Differences Between Magnitudes and Health Impacts of BC Emissions Across the United States Using 12 km Scale Seasonal Source Apportionment


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Supporting Information

ABSTRACT: Recent assessments have analyzed the health impacts of PM2.5 from emissions from different locations and sectors using simplified or reduced-form air quality models. Here we present an alternative approach using the adjoint of the Community Multiscale Air Quality (CMAQ) model, which provides source—receptor relationships at highly resolved sectoral, spatial, and temporal scales. While damage resulting from anthropogenic emissions of BC is strongly correlated with population and premature death, we found little correlation between damage and emission magnitude, suggesting that controls on the largest emissions may not be the most efficient means of reducing damage resulting from anthropogenic BC emissions. Rather, the best proxy for locations with damaging BC emissions is locations where premature deaths occur. Onroad diesel and nonroad vehicle emissions are the largest contributors to premature deaths attributed to exposure to BC, while onroad gasoline emissions cause the highest deaths per amount emitted. Emissions in fall and winter contribute to more premature deaths (and more per amount emitted) than emissions in spring and summer. Overall, these results show the value of the high-resolution source attribution for determining the locations, seasons, and sectors for which BC emission controls have the most effective health benefits.

INTRODUCTION

Exposure to fine particulate matter (PM2.5) in ambient air has been shown to be a cause of various adverse health effects, such as cardiopulmonary disease and lung cancer, leading to an estimated 130 000 premature deaths in 2005 in the United States alone. Recent work has focused on species-specific and source-specific analyses of PM health impacts. Many studies have associated increased premature deaths with proximity to major roadways. Other studies have found associations of

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increased premature deaths and morbidity to concentrations of black carbon (BC).15−20 Bell et al.21 analyzed source-specific effects of PM species on both cardiovascular and respiratory diseases and found that increases in concentrations of BC and vanadium resulted in the largest increase in these diseases. Reviews of the health effects of exposure to BC have suggested that there is a causal relationship between exposure to BC and premature death.22,23 There is additional interest in BC owing to potential near-term climate benefits of reductions of BC emissions.24−29

Since studies implicate exposure to ambient PM2.5 in causing a large number of premature deaths, it is further important to determine which emissions, in terms of both location and source sectors, have the largest effect on human health. Even if one assumes uniform toxicity per unit mass of PM2.5, the health effects of emissions can vary greatly between locations and sectors due to differences in spatial distributions of emissions and population. Muller et al.30 used a dispersion model to estimate air pollution damage (from both health and climate effects) associated with emissions of six gaseous or particulate pollutants across a wide range of sectors and found that the utility sector contributes to the most air pollution damage in the United States. Fann et al.31 estimated the PM2.5-related health benefits associated with emission reductions for 17 sectors across the United States and found that the largest benefits per ton of emission reductions come from reducing directly emitted PM2.5. Tainio et al.32 estimated that traffic accounts for approximately 30% of emissions of PM2.5, yet exposure to traffic emissions accounts for approximately 50% of adverse health effects associated with exposure to PM2.5 in Finland. Human health benefits per ton can also vary substantially across emissions from different locations and source sectors. Using a reduced-form air quality model to evaluate the benefits per ton estimates for the same pollutant across various sources and 9 urban areas, Fann et al.33 estimated benefits per ton estimates ranging from $65 000/ton for carbon electric generating utility (EGU) and non-EGU emissions in Salt Lake City to $2 500 000/ton for area source carbon emissions in Phoenix (2006).

