emissions. NO_x becomes more efficient in reducing $PM_{2.5}$-related health risk with simultaneous control of NH_3, as illustrated by trends in Europe. However, in conditions of declining NO_X but increasing NH_3 emissions, such as in high-income North America, negligible changes in associated $PM_{2.5}$-mortalities to these species is noted. Thus, simultaneous control of NH_3 is necessary to maximize the health benefit from NO_x emission mitigation.

An assessment of VOC emission controls was not considered in this study due to the likelihood that secondary organic aerosols (SOA) were underestimated in the underlying WRF-CMAQ simulations and also because anthropogenic/biogenic splits are highly uncertain. For example, previous studies suggest that the model may underestimate the SOA formation by a factor of 2-6 (Carlton et al. 2010; Foley et al. 2010; Baek et al. 2011). Nevertheless, the importance of VOC mitigation for health benefit should be explored in the future as improvements in the model's ability to simulate SOA are implemented.

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Table 1 Estimated $PM_{2.5}$-mortality due to long-term exposure to $PM_{2.5}$ in 1990 and 2010 (unit: thousand)

Year	Case	East Asia	South Asia	High-income North America	Europe	North Africa and the Middle East	Eastern and Western Sub-Saharan Africa
1990	Lim et al. (2012)[a]	947	538	163	729	136	97
	This study [b]	866	578	122	418	124	96
2010	Lim et al. (2012)	1271	771	110	420	176	105
	This study	1048	1068	51	137	171	147
Incr $=(Y_{2010}/Y_{1990}-1)$	Lim et al. (2012)	34%	43%	-33%	-42%	29%	8%
	This study	21%	85%	-58%	-67%	38%	53%

[a] Data obtained from the http://vizhub.healthdata.org/gbd-cause-patterns/ (last access data: 9/2/2015)
[b] Estimated $PM_{2.5}$-mortality in this study

Figure 1 The northern hemisphere model simulation domain and the six selected analysis sub-regions: East Asia, High-income North America, Europe, South Asia, North Africa and the Middle East, Eastern and Western Sub-Saharan Africa. Map used in this figure was created using ArcGIS software by Esri (www.esri.com).

Figure 2 Changing population exposure to ambient $PM_{2.5}$ levels across the six analysis sub-regions of the northern hemisphere (shown in Figure 1), for the years 1990 (blue dash line) and 2010 (red solid line). Model estimated surface $PM_{2.5}$ concentration across grid cells in a region are grouped by population distributions during 1990 and 2010. d(Population)/d(log([$PM_{2.5}$])) represents the population per unit $PM_{2.5}$ section in log scale. The area below a curve represents the total population for that region for that year.

Figure 3 Comparison of long-term trends (1990-2010) of population-weighted and regional average $PM_{2.5}$ concentrations for the six analysis regions (a-f) and the population scale factor (g). Units for both the population weighted and regional average $PM_{2.5}$ concentrations is $\mu g\ m^{-3}$. The population scale factor is the ratio of the population-weighted average $PM_{2.5}$ to regional average $PM_{2.5}$.

Figure 4 Relative contributions of the baseline incidence rate, changes in population, and changes in $PM_{2.5}$ concentrations to the estimated $PM_{2.5}$-mortality changes during 1990 to 2010 in the different regions across the northern hemisphere. Positive numbers indicate an increase in estimated mortality due to exposure to ambient $PM_{2.5}$, while negative numbers represent a reduction.

Figure 5 Estimated relative changes in $PM_{2.5}$-mortality associated with changes in SO_2, NO_x, NH_3 and primary PM emissions during 1991 to 2010 relative to the 1990 values.

Figure 6 Relationship between changes in $PM_{2.5}$-mortality and changes in emissions in three regions (a) East Asia, (b) high-income North America and (c) Europe. Each data point represents the change for each year during 1991-2010 relative to 1990. The slope of the linear regression between the variables represents the emission mitigation efficiency, i.e., EME.

Figure 1.

