Long-term (2001–2012) concentrations of 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 5100 individuals per year 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 15 per 10,000 person-years, 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 (PM) 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 (LC), 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 estimate mortality due to PM$_{2.5}$ from anthropogenic sources. Globally, air pollution has been estimated to represent a significant fraction 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 ranked fourth 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 megalopolis 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 km². 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., 2014; 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. Sun et al. (2004), based on aerosol samples from 2002 to 2003 in Beijing, showed that coal burning and traffic exhausts, plus the dust through long-range transport, could be the major sources of the aerosol pollution in Beijing. The winter heavy fog in Beijing is correlated not only with local pollution emission but also with long distance pollution transport from the surrounding areas of Beijing, such as Tianjin city and Hebei and Shandong provinces. (Ma et al., 2010; Shi and Xu, 2012). R. Zhang et al. (2013), based on 121 daily PM$_{2.5}$ samples collected in Beijing, showed that soil dust, coal combustion, biomass burning, traffic and waste incineration emission, industrial pollution, and secondary inorganic aerosol are the six main sources of PM$_{2.5}$ aerosol speciation and demonstrated that regional sources could be crucial contributors to PM pollution in Beijing. Regarding PM in Beijing, both PM$_{10}$ and PM$_{2.5}$ have been extensively studied (Hu et al., 2013; Z. Li et al., 2013; Sun et al., 2006; A. Zhang et al., 2013; P. S. Zhao et al., 2013; X. J. Zhao et al., 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 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 (X. J. 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; Z. F. 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 (RH).

Epidemiological research using time series methods has shown the relationship between PM concentrations and human health in Beijing associated with mortality and morbidity (P. Li et al., 2013; J. Y. Zhang et al., 2012; Y. J. Zhang et al., 2012). 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 them in the calculation of air quality index (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 support policy decisions aimed at reducing health impacts of PM$_{2.5}$. However, long-term continuous measurements of PM$_{2.5}$ for the period 2001–2012 in Beijing are not available, 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 Aerosol Robotic Network (AERONET) AOD and analyze its seasonal variability. A linear regression model for PM$_{2.5}$ has been established based on AOD considering boundary layer height (BLH) and 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 RH observations from the US embassy in Beijing, AERONET, and the China Meteorological Data Sharing Service System respectively. The map of Beijing with AERONET and PM$_{2.5}$ stations is shown in Fig. 1. 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 criteria: 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 data set covers a 423-day period.

The AOD observations are obtained from the 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 start on 7 March 2001 and end on 19 September 2012 and encompass the AOD at the four wavelengths 1020, 870, 675, and 500 nm. The AERONET data provide 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.nmic.cn/home.do). Beijing station lies in the centre of Beijing city.

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, windy weather due to cold air advected by 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. J. Wang et al. (2010) who showed that AOD is highest from June to August.

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):

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

Figure 2. Daily AOD at 550 nm wavelength from AERONET in Beijing from 2001 to 2012.

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

$$\beta = \frac{\tau(\lambda_1)}{\lambda_1^{-\alpha}},$$

where $\lambda$ refers to the wavelength, $\tau(\lambda)$ represents the AOD at wavelength $\lambda$, $\beta$ is the Ångström turbidity coefficient which equals the AOD at $\lambda = 1$ µm, and $\alpha$ is the Ångström exponent.

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). The daily AOD at 550 nm wavelength is shown in Fig. 2. For the entire data set 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.
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 Multi-angle 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 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 that impacts aerosol extinction coefficient. These two factors are related to atmospheric profiles, ambient conditions, as well as the size distributions and chemical compositions of aerosols, and they may have large spatial and temporal variations (Z. F. Wang et al., 2010). In order to reduce the uncertainties, 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; Z. F. 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 $\lambda$. 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 $\lambda$, 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).

