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The Effects of Urban Form on Ambient Air Pollution and Public Health Risk: A Case Study in Raleigh, North Carolina

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Abstract

Since motor vehicles are a major air pollution source, urban designs that decrease private automobile use could improve air quality and decrease air pollution health risks. Yet, the relationships among urban form, air quality, and health are complex and not fully understood. To explore these relationships, we model the effects of three alternative development scenarios on annual average fine particulate matter ($PM_{2.5}$) concentrations in ambient air and associated health risks from $PM_{2.5}$ exposure in North Carolina's Raleigh-Durham-Chapel Hill area. We integrate transportation demand, land-use regression, and health risk assessment models to predict air quality and health impacts for three development scenarios: current conditions, compact development, and sprawling development. Compact development slightly decreases (−0.2%) point estimates of regional annual average $PM_{2.5}$ concentrations, while sprawling development slightly increases (+1%) concentrations. However, point estimates of health impacts are in opposite directions: compact development *increases* (+39%) and sprawling development *decreases* (−33%) $PM_{2.5}$-attributable mortality. Further, compactness increases local variation in $PM_{2.5}$ concentrations and increases the severity of local air pollution hotspots. Hence, this research suggests that while compact development may improve air quality from a regional perspective, it may also increase the concentration of $PM_{2.5}$ in local hotspots and increase population exposure to $PM_{2.5}$. Health effects may be magnified if compact neighborhoods and $PM_{2.5}$ hotspots are

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spatially co-located. We conclude that compactness alone is an insufficient means of reducing the public health impacts of transportation emissions in automobile-dependent regions. Rather, additional measures are needed to decrease automobile dependence and the health risks of transportation emissions.

Keywords

Built Environment; Transportation; Public Health

1. INTRODUCTION

1.1 Background

Globally, ambient air pollution is one of the ten leading causes of premature mortality and preventable disease.[1] In urban areas, motor vehicle emissions are a leading air pollution source and an important public health risk factor.[2] However, under current practice, transportation and urban planners often do not account for air quality-related health effects when evaluating alternative transportation infrastructure investments and policies.[3] Recently, public health specialists have promoted formal health impact assessment (HIA) as a process for encouraging transportation and city planners to consider the health impacts of their decisions.[4] Yet, tools to support HIAs of transportation and urban planning projects are not readily available or easy to use. Hence, most HIAs of transportation projects have employed qualitative assessments.[5] Inadequate understanding of the complex relationships linking urban form, air quality, and public health presents a barrier to improved HIA practice for transportation projects.

The relationships among urban form, air quality, and health are complex and not fully understood. Compact and walkable urban forms have been associated with lower per-capita vehicle kilometers travelled (VKT) and thus lower emissions of transportation-related air pollutants.[6–13] While one might expect decreases in VKT and the associated air quality benefits to improve public health, recent research has raised concerns that compact urban forms may increase population exposure to poor air quality and hence elevate incidence rates of air-quality-associated illnesses due in part to elevated environmental concentrations of pollutants resulting from more compact urban forms.[14–19] Further, while urban form interventions, such as increased residential density and land use diversity, have shown promise in decreasing transportation-related air pollutant emissions, some studies have shown that innovations in the vehicle fleet, such as the adoption of hybrid vehicle technology, are more effective policy levers in improving air quality.[18, 19] Previous research conducted in our study region has demonstrated the efficacy of vehicle fleet innovations in reducing mobile-source emissions but has not considered the effects of urban form directly. [20]

To better understand the manner in which urban form, air quality, and public health interact, we employ an innovative approach for modeling the relationships between urban form and all-cause mortality associated with chronic exposure to fine particulate matter ($PM_{2.5}$) in ambient air. We examine the effects of three alternative land development scenarios—a compact development scenario, a decentralized development scenario, and an intermediate scenario representing current conditions—on annual average $PM_{2.5}$ concentrations in air and related health effects in the Raleigh-Durham-Chapel Hill area of North Carolina (NC), a region collectively referred to as "The Research Triangle." Our modeling approach links a transportation demand model that predicts automobile traffic patterns to a land use regression (LUR) model that predicts $PM_{2.5}$ concentrations in air. In turn, the LUR model links to a health risk assessment model (Figure 1). To our knowledge, this is the first study to link a transportation demand model, a LUR model for $PM_{2.5}$ concentrations, and a health risk assessment model to explore how urban development patterns might influence traffic patterns, air quality, and public health. The modeling framework we employ may be applicable to urban planners, environmental regulators, and public health practitioners in other regions considering land-use and transportation policies as means to improve health outcomes. This research is timely in providing a further exploration, using a new modeling approach, of the relationships among urban form, air quality, and health in a rapidly growing area of study.

