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Long-term (2001–2012) fine particulate matter ($PM_{2.5}$) and the impact on human health in Beijing, China

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Abstract

Beijing, the capital of China, is a densely populated city with poor air quality. The impact of high pollutant concentrations, in particular of aerosol particles, on human health is of major concern. The present study uses Aerosol Optical Depth (AOD) as proxy to estimate long-term $PM_{2.5}$, and subsequently estimates the premature mortality due to $PM_{2.5}$. We use the AOD from 2001 to 2012 from the Aerosol Robotic Network (AERONET) site in Beijing and the ground-based $PM_{2.5}$ observations from the US embassy in Beijing from 2010 to 2011, to establish a relationship between $PM_{2.5}$ and AOD. By including the atmospheric boundary layer height and relative humidity in the comparative analysis, the correlation (R^2) increases from 0.28 to 0.62. We evaluate 12 years of $PM_{2.5}$ data for the Beijing central area using an estimated linear relationship with AOD, and calculate the yearly premature mortality by different diseases attributable to $PM_{2.5}$. The estimated average total mortality due to $PM_{2.5}$ is about 6100 individuals yr^{-1} for the period 2001–2012 in the Beijing central area, and for the period 2010–2012 the per capita mortality for all ages due to $PM_{2.5}$ is around 17.9 per 10 000 person-year, which underscores the urgent need for air pollution abatement.

1 Introduction

Air pollution has intensified strongly since the industrial revolution, i.e., during the epoch known as the Anthropocene (Crutzen, 2002). Ground-level fine particulate matter with a diameter < 2.5 µm ($PM_{2.5}$) has increased substantially, not only in most urbanized and industrialized areas but also in rural and even remote regions (Akimoto, 2003; Anenberg et al., 2010; Schulz et al., 2006). Aerosols have extensive impacts on our climate and environment (Kaufman et al., 2002). $PM_{2.5}$ can have serious health impacts by cardiovascular and respiratory disease and lung cancer, and especially chronic exposure is associated with morbidity and premature mortality (Dockery et al., 1993; McDonnell et al., 2000; Pope III et al., 2009). Concentration-response functions have been used to

ACPD
14, 28657–28684, 2014

estimate mortality due to $PM_{2.5}$ from anthropogenic sources. Globally, air pollution has been estimated to represent 1.4 % of the total mortality attributable to 26 risk factors assessed by the World Health Organization (WHO) global burden of disease project (GBD) (Ezzati et al., 2002). Cohen et al. (2004) estimated that urban $PM_{2.5}$ exposure is responsible for approximately 712 000 cardiopulmonary disease (CPD) and 62 000 lung cancer deaths in 2000. Anenberg et al. (2010) found that anthropogenic $PM_{2.5}$ is associated with 3.5 million CPD and 220 000 lung cancer mortalities annually. Evans et al. (2012) undertook a global assessment of mortality associated with long-term exposure to fine particulate air pollution using remote sensing data and found that the global fraction of adult mortality attributable to the anthropogenic component of $PM_{2.5}$ is 8.0 % for CPD and 12.8 % for lung cancer. The GBD for 2010 indicates that outdoor air pollution in the form of fine particles is a much more significant public health risk than previously assumed (Lim et al., 2012). In China, the GBD estimates 1.2 million premature deaths. Outdoor air pollution ranks number 4 among leading risk factors contributing to deaths in China in 2010.

China has undergone very rapid economic growth since the economic reform beginning in 1978. This has resulted in an increase in energy consumption, air pollution and associated health problems (HEI International Oversight Committee, 2004). Beijing, as a megacity and the capital of China, is one of the most populous cities in the world with 20 million inhabitants (in 2011) over an area of 16 800 km^2. It faces serious air pollution and associated human health problems. Several studies on the characteristics of aerosols in Beijing have been carried out (Cao et al., 2002; Han et al., 2013; Sun et al., 2012; Winchester and Mu-Tian, 1984; Yang et al., 2000), showing that industrial emissions, vehicle exhausts, dust and coal burning are major causes of particulate pollution in Beijing. Regarding particulate matter (PM) in Beijing, both PM_{10} and $PM_{2.5}$ have been extensively studied (Hu et al., 2013; Li et al., 2013b; Sun et al., 2006; Zhang et al., 2013; Zhao et al., 2013, 2009; Zhu et al., 2011). The highest PM_{10} concentrations in Beijing typically occur in April and October according to the records from 2003 to 2009 (Zhu et al., 2011). From the daily PM_{10} concentration measurements collected

ACPD
14, 28657–28684, 2014

at 27 stations in Beijing over a 5 year period, it is found that the overall trend of PM_{10} is generally negative, which applies in particular to summer and winter, while in spring the concentration has increased in recent years (Hu et al., 2013). Pronounced seasonal variation of $PM_{2.5}$, measured from 2005 to 2007 at 5 min time resolution, occurs in the urban area in Beijing, with the highest concentrations typically in the winter and the lowest in the summer (Zhao et al., 2009).

