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Finely Resolved On-Road PM_{2.5} and Estimated Premature Mortality in Central North Carolina

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Abstract

To quantify the on-road PM_{2.5} -related premature mortality at a national scale, previous approaches to estimate concentrations at a 12-km × 12-km or larger grid cell resolution may not fully characterize concentration hotspots that occur near roadways and thus the areas of highest risk. Spatially resolved concentration estimates from on-road emissions to capture these hotspots may improve characterization of the associated risk, but are rarely used for estimating premature mortality. In this study, we compared the on-road PM_{2.5} -related premature mortality in central North Carolina with two different concentration estimation approaches-(i) using the Community Multiscale Air Quality (CMAQ) model to model concentration at a coarser resolution of a 36-km × 36-km grid resolution, and (ii) using a hybrid of a Gaussian dispersion model, CMAQ, and a space-time interpolation technique to provide annual average PM_{2.5} concentrations at a Census-block level (~105,000 Census blocks). The hybrid modeling approach estimated 24% more on-road PM_{2.5} -related premature mortality than CMAQ. The major difference is from the primary on-road PM_{2.5} where the hybrid approach estimated 2.5 times more primary on-road PM_{2.5} -related premature mortality than CMAQ due to predicted exposure hotspots near roadways that coincide with high population areas. The results show that 72% of primary on-road PM_{2.5} premature mortality occurs within 1,000 m from roadways where 50% of the total population resides, highlighting the importance to characterize near-road primary PM_{2.5} and suggesting that previous studies may have underestimated premature mortality due to PM_{2.5} from traffic-related emissions.

Keywords

Air pollution; PM_{2.5}; R-LINE; fine-resolution modeling; traffic-related mortality

1. INTRODUCTION

Traffic-related air pollutants (TRAPs) can cause adverse health effect on human health, including decreased lung function,¹ coronary heart disease,² asthma,³ 4 thrombosis,⁵ and

tuberculosis.⁶ In the United States, 19% of the population lives near heavy-traffic roads,⁷ thus understanding the burden of disease due to exposure to TRAPs is important.

Traditionally, the burden of disease due to exposure to air pollutants is estimated by combining chemical-transport air quality models (CTM) and health impact functions (HIFs). CTMs incorporate emission data and current knowledge about physical and chemical processes in the atmosphere to predict pollutant concentrations. The estimated concentrations can then be used to estimate the resultant mortality or morbidity with HIFs.⁸ Global CTMs have been used previously for estimating the mortality due to total anthropogenic emissions,⁹ climate change,¹⁰ total outdoor air pollutants,¹¹ emissions reduction,^{12, 13} and sector-specific emissions.¹⁴ In the sector-specific study, Lelieveld *et al.* estimated that traffic accounts for only 5% of premature mortality from PM_{2.5} globally, but could be up to 20% in the United States and Germany. Further, for these two countries if the differential toxicity of the PM_{2.5} chemical component is considered, traffic-related emissions can cause 36% of PM_{2.5}-associated premature mortality and are the largest contributor to burden of disease.¹⁴ Global CTMs have also been used for estimating premature mortality from surface transportation emissions by nation.¹⁵ Chambliss *et al.* have summarized that surface transportation can cause 8.5% of PM_{2.5}-related premature mortality globally but up to 23.5% and 22.8% in North America and Western Europe. One limitation of the global CTM for estimating premature mortality due to traffic-related emissions is the limited spatial resolution. The spatial resolution of the global CTM (which models concentration in 110-km × 110-km “grids”) cannot capture the effects of finer-scale variation of traffic pollution; thus the effect from traffic-related emissions could be underestimated.¹⁶

Regional CTMs, compared to global CTMs, can estimate the concentration at a finer spatial resolution. In the United States, Fann *et al.* used a detailed regional CTM and estimated that traffic sources can cause ~29,000 ozone- and PM_{2.5}-related premature deaths.¹⁷ Also in the United States, Caiazzo *et al.* used another CTM but a different health impact function and estimated 53,000 PM_{2.5}-related premature deaths.¹⁸ Consistent with the global study by Lelieveld *et al.*,¹⁴ these two studies concluded that traffic source in the United States was either the largest or second largest emission sector to cause premature mortality. While these analyses were able to predict some compelling risk estimates, the finer spatial resolutions (36-km × 36-km and 12-km × 12-km) may still not capture the important spatial distribution of concentration gradients of TRAPs. Some of the TRAPs, as shown in a previous study based on field measurement, drop by more than 50% within 150 m from roadways.¹⁹ Although regional CTMs can be run at such a spatial resolution, the associated computational burden can be too great to implement. With a 36-km × 36-km or 12-km × 12-km resolution, the ability to determine the locations of specific high-risk areas in population risk assessments can be limited.^{20, 21} In addition, quantifying the contribution from a single source would generally require running the model multiple times (with and without the emission of interest), which further increases the computational burden.