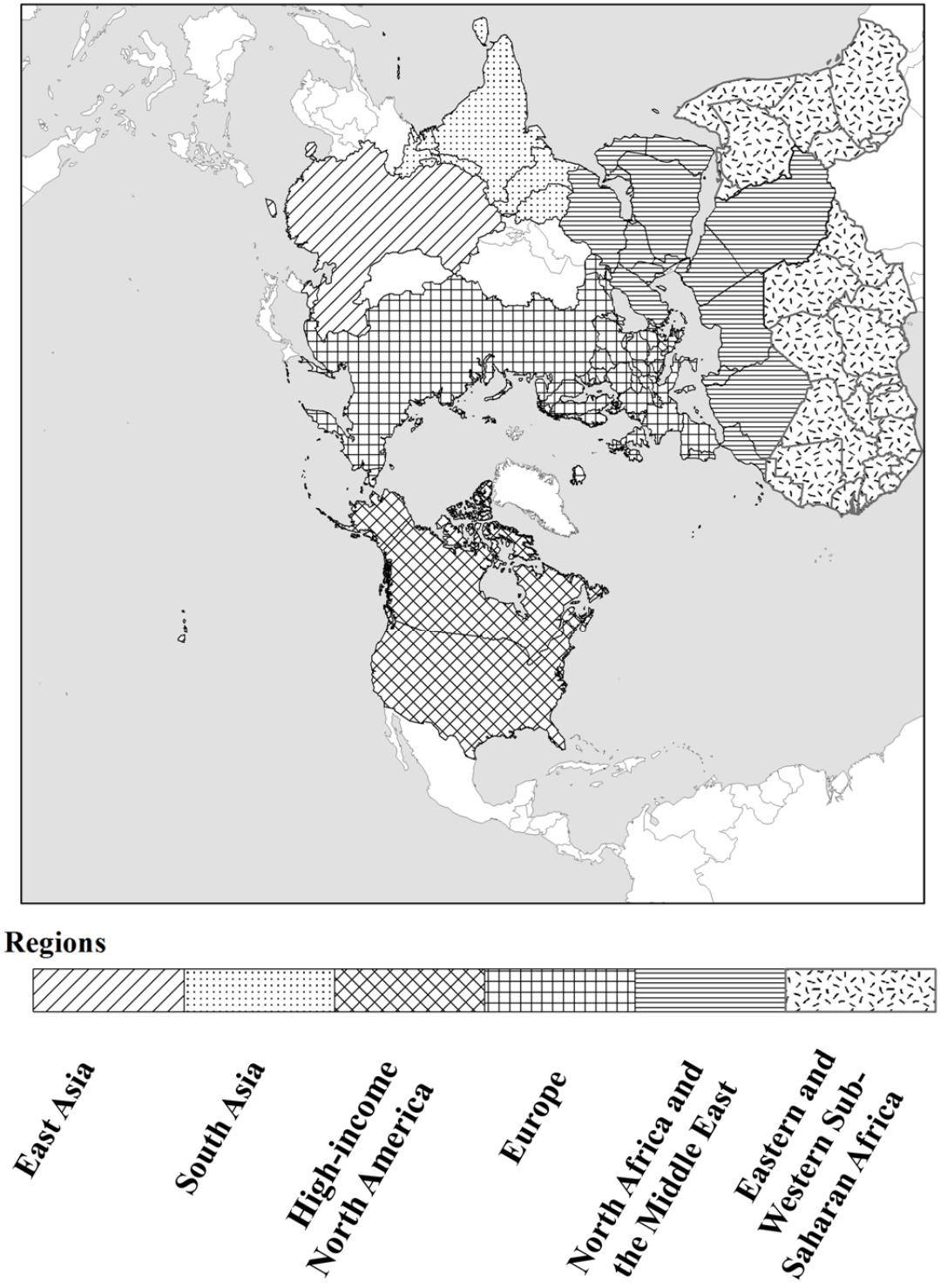

Regions
East Asia
South Asia
High-income North America
Europe
North Africa and the Middle East
Eastern and Western Sub-Saharan Africa

Figure 2.