Satellite derived aerosol optical depth (AOD) and aerosol concentrations at the surface ($PM_{2.5}$, PM_{10}) have been analyzed, and high correlations have been found (Li et al., 2005; Gupta et al., 2006; Wang et al., 2010; Zheng et al., 2014). These correlations are partly based on models to infer the surface data from column satellite data, and are strongly influenced by the assumed vertical distribution of aerosols and the relative humidity. AOD and PM are related to atmospheric profiles, ambient conditions, as well as the chemical composition of aerosols. Epidemiological research using time series methods has shown the relationship between PM concentrations and human health in Beijing associated with mortality and morbidity (Li et al., 2013a; Zhang et al., 2012a, b). Nevertheless, these studies have focused on particular periods of a few years or less. In addition, most of these epidemiological studies are based on limited ground-based $PM_{2.5}$ and PM_{10} measurements, which may not represent the city. Since 2013 the Beijing Municipal Environmental Monitoring Centre has started to publish $PM_{2.5}$ data and has included it in the calculation of air quality index (AQI) (Zheng et al., 2014). In addition to air pollution, the population in Beijing has steadily increased over the past decades, being 13 million in 2000 and growing to 21 million in 2013. The long term $PM_{2.5}$ and premature mortality estimation will help with policy decisions aimed at reducing health impacts of $PM_{2.5}$. However, ground-based $PM_{2.5}$ is not available for the period 2001–2012 in Beijing, let alone the premature mortality due to $PM_{2.5}$.

In the present study, we use AOD as proxy to estimate long term $PM_{2.5}$, and then estimate the premature mortality due to $PM_{2.5}$ to assess to what degree $PM_{2.5}$ affects human health in Beijing. We collect the long term AERONET AOD, and analyze its seasonal variability. A linear regression model for $PM_{2.5}$ has been established based on

ACPD
14, 28657–28684, 2014

AOD, considering boundary layer height (BLH) and relative humidity (RH) corrections, allowing the reconstruction of $PM_{2.5}$ concentrations for the last decades. Furthermore, the annual premature mortality attributable to different diseases caused by $PM_{2.5}$ has been estimated by employing concentration-response functions based on epidemiological cohort studies.

2 Data

In this work we use ground-based $PM_{2.5}$, AOD and relative humidity (RH) observations from the US embassy in Beijing, AERONET and the China Meteorological Data Sharing Service System, respectively. We have adopted daily $PM_{2.5}$ data from the US embassy monitoring station as published by Wang et al. (2013). The US embassy is located in the Chaoyang district. Hourly $PM_{2.5}$ concentrations are reported by the US embassy and made available via the Internet. The US embassy monitors the energy decay of beta rays to assess the concentration of particles in the atmosphere. The results obtained from beta ray measurements are usually at least 15 % higher than those collected by oscillating microbalance, according to data on the website of the China National Environmental Monitoring Center (http://usa.chinadaily.com.cn/epaper/2012-10/30/content_15856991.htm). Wang et al. (2013) gathered $PM_{2.5}$ at the US embassy station in Beijing from 10 May 2010, to 6 December 2011. Days with extended periods of missing $PM_{2.5}$ (hourly) data were discarded based on the following criterion: during a day there are consecutive data gaps of more than 3 h or the cumulative amount of missing data exceeds 12 h. The final dataset covers a 423 day period.

The AOD observations are obtained from the Aerosol Robotic Network (AERONET) program, which is a federation of ground-based remote sensing aerosol networks to measure aerosol optical properties (Holben et al., 1998). We use the AERONET level 2.0 data, which are cloud screened and quality assured. The AERONET data for the Beijing site starts on 7 March 2001, and ends on 19 September 2012, and encompasses the AOD at the four wavelengths 1020, 870, 675 and 500 nm. The AERONET

ACPD
14, 28657–28684, 2014

data provides AOD in the form of all points, daily averages, and monthly averages. The daily average AOD is used in this study. The daily RH at the Beijing national meteorological station has been taken from the China Meteorological Data Sharing Service System (http://cdc.cma.gov.cn/home.do). Beijing station lies in the center of Beijing city. The BLH is from ERA-Interim by the European Centre for Medium-Range Weather Forecasts (ECMWF) (Persson and Grazzini, 2005). Daily BLH is the average of BLH values at 00:00, 03:00, 06:00, 09:00, 12:00, 15:00, 18:00, and 21:00 within one day.

3 Analyzing AERONET AOD

The AOD at 550 nm is estimated using the spectral dependence of the AOD at the two nearest wavelengths, generally 500 and 675 nm with the following equations (Ångström, 1964):

$$\tau(\lambda) = \beta\lambda^{-\alpha} \tag{1}$$

$$\alpha = -\frac{\ln(\tau(\lambda_1)/\tau(\lambda_2))}{\ln(\lambda_1/\lambda_2)} \tag{2}$$

$$\beta = \frac{\tau_\alpha(\lambda_1)}{\lambda_1^{-\alpha}} \tag{3}$$

where λ refers to the wavelength, $\tau(\lambda)$ represents the AOD at wavelength λ, and β the Ångström turbidity coefficient which equals the AOD at $\lambda = 1\,\mu m$, and α is the Ångström exponent (AE).