When compared to a CTM, a Gaussian plume dispersion model is relatively simple, and computationally efficient. Compared to CTMs, dispersion models do not require allocating emissions from roadways to grids that are consistent with CTMs’ modeling grids. In a dispersion model, the emission source is allocated in a much finer spatial resolution more

realistically retaining the shape and physical characteristics of the emitted plume. For example, dispersion models can model roadways as adjacent area sources^{22–24} or line sources^{25–27} and thus the sharp concentration gradients from roadways can be captured with a higher spatial resolution.^{28, 29} Modeling TRAPs with dispersion models can still be challenging due to the requirement to characterize a temporally refined emission for each roadway. This link-based emission can be obtained with a “bottom-up” approach,^{24, 30, 31} where detailed information including geometry of the road network, traffic activity, temporal allocation factor,³² emission factors of pollutants, and meteorology is required. Because of the need for these detailed input, and data, this kind of technique, to our knowledge, has only been applied in smaller geographical areas such as at city^{33, 34} or community³⁵ level or subject-specific health studies.³⁶ Limited studies have applied the bottom-up approach over a large spatial domain for a burden of disease assessment. These results, however, were not compared with the traditional CTM approach.³⁷ Further, unlike a CTM, a Gaussian dispersion model is unable to predict secondary pollutants such as secondary PM_{2.5} formed from atmospheric chemical reactions. Because the majority of ambient PM_{2.5} is secondary,^{38–41} using the Gaussian plume dispersion model may underestimate the total impact of on-road PM_{2.5}.

This work describes a new hybrid approach that combines the Research LINE source dispersion model (R-LINE,⁴² a Gaussian dispersion model), Community Mutiscale Air Quality Model⁴³ (CMAQ, a CTM), and space–time kriging technique⁴⁴ to model on-road and total annual average PM_{2.5} concentration fields at a Census-block level. Using this hybrid approach we can greatly increase the spatial resolution and permit a better characterization of PM_{2.5} concentrations due to traffic-related emissions. We hypothesize that the more finely resolved concentration field from the hybrid approach results in a higher burden of disease estimate for PM_{2.5} primarily due to on-road emissions.

2. METHODOLOGY

In this study, we used a traditional CTM approach at 36-km × 36-km grid resolution and a Gaussian dispersion model based hybrid approach at a Census-block level to estimate the burden of disease of on-road PM_{2.5} in 2010 in central North Carolina. This region, called the Piedmont region, contains 42 counties and features three major metropolitan areas with several cities with population greater than 200,000 (Fig. 1). The resultant concentration fields from both approaches were then used as input to two HIFs to estimate the burden of disease.

2.1. Health Impact Function

Health impact function relates change in mortality to changes in pollutant concentrations. The change in concentration here is defined as the PM_{2.5} contribution from on-road vehicles as simulated by CMAQ (with the CTM approach) or R-LINE (with the hybrid approach). A key component in HIF is the concentration–response function (CRF), which describes the relationship between relative risk (RR) and change in concentration based on epidemiological studies.^{45, 46} In this study, we used two commonly used CRFs to estimate

the premature mortality. This allows the comparison between these two different CRFs. The first one describes RR and change in concentration with a log-linear relationship:

$$RR = \exp^{\beta \Delta x}, \quad (1)$$

where β is the concentration–response factor (the slope of the log-linear relationship between concentration and RR) and Δx is the change in concentration. In this study, Δx was defined as the $PM_{2.5}$ from on-road vehicles.

The second CRF is the integrated risk function (IER) developed for estimating the burden of acute lower respiratory infection for children and ischemic heart disease, chronic obstructive pulmonary disease, stroke, and lung cancer (LC) for adults age greater than 25. The objective of the IER is to avoid the overestimation of mortality under a high-exposure scenario because of extrapolating results from epidemiologic studies with low ambient $PM_{2.5}$ exposure (i.e., $<50 \mu g/m^3$).⁴⁵ Additional details of this method can be found in the Supplementary Information.