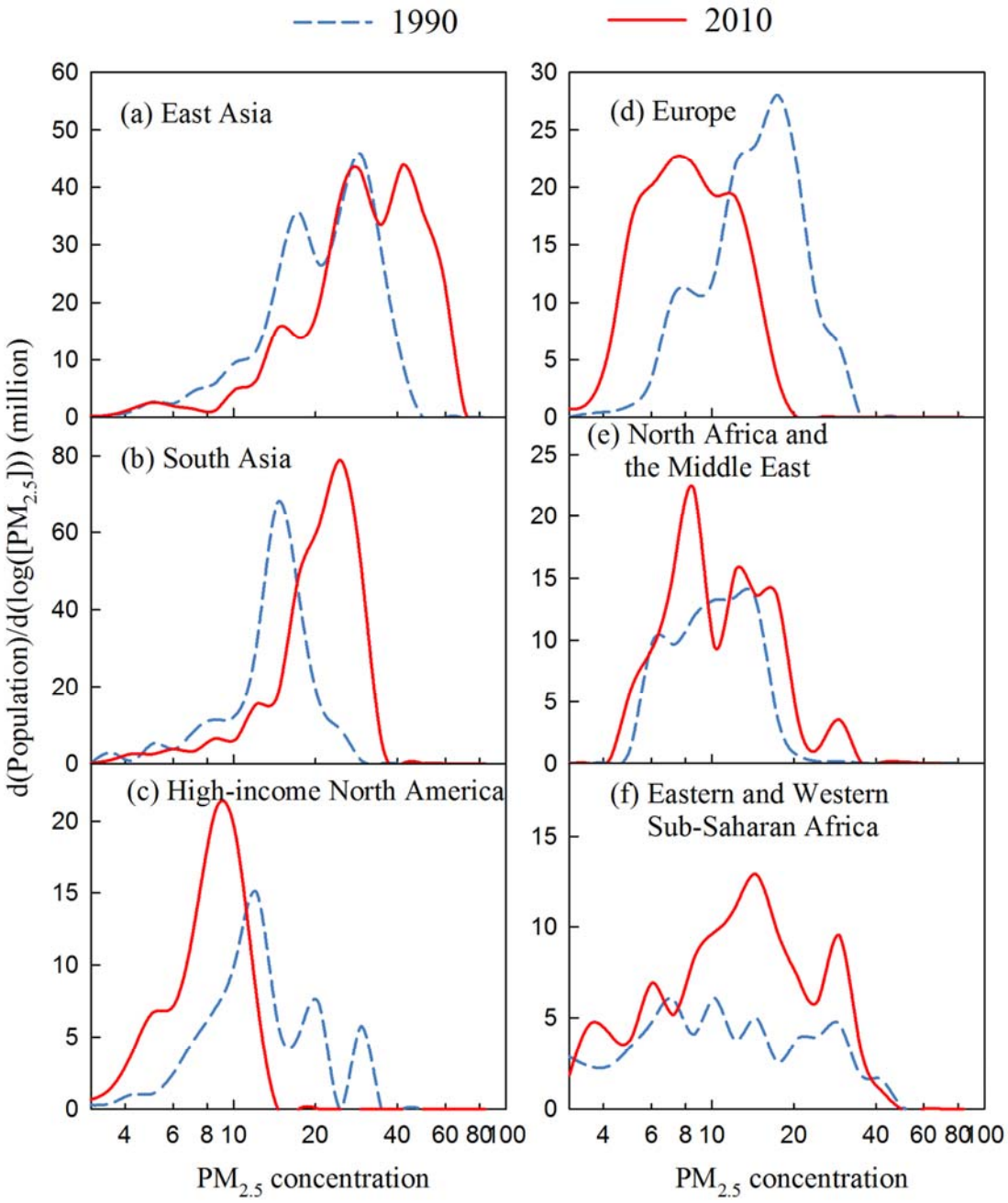

1990
2010
(a) East Asia
(b) South Asia
(c) High-income North America
(d) Europe
(e) North Africa and the Middle East
(f) Eastern and Western Sub-Saharan Africa
d(Population)/d(log([PM2.5])) (million)
PM2.5 concentration
PM2.5 concentration

Figure 3.