$$\tau(\lambda) = \int_{0}^{\infty} k_{a}(\lambda, Z) dZ$$  \hspace{1cm}  (4)

$$k_{a}(\lambda, Z) \approx k_{a,0}(\lambda) e^{-\frac{Z}{H}}$$  \hspace{1cm}  (5)

$$\tau(\lambda) \approx k_{a,0}(\lambda) \int_{0}^{\infty} e^{-\frac{z}{H}} dz = k_{a,0}(\lambda) \times H$$  \hspace{1cm}  (6)

Based on the previous studies (Im et al., 2001; Li et al., 2005; Z. F. Wang et al., 2010), the RH correction, $f(RH)$, can be represented as Eq. (7).

$$f(RH) = (1 - RH/100)^{-g},$$  \hspace{1cm}  (7)

where $g$ is an empirical fit coefficient and 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. 3a. The correlation between the two data sets 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$_{2.5}$ × $f(RH)$) has a much higher correlation with the vertically corrected AOD (AOD/BLH), with an $R^2$ of 0.62, as shown in Fig. 3b. 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 (Engel-Cox et al., 2004; Koelemeijer et al., 2006; Z. F. 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. 3b, 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. 4a, and the average and standard deviation for estimated PM$_{2.5}$ during these 12 years is 100.39 and 55.67 µg m$^{-3}$ respectively. For the monthly data, the mean of PM$_{2.5}$ is highest in January and December, with the values of around 100 µg m$^{-3}$. The yearly averaged PM$_{2.5}$ from 2001 to 2012 is shown in Fig. 4b, and it is highest in the year 2006 with the value of 111.4 µg m$^{-3}$. Clearly an increasing trend is present between 2001 and 2012, although no clear trends are detectable after the year 2004. With regard to the uncertainties of the estimated PM$_{2.5}$, we compared the estimated PM$_{2.5}$ to the ground-based PM$_{2.5}$ from the US embassy in Beijing from 10 May 2010 to 6 December 2011. The average of the estimated PM$_{2.5}$ and ground-based PM$_{2.5}$ is 104 and 93 µg m$^{-3}$ respectively. Han et al. (2007) investigated PM$_{2.5}$ concentrations in Beijing from 2001 to 2004 and found that it was 79.6, 111.6, and 107.3 µg m$^{-3}$ respectively during summer of 2002 and spring and autumn of 2003. In this study, the estimated PM$_{2.5}$ during summer of 2002 and spring and autumn of
Figure 3. 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.

Figure 4. Estimated PM$_{2.5}$ from 2001 to 2012 in Beijing using AERONET AOD with BLH and RH correction: (a) daily PM$_{2.5}$ and (b) yearly averaged PM$_{2.5}$.

2003 was 73.7, 99.9, and 78 µg m$^{-3}$ respectively. H. L. Wang et al. (2009) found in summer and winter during 2005–2007 in Beijing that the average PM$_{2.5}$ was 102 µg m$^{-3}$. During the same period, our estimated PM$_{2.5}$ was 99 µg m$^{-3}$.

\[ \text{PM}_{2.5} = \left( 97.569 \times \frac{\text{AOD}}{\text{BLH}} + 86.357 \right) / f(\text{RH}) \] (8)

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 six ground stations in these three districts from 8 October 2012 to 13 November 2012 from the study by A. Zhang et al. (2013). Figure 5 shows the daily PM$_{2.5}$ in the Dongsi and Tiantan stations in Dongcheng district, the Guanyuan and Wanshouxi-gong stations in Xicheng district, and the Ao-tizhongxin 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. 6. 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 (IHD) 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.

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 µg 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
5.3 Mortality estimation and discussion

Long-term exposure to PM$_{2.5}$ is associated with increased mortality from ischemic heart disease, cerebrovascular disease (stroke, CEV), chronic obstructive pulmonary disease (COPD), and lung cancer, and increased incidence of acute lower respiratory infections (ALRI). Unfortunately, long-term cohort data from Beijing are not yet available. Therefore, we used the relationship between PM$_{2.5}$ and RR,

\[(y = 0.961x + 2.5511, \quad R^2 = 0.99)\]

\[(y = 0.952x + 3.8915, \quad R^2 = 0.98)\]

\[(y = 0.999x + 3.2674, \quad R^2 = 0.99)\]

\[(y = 0.047x + 2.652, \quad R^2 = 0.97)\]

\[(y = 0.9245x + 0.8456, \quad R^2 = 0.98)\]