While variability in emission damage across locations and sectors is clearly significant, incorporating this type of analysis into air quality control strategies is often hindered by the computational expense of quantifying damage for numerous sources. Previous works have used simplified,30,34 reduced-form, or coarser models31,33,35 and considered a limited number of sources. An alternative approach is using adjoint modeling, which provides a means to analyze detailed source−receptor relationships at a highly resolved sectoral, spatial, and temporal scale.36,37 Koo et al.38 used adjoint modeling for evaluating PM2.5 health impacts from the aviation sector in a global model, and other studies have applied this approach to evaluate PM2.5 health impacts from a range of sectors at global39 and regional scales.40 Pappin and Hakami41 used adjoint modeling for estimation of mortality due to short-term exposure to gas-phase pollutants, and this approach has been further applied for estimation of ozone health impacts.42−44 In this work, we introduce and apply a new CMAQ adjoint model to quantify the individual role of more than 10⁶ emissions (13 sectors × 97 416 locations × 4 seasons) on premature deaths attributed to exposure to BC in the United States at much lower computational cost than the more than 10⁶ simulations that would be needed to obtain the same results without this tool.

### MATERIALS AND METHODS

**Forward Model.** BC concentrations are estimated using the Community Multiscale Air Quality (CMAQ) model v4.7.1.45 The modeling domain covers the continental United States, northern Mexico, and southern Canada at a 12 km × 12 km resolution (396 × 246). The vertical domain consists of 24 terrain-following layers in sigma-pressure coordinates. Meteorological inputs were obtained from version 3.1 of the Weather Research Forecasting model (WRF).46,47 Boundary conditions and initial condition profiles were obtained by running GEOS-Chem version 8.02.03 with a grid resolution of 2.0° × 2.5° (latitude × longitude) and 47 vertical layers.48

We use anthropogenic emissions from version 2 of the 2008 National Emission Inventory (2008 NEI).49 Canada’s 2006 inventory, and Mexico’s Phase III 2008 inventory. The 2008 NEI includes five sectors: nonpoint, point, nonroad mobile, onroad mobile, and fires. Biogenic emissions are obtained from the BEIS3.14 model.50 Wildfire emissions were calculated using the SMARTFIRE2 system.51 Sectoral emission profiles were obtained by running the Sparse Matrix Operator Kernel Emissions (SMOKE) modeling system52 v3.5.1 for the following sectors: onroad mobile, nonroad mobile, fire, EGU, non-EGU, commercial marine, non-U.S. point, dust, rail, nonpoint, non-U.S. onroad, and non-U.S. nonpoint, and nonroad. The onroad mobile sector was further differentiated by fuel type based on the source classification code in SMOKE. The nonpoint sector (called “stationary area” in previous NEIs) includes sources such as residential heating, gas stations, dry cleaning, commercial cooking, etc. The non-EGU sector includes large industrial combustion sources that are not categorized as EGUs. The “non-U.S.” sectors contain emission sources that are located outside of the continental United States (i.e., Southern Canada and Northern Mexico).

We use CMAQ to estimate national premature deaths from exposure to BC using the following health impact function:

\[
J = \sum_{i=1}^{N} M_i (1 - e^{-\beta C_{av,i}})
\]

where \(M_i\) is the 2010 gridded annual nonaccidental premature deaths in the United States for people age 30 or older, \(C_{av,i}\) is the gridded annual average BC concentration, \(i\) is the grid cell index, \(N\) is the number of grid cells, and \(\beta\) is the concentration response factor. We use a concentration response factor of \(0.005827\) (response factor is calculated from the relative risk estimate presented in Krewski et al.,1 1.06 (1.04−1.08)). Mortality rates for 2004−2006 are from the Centers for Disease Control and National Center for Health Statistics.54 Our linear approximation of this health impact function yields a high bias of approximately 15%.