There are 2590 days with valid AOD data from the Beijing site of AERONET during the period 2001 to 2012, and we estimated the daily AOD at 550 nm wavelength for these 2590 days using Eqs. (1)–(3). For the entire dataset the mean value is 0.66 ranging between 0.05 and 4.46. For the monthly data, both the mean and median of AOD values are highest in June, while both the maximum and minimum are highest in April. From April to August the AOD means exceed 0.7.

ACPD
14, 28657–28684, 2014

Beijing has a typical continental monsoon climate with four distinct seasons. Spring (March–May) experiences dust episodes from the Kumutage and Taklimakan deserts in western China and northerly winds from the Mongolian deserts (Sun et al., 2001). Summer (June–August) is characterized by relatively hot and humid weather with southerly winds. Autumn (September–November) is characterized by relatively clear weather. Winter (December–February) is dominated by cold, dry, and windy weather due to cold air from the west Siberian anticyclone (Yu et al., 2013). High AOD values imply very high levels of air pollution and associated negative impacts on human health, while low AOD values represent good air quality. High AOD observed in spring (March–May) is mainly due to dust events over Beijing (Cao et al., 2014). The highest AOD occurs in June despite the aerosol removal by monsoon precipitation, corroborating previous studies, e.g., Wang et al. (2010) who showed that AOD is highest from June to August.

4 Estimating $PM_{2.5}$

4.1 Influence of the BLH and ambient RH

Based on the ground-based $PM_{2.5}$ observations from the US embassy in Beijing from 10 May 2010, to 6 December 2011, a relationship with the observed AOD can be found. The relationship between AOD and $PM_{2.5}$ concentration has been investigated by many researchers. For example, Engel-Cox et al. (2004) developed simple empirical relationships between these two variables over the United States. The direct correlation between the moderate resolution imaging spectroradiometer (MODIS) AOD and $PM_{2.5}$ has been applied to estimate $PM_{2.5}$ across the global urban areas spread over 26 locations, and the results show that the relationship between $PM_{2.5}$ and AOD strongly depends on aerosol concentrations and ambient relative humidity (Gupta et al., 2006). Van Donkelaar et al. (2010) compared the original MODIS and multiangle imaging spectroradiometer (MISR) total-column AOD with ground-based measurements of daily mean $PM_{2.5}$, and both the MODIS and MISR instruments indicate some relationship

ACPD
14, 28657–28684, 2014

between AOD and $PM_{2.5}$, both with spatial correlation coefficients R of 0.39. However, the AOD reflects aerosol optical extinction of the total column, while the $PM_{2.5}$ concentration measurements are at the surface. The correlation between AOD and $PM_{2.5}$ is strongly influenced by the vertical distribution of aerosols and the RH. In order to reduce the uncertainties caused by atmospheric profiles and ambient conditions, the atmospheric BLH and ambient RH have been introduced into the correlation analysis (Koelemeijer et al., 2006; Li et al., 2005; Liu et al., 2005; Wang et al., 2010).

Under the assumption of a plane parallel atmosphere, AOD is the integral of the k_a at all altitudes along the vertical orientation, shown in Eq. (4). $k_a(\lambda, Z)$ represents the aerosol extinction coefficient at altitude Z and wavelength λ. In addition, assuming the vertical distribution of $k_a(\lambda, Z)$ as the negative exponent form is shown in Eq. (5). $k_{a,0}(\lambda)$ refers to the surface level aerosol extinction coefficient at wavelength λ, and H stands for the scale height of the aerosol. Substituting Eq. (5) to Eq. (4) we get Eq. (6). The $k_{a,0}(\lambda)$ could be calculated from AOD and H, and H could be approximately replaced by the atmospheric BLH. Therefore, the vertical correction, AOD/BLH, can reflect aerosol optical extinction at the surface level (Liu et al., 2005).

$$\tau(\lambda) = \int_0^\infty k_a(\lambda, Z)\mathrm{d}z \tag{4}$$

$$k_a(\lambda, z) \approx k_{a,0}(\lambda) e^{-\frac{Z}{H}} \tag{5}$$

$$\tau(\lambda) \approx k_{a,0}(\lambda) \int_0^\infty e^{-\frac{Z}{H}}\mathrm{d}z = k_{a,0}(\lambda) \times H \tag{6}$$

The correlation between aerosol extinction coefficient and $PM_{2.5}$ concentration is influenced by the chemical components of particles and RH of the ambient air. Based on the previous studies (Im et al., 2001; Li et al., 2005; Wang et al., 2010), the RH

ACPD
14, 28657–28684, 2014

correction, $f(\mathrm{RH})$, can be represented as Eq. (7).

$$f(\mathrm{RH}) = (1 - \mathrm{RH}/100)^{-g} \quad (7)$$

Where g is an empirical fit coefficient, and it equals 1 in this study.