The attributable fraction (AF) of the disease burden due to exposure to air pollutants is defined as:

$$AF = \frac{RR - 1}{RR}, \quad (2)$$

Multiplying the AF with baseline mortality (y_0) and population (pop) would yield the mortality attributable to exposure to $PM_{2.5}$ ($\Delta Mort$):

$$\Delta Mort = y_0 \times AF \times pop, \quad (3)$$

For the log-linear CRF, substituting RR in Equation 3 with Equation 1 would yield:

$$\Delta Mort = y_0 \times (1 - \exp^{-\beta \Delta x}) \times pop, \quad (4)$$

We back calculated β using RRs for cardiopulmonary disease ($RR = 1.128$ for $10 \mu g/m^3$ $PM_{2.5}$ increase, 95% confidence intervals 1.077–1.182) and LC ($RR = 1.142$ for $10 \mu g/m^3$ $PM_{2.5}$ increase, 95% confidence intervals 1.057–1.234) for adults age greater than 30 from Krewski *et al.*,⁴⁶ which reanalyzed the cohort data from the American Cancer Society's $PM_{2.5}$ studies.⁴⁷ The shape of the two HIFs are similar at the exposure level in this study except that IER assumes a noneffect threshold at the lower end of the concentration. We only present results using the log-linear approach here, while results using the IER approach are summarized in the Supplementary Information.

We calculated mortality counts due to cardiopulmonary diseases and LC. In this part of the analysis, we assumed that RR does not vary by population age.^{9, 10} To be consistent with these selected epidemiological studies, we used model-estimated annual average primary and secondary on-road $PM_{2.5}$ as well as total $PM_{2.5}$ concentration as inputs for both CRFs.

For IER, because primary $PM_{2.5}$ concentration can be lower than the noneffect threshold value in many locations, we used total annual $PM_{2.5}$ as input to estimate the premature mortality, and then apportioned the impact from primary and secondary on-road $PM_{2.5}$ based on their percentage contribution to the total $PM_{2.5}$ at a given Census block or modeling grid.

2.2. CMAQ Modeling

We used CMAQ 48 model version 5.0.2 to model annual $PM_{2.5}$ concentration for 2010 with 148×112 horizontal grids and 34 vertical layers covering the continental United States at a $36\text{-km} \times 36\text{-km}$ resolution. We used an updated carbon bond (CB05)49 multipollutant mechanism with explicit air toxics chemistry (CB05TUMP_AE6_AQ).50 The meteorological inputs were generated with the Weather Research and Forecasting model51 using downscaled input raw data from NASA's modern-era retrospective analysis for research and applications (MERRA).52 The initial and boundary conditions were downscaled from a global CTM: CAM-Chem.53 The 2010 emission data were generated with the Sparse Matrix Operator Kernel Emissions model54 interpolated from U.S. EPA's 2005 National Emission Inventory (NEI)55 and a projected-year emissions inventory for the year 2012. We also included the lightning NOx feature56 to generate lightning NO emissions in CMAQ. To be consistent with the emissions inputs in the hybrid approach (described further in Section 2.3.), the on-road emissions were generated using emission factor tables from the MOBILE Vehicle Emission Simulator 2010b (MOVES 2010b).57 The model evaluation against ambient observations can be found in the Supplementary Information.

To obtain the on-road $PM_{2.5}$ contribution, we generated two sets of emissions data. The first set contained the total emissions (named base case) and the second set kept all other emissions but removed the emissions from on-road vehicles (named sensitivity case). These two cases of emission were then used as input for the CMAQ model to obtain the concentrations for both base case and sensitivity case. The total contribution from on-road vehicles ($PM_{2.5\text{onroad}}$) was calculated by subtracting sensitivity case ($PM_{2.5\text{sens}}$) from the base case ($PM_{2.5\text{base}}$):

$$PM_{2.5\text{onroad}} = PM_{2.5\text{base}} - PM_{2.5\text{sens}}, \quad (5)$$

A similar approach has been used in other studies to investigate impact from sectoral emission17, 18 or single emission source.58 We also separated $PM_{2.5}$ by its primary (i.e., directly emitted) and secondary (i.e., formed through atmospheric chemical process) components to allow direct comparison with the hybrid modeling approach (see Section 2.3.). The primary and secondary on-road $PM_{2.5}$ were calculated as follows:

$$PM_{2.5\text{PRIM}} = AEC + APOC + 0.01 \times ASO_4, \quad (6)$$

where $PM_{2.5\text{PRIM}}$ is the primary $PM_{2.5}$, AEC is the primary elemental carbon, APOC is the primary organic carbon, and ASO_4 is the PM sulfate. These species are available from CMAQ output. We assign 1% of ASO_4 to be primary based upon the ratio of primary sulfate emissions and SO_2 emission. This function applies for both base case and sensitivity case.

