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Potential health benefits of controlling dust emissions in Beijing☆

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ABSTRACT

Although the adverse impact of fine particulate matter (i.e., $PM_{2.5}$) on human health has been well acknowledged, little is known of the health effects of its specific constituents. Over the past decade, the annual average dust concentrations in Beijing were approximately ~14 μg m^{-3}, a value that poses a great threat to the city's 20 million residents. In this study, we quantify the potential long-term health damages in Beijing resulting from the dust exposure that occurred from 2000 to 2011. Each year in Beijing, nearly 4000 (95% CI: 1000–7000) premature deaths may be associated with long-term dust exposure, and ~20% of these deaths are attributed to lung cancer. A decomposition analysis of the inter-annual variability of premature deaths in Beijing indicates that dust concentrations determine the year-to-year tendency, whereas population growth and lung cancer mortality rates drive the increasing tendency of premature death. We suggest that if Beijing takes effective measures towards reducing dust concentrations (e.g., controlling the resuspension of road dust and the fugitive dust from construction sites) to a level comparable to that of New York City's, the associated premature deaths will be significantly reduced. This recommendation offers "low-hanging fruit" suggestions for pollution control that would greatly benefit the public health in Beijing.

1. Introduction

Over the past two decades, extensive epidemiological studies (either time-series or cohort studies) have shown that exposure to fine particulate matter with an aerodynamic diameter of 2.5 μm or less ($PM_{2.5}$) is associated with elevated rates of morbidity and mortality (Bell et al., 2011; Buggiano et al., 2015; Ceretti et al., 2015; Gray et al., 2015; Kim et al., 2015; McEachran et al., 2015; Pope et al., 2002, 2004; Rohr and Wyzga, 2012; Schwartz et al., 2002a, 2008a; Wyzga and Rohr, 2015). However, this concentration-mortality relationship usually is based on $PM_{2.5}$ mass, which is a mixture of sulfate, nitrate, organics, elemental carbon, crustal materials and other trace elements (Cheung et al., 2011; Han et al., 2014; Kampa and Castanas, 2008). Recently, efforts have been made to evaluate variations in the health damages related to changes in a specific $PM_{2.5}$ constituent (Cao et al., 2012; Dai et al., 2014; Franklin et al., 2008; Rohr and Wyzga, 2012). Certain crustal elements (such as aluminum, silicon etc.) may increase the adverse effects of $PM_{2.5}$ on mortality (Dai et al., 2014; Franklin et al., 2008; Huang et al., 2012; Ostro et al., 2007, 2010; Zhou et al., 2011). Other toxicological studies have found plausible biological mechanisms for the inflammatory effects of road dust containing aluminum and/or silicon (Becker et al., 2005; Clarke et al., 2000; Ostro et al., 2010). All of these studies support the hypothesis that the health damages of exposure to 10 μg m^{-3} dust particles are comparable or even larger than the average effect of exposure to 10 μg m^{-3} $PM_{2.5}$, which contains many chemical constituents in addition to dust (Dai et al., 2014).

Dust particles typically contain various trace elements, including silicon, aluminum, potassium, etc. (Han et al., 2014; Kampa and Castanas, 2008). A number of these elements, especially the trace metals, are insoluble and toxic and associated with spatially heterogeneous mortality and morbidity rates (Goudie, 2014). A high proportion of dust would significantly enhance the

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total health effects of $PM_{2.5}$ (Dai et al., 2014; Franklin et al., 2008). An increasing number of studies, especially in East Asia, have shown correlations between dust events and a range of human health issues, such as cardiovascular complaints and respiratory problems (Bennion et al., 2007; Chang et al., 2006; Cheng et al., 2008; Chien et al., 2012; Goudie, 2014). Thus, in populous urban areas, high dust loading will pose a threat to both air quality and public health.

Natural dust particles primarily emanate from barren regions, such as dry lakes or deserts (Zender et al., 2003); however, dust particles produced over urban areas from traffic, construction and soil erosion can also cause substantial dust emissions. Globally, the total dust sources range from 1000 to 5000 million tons per year (Huneeus et al., 2011), with human activities (e.g., transportation, construction and industrial activities) accounting for ~30% (Ginoux et al., 2012). Because of differences in land coverage and road conditions, mineral dust concentrations vary considerably across cities (Goudie, 2014) and are particularly severe in cities in developing countries. Urban sprawl-induced construction activities and road traffic account for a significant portion of dust sources.

Beijing is the capital of China and has nearly 20 million residents, and it has suffered frequent episodes of haze over the past decade (Zhang et al., 2013). The annual mean $PM_{2.5}$ concentration has climbed to 100 $\mu g\ m^{-3}$, which is nearly 2-fold higher than what is allowed under China's new air quality standard (to be fully implemented in 2016) and 6-fold higher than the US standard for $PM_{2.5}$ (Zhou et al., 2009). Mineral components typically account for 10–20% of the $PM_{2.5}$ total mass in Beijing, although they could increase to considerably high levels during certain periods. For example, in January 2010, nearly 40 $\mu g\ m^{-3}$ or 30% of the $PM_{2.5}$ mass in Beijing was mineral dust, and 50% was attributed to resuspended road dust (Zhang et al., 2013).