**Adjoint Model.** In contrast to forward model sensitivity analysis, adjoint modeling provides receptor-based sensitivities. The adjoint method has two main advantages: First, the adjoint model calculates sensitivities with respect to all model parameters simultaneously, requiring significantly less run time than forward model sensitivity analysis. Second, the computed gradient is numerically precise when using the adjoint model, whereas forward model perturbation methods are more subject to roundoff and truncation errors. While most adjoints of Eulerian chemical transport models55 have been developed and used for inverse modeling of gas-phase species,56,57 several studies have addressed aerosols.58,59 An adjoint of a fixed size aerosol model has been developed for a
global coupled chemistry–aerosol model, and a box model adjoint of aerosol dynamics has been studied. Hakami et al. applied the adjoint of the STEM-2k1 chemical transport model to constrain BC emissions during the Asian Pacific Regional Aerosol Characterization Experiment. Dubovik et al. developed the adjoint of the GOCART aerosol transport model to retrieve global aerosol source emissions from satellite observations. Huneus et al. developed a simplified global aerosol model and its adjoint to optimize aerosol and aerosol precursor emissions using variational data assimilation.

Inclusion of aerosols in the existing CMAQ-ADJ results in the first coupled gas–aerosol, regional adjoint model to explicitly describe aerosol mass composition and size distribution (see Supporting Information for adjoint development details). For this project, the receptor function of interest is national premature death attributed to BC exposure (eq 1). The adjoint model calculates the sensitivity of this function with respect to emissions at highly resolved scales. The resulting sensitivities quantify the effects of location, time, and sector of emissions of BC on human health in the United States.

Calculation of the health impact function (including checkpointing) and its gradients takes approximately 3.9 times the computational cost of the health impact function alone (without checkpointing). The updated CMAQ adjoint model has been validated against both complex variable and alone (without checkpointing). The updated CMAQ adjoint times the computational cost of the health impact function checkpointing) and its gradients takes approximately 3.9

Results and Discussion

Forward Model Simulations. CMAQ was run from December 21, 2006 to December 31, 2007 to generate gridded annual average concentrations. The annual average BC concentrations were then used in the health impact function (eq 1) to estimate the premature deaths attributed to exposure to BC in 2007. For computational expediency, CMAQ model configurations did not include gas-phase chemistry, which has a negligible effect on simulated BC concentrations.

We first evaluate simulated annual average BC concentrations via comparison to observations from the Interagency Monitoring of Protected Visual Environments (IMPROVE) and Chemical Speciation Network (CSN) monitoring networks (Figure 1). The IMPROVE and CSN monitoring networks use different protocols to measure aerosol carbon fraction; therefore, we convert the CSN measurements to IMPROVE-equivalent values using the seasonal conversion factors from Hu et al. (this conversion is not necessary for recent years due to the recent update to the CSN protocol).

While a majority of locations show similar concentrations between model and observations, some points have substantial differences. Simulated annual average BC concentrations tend to overestimate the largest concentrations (simulated annual average concentrations > 1.5 μg/m³) when compared to IMPROVE data (mean bias of 0.09 μg/m³, see Table S1, Supporting Information). However, at lower concentrations (simulated annual average concentrations < 1 μg/m³) CMAQ tends to compare well with IMPROVE, as shown by the coefficient of determination of 0.65 and the root mean squared error (RMSE) of 0.14 μg/m³ (mean absolute error of 0.13 μg/m³). Comparisons to the adjusted CSN measurements, on the other hand, tend to be worse. While CMAQ and CSN concentrations are similar for smaller concentrations, there is a large amount of scatter in the higher CSN values not captured by the model, likely owing to subgrid variability in concentrations near the urban CSN sites. However, as with the comparison to IMPROVE observations, the RMSE is small (0.45 μg/m³, mean absolute error = 0.50 μg/m³) for comparisons between CMAQ and CSN concentrations.

Further, while the coefficient of determination is small when comparing to CSN observations (R² = 0.19), the simulated concentrations have a small normalized mean bias of −17% (mean bias of −0.22 μg/m³).
By combining the forward model BC concentrations with gridded baseline mortality rates in the continental United States using eq 1, we estimate approximately 12 600 (8700−16 500, see Supporting Information for uncertainty calculation details) national premature deaths attributed to exposure to BC for 2007. This is consistent with previous studies, taking into account that about 5−10% of PM2.5 mass is BC.69 As one might expect, a majority of premature deaths attributed to exposure to BC occur around major cities (Figure S8, Supporting Information).