2. METHODS

2.1 Study Region

The Research Triangle, consisting of Raleigh, Durham, Cary, Chapel Hill, and a number of smaller municipalities, is a sprawling urban agglomeration in central NC with a 2010 population of 1,589,853 spread over 3,380 square miles. (21) Over the past decade, the Research Triangle was the second-fastest-growing metropolitan region in the United States. (22) Smart Growth America has ranked Raleigh third worst in the United States on an index of urban sprawl and as a result the city earned the dubious nickname "Sprawleigh." (23) This rapid growth and sprawling development pattern typify urban areas throughout the southeastern United States. The region is highly auto-dependent, with nearly 90% of trips occurring in private automobiles. (24) 38% of the study area population lives within one-half of a mile of a freeway or major arterial. Due in part to automobile emissions, the Research Triangle suffers from intermittent poor air quality. In 2010, 117 days were recorded with a moderate Air Quality Index and five days were considered "unhealthy for sensitive groups." (25)

2.2 Transportation Demand Model

Transportation planners in the study area use a regional transportation demand model—the Triangle Regional Model (TRM)—to predict the number, length, and types of trips in the region and the modes (private cars, public transit, and non-motorized) used for each trip. These predictions are based on structural features of the built environment, including land-use patterns and transportation infrastructure, and household socioeconomic data. The TRM is a macroscopic model (in other words, it simulates the whole traffic system rather than individual vehicles) calibrated using household transportation surveys and validated using work trip distributions estimated by the 2000 Census Transportation Planning Package and the 2006–2008 American Community Survey. The TRM covers 12 counties and divides the study region into 2,678 transportation analysis zones (TAZs)—the geographic units used in transportation demand modeling and constructed by the Census Bureau based on census block information. Of the 2,678 TAZs in the study region, 99 are “point TAZs” used to represent a single employment node, resulting in 2,579 TAZs with a defined spatial extent. [21]

The TRM employs four steps typical of traditional transportation demand models: 1) trip generation, 2) trip distribution, 3) mode choice, and 4) trip assignment. The output includes estimates of VKT during different time intervals on 19,575 transportation network links in the study region. The TRM explicitly models traffic on freeways and major roadways (major and minor arterials) between TAZs; however, it does not model traffic on local streets or trips that begin and end within the same TAZ. We use the TRM to estimate VKT during the weekday morning peak traffic period (6:00 AM–10:00 AM) for the study year (2010). The TRM output consists of a line file containing all links in the transportation system and accompanying traffic counts per link. While the TRM does not provide uncertainty associated with traffic count estimation, we use a coefficient of variation derived in a recent study of traffic demand modeling output sensitivity to uncertainties in input variables to develop distributions for each TRM output variable (see Appendix 1). [26] The TRM was executed using TransCAD version 5.0 build 1880.

2.3 Development Scenarios

We model the impacts of three regional development scenarios on air quality and public health: current (base case) conditions plus two alternative scenarios, one emphasizing compact development and the other urban sprawl. Rather than projecting growth to the future, we consider two alternative presents, given a hypothetical history of different land-use and transportation policy choices. In effect, this modeling approach enables a controlled experiment to test the effects of alternative land use patterns alone on air quality and population health. In the sprawl and compact development scenarios, we alter the spatial distribution of population and housing while holding constant aggregate population, household socioeconomic characteristics, and transportation system infrastructure based on 2010 data.

Scenario 1—*Base Case* uses observed land-use, population, and employment data and modeled travel patterns in the Triangle in 2010. While the base case contains several dense urban cores, the region as a whole is relatively dispersed, with low population densities in large portions of the study area, shown in the top left corner of Figure 2. This dispersed land use pattern resulted from historic policy choices in the Triangle, including poor investment in mass transit, pedestrian, and bicycle infrastructure; limited jurisdictional cooperation in enacting land development controls; and strong incentives for economic development in suburban research campuses and industrial parks near the region’s geographic center.