Primary on-road $PM_{2.5}$ ($PM_{2.5\text{onroad, PRIM}}$) was obtained by subtracting primary $PM_{2.5}$ of sensitivity case $PM_{2.5\text{sens, PRIM}}$ from that of base case $PM_{2.5\text{base, PRIM}}$:

$$PM_{2.5\text{onroad, PRIM}} = PM_{2.5\text{base, PRIM}} - PM_{2.5\text{sens, PRIM}}, \quad (7)$$

The secondary on-road $PM_{2.5}$ ($PM_{2.5\text{onroad, SEC}}$) was then obtained by subtracting $PM_{2.5\text{onroad, PRIM}}$ from $PM_{2.5\text{onroad}}$:

2.3. Hybrid Modeling

We refined a previously developed novel hybrid approach that now combines dispersion modeling (R-LINE), CMAQ, and space–time ordinary kriging (STOK) technique to model primary and secondary on-road $PM_{2.5}$ as well as total $PM_{2.5}$ at Census block centroids (~105,000 Census blocks). The detail of the R-LINE and STOK hybrid framework is described elsewhere.^{28, 29} We further improved the approach to capture secondary on-road $PM_{2.5}$ by utilizing CMAQ predictions (see Section 2.3.2.).

2.3.1 Primary On-Road $PM_{2.5}$ —Primary on-road $PM_{2.5}$ was modeled with R-LINE. R-LINE treats roadways as line sources and parameterizes the horizontal and vertical spread of the plume with an updated mathematical formula.^{42, 59} Link-based emission for the entire central North Carolina was developed with a bottom-up approach that required transportation data, vehicular emission factors, and meteorological data. The transportation data were gathered from multiple sources, including the Federal Highway Administration's (FHWA) Freight Analysis Framework Version 3 (FAF3),⁶⁰ Highway Statistics,⁶¹ and U.S. EPA's NEI, producing information on: road networks (individual road segment locations), traffic activity (number of vehicles for each road segment over a period of time and the fleet distribution), and vehicle speed. The meteorological data were obtained from 14 National Weather Service stations covering the study domain (Table SI1). The emission factors were obtained from MOVES 2010b that are consistent with Section 2.2.. This would allow direct comparison between CMAQ predictions and the hybrid approach.

To reduce the computational burden for estimating annual concentration at a Census-block level, we used the METeorologically Averaging for Risk and Exposure (METARE)²⁹ approach. With the METARE method, selected meteorological hours are used to represent those with similar dispersion-related parameters, including wind direction, wind speed, and Monin–Obukhov length. The resultant simulated concentrations were then weighted and summed to yield the annual average concentration. A detailed description of METARE and the approach for developing the emissions inputs can be found in an earlier study.²⁹

2.3.2. Secondary On-Road $PM_{2.5}$ —To improve the model prediction for secondary on-road $PM_{2.5}$ at a Census-block level, we combined CMAQ outputs, observational data from U.S. EPA's Air Quality System (AQS),⁶² and STOK to capture secondary on-road $PM_{2.5}$. STOK is a Bayesian maximum entropy (BME)-based geospatial technique that uses observational data to estimate the concentration at locations without monitors. This technique assumes that the concentration value at each estimation point is a linear

combination of nearby observed data. The method is similar to Arunachalam *et al.*,⁶³ which estimated urban background concentrations using both *hard data* (i.e., exact measurements) and *soft data* (i.e., data that is described by a statistical distribution). In our approach, the estimation was based solely on soft data because the exact measurement for secondary on-road PM_{2.5} is not available. We used two methods to generate soft data. The first kind of soft data on secondary on-road PM_{2.5} was obtained by multiplying the observed total PM_{2.5} by a random variable, $R_{\text{secondary_on-road/Total}}$, representing the ratio of secondary on-road PM_{2.5} to total PM_{2.5}. This random variable is assumed to be normally distributed with a mean μR and variance $\sigma^2 R$ obtained from the CMAQ-predicted secondary on-road PM_{2.5} and total PM_{2.5} (Section 2.2.). The ratio of secondary on-road to total PM_{2.5} was calculated for each hour of the study period and μR and $\sigma^2 R$ were obtained for each monitoring site as the mean and variance of the ratios by hour of day, weekday or weekend, and season. The μR and $\sigma^2 R$ were calculated for such periods because traffic pattern is strongly correlated with time,⁶⁴ and using the mean and variance for the entire modeling period would yield greater $\sigma^2 R$ and result in concentration estimates with larger uncertainty. The observed total PM_{2.5} was retrieved from 106 AQS monitoring sites in North Carolina and surrounding states, including Alabama, Florida, Georgia, Kentucky, South Carolina, Tennessee, Virginia, and West Virginia. Hourly secondary on-road PM_{2.5} was estimated at Census blocks in this region.