4.2 Correlation analysis

We compared the direct relationship between daily AERONET AOD at 550 nm wavelength and $PM_{2.5}$ from 10 May 2010 to 6 December 2011, as shown in Fig. 1a. The correlation between the two datasets is rather poor with an R^2 of 0.28. After considering the influence of the BLH and ambient RH, the former obtained from the ECMWF assimilated analysis model (Persson and Grazzini, 2005) and the latter from the meteorological station, we find that the RH corrected $PM_{2.5}$ ($PM_{2.5} \times f(\mathrm{RH})$) has a much higher correlation with the vertically corrected AOD (AOD/BLH), with an R^2 of 0.62, as shown in Fig. 1b. In addition, we compared the correlation coefficient (R^2) between AOD and $PM_{2.5}$ in this study to that established in some of the previous studies (Engle-Cox et al., 2004; Koelemeijer et al., 2006; Wang et al., 2010; Xin et al., 2014). It is found that our correlative model yields a higher correlation coefficient compared to these studies.

Based on the linear correlation in Fig. 1, Eq. (8) coefficients were derived and Eq. (8) was then used to calculate the daily $PM_{2.5}$ in Beijing from 2001 to 2012 from the AERONET AOD. The results are shown in Fig. 2, and the average and SD for estimated $PM_{2.5}$ during these 12 years is 100.39 $\mu g\,m^{-3}$, and 55.67 $\mu g\,m^{-3}$, respectively.

$$PM_{2.5} = (97\,569 \times \mathrm{AOD}/\mathrm{BLH} + 86.357)/f(\mathrm{RH}) \quad (8)$$

ACPD
14, 28657–28684, 2014

5 Health effects

5.1 Beijing central area

The districts Chaoyang, Dongcheng and Xicheng in Beijing are adjacent and here collectively defined as the Beijing central area. The US embassy is located in the Chaoyang district, and is also close to the Dongcheng and Xicheng districts. We have collected daily $PM_{2.5}$ data of 6 ground stations in these three districts from 8 October 2012, to 13 November 2012, from the study by Zhang et al. (2013). Figure 3 shows the daily $PM_{2.5}$ in the Dongsi and Tiantan stations in Dongcheng district, the Guanyuan and Wanshouxigong stations in Xicheng district, and the Aotizhongxin and Nongzhanguan stations in Chaoyang district. There is no obvious difference among the daily data in these six stations, and the high correlation between Dongsi station and other stations is shown in Fig. 4. Therefore, we have used the daily $PM_{2.5}$ from the US embassy station to represent the $PM_{2.5}$ concentration in the Beijing central area.

5.2 Concentration-response functions

Health effects of $PM_{2.5}$ have been derived from epidemiological cohort studies in a variety of geographical (principally urban) locations, mostly in the USA. Lelieveld et al. (2013) applied an epidemiological health impact function to calculate cardiopulmonary disease and lung cancer mortality attributable to air pollution in 2005. In the function, the concentrations of $PM_{2.5}$ are the yearly average in 2005, and the global population is also for the year 2005. Evans et al. (2012) used a concentration-response function for the association between $PM_{2.5}$ and mortality to calculate the lung cancer, cardiopulmonary disease, and ischemic heart disease mortality. Since we estimated $PM_{2.5}$ from 2001 to 2012 in the Beijing central area, we have calculated the yearly premature mortality caused by $PM_{2.5}$ using concentration-response functions that relate changes in pollutant concentrations to changes in mortality.

ACPD
14, 28657–28684, 2014

No epidemiologic study has estimated the association of long-term exposure to direct measurements of $PM_{2.5}$ with mortality from chronic cardiovascular and respiratory disease in Asia and other developing and emerging countries where annual average $PM_{2.5}$ exposures can exceed 100 $\mu g\,m^{-3}$ (Brauer et al., 2012). Previously, the functions for $PM_{2.5}$ have been based on the relationship between relative risk (RR) and concentrations defined by epidemiology studies where a log-linear (Ostro, 2004) and a linear model (Cohen et al., 2004) were used to calculate RR. However, the coefficients of these models are based on information from a single US cohort study – American Cancer Society Cancer Prevention II, with annual mean exposure levels below 22 $\mu g\,m^{-3}$. The form of the models used for global burden assessment was motivated largely by the concern that linear extrapolation would produce unrealistically large estimated of RR. The Integrated Exposure-Response (IER) model that covers the global range of exposure is developed by integrating RR information from different combustion types that generate emissions of particulate matter, and it was able to take shapes similar to previous models such as linear and log-linear and a power function. In addition to these shapes, it also has the feature of flattening at high exposures. It was shown that it is a superior predictor especially for high $PM_{2.5}$ concentrations (Burnett et al., 2014).

5.3 Mortality estimation and discussion

Long-term exposure to $PM_{2.5}$ is associated with increased mortality from ischemic heart disease (IHD), cerebrovascular disease (stroke, CEV), chronic obstructive pulmonary disease (COPD), and lung cancer (LC), and increased incidence of acute lower respiratory infections (ALRI). Unfortunately, long-term cohort data from Beijing are not yet available. Therefore, we used the IER model to calculate RR over the Beijing central area for causes of premature mortality in adults (> 30 yrs): IHD, CEV, COPD and LC. In addition, the RR for ALRI was also calculated for infants (< 5 yrs). The IER model