Scenario 2—*Compact Development* emulates how land use in the Triangle might be distributed today if growth management and land conservation policies had been implemented in the past. Examples of such policies include density incentives, transfer of development rights, urban growth boundaries, and targeted investment to concentrate growth in urban cores instead of rural areas. To create this scenario, we first calculate the base case population density in each TAZ in the study region. We then classify TAZs into quintiles based on population density. Next, we reallocate evenly all households and employment locations in TAZs in the two lowest density quintiles across all TAZs in the highest two quintiles. The middle quintile is unchanged from the base case. Thus, households and workplaces are transferred from non-core areas to urban cores, increasing the population and employment density of core urban areas and leaving many non-core areas uninhabited. Given the low population density across much of the study region, reallocating the lowest density TAZs to existing urban cores increases the average density of already dense areas by only a small amount, even though the percentage of the population living in high-density TAZs increases substantially, as shown in the map in the top center of Figure 2 and the histogram of population density in the center of the right-most column of Figure 2. Because the density increase in urban cores is modest, this scenario is conservative and may understate potential reductions in motor vehicle travel that could occur given more aggressive policies to discourage sprawl.

Scenario 3—*Increased Sprawl* amplifies the "hollowing-out" of urban areas typical of post-1950's urban development in the United States. This scenario represents a history of policies supporting increased decentralization, including limited land development controls and rapid expansion of utility service areas. To develop this scenario, we first apply a density cap of 1,121 persons/mi^2 (1.75 persons/acre) – the 60th percentile of TAZ household density in the base case. We then relocate all households in TAZs with densities above the cap to TAZs with densities below the cap but having non-zero populations in the base case without moving employment locations to increase the spatial mismatch between housing and employment locations.

Let ρ=1,121 people/mi^2 (the density threshold). To reallocate population from TAZs with densities above ρ, we employ the following procedure. First, we divide the TAZs into three groups: (1) those with density>ρ, (2) those with density≤ρ, and (3) those with zero population. Let M_i denote the population in TAZ$_i$ from within the first group of TAZs. Let R_j represent the population that TAZ$_j$ (from within the second group of TAZs) could receive without exceeding a density of ρ. Then for each TAZ$_i$ in group 1, we remove M_i persons, and into each TAZ$_j$ in group 2, we add an additional number of persons equal to:

$$R_j \times \frac{\sum_i M_i}{\sum_j R_j} \quad (1)$$

Figure 2 maps the resulting population spatial distributions (*left*) and histograms of TAZ population densities (*right*) for the three scenarios. In the sprawl scenario, a relatively high percentage of the population resides in TAZs with relatively low population densities whereas in the compact growth scenario, a much higher percentage of the population lives in TAZs with relatively high population density. Comparing the population density histograms between scenarios (*right*), a significant portion of the base case population is shifted to higher density TAZs in the compact scenario and to lower density TAZs in the sprawl scenario. We enter new TAZ population and employment information into the TRM to estimate traffic for each scenario.

2.4 LUR Model

To quantify the effects of land-use changes on $PM_{2.5}$ concentrations, we develop and calibrate a LUR model for the study region. Previous studies have found that LUR models perform well in estimating $PM_{2.5}$ and other air pollutant concentrations at fine spatial scales, with R^2 values of 0.6–0.7.[27–32] At fine spatial scales, LUR models have significant advantages over more complex chemical transport models due to their computational simplicity.[33–35] Additionally, LUR models can represent pollutant spatial heterogeneity that may not be accounted for by geostatistical techniques (such as kriging) in cases where monitoring station locations mask spatial heterogeneity due to their intentional siting away from major local pollution sources. [36] A meta-analysis of studies assessing the spatial extent of pollutants from mobile sources found that significant spatial gradients exist in $PM_{2.5}$ concentrations above regional background levels in urban areas. [37] Thus, LUR is an appropriate and demonstrated technique to capture expected spatial variations in urban $PM_{2.5}$ concentrations above regional background concentrations.

We rely on previous LUR studies to identify explanatory variables most likely to significantly predict $PM_{2.5}$ concentrations in urban areas. While the study region contains a limited number (eight) of fixed-site air quality monitors, studies investigating the region-to-region transferability of LUR models suggest that observations from as few as ten monitoring stations are sufficient to calibrate a LUR model that has previously been successfully calibrated in an urban area with similar cultural and regulatory environments.[27, 38–40] Furthermore, research suggests that the variation between stations is more important for model calibration than the number of stations.[41] While our study region contains only one rural monitor, the outcome variable and all explanatory variables tested with the exception of industrial land use vary significantly among monitors (Table II).

