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Assessing public health burden associated with exposure to ambient black carbon in the United States

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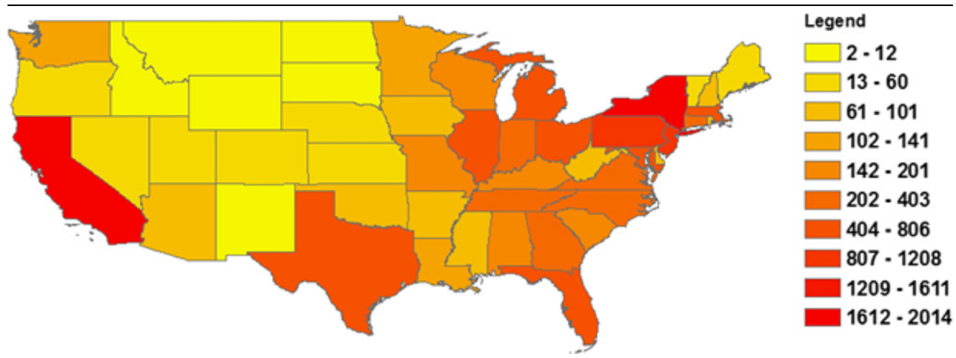
HIGHLIGHTS

- Black carbon (BC) is a strong climate warming forcing agent.
- This study quantifies the public health burden attributed to BC exposure in the US.
- We use an integrated assessment approach to estimate BC-related health impacts.
- Our findings suggest substantial health benefits to be resulted from BC control.

GRAPHICAL ABSTRACT



Annual premature mortality by State attributed to exposure to ambient black carbon in the United States in 2010.

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ABSTRACT

Black carbon (BC) is a significant component of fine particulate matter ($PM_{2.5}$) air pollution, which has been linked to a series of adverse health effects, in particular premature mortality. Recent scientific research indicates that BC also plays an important role in climate change. Therefore, controlling black carbon emissions provides an opportunity for a double dividend. This study quantifies the national burden of mortality and morbidity attributable to exposure to ambient BC in the United States (US). We use GEOS–Chem, a global 3-D model of atmospheric composition to estimate the 2010 annual average BC levels at $0.5 \times 0.667°$ resolution, and then re-grid to 12-km grid resolution across the continental US. Using $PM_{2.5}$ mortality risk coefficient drawn from the American Cancer Society cohort study, the numbers of deaths due to BC exposure were estimated for each 12-km grid, and then aggregated to the county, state and national level. Given evidence that BC particles may pose a greater risk on human health than other components of $PM_{2.5}$, we also conducted sensitivity analysis using BC-specific risk coefficients drawn from recent literature. We estimated approximately 14,000 deaths to result from the 2010 BC levels, and hundreds of thousands of illness cases, ranging from hospitalizations and emergency department visits to minor respiratory symptoms. Sensitivity analysis indicates that the total BC-related mortality could be even significantly larger than the above mortality estimate. Our findings indicate that controlling BC emissions would have substantial benefits for public health in the US.

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1. Introduction

Black carbon (BC) is a significant component of ambient fine particulate matter (PM <= 2.5 μm in aerodynamic diameter; $PM_{2.5}$) air pollution. Recent scientific evidence has indicated that BC is the most strongly light-absorbing component of $PM_{2.5}$. BC absorbs solar radiation, influences cloud processes, and alters the melting of snow and ice cover, and thus plays an important role in the Earth's climate system (Bond et al., 2013). In addition to its climate effects, BC has been associated with adverse effects on human health (e.g. Janssen et al., 2011; Laden et al., 2006). Some suggested that BC may pose greater health risk as indicated by the higher effect estimates per mass unit for BC particles compared with PM mass as a whole (Janssen et al., 2011, 2012). Therefore, mitigating climate change through controlling BC emissions is likely to generate substantial co-benefits for human health.

Black carbon is emitted from a variety of combustion processes, mainly the incomplete combustion of fossil fuels, biofuels, and biomass (EPA, 2012). The US contributes about 8% of the global emissions of BC (EPA, 2012). Within the US, BC is estimated to account for approximately 12% of all direct $PM_{2.5}$ emissions in 2005, and transport was the largest source of BC emissions in that year, which contributed to about 52% of the total BC emissions in the US, followed by open biomass burning (35%) (EPA, 2012).

Given the significance of BC both as a health effect agent and a climate forcing pollutant, the total health burden of BC would provide valuable information in developing climate and air pollution strategies. BC emissions will be substantially reduced by 2030 due to recently promulgated regulations, including the emissions standards for new engines and the retrofit programs for in-use mobile diesel engines (EPA), 2012). The present study aims to quantify the public health burden attributable to the ambient BC levels within the continental US in 2010 prior to the expected improvement in BC levels in the future. Earlier national-level particle-related health impacts assessment have focused on total, undifferentiated $PM_{2.5}$ mass. For example, using the photochemical Community Multiscale Air Quality (CMAQ) model results in conjunction with ambient monitored data, Fann et al. (2012) estimated 130,000 $PM_{2.5}$-related deaths in 2005. Based on observational data, it has been estimated that BC comprises approximately 5–10% of average urban $PM_{2.5}$ mass in the US (EPA, 2012). However, it is difficult to estimate the BC-related health outcomes based on the $PM_{2.5}$-related estimates owing to the fact that spatial variability in concentrations for BC is often larger than for $PM_{2.5}$, particularly in urban and populous areas. Also, given recent evidence that BC particles may pose a greater risk on human heath than other components of $PM_{2.5}$, it is important to assess the national health impacts of BC separately.