ACPD
14, 28657–28684, 2014

has the following form:

$$\begin{aligned} &\text{For } x < x_0, \quad \mathrm{RR} = 1 \\ &\text{For } x \geq x_0, \quad \mathrm{RR} = 1 + a \times \left\{1 - \exp\left[-B \times (x - x_0)^p\right]\right\} \end{aligned} \tag{9}$$

where x is the exposure to $PM_{2.5}$ in $\mu g\,m^{-3}$, and x_0 is the threshold concentration below which it is assumed there is no additional risk. For very large x, RR $\sim 1 + a$. The power of $PM_{2.5}$, p, is used to predict risk over a very large range of concentrations. The parameters (a, B, p) are from the work of Burnett et al. (2014). Table 1 shows details of the IER model for estimating RR for five different diseases (IHD, CEV, COPD, LC, and ALRI). We calculated the value of x as the yearly average $PM_{2.5}$ concentrations at the US embassy monitor station from 2001 to 2012. The threshold concentrations for different diseases are shown in Table 1. Hence, we acquired the yearly RR of these five disease categories caused by $PM_{2.5}$. The fraction of the disease burden attributable to the risk factor (the attributable fraction), AF, is defined as (Anenberg et al., 2010; Ostro, 2004)

$$\mathrm{AF} = (\mathrm{RR} - 1)/\mathrm{RR} \tag{10}$$

To calculate the number of premature mortality cases due to pollution $PM_{2.5}$, the AF is applied to the total number of deaths

$$\Delta\mathrm{Mort} = y_0 \times \mathrm{Pop} \times \mathrm{AF} \tag{11}$$

where ΔMort is the change in annual mortality due to a pollutant (in our study $PM_{2.5}$). Pop is the total population with an age of > 30 yrs or < 5 yrs exposed to the pollutant. y_0 is the baseline mortality rate (BMR) for a given population and a specific disease. The household population and age distribution was obtained by the Beijing statistical yearbook for every year from 2001 to 2012 (see Fig. 5), and the household registration record officially identifies a person as a resident of an area. Since 2010 there is an obvious increase of population in the Beijing central area, which does

ACPD
14, 28657–28684, 2014

not really reflect the real population growth, but rather statistical data collection, because China carried out the fifth and sixth census in the year 2000 and 2010, respectively. The population record has been updated since the sixth census and therefore data before the year 2010 are not considered very accurate. Regarding BMR, we downloaded regional cause-specific mortality estimates (http://www.who.int/healthinfo/statistics/mortality_rawdata/en/index.html), and calculated them for IHD, CEV, COPD, LC, and ALRI in China. It should be emphasized that the calculations scale linearly with the BMR, so countries and regions with relatively high baseline mortality rates have proportionally higher mortality attributed to air pollution.

Based on the health impact function, Eq. (11), we have calculated the yearly premature mortality by IHD, CEV, COPD and LC for people > 30 yrs, and ALRI for infants < 5 yrs in the Beijing central area from 2001 to 2012, which is shown in Fig. 6. The premature mortality due to CEV (> 2580 deaths year^{-1}) is highest among the five diseases and the premature mortality by COPD (> 940 deaths year^{-1}) is the second highest. In addition, the premature mortality by ALRI exceeds 35 deaths year^{-1}. The annual premature mortality attributable to air pollution in the Beijing central area is shown in Fig. 7, as well as the corresponding per capita mortality for all ages. The annual premature mortality is more than 4900 deaths year^{-1}, and shows an increasing trend during 2001–2012, with the highest value of 7783 deaths in 2012. The average premature mortality attributable to $PM_{2.5}$ is around 6100 deaths year^{-1} for the period 2001–2012. The per capita mortality for all ages is 18.0 per 10 000 person-year in 2012, higher than that of 16.6 per 10 000 person-year in 2001. We calculate that the highest per capita mortality (18.6 per 10 000 person-year) occurred in 2004. The per capita mortality for all ages due to $PM_{2.5}$ is around 18.0 per 10 000 person-year for the period 2001–2012. Since the population data are more accurate for the period 2010–2012, the sum of the premature mortality related to each disease and the corresponding ratio to the population section is shown in Table 2 for the years 2010–2012. It is found that the annual average premature mortality attributable to $PM_{2.5}$ is around 7627 deaths year^{-1} for the period 2010–2012 in the Beijing central area. The per capita mortality under adults

ACPD
14, 28657–28684, 2014

(> 30 yrs) in the Beijing central area in 2010–2012 attributable to $PM_{2.5}$ by CEV (15.2 per 10 000 person-year) is higher than any other disease. Further, the per capita mortality (for people > 30 yrs and < 5 yrs) attributable to $PM_{2.5}$ is around 27.6 per 10 000 person-year for the period 2010–2012. The per capita mortality for all ages attributable to $PM_{2.5}$ by CEV (9.3 per 10 000 person-year) is higher than by the other diseases as shown in Fig. 6. For the period 2010–2012 the per capita mortality for all ages due to $PM_{2.5}$ is around 17.9 per 10 000 person-year.