The second set of soft data was used to capture the potential hotspots where AQS monitors are not available. Here we assume that the CMAQ-predicted secondary on-road PM_{2.5} is a random variable that can be described by a normal distribution with mean and variance estimated from the observational data.^{65, 66} To obtain the mean and variance, we relied on the observation-prediction pairs where CMAQ predicted secondary on-road PM_{2.5} is paired with the available AQS observational data in space and time. Because direct measurement of secondary on-road PM_{2.5} is not available, we assume that the multiplication of observed total PM_{2.5} and $R_{\text{secondary_on-road/Total}}$ is the observed secondary on-road PM_{2.5}. These observation–prediction pairs were then stratified into 68 bins based on the modeled concentration, each containing approximately 1.5% of the data points. For each bin, the mean and variance of the observed secondary on-road PM_{2.5} were calculated to represent the corresponding mean and variance of the random variable. Then, the mean and variance corresponding to each given CMAQ prediction is simply the interpolation between the 68 mean values obtained in each bin. These means and variances are then merged with the first set of soft data as input to STOK to estimate secondary on-road PM_{2.5}. Additional description of the STOK methodology implemented here and corresponding evaluation can be found in the Supplementary Information.

2.3.3. Total PM_{2.5}—Since IER requires total PM_{2.5} to estimate the burden of the diseases (Section 2.1.) and since Sections 2.3.1 and 2.3.2. only estimate primary and secondary on-road concentrations, we also need to capture the contribution from other sources, referred to as “urban background.” Here, we followed the method developed by Arunachalam *et al.*⁶³ and further used in Chang *et al.*²⁹ and use STOK with monitoring data from AQS sites as hard data to estimate the urban background. We assume that the urban background

measurements capture regional background concentration so the total concentration is the sum of urban background and primary on-road PM_{2.5}.²⁹

3. RESULTS AND DISCUSSION

3.1. CMAQ and Hybrid Modeled Concentration

Boxplots for total and primary and secondary components of predicted on-road PM_{2.5} are shown in Fig. 2. For total PM_{2.5}, both CMAQ and hybrid approaches estimated similar concentration levels with a median of 12.5 $\mu\text{g}/\text{m}^3$. For primary on-road PM_{2.5}, the hybrid approach yielded a higher estimate (median: 0.53 $\mu\text{g}/\text{m}^3$) than CMAQ (median: 0.32 $\mu\text{g}/\text{m}^3$). Also, the range of concentrations is wider in the hybrid approach (90% range: 0.07–2.94 $\mu\text{g}/\text{m}^3$) than CMAQ (90% range: 0.19–0.6 $\mu\text{g}/\text{m}^3$). For secondary on-road PM_{2.5}, the hybrid approach yielded a lower estimate (median: 0.98 $\mu\text{g}/\text{m}^3$) than the CMAQ approach (median: 1.13 $\mu\text{g}/\text{m}^3$) and a smaller variation (90% range for hybrid: 0.87–1.04 $\mu\text{g}/\text{m}^3$, 90% range for CMAQ: 0.70–1.36 $\mu\text{g}/\text{m}^3$).

The comparable total PM_{2.5} estimates from the two approaches indicate a good agreement for estimating regional PM_{2.5} concentration. comparable total PM_{2.5} estimates from the two approaches 3b), however, indicate that the hybrid approach is able to capture concentration hotspots near roadways, especially along interstate highways. These detailed features cannot be captured by CMAQ (Fig. 3a) because after it is emitted, the primary on-road PM_{2.5} is immediately diluted to the modeling grid cell resolution of 36 km $\times$ 36 km. Although the primary on-road PM_{2.5} concentration estimated by CMAQ still follows the location of interstate highways (Fig. 3c), the concentration hotspot near roadways cannot be captured by CMAQ when compared to the hybrid approach (Fig. 3d). The majority of on-road PM_{2.5} predicted by CMAQ is secondary (~65%), with high concentrations spanning across major cities and the domain (Fig. 3e). The secondary on-road PM_{2.5} estimated by hybrid approach is relatively lower than the CMAQ prediction because the kriging technique adjusts for the overprediction under a high-concentration scenario. Nevertheless, hybrid-estimated secondary on-road PM_{2.5} is still higher than hybrid-estimated primary on-road PM_{2.5} except for locations near roadways.