Sensitivity of BC Health Effects to Anthropogenic Emissions. A comparison of yearly average BC concentrations to average BC concentrations calculated from 12 weeks (Figure S7, Supporting Information) shows that results from these 12 weeks provide an accurate ($R^2 = 0.88$, RMSE = 0.07 μg/m$^3$) representation of the annual average. To reduce the computational cost of this analysis, adjoint simulations were run (in parallel) for 12 1 week periods from the first to the seventh of each month. For each 1 week adjoint simulation, we used a 4 day spin-up period. Seminormalized sensitivities (\[(\partial J)/(\partial E)\]E, Figure 2a) with respect to emissions (E) were accumulated throughout the entire week of the adjoint run, and the resulting sensitivities were summed over the 12 individual simulations and scaled to represent a yearly value.

We refer to seminormalized sensitivities as contributions, because they quantify the contribution of the BC emissions in each grid cell to the model estimate of total national premature deaths. For example, our analysis estimates that emissions in the grid cell that contains New York City result in 174 premature deaths associated with exposure to BC in the continental United States. Therefore, emissions in this grid cell have a contribution of 174 premature deaths to the national total. The contribution percentage is defined as the percent of the total continental U.S. deaths owing to BC emissions from a single grid cell. For example, the contribution percentage of the grid cell that contains New York City is 174/(12 200) × 100% = 1.4%. From our analysis, we estimate that anthropogenic emissions of BC account for 12 200 (8400−16 000) premature deaths (approximately 95% of total premature deaths from exposure to BC). It has often been assumed that the damage (contribution per unit emission) of BC emissions is constant or proportional to the magnitude of emissions across large domains.30 However, our analysis shows that damage varies greatly by region (see Figure S9, Supporting Information), with damage estimates ranging 2 orders of magnitude and being largest in urban centers.

We generated plots (Figures 2b−d) for various ratios on a log scale. When generating these ratio plots, only a fraction of the grid cells was included in order to emphasize the cells with larger values while also omitting cells that might have large ratios but small overall impacts. Of the ∼97 000 grid cells in the domain, data from only ∼10 000 grid cells were used. For each ratio plot, the largest values for the numerator and denominator (summing to approximately 95% for each) are displayed in the plot.

To analyze the spatial differences between the magnitude of emissions and the magnitude of exposure on a grid-cell by grid-cell basis, we plotted the ratio of premature death percentage to anthropogenic emission percentages (Figure 2b). Warm (cool) colors indicate locations with a larger (smaller) premature death percentage than the corresponding anthropogenic emission percentage. Highly populated urban areas have larger premature death percentages than anthropogenic emission percentages. In contrast, grid cells along many interstates in rural areas have larger anthropogenic emission percentages than premature death percentages. This is expected, as trans-
portation emissions account for a majority of anthropogenic U.S. BC emissions (~80%), and one would not expect many premature deaths attributed to BC exposure to occur near interstate highways in rural areas. However, near highways passing through urban areas (e.g., Highway 99 in Modesto and Fresno, CA) there are larger premature death percentages than anthropogenic emission percentages due to the proximity of populous areas to the highway.

Next, the ratio of contribution percentage to anthropogenic emission percentage (Figure 2c) demonstrates the variability in the difference between the magnitude of emissions and their marginal damages (the incremental change in damage resulting from an increase or decrease in emissions). Warm (cool) colors show locations with higher (lower) contribution percentages than anthropogenic emission percentages. Highly populated urban areas tend to have larger contribution percentages than anthropogenic emission percentages. However, in rural areas, such as eastern Texas and Tennessee, some major roadways have larger anthropogenic emission percentages than contribution percentages. The fact that rural roadways often have higher emission percentages than contribution percentages in many parts of the country suggests that additional restrictions on vehicle emissions would not be the most efficient means of reducing national premature deaths attributed to exposure to BC. However, Figure 2c does not provide sufficient information to weigh this against the impact of vehicle emissions near the urban areas. Further analysis of net sectoral impacts is provided in the Seasonal and Sectoral Trends section.