There are few long-term cohort studies for chronic cardiovascular and respiratory disease and lung cancer in East and South Asia, where ambient exposures are often higher than in other parts of the world. The IER model by Burnett et al. (2014) is very useful in this respect as it extends the risk estimates to higher exposures. While the IER model yields state-of-the-science predictions of the risk over a range of concentrations that prevail in China, some limitations of this approach remain. There are uncertainties due to lack of information on actual exposure to $PM_{2.5}$ for some source-specific RRs used to fit the model. Additionally, the IER model is developed for the entire global exposure to air pollution, not specific to Beijing, hence assuming that the toxicity of particulates is the same everywhere. Fortunately, the IER model considers the shape of the mortality RR functions at high ambient concentration, which is suitable for Beijing. Therefore, it is a useful approach to estimate the mortality attributable to $PM_{2.5}$ by IHD, CEV, COPD, and LC, and ALRI in Beijing.

6 Conclusion

We have analyzed the seasonal distribution and characteristics of AOD at 550 nm wavelength in Beijing during the decadal period 2001–2012. Long-term $PM_{2.5}$ concentrations were calculated using an estimated linear relationship with AOD. The average and SD of the estimated $PM_{2.5}$ from 2001 to 2012 is 100.39 $\mu g\,m^{-3}$, and 55.67 $\mu g\,m^{-3}$, respectively. Using concentration-response functions based on epidemiological cohort studies, we estimated the yearly mortality attributable to $PM_{2.5}$ by IHD, CEV, COPD

ACPD
14, 28657–28684, 2014

and LC among people > 30 yrs and that by ALRI among infants < 5 yrs in the Beijing central area from 2001 to 2012. The estimated total mortality in central Beijing is 7627 deaths year^{-1} (average 2010–2012), and the per capita mortality for all ages is around 17.9 per 10 000. Due to the growing population rate of Beijing this study corroborates the urgency of air pollution abatement strategies within its urban area.

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ACPD
14, 28657–28684, 2014

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ACPD
14, 28657–28684, 2014

Table 1. The IER model for estimating RR associated with long-term exposure to $PM_{2.5}$.

Disease	Age	Parameters			
Ischemic Heart Disease (IHD)	> 30 yrs	$a = 1.65$	$B = 0.0483$	$p = 0.467$	$X = 7.45$
Cerebrovascular Disease (CEV)	> 30 yrs	$a = 1.31$	$B = 0.0120$	$p = 1.274$	$X = 7.36$
Chronic Obstructive Pulmonary Disease (COPD)	> 30 yrs	$a = 22.16$	$B = 0.00110$	$p = 0.697$	$X = 7.34$
Lung Cancer (LC)	> 30 yrs	$a = 159.22$	$B = 0.00020$	$p = 0.759$	$X = 7.35$
Acute Lower Respiratory Infections (ALRI)	< 5 yrs	$a = 2.38$	$B = 0.00380$	$p = 1.193$	$X = 7.30$

ACPD
14, 28657–28684, 2014

Table 2. Annual mortality attributable to air pollution by disease category, and the corresponding per capita mortality (IHD, CEV, COPD, and LC for people > 30 yrs, and ALRI for infants < 5 yrs) in 2010–2012 in the Beijing central area.

Disease	IHD	CEV	COPD	LC	ALRI	Total
Annual mortality	1322	3967	1495	773	71	7627
Per capita mortality (per 10 000 person-yr)	5.1	15.2	5.7	3.0	4.4	27.6
Per capita mortality for all ages (per 10 000 person-yr)	3.1	9.3	3.5	1.8	0.2	17.9

ACPD
14, 28657–28684, 2014

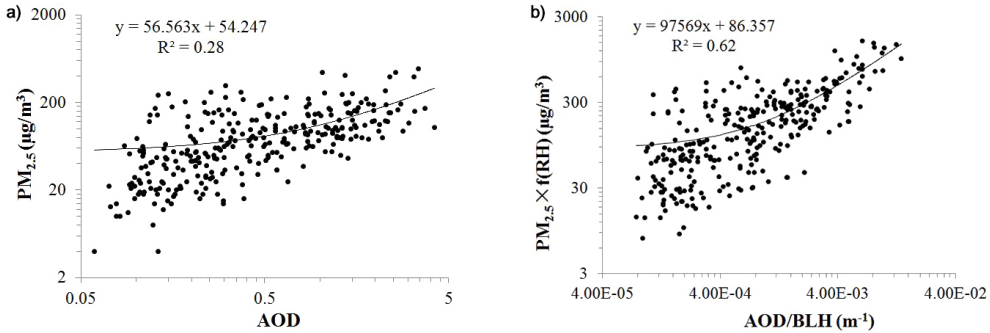

Figure 1. Relationship between daily AERONET AOD and $PM_{2.5}$ from 10 May 2010 to 6 December 2011 in Beijing, **(a)** without BLH and RH correction, **(b)** with BLH and RH correction.