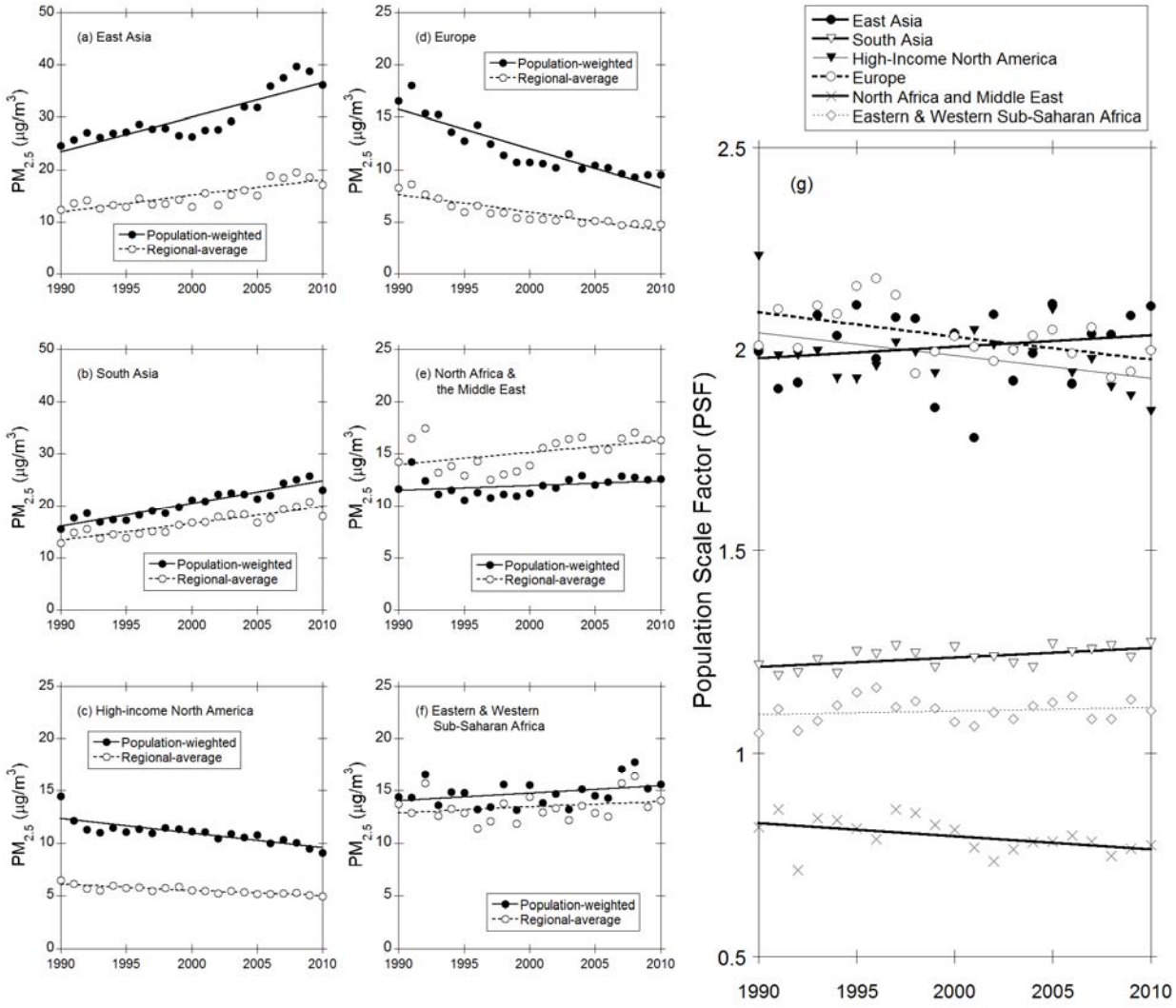

(a) East Asia
(b) South Asia
(c) High-income North America
(d) Europe
(e) North Africa & the Middle East
(f) Eastern & Western Sub-Saharan Africa
Population-weighted
Regional-average
PM2.5 (μg/m3)
East Asia
South Asia
High-Income North America
Europe
North Africa and Middle East
Eastern & Western Sub-Saharan Africa
(g)
Population Scale Factor (PSF)

Figure 4.