3.2. Health Impact Estimates

We estimated the on-road air-pollution-related premature mortality during the year 2010 for the population in 42 counties in the Piedmont region of central North Carolina shown in Fig. 1. The hybrid approach estimated 24% more on-road-related premature mortalities (295 vs. 237) than CMAQ (Table I). The major difference is from the primary on-road PM_{2.5}. With the hybrid approach, primary on-road PM_{2.5} was estimated to cause 135 (with log-linear CRF) mortality, which is 2.3 times higher than CMAQ-predicted mortality (60 with log-linear). For the secondary on-road PM_{2.5}, the hybrid approach estimated approximately 9.5% less mortality than CMAQ (160 vs. 177 with log-linear CRF). The slightly lower mortality estimate associated with secondary on-road PM_{2.5} is because the STOK component in the hybrid approach adjusted the overprediction from CMAQ under low-concentration ranges and the underprediction under high-concentration ranges. For example, the high-concentration region at the southwest domain (Fig. 3e) was adjusted to a lower

level (Fig. 3f) and the low-concentration region at the north domain was adjusted to a higher level. Results from IER approach are summarized in the Supplementary Information.

Regarding the contribution from primary or secondary on-road $PM_{2.5}$ to mortality, CMAQ predicts smaller contribution from primary on-road $PM_{2.5}$ (~25%) than the hybrid approach (~45%). This suggests that primary emitted $PM_{2.5}$ plays an important role regarding on-road-related premature mortality.

3.3. Mortality Estimate by Region Age and Disease

The total population in the central North Carolina region is approximately 4.5 million, with higher population density at urban areas, including Charlotte, Winston-Salem, Greensboro, and Raleigh, and also along the interstate highways (Fig. 4a). As a result, the spatial distribution of premature mortality would follow this pattern. For CMAQ (Fig. 4b), the estimated mortality associated with primary on-road $PM_{2.5}$ is concentrated at the three cities mentioned above but not obvious along the interstate highways. With the hybrid approach (Fig. 4c), the estimated premature mortality is also concentrated in the four cities and along the interstate highways. This is because the high concentration adjacent to roadways can be captured by the hybrid approach. For secondary on-road $PM_{2.5}$, both CMAQ (Fig. 4d) and the hybrid approach (Fig. 4e) yielded premature mortality estimates with a similar spatial pattern because the concentration fields from the two approaches are similar. Because secondary $PM_{2.5}$ is more likely to be regionally homogenous (i.e., with less spatial variation), the estimated mortality would be distributed similarly as the population patterns.

To understand the spatial pattern of population, primary on-road $PM_{2.5}$ concentration, and its associated mortality, we further organize these parameters as a function of distance from the roadways for the CMAQ and hybrid modeling (Figs. 5a and 5b). The population (blue lines in Figs. 5a and 5b) in this region decreases as the distance from roadways increases. Compared to the maximum (i.e., locations at approximately 500 m from roadways), the population reduces to 20% at 2,000 m from roadways. With CMAQ, because the concentration (green line in Fig. 5a) only reduced by 20% at 5,000 m from roadways, the population was exposed to a similar level of primary on-road $PM_{2.5}$ and thus the estimated mortality's pattern (red line) overlaps with the population. The accumulated mortality as a function of distance from roadways (Fig. 5c) indicates that 50% of the population within this region lives within 1,000 m from the roadways and also 50% of the primary on-road $PM_{2.5}$ -related premature mortality is seen within the same distance. With the hybrid approach, the concentration reduced by 80% within 500 m from roadways (green line in Fig. 5b) and thus the mortality reduces by 80% within 1,000 m from roadways (red line in Fig. 5b). Further, the concentration hotspot near roadways also coincides with high-population areas, resulting in 72% of the primary on-road $PM_{2.5}$ -related premature mortality occurring within 1,000 m from roadways (Fig. 5d).

The counties with the most on-road $PM_{2.5}$ -related premature mortality are those containing major cities with population greater than 200,000. The top five counties with the most premature mortality estimated using the log-linear CRF and hybrid approach are Mecklenburg (Charlotte), Wake (Raleigh), Guilford (Greensboro), Forsyth (Winston Salem), and Gaston (Charlotte) (Table II). In these counties, the percentage of cardiopulmonary

disease and LC deaths attributable to on-road PM_{2.5} ranges between 1.9% for Forsyth County and 3.8% for Mecklenburg County using the hybrid approach with the log-linear CRF. These five counties when combined comprise 50% of on-road PM_{2.5}-related premature mortality in the central North Carolina. This ranking is consistent in both CMAQ and the hybrid approach. For Davidson, Randolph, and Union counties, CMAQ estimated more premature mortality than the hybrid approach because the overestimation for secondary on-road PM_{2.5} was adjusted. Ninety percent of mortality is seen in population aged above 55 for both cardiopulmonary diseases and LC (Table III).