Given the short lifetime of BC, one might expect that contributions might be reduced most efficiently through reductions to emissions in highly populated areas. Adjoint sensitivity analysis quantitatively separates the influence of emission location and population distributions from atmospheric transport, which is shown by the ratio of gridded contribution percentage over gridded premature death percentage (Figure 2d). Warm (cool) colors show locations with higher (lower) contribution percentage than premature death percentage. Many of the major roadways have contribution percentages greater than premature death percentages. However, along some major roadways (such as Interstate 90 in New York) there are larger premature death percentages than contribution percentages. Areas downwind of major emission locations have more premature deaths than accounted for by emissions in the corresponding grid cells (e.g., Connecticut, Rhode Island, and eastern Massachusetts are downwind of emissions from northern New Jersey and show larger premature death percentages than contribution percentages). Additionally, many of the areas with a higher contribution percentage than premature death percentage are upwind of highly populated areas (northern New Jersey, west-southwest of New York City). As with the northeastern United States, the California region shows higher premature death percentages downwind of high emission areas.

We next compare premature death and contribution percentages to population and anthropogenic emission percentages across the nation to determine national-scale trends (Figures 3 and S9, Supporting Information). Locations with the highest anthropogenic emissions do not necessarily correspond to the highest contributions for BC premature deaths (see Figure 3). In fact, the second largest anthropogenic emissions considered (0.18%) has a corresponding contribution percentage of only 0.02%. These values correspond to a grid cell that encompasses Gary, IN. As such, the emissions in this grid cell are in and near locations of high population, and the large discrepancy between emissions and contributions cannot be attributed to large emissions occurring in rural, low-populated areas. Additionally, 21 of the 50 largest emissions are in the lowest 50% of contributions. As one might expect, contribution is strongly correlated with population ($R^2 = 0.79$) and premature death ($R^2 = 0.88$). Premature deaths are also highly correlated with population, with a coefficient of determination of approximately 0.85. This is expected, as population is the most spatially variable factor in our definition of the impact function (eq 1). It might be expected that anthropogenic emissions would be highly correlated with population; however, our analysis shows a coefficient of determination of only 0.41. When comparing anthropogenic emissions with contribution and premature death, we obtain $R^2$ values of 0.46 and 0.35, respectively. Regression of premature death on emission shows that 83 of the 200 largest premature deaths are in the lowest 50% of emissions. The grid-scale contributions calculated with the CMAQ adjoint identify locations where BC control strategies would have the greatest impact on national premature deaths. However, it is interesting to also consider the efficiency of strategies developed without such results. Our analysis shows that the best proxy for identifying locations where BC emissions have the highest contributions (i.e., the most effective locations to target for reducing health impacts) is areas with the largest premature deaths, rather than locations with the highest emissions or population. U.S. National Ambient Air Quality Standards (NAAQS) focus on regions with concentration exceedences. Therefore, the most effective means of reducing health impacts is being addressed only where high concentrations coincide with high premature deaths.

**Sensitivity of BC Health Effects to Anthropogenic Emissions, Summed by State.** In this section, we aggregate our grid-level sensitivity results on a per-state basis, following Hakami et al.56 While the results presented here generally emphasize the importance of transport of BC, this type of analysis, if repeated for health impact functions focusing on individual states, could be used to quantify interstate transport of BC. All grid cells were included for these plots, and we consider differences in state-level results (as opposed to the previous ratio analysis) since values in every state are of the same order of magnitude. Figure S11a, Supporting Information,
shows the contribution percentage on a per-state basis. A majority of contributions are attributed to emissions from California (with approximately 15.8%, or 2000 premature deaths), followed by Illinois, New York, and Texas (each with approximately 6.7% or 800 premature deaths).