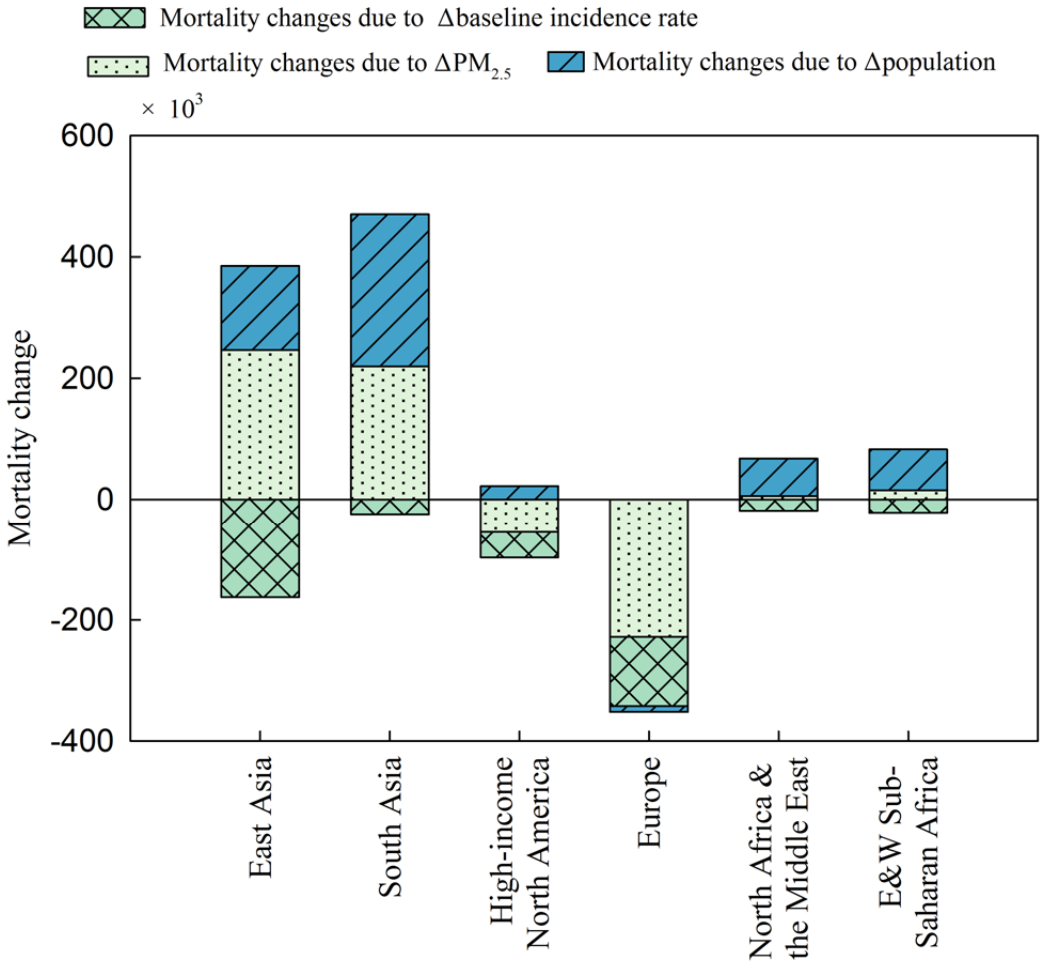

Mortality changes due to Δbaseline incidence rate
Mortality changes due to ΔPM2.5
Mortality changes due to Δpopulation
× 10^3
600
400
200
0
-200
-400
Mortality change
East Asia
South Asia
High-income North America
Europe
North Africa & the Middle East
E&W Sub-Saharan Africa

Figure 5.