4. CONCLUSIONS AND LIMITATIONS

We compared the burden of disease associated with on-road PM_{2.5} in central North Carolina using a hybrid modeling approach that characterizes concentration at the Census-block level and a traditional CTM (CMAQ) approach that estimates concentration at 36-km × 36-km grid resolution. The results show that the hybrid approach predicts 24% more premature mortality than the CTM approach. Compared to Fann *et al.*, 17 in which the Comprehensive Air Quality Model with Extensions, CAMx—another CMAQ-like CTM—was used with the same log-linear CRF as in this study but with all-cause mortality, the on-road PM_{2.5}-related mortality for the entire North Carolina was estimated to be 263. Although our study only focuses on central North Carolina and hence direct comparison to the prediction by Fann *et al.* is not feasible, the hybrid approach with the same CRF still estimated more premature mortality (295). Although comparison to an observed on-road PM_{2.5}-related premature mortality is not viable, our results suggest the potential for previous studies to have underpredicted premature mortality from on-road PM_{2.5}.

The major difference between the CTM approach and the hybrid approach is with the primary on-road PM_{2.5}. The primary on-road PM_{2.5} from the hybrid approach was estimated to cause 2.3 times more mortality than the CTM approach using log-linear CRF. We have demonstrated that the cause is the overlapping of high concentration and high population area near roadways, resulting in 72% of primary on-road PM_{2.5}-related mortality within 1,000 m from roadways where 50% of the total population in the study domain resides. With CMAQ, the on-road PM_{2.5}-related mortality attributable to primary PM_{2.5} is 25%, whereas it is 45% with the hybrid approach. This highlights the importance to capture the sharp concentration gradient of primary PM_{2.5} adjacent to roadways.

There are several potential limitations in this study. First, there is uncertainty in the air quality model's ability to characterize both primary and secondary PM_{2.5}. For primary PM_{2.5}, the METARE approach underpredicts the annual average PM_{2.5} concentration by 13% compared to an explicit model run using one year of hourly meteorological data.²⁹ Further, the error tends to be greater than 30% for locations away from roadways. Nevertheless, the impact would be small because these areas with high error are in regions with concentrations in the range of 0.1–0.4 µg/m³, which would not cause much mortality. The secondary PM_{2.5} predicted by CMAQ represents the state-of-the-science in chemical transport modeling. There are, however, several limitations that might affect the results. For example, a new version of CMAQ (version 5.1) was released with an updated secondary organic aerosol (SOA) formation mechanism including isoprene, PAH, and long alkanes.

This could increase the estimated secondary on-road PM_{2.5} in this study. The overall conclusion, however, might not change as a previous study has used CMAQ to investigate the component of PM_{2.5} by emission sector and concluded that SOA only contribute to 6% of on-road PM_{2.5} exposure.⁶⁷ Furthermore, we adjusted the bias in CMAQ with the BME-based STOK framework in the hybrid approach. Even if an improved CMAQ can more accurately predict PM_{2.5}, this would not impact the predicted secondary on-road PM_{2.5} for the hybrid approach.

Besides the potential uncertainty in the air quality models, there are also uncertainties in the input data. For example, the Freight Analysis Framework version 3 (FAF3) data set only contains primary and secondary roads but not local roads. Neglecting local roads might result in the underestimation of premature mortality, although local roads usually have a lower volume of traffic. Also, fleet distribution plays an important role in estimating concentration level.²⁹ The fleet distribution data used in this study are from FHWA at a state level. As the fleet distribution might vary within a state, a link-based fleet distribution might help to improve the estimate.

Another potential source of uncertainty is with the assumption in health impact function that all PM_{2.5}, regardless of the composition, can cause the same impact on mortality. Nevertheless, several previous studies have shown that PM_{2.5} with different composition may adjust the impact on mortality^{68, 69} and thus using only the mass concentration to evaluate health outcome may be insufficient. Future epidemiological studies focusing on source or composition-associated mortality might help reduce the uncertainty.

In spite of these limitations, this study, to our knowledge, is the first to quantify the potential error in estimating on-road PM_{2.5}-related mortality due to resolution of air quality models in a large spatial domain. We demonstrated the possibility for prior studies to have underestimated on-road PM_{2.5}-related premature mortality, especially the primary PM_{2.5}, which is a key component in the near-road environment. The same approach from this study can be expanded to a national scale to evaluate the total impact from on-road PM_{2.5} on premature mortality to provide insights for policymakers for emissions control strategies.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.
The central North Carolina (NC Piedmont) region. The major cities (yellow stars) from left to right: Charlotte, Winston-Salem, Greensboro, and Raleigh

Figure 2.