The rapid industrialization and urbanization of China has generally followed a trajectory similar to that of developed nations (Seinfeld, 2004). Historically, developed countries have experienced extreme air pollution episodes, such as in Belgium in 1930 (Nemery et al., 2001), Donora (Pennsylvania, USA) in 1948 (Zender et al., 2003), and London in 1952 (Lu et al., 2015). Currently, western European countries and the United States have gained significant and visible improvements in air quality in recent decades by implementing air pollution control measures. For example, the average $PM_{2.5}$ concentrations in New York City (NYC), the most populous city in the United States, were approximately 100 $\mu g\ m^{-3}$ in the 1970s (Lippmann et al., 1977) but are less than 15 $\mu g\ m^{-3}$ today (Qin et al., 2006). Unfavorable meteorological conditions also contribute to Beijing's haze by trapping pollution released from residential, industrial and traffic activities (Bell and Davis, 2001; Bell et al., 2004). A comparison of aerosols in China and the United States will show what China can learn from the United States and achieve through science-based pollution control, thereby indicating the potential health benefits that may be obtained in China.

Controlling $PM_{2.5}$ emissions from fossil fuel combustion (i.e., removing primary aerosols, such as organic carbon (OC) and element carbon (EC), as well as $PM_{2.5}$ precursors, such as sulfur dioxide (SO_2), ammonia (NH_3) and nitrogen oxides (NO_x)) is important but costly because it requires installing expensive equipment (e.g., scrubbers to mitigate industrial SO_2 or catalytic converters or filters to control vehicle exhaust) and maintaining a complicated supervision system to guarantee scrubber activity and forestall cheating. This process usually requires a massive investment, and it may take a long time to gain sufficient regulatory experience.

In this regard, we have reviewed the concentration and composition of $PM_{2.5}$ in Beijing during the 2000s and employed the epidemiological exposure-response function to quantify the potentially avoidable premature mortalities and years of life lost, with the goal of promoting effective dust control measures.

2. Methods

2.1. Quantifying premature mortality

The association between increased mortality and elevated $PM_{2.5}$ concentrations has been shown by either cohort or time-series studies (Dai et al., 2014; Franklin et al., 2008; Rohr and Wyzga, 2012; Wyzga and Rohr, 2015). Cohort studies track the health status of a group of individuals for many years and estimates the health risk from their cumulative exposure to $PM_{2.5}$. Several cohort studies in the United States and Europe have evaluated the long-term effects of $PM_{2.5}$ exposure (Dockery et al., 1993; Hoek et al., 2002; Laden et al., 2006; Pope et al., 1995, 2002) and found a strong association between air pollution exposure and an increased risk of all-cause mortality, lung cancer and cardiopulmonary mortality.

The concentration-response (CR) function quantitatively describes the enhanced health damages caused by elevated PM exposure, with these damages primarily caused by the total $PM_{2.5}$ mass. To our knowledge, the CR relationship for specific $PM_{2.5}$ chemical constituents cannot be directly estimated. In this study, we assume that all of the chemical constituents in $PM_{2.5}$ have equal effects on human health. A similar assumption was made by Liu et al. (2009), who estimated the global premature mortalities resulting from the inter-continental transport of fine PM, and by Anenberg et al. (2012), who quantified the global health impact of black carbon aerosols. As few studies have estimated all the components in the mineral dust, the most commonly used method is by employing an indicator element, i.e. aluminum, to calculate mineral dust concentrations (Arimoto et al., 2004; Chester et al., 1989; Choi et al., 2001; Guieu et al., 2002). We adopted the method to estimate dust aerosols from the aluminum element:

$$[\text{Mineral}] = [\text{Al}]/0.07 \quad (1)$$

where 0.07 is the average aluminum content in dust reported by Zhang et al. (2003)). Numerous studies have applied this dust-aluminum ratio to estimate dust concentrations (Fan, 2013; Ho et al., 2006; Hsu et al., 2010; Zhang et al., 2013). To link the air pollution concentration to the adverse health effects of the concentration, we use the following exposure-response function:

$$\text{Death} = \text{Pop}_{30} \cdot \text{Mb} \cdot (1 - \exp(-\text{r} \cdot (\text{C} - C_0))) \quad (2)$$

where Death is the number of premature mortalities resulting from dust exposure that increases from C to C_0; Pop_{30} is the total population, which only includes adults 30 years or older and is consistent with previous assessments (Fang et al., 2013; Liu et al., 2009); Mb is the baseline mortality rate of the population; r represents the CR function, which refers to increases in the logarithm of the relative risk of premature mortality associated with a unit increase in $PM_{2.5}$ concentrations; and C_0 is the threshold below which the function is assumed invalid and a sensitive parameter for determining the health effects of pollution. However, there is little evidence for a threshold in the association between exposure to fine particles and the risk of death in both cohort (Schwartz et al., 2008b) and time-series studies (Chuang et al., 2001; Daniels et al., 2000; Koop and Tole, 2006; Schwartz et al., 2002b; Schwartz and Zanobetti, 2000). Thus, the threshold (C_0) for the health effects of $PM_{2.5}$ is assigned as zero in this study.

We apply the CR value described in Pope et al. (2002) to the