We next consider which states are experiencing more than their share (when compared to emissions) of national premature deaths (see Figure S11b, Supporting Information). There are many states that have higher anthropogenic emission percentages than premature death percentages, such as California (by 6.9%) and New York (by 6.3%). However, while the maximum positive difference is 6.9% (California), a majority of the states with larger anthropogenic emission percentages than premature death percentages have a negative difference smaller than −1%.

In addition, we compare contribution percentages to anthropogenic emission percentages (Figure 4). Figure 4 shows that emissions originating in California account for a smaller percent of national BC emissions than the percent of national premature deaths. While there are many states with a higher anthropogenic emission percentage than contribution percentage, such as Texas and Louisiana, a majority of these states have a difference below 1%.

To determine states that have emissions that contribute to more premature deaths than there are in that state, we analyze the differences between contribution and premature death percentages for each state (Figure S11c, Supporting Information). This type of analysis allows us to estimate which states contribute disproportionately to the national health burden (red) and which would benefit the most from national-scale uniform emissions reductions (blue). A majority of states in the southeastern United States have higher premature death percentages than contribution percentages. Most states west of the Mississippi have higher contribution percentages than premature death percentages. Emissions originating in California contribute to more premature deaths than there are premature deaths in California (however, this analysis does not allow us to specify where the surplus premature deaths occur), while there are more premature deaths attributed to exposure to BC in New York than can be accounted for by BC emissions in New York. This suggests that, even for short-lived species, transport processes influence the distribution of damages from pollutant emissions at national scales. Still, despite these potentially interesting differences, overall differences across states are small, particularly compared to the order of magnitude variability in the grid-scale analysis. Therefore, there is more room for enhancing the effectiveness of BC control strategies within states or particular sectors (see next section) rather than developing state-specific caps to BC emissions.
Seasonal and Sectoral Trends. Here we perform an analysis of the seasonal and sectoral trends of emissions and the corresponding contributions (Figure 5). The sectors are sorted by contribution percentage, with the largest contributions being from onroad diesel emissions. Emissions from the fire sector have been halved for scale, while contributions from the fire sector are unaltered.

Seasonal trends in emissions do not necessarily correspond to similar seasonal trends in contributions for most sectors. For example, onroad diesel emissions are largest in the JJA months, followed by MAM, SON, and DJF. However, the contributions from these emissions are largest in the MAM season, followed closely by DJF. While onroad diesel emissions in the winter months account for the smallest percentage of annual onroad diesel emissions, their emissions in winter contribute nearly the most of any season to annual premature deaths. This is most likely attributed to the lower planetary boundary layer in the winter, which results in larger ground-level concentrations per unit emitted.

In addition to seasonal variability in damage, Figure 5 shows that the magnitude of emissions from a sector is not necessarily an accurate predictor of the magnitude of contributions from those emissions. Fire emissions exemplify this behavior. The percent of BC emitted by fires is nearly 40%, but only 3.2% of contributions come from fire emissions, which can largely be explained by the rural location of the majority of these emissions. Onroad gasoline emissions also support our finding that the magnitude of emissions is not always a good predictor of the magnitude of contributions. Onroad gasoline emissions account for approximately 2.1% of emissions yet result in approximately 5% of premature deaths attributed to exposure to BC. Non-EGU (∼3.1%) and EGU (∼2.9%) sources both have larger emissions than onroad gasoline, yet the contributions from non-EGU (∼3.3%) and EGU (∼2.5%) sources are smaller than those from onroad gasoline emissions. However, this trend does not hold for the largest contributors. Onroad diesel and nonroad mobile sectors are the two largest contributors (with 38% and 28%, respectively) as well as the two largest emitters (with 19% and 15%, respectively) excluding fire emissions. While the results presented in Figure 2c suggest that additional restrictions on vehicle emissions along some highways might not be the most efficient means of reducing national premature deaths attributed to exposure to BC, Figure 5 includes sectoral contribution information from the urban areas and suggests that the transportation sector is indeed an effective target for emissions controls.