Boxplots for total, on-road primary, and on-road secondary predicted annual concentrations of PM_{2.5} estimated with the CMAQ model and the hybrid approach. Bottom and top of box represents 25th and 75th percentiles, the line in the middle of the box is the median, the ends of the whisker are the 5th and 95th percentiles, and the dot on the whisker is the mean.

Figure 3.

Spatial map of annual average total (a and b), on-road primary (c and d), and on-road secondary (e and f) $PM_{2.5}$ concentrations for 2010 estimated with CMAQ model (left column) and the hybrid approach (right column). The color bar represents concentration level in $\mu g/m^3$.

Figure 4.

Spatial map of population (a) and premature mortality from on-road primary PM_{2.5} estimated from CMAQ model (b) and hybrid approach (c); on-road secondary PM_{2.5} estimated from CMAQ (d) and hybrid approach (e) using the log-linear CRF. The color bars represent estimates of population (a) and premature mortality (b–e).

Figure 5.

Normalized on-road primary annual averaged $PM_{2.5}$ concentrations (in green) predicted by the CMAQ model (a) and the hybrid approach (b). Also shown is mortality by Krewski *et al.* (in red) and population (in blue) by distance from the roadways. The normalized cumulative mortality and population by distance from roadways are shown for CMAQ (c) and hybrid (d). *Mortality (*K*) represents the estimation using log-linear CRF with RR from Krewski *et al.* (2009). Mortality (IER) represents the estimation using IER.

Table I.

Total Estimated Premature Mortality in Central North Carolina (Fig. 1) Due to Estimated Exposure to On-Road PM_{2.5} Estimated by CMAQ and Hybrid

	Log-Linear
CMAQ approach	
Primary	60 (34–85)
Secondary	177 (102–252)
Total	237 (136–337)
Hybrid approach	
Primary (R-LINE)	135 (78–192)
Secondary (STOK)	160 (92–228)
Total	295 (170–420)

Note: The number in the parentheses represents 95% confidence intervals of the estimate. STOK is space–time ordinary kriging and R-LINE is research line source dispersion model.

* The uncertainty bounds for the log-linear function were determined using the Monte Carlo approach that repeats the risk calculation by sampling the RR value from the reported range (Krewski et al., 2009) for 1,000 times.

Table II.

For the Top 15 Counties in Central North Carolina the Estimated On-Road PM_{2.5}-Associated Premature Mortality and its Percentage of All Disease-Specific Deaths in Parentheses Based on Log-Linear CRF (Krewski *et al.*, 2009)

County	FIPS	Log-Linear CRF	
		CMAQ	Hybrid
MECKLENBURG	37119	34 (2.6)	50 (3.8)
WAKE	37183	23 (1.9)	38 (3.2)
GUILFORD	37081	22 (2.2)	24 (2.4)
FORSYTH	37067	16 (1.6)	19 (1.9)
GASTON	37071	12 (1.9)	17 (2.7)
ALAMANCE	37001	7 (2.6)	12 (4.5)
CABARRUS	37025	9 (1.6)	11 (2)
DURHAM	37063	8 (1.8)	11 (2.5)
DAVIDSON	37057	11 (2.3)	10 (2.1)
IREDELL	37097	8 (1.7)	10 (2.1)
ROWAN	37159	9 (2)	10 (2.2)
JOHNSTON	37101	8 (2)	9 (2.3)
RANDOLPH	37151	9 (2.1)	8 (1.9)
MOORE	37125	5 (1.1)	6 (1.4)
UNION	37179	8 (1.8)	6 (1.4)

Note: PM_{2.5} exposures estimated using the CMAQ model and the hybrid approach.

* The causes of death for the log-linear CRF are cardiopulmonary disease and lung cancer (LC) for adults greater than 30 years old (Krewski et al., 2009).

Table III.

Estimated On-Road PM_{2.5}-Associated Premature Mortality by Age and Disease Using Log-Linear CRF with Relative Risk (Krewski *et al.*, 2009) in Central North Carolina

Age	Cardiopulmonary		LC	
	CMAQ	Hybrid	CMAQ	Hybrid
30 to 34	1	1	0	0
35 to 39	2	3	0	1
40 to 44	2	3	0	0
45 to 49	7	9	2	3
50 to 54	7	8	2	2
55 to 59	14	18	6	7
60 to 64	13	15	5	6
65 to 69	25	31	9	11
70 to 74	18	22	7	8
75 to 79	40	50	7	9
80 to 84	30	38	5	7
Over 85	28	36	5	6
Total	187	234	48	60

LC = lung cancer.