We use an integrated procedure that combines exposure assessment, exposure-response relationships and baseline health statistics to quantify the public health burden attributed to BC exposure. Our methods are consistent with those used previously by the U.S. Environmental Protection Agency (U.S. EPA) to assess the public health burden attributed to criteria air pollutants in the US, such as particulate matter and ozone (Fann et al., 2012; Hubbell et al., 2005), and also those used to assess the global burden of disease (Anenberg et al., 2010; Cohen et al., 2005; Ostro, 2004). To estimate ambient BC concentrations, we use GEOS–Chem, a global 3-D model of atmospheric composition with a horizontal resolution of 0.5° latitude × 0.667° longitude (approximately 55 km × 55 km) over North America. Since observational data for BC are only available at limited monitoring locations, using simulated concentrations allows us to capture the BC impacts across the entire continental US.

We quantify the excess mortality and morbidity impacts associated with anthropogenic BC emissions nationwide and also the effects at the State and county level. In addition, previous health risk assessments of $PM_{2.5}$ generally assume that all constituents of $PM_{2.5}$, including BC, are equally toxic (Levy et al., 2012). We conducted a literature review of epidemiologic studies that investigate the health risks from both BC and total $PM_{2.5}$ mass. In this analysis, we use both the concentration–response (CR) functions for $PM_{2.5}$ and several BC-specific CR functions to indicate the possible species potency of BC. Finally, we discuss the significant sources of uncertainty associated with the health impact assessment and quantify their impacts on incidence estimates.

The remainder of the paper is organized as follows. Section 2 reviews the current epidemiologic literature on the health effects of BC and describes the methods used to quantify the public health burden (mortality and morbidity) associated with BC exposure in the US. Section 3 presents our estimates of BC-related premature mortality at the nation, State and county level, as well as the national estimates of various morbidity effects. Section 3 also discusses the uncertainty in our estimates. Section 4 summarizes the major conclusions of this study and issues to be further investigated in future work.

2. Methods

2.1. Health effects of ambient black carbon pollution

The negative impacts of $PM_{2.5}$ on public health have been well documented by epidemiological studies. Time-series studies of the short-term effects of air pollutants, conducted around the world, have consistently reported significant associations between daily mortality and daily exposure to $PM_{2.5}$ (Ostro, 2004). Cohort studies usually aim at investigating possible long-term chronic effects of exposure to air pollution. Two large-scale cohort studies conducted in the U.S. – the Harvard Six Cities Study and the American Cancer Society (ACS) study – both reported increased mortality associated with an increase in annual average $PM_{2.5}$ levels (Dockery et al., 1993; Krewski et al., 2000, 2009; Laden et al., 2006; Pope III et al., 2002, 1995).

While existing epidemiological studies provide compelling evidence that $PM_{2.5}$ increases mortality rates, it is uncertain whether some components of $PM_{2.5}$ have a stronger relationship with mortality than other components. Some studies report that traffic-related fine particulate air pollution, indicated by black carbon, may pose greater risk on human health than $PM_{2.5}$ from other sources (Gan et al., 2011; Laden et al., 2006; Peng et al., 2009), while others report that some other constituents of $PM_{2.5}$, such as sulfates or organic carbon have stronger or more robust associations with adverse health effects (Ostro et al., 2010; Smith et al., 2010). However, in spite of this evidence, U.S. EPA maintains that "there is insufficient information at present to differentiate the health effects of the various constituents of $PM_{2.5}$" and "the limited scientific evidence that is currently available about the health effects of BC is generally consistent with the general $PM_{2.5}$ health literature" (EPA, 2012).

A recent systematic review on health effects of black carbon performed by the World Health Organization Regional Office for Europe found that effect estimates from both short- and long-term studies are much higher for BC compared to PM_{10} and $PM_{2.5}$ when the particulate measures are expressed per μg/m^3 (Janssen et al., 2012). This report suggests that BC might be a better indicator of harmful particulate substances from combustion sources (especially traffic) than undifferentiated $PM_{2.5}$ mass (Janssen et al., 2012). Moreover, Janssen et al. (2011) conducted a systematic review and meta-analysis of health effects of BC compared with $PM_{2.5}$ based on data from studies that measured both exposures. The study found that the pooled estimates of relative risk per 1 μg/m^3 for all-cause mortality related to long-term exposure to $PM_{2.5}$ and BC are 1.007 and 1.06, respectively, suggesting that the concentration–response coefficient β for BC could be up to ten times larger than that for $PM_{2.5}$.

Given the uncertainty with regard to the species potency of BC, we reviewed existing epidemiological studies that report the health effects of both $PM_{2.5}$ and BC. Both time-series studies of the short-term effects and cohort studies of long-term chronic effects were reviewed. As shown in Tables AI and AII in the Appendix A, our review indicates that effect estimates for BC are mostly higher than those for PM in terms of effect per μg/m^3 particulate. Among the studies we found that report the effects of both $PM_{2.5}$ and BC in the same analysis, ratios