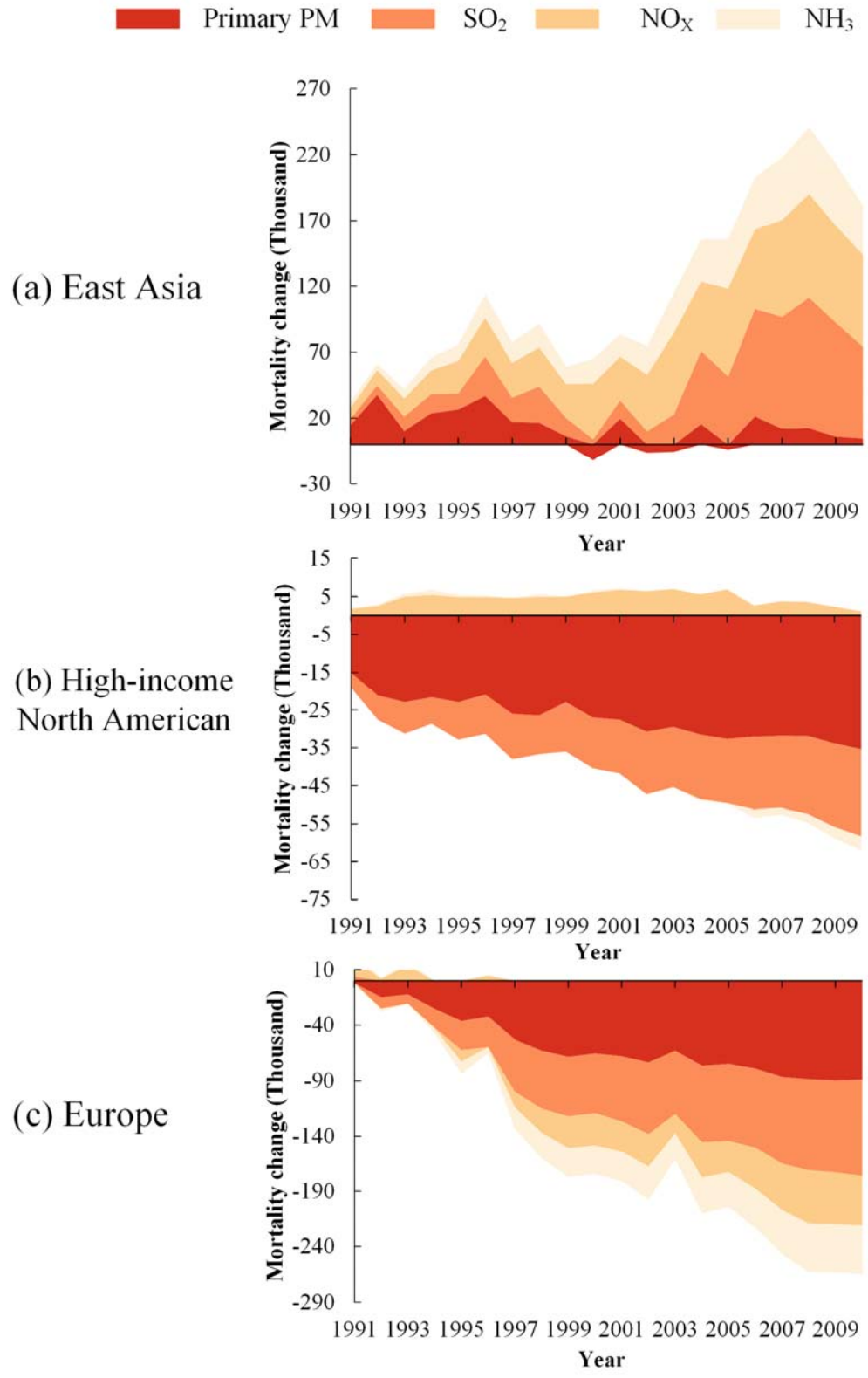
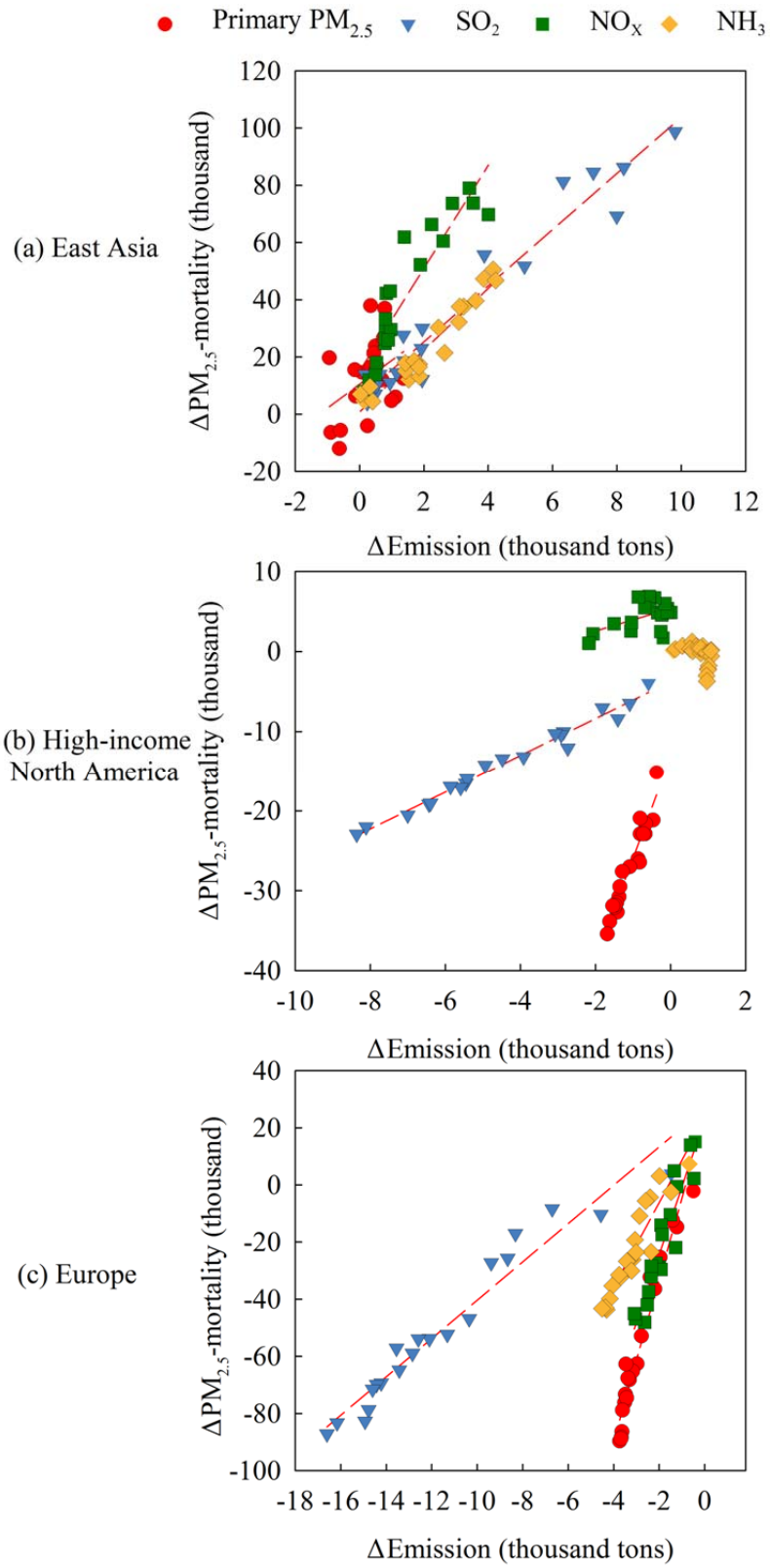

Primary PM
SO_2
NO_X
NH_3
(a) East Asia
Mortality change (Thousand)
270
220
170
120
70
20
-30
1991 1993 1995 1997 1999 2001 2003 2005 2007 2009
Year
(b) High-income North American
Mortality change (Thousand)
15
5
-5
-15
-25
-35
-45
-55
-65
-75
1991 1993 1995 1997 1999 2001 2003 2005 2007 2009
Year
(c) Europe
Mortality change (Thousand)
10
-40
-90
-140
-190
-240
-290
1991 1993 1995 1997 1999 2001 2003 2005 2007 2009
Year

Figure 6.



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