ACPD
14, 28657–28684, 2014

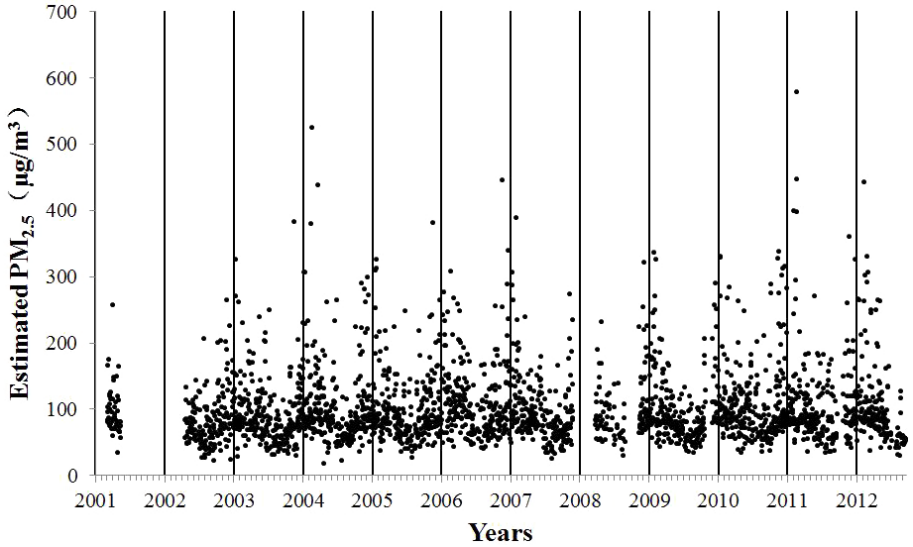

Figure 2. Estimated daily $PM_{2.5}$ from 2001 to 2012 in Beijing using AERONET AOD with BHL and RH correction.

ACPD
14, 28657–28684, 2014

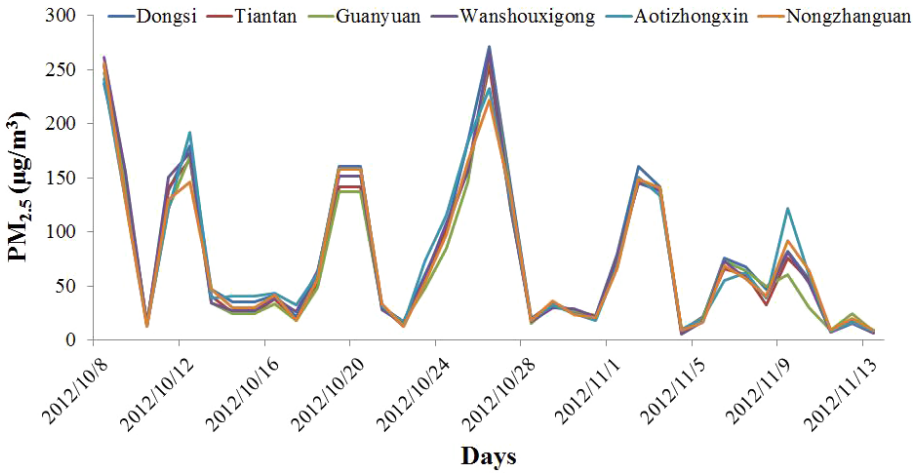

Figure 3. Daily $PM_{2.5}$ from six ground stations in the Chaoyang, Dongcheng, and Xicheng districts.

ACPD
14, 28657–28684, 2014

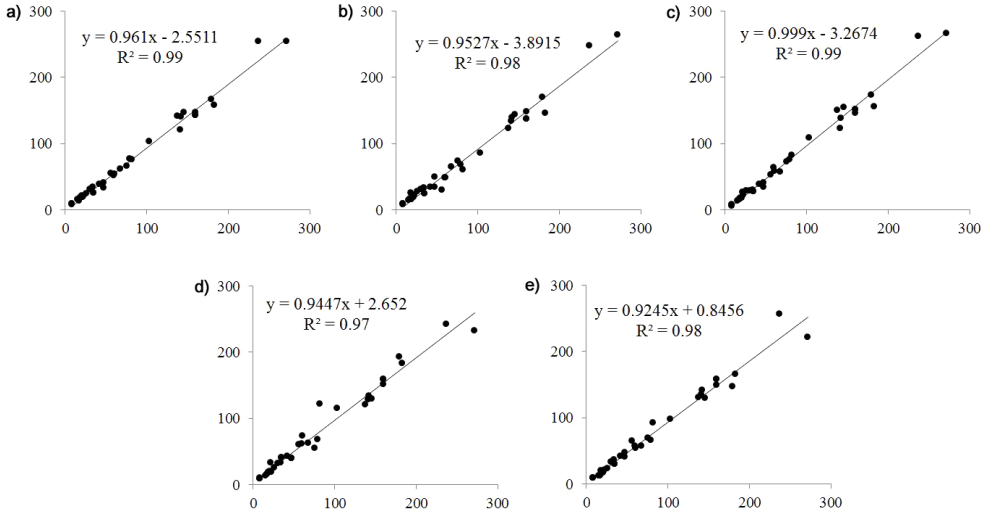

Figure 4. Correlation of $PM_{2.5}$ between Dongsi station and the other five stations. **(a)** Dongsi and Tiantan, **(b)** Dongsi and Guanyuan, **(c)** Dongsi and Wanshouxigong, **(d)** Dongsi and Aotizhongxin, **(e)** Dongsi and Nongzhanguan.