\(\text{(Cohen et al., 2004)}\) 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 µg 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 RR.

\[\text{Figure 5. Daily PM$_{2.5}$ from six ground stations in the Chaoyang, Dongcheng, and Xicheng districts.}\]

\[\text{Figure 6. Correlation of PM$_{2.5}$ between Dongsi station and the other five stations: (a) Dongsi and Tiantan, (b) Dongsi and Guangyuan, (c) Dongsi and Wanshouxigong, (d) Dongsi and Aotizhongxin, (e) Dongsi and Nongzhanguan.}\]

\[\text{Figure 7. Yearly registered population for all ages and of > 30 years and < 5 years in the Beijing central area.}\]

which are organized in bins from global burden of disease study 2010 (http://ghdx.healthdata.org/record/global-burden-disease-study-2010-gbd-2010-ambient-air-pollution-...
risk-model-1990-2010), to calculate RR over the Beijing central area for causes of premature mortality in adults (> 30 years): IHD, CEV, COPD, and LC. In addition, the RR for ALRI was also calculated for infants (< 5 years).

We calculated the yearly average PM$_{2.5}$ concentrations at the US embassy monitor station from 2001 to 2012, and it is shown in Fig. 4b. 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)

$$AF = (RR - 1) / RR.$$  \hspace{1cm} (9)

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

$$\Delta\text{Mort} = y_0 \times Pop \times AF,$$  \hspace{1cm} (10)

where $\Delta\text{Mort}$ 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 years or < 5 years 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. 7), 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 reflects not 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. As for the uncertainties of the mortality estimation, we mainly use the lower and upper bound of RR to calculate a minimum and maximum AF and to further calculate a minimum and maximum mortality by Eq. (10).

Based on the health impact function, Eq. (10), we have calculated the yearly premature mortality by IHD, CEV, COPD, and LC for people > 30 years, and ALRI for infants < 5 years, as well as the corresponding uncertainties of the mortality estimation in the Beijing central area from 2001 to 2012, which is shown in Fig. 8. The premature mortality due to CEV (> 2225 deaths per year) is highest among the five diseases and the premature mortality by IHD (> 788 deaths per year) is the second highest. In addition, the premature mortality due to COPD and LC are also higher than the rest. The premature mortality due to ALRI is relatively low, which is consistent with the limited evidence of air pollution and ALRI in China.
mortality by ALRI exceeds 30 deaths per year. The annual premature mortality attributable to air pollution in the Beijing central area is shown in Fig. 9, as well as the uncertainties of the estimation and the corresponding per capita mortality for all ages. The annual premature mortality is more than 4102 deaths per year, and shows an increasing trend during 2001–2012, with the highest value of 6495 deaths in 2012. The average premature mortality attributable to PM$_{2.5}$ is around 5098 (2463–6621) deaths per year for the period 2001–2012. The per capita mortality for all ages is 15.0 per 10 000 person-years in 2012, higher than that of 13.8 per 10 000 person-years in 2001. We calculate that the highest per capita mortality (15.6 per 10 000 person-years) occurred in 2004. The per capita mortality for all ages due to PM$_{2.5}$ is around 15.0 (7.3–19.5) per 10 000 person-years 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 1 for the years 2010–2012.