Figure 5 also shows that the efficiency of BC emissions to result in premature deaths (right column) is consistently greater in the winter months. With the exception of fire emissions and non-U.S. emissions, every sector shows significantly larger contributions per unit emission in the SON and DJF months than the JJA and MAM months. Additionally, while there are approximately 8 times more contributions from onroad diesel emissions than onroad gasoline emissions, onroad gasoline emissions of BC result in more premature deaths per unit emission than any other sector.

While previous source attribution studies of PM$_{2.5}$ health impacts have used simplified, reduced-form, or coarser models, we used an alternative approach (using the adjoint of CMAQ) that provides source–receptor relationships at highly resolved sectoral, spatial, and temporal scales. Many previous studies that analyzed damage associated with air pollution assumed a single damage estimate across multiple locations and sources; however, our analysis allows us to identify the damage of emissions for all locations, which are highly variable. Through adjoint-based analysis, we have shown that highly populated urban areas have larger premature death percentages than BC emission percentages. Additionally, we compared damage percentages (percent of premature deaths resulting from emissions in a specific location) to premature death, population, and emission percentages. We found that while damage percentages are highly correlated with premature death and population percentages, there is little correlation between damage percentages and emission percentages, suggesting that controls on the largest emitters would not be an efficient means of reducing damage associated with exposure to BC. In addition to the gridded analysis, we performed a state-level analysis of the results. However, the overall differences in emission, damage, and premature death percentage are small at these scales, suggesting that optimal control strategies for BC need to be developed at a state and sector level. While our analysis provides source–receptor relationships at high sectoral, spatial, and temporal scales, there are some drawbacks. Our analysis utilized a single chemical transport model, as opposed to other studies in the broader field of air quality modeling that have used an ensemble of simulations to minimize uncertainty owing to any single model.**70–73** There is also uncertainty associated with the health impact function. We chose to use a concentration response factor that corresponds to the relative risk of exposure to PM$_{2.5}$ as opposed to using a concentration response factor specific to exposure to BC. Additionally, our analysis exclusively considered premature deaths attributed to pollutant exposure for the entire adult population, as opposed to focusing on certain demographics that have been shown to be at a higher risk.**74–77** While our analysis uses emission profiles from 2007, mobile emissions have been declining in the United States.78 This decrease in mobile emissions would most likely result in fewer estimated health impacts. Also, our estimate of health impacts does not include premature deaths in Canada or Mexico, yet emissions of BC in the United States can result in premature deaths in neighboring countries. Furthermore, previous studies have shown that premature death estimates from forward model studies can vary largely for different horizontal grid resolutions,**39,79,80** with premature death estimates decreasing at coarser grid resolutions. On the basis of these studies, we estimate that our analysis (performed at the 12 km resolution) has a low bias relative to higher resolution simulations (4 km) of only a few percent. However, our estimate would likely present a lower bound for this bias as neither 12 nor 4 km simulations are at a fine enough resolution to accurately resolve near-roadway gradients in BC concentrations. Finally, while this paper’s focus is on premature deaths in the United States associated with exposure to BC, this work could be extended to analyze the effects of BC emissions on premature death within a specified region, state, or socioeconomic demographic. This work can also be extended to analyze sources contributing to premature death owing to exposure of total PM$_{2.5}$, similar to previous studies that used adjoints sensitivities from coarser models.**40**

**ASSOCIATED CONTENT**

5 Supporting Information
Full documentation of the adjoint validation as well as additional figures. This material is available free of charge via the Internet at http://pubs.acs.org.

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