ACPD
14, 28657–28684, 2014

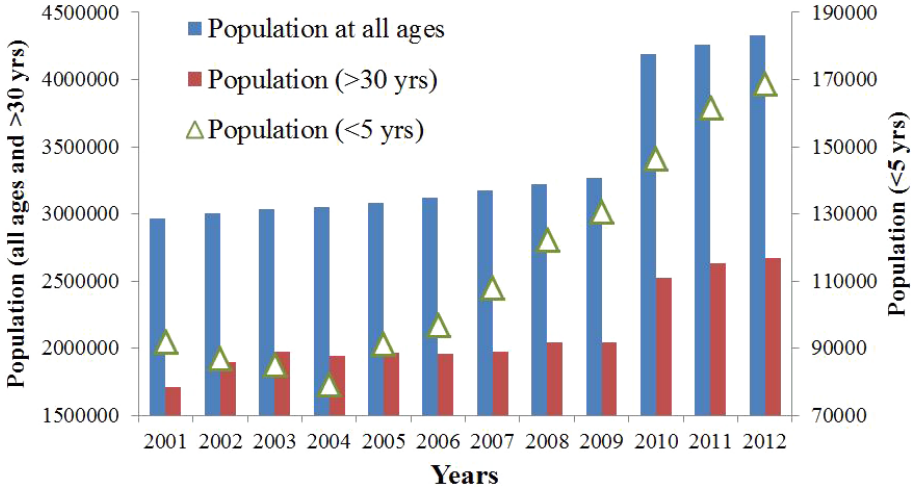

Figure 5. Yearly registered population for all ages and of > 30 yrs and < 5 yrs in the Beijing central area.

ACPD
14, 28657–28684, 2014

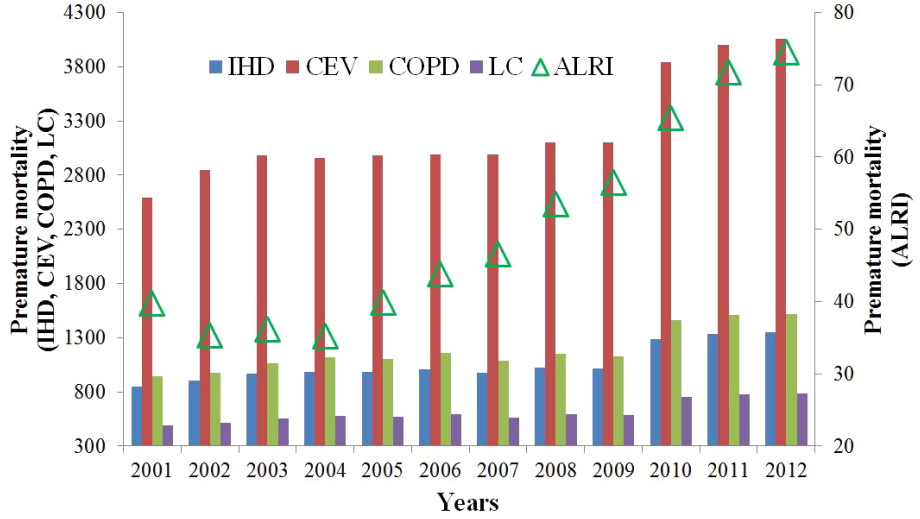

Figure 6. Yearly premature mortality attributable to IHD, CEV, COPD and LC for people > 30 yrs, and ALRI for infants < 5 yrs in the Beijing central area.

ACPD
14, 28657–28684, 2014

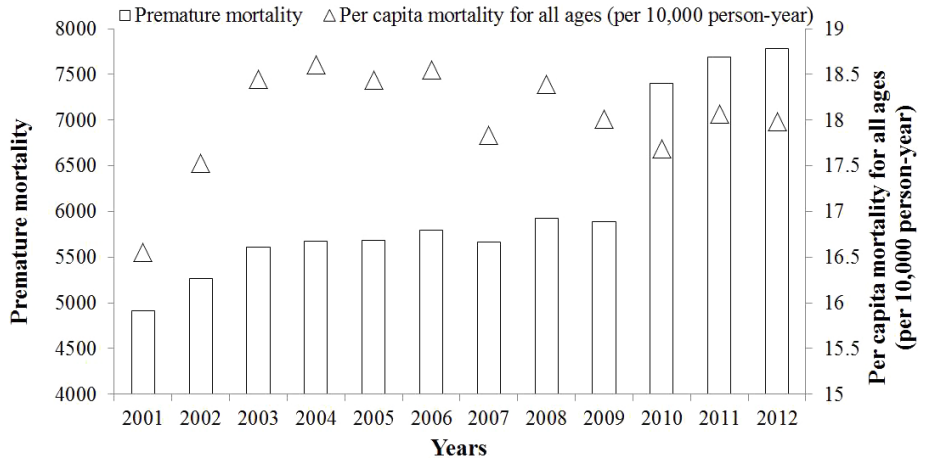

Figure 7. Annual premature mortality attributable to $PM_{2.5}$ and the corresponding per capita mortality for all ages for the period 2001–2012 in the Beijing central area.

ACPD
14, 28657–28684, 2014