It is found that the annual average premature mortality attributable to PM$_{2.5}$ is around 6382 (3130–8254) deaths per year for the period 2010–2012 in the Beijing central area. The per capita mortality under adults (> 30 years) in the Beijing central area in 2010–2012 attributable to PM$_{2.5}$ by CEV (13.1 per 10 000 person-years) is higher than any other disease. Furthermore, the per capita mortality (for people > 30 years and < 5 years) attributable to PM$_{2.5}$ is around 23.1 per 10 000 person-years for the period 2010–2012. The per capita mortality for all ages attributable to PM$_{2.5}$ by CEV (8.0 per 10 000 person-years) is higher than by the other diseases as shown in Fig. 8. For the period 2010–2012 the per capita mortality for all ages due to PM$_{2.5}$ is around 15.0 (7.4–19.4) per 10 000 person-years. Although the average PM$_{2.5}$ from 2010 to 2012 (105.03 µg m$^{-3}$) is a little higher than during the period 2001 to 2012 (100.39 µg m$^{-3}$), the per capita mortality for all ages due to PM$_{2.5}$ for the period 2010–2012 is the same as that for the period 2001–2012. This is mainly because the per capita mortality is also influenced by the ratio of the population with an age of > 30 years or < 5 years and the population with all ages, and the ratio is 0.65 and 0.66 for the period 2010–2012 and 2001–2012 respectively.

We have compared the estimation of the mortality due to PM$_{2.5}$ with past studies. Lelieveld et al. (2013) calculated megacity premature mortality due to air pollution and found that the per capita mortality for all ages attributable to PM$_{2.5}$ is 11.8 per 10 000 person-years in Beijing. Anenberg et al. (2010) estimated the global premature mortality attributable to PM$_{2.5}$. According to the study of Anenberg et al. (2010), the per capita mortality for all ages attributable to PM$_{2.5}$ is about 10.8 per 10 000 person-years in Beijing. Since the study area addressed here is located in the Beijing central area with relatively high PM$_{2.5}$ concentrations, the per capita mortality (15.0 per 10 000 person-years) may be somewhat higher than in the entire city. When we assume that the range found in these studies, i.e. 11–15 per 10 000 person year, is representative for Beijing with a population of 20 million, it follows that the annual premature mortality rate due to PM$_{2.5}$ is approximately 22 000–30 000 per year. In addition, the few long-term cohort studies for chronic cardiovascular and respiratory disease and lung cancer in East and South Asia show that ambient exposures are often higher than in other parts of the world. 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, showing that mortality due to long-term exposure to air pollution is a severe problem and that air pollution abatement is needed urgently.

### 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 standard deviation of the estimated PM$_{2.5}$ from 2001 to 2012 is 100.39 and 55.67 µg 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, and LC among people > 30 years and that by ALRI among infants.

### Table 1. Annual mortality attributable to air pollution by disease category and the corresponding per capita mortality (IHD, CEV, COPD, and LC for people > 30 years and ALRI for infants < 5 years) in 2010–2012 in the Beijing central area, as well as the uncertainty ranges of the total mortality estimation.

<table>
<thead>
<tr>
<th>Disease</th>
<th>IHD</th>
<th>CEV</th>
<th>COPD</th>
<th>LC</th>
<th>ALRI</th>
<th>Total</th>
<th>Uncertainty ranges of the total mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Annual mortality</td>
<td>1224</td>
<td>3426</td>
<td>1129</td>
<td>542</td>
<td>62</td>
<td>6382</td>
<td>3130</td>
</tr>
<tr>
<td>Per capita mortality (per 10 000 person-years)</td>
<td>4.7</td>
<td>13.1</td>
<td>4.3</td>
<td>2.1</td>
<td>3.9</td>
<td>23.1</td>
<td>11.3</td>
</tr>
<tr>
<td>Per capita mortality for all ages (per 10 000 person-years)</td>
<td>2.9</td>
<td>8.0</td>
<td>2.7</td>
<td>1.3</td>
<td>0.1</td>
<td>15.0</td>
<td>7.4</td>
</tr>
</tbody>
</table>
< 5 years in the Beijing central area from 2001 to 2012. The estimated total mortality in central Beijing is 6382 deaths per year (average 2010–2012), and the per capita mortality for all ages is around 15.0 (7.4–19.4) per 10 000 person-years. If we assume the range of our and previous studies to be representative of the city of Beijing, this implies a mortality attributable to PM<sub>2.5</sub> of about 22 000–30 000 persons per year. Considering the growing population of Beijing and the continued high levels of air pollution, this study corroborates the urgency of air pollution abatement strategies.

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