2.5 Calibration Data

Figure 3 shows the locations of the eight air quality monitoring stations in the study area. Seven stations track both $PM_{2.5}$ and PM_{10} mass concentration over time, with most stations tracking daily concentrations since 1999. The eighth station tracks only PM_{10} concentrations and has records dating back to 1990. Consistent with previously demonstrated methods, annual average $PM_{2.5}$ concentrations are estimated at the latter monitoring station using the average $PM_{2.5}$:PM_{10} ratio observed at the other seven monitoring stations.[42] Four monitoring stations stopped reporting $PM_{2.5}$ concentrations before 2010, but time series data from prior years are available (Table I). For these stations, we developed station-specific linear regression models including all available time series data in order to predict 2010 $PM_{2.5}$ concentrations. Factors that influence $PM_{2.5}$ concentrations, including urban form, transportation demand, and vehicle fleet characteristics, vary over time; thus, we must impute missing 2010 data to capture changes in these factors over time. Table I shows the resulting regression models and their significance levels. Three of the four models (monitors 183-0015, 135-0007, and 063-0001) are statistically significant at the 95% confidence level. The fourth model (183-0003) is significant only at the 85% confidence level; however, we retain this station due to limited data in the study region. Additionally, cross-validation (Section 3.1) showed that inclusion of station 183-003 in the analysis did not significantly change the parameters of our LUR model, supporting the decision to include these data in our analysis.

Common explanatory variables in studies predicting $PM_{2.5}$ concentrations include traffic density and/or intensity, land use classification, population density, and elevation.[(28–32)] While these model parameters capture most significant anthropogenic sources of $PM_{2.5}$, they do not capture factors such as the fuel mix (such as the proportion of diesel and gasoline vehicles). We fit a linear LUR model to the study area by testing combination of the following explanatory variables: weekday AM peak VKT, population density, and total industrial land use as defined in the most recent available land use data. [(43–49)] The value of each explanatory variable is calculated within four circular buffers (0.5, 1, 1.5, and 2 km) around each monitoring station. Elevation differences between monitoring stations used to calibrate the LUR model are minimal, as the last column of Table II shows; therefore we do not include elevation as an explanatory variable. While variation in elevation around a station may increase vehicle emissions (due to increased emission rates when vehicles travel uphill), we did not identify previous LUR studies in the literature that found variation in elevation around air quality monitoring stations a significant predictor of annual average $PM_{2.5}$ concentrations. Table II summarizes all explanatory data within all buffer sizes used for model calibration.

2.6 Risk Assessment Model

As the first step in assessing the health risks of $PM_{2.5}$ air pollution under each development scenario, we impose a 1 km × 1 km grid containing 9,185 cells over the study area. As Figure 1 illustrates, the LUR model predicts annual average $PM_{2.5}$ concentration, denoted as C_{ik} for grid cell k within TAZ i. Then, for each TAZ i, we compute the spatial average $PM_{2.5}$ concentration, denoted as C_i, as the spatially-weighted mean value of the C_{ik}'s. Like numerous previous studies quantifying impacts of $PM_{2.5}$ on health,[(42, 50–54)] the following health impact function translates C_i into an estimate Δy_i of the number of premature deaths in 2010 in TAZ i attributable to $PM_{2.5}$:

$$\Delta y_i = y_{i0} \times AF_i = y_{i0} \times \frac{\int_{C_i=0}^{\infty} RR(C_i)P(C_i)dC_i - \int_{C_i=0}^{\infty} RR(C_i)P'(C_i)dC_i}{\int_{C_i=0}^{\infty} RR(C_i)P(C_i)dC_i} \quad (2)$$

Where:

Δy_i = Deaths per year attributable to $PM_{2.5}$ in ambient air in TAZ i

y_{i0} = Total deaths in TAZ i in the year 2010

AF_i = Fraction of deaths attributable to $PM_{2.5}$ in ambient air in TAZ i

C_i = Annual average $PM_{2.5}$ concentration in TAZ i (in μg/m^3)

$RR(C_i)$ = Relative risk of premature mortality when exposed to $PM_{2.5}$ at an annual average concentration C_i; equal to $1 + 0.06C_i/(10\ \mu g/m^3)$ *(from reference* 56)

$P(C_i)$ = Probability distribution of population exposure to $PM_{2.5}$ in TAZ i under the scenario being modeled

$P'(C_i)$ = Counterfactual exposure distribution—assumed to represent the case in which all $VKT=0$

Total deaths from all-cause mortality in 2010 are calculated for each TAZ using 2010 population and county-level death rates. [55] Because the relative risk function is assumed to be linear and the exposure concentration in each TAZ is assumed to be constant across the population, equation 2 simplifies to the following:

$$\Delta y_i = y_{i0} \times AF_i = y_{i0} \times \frac{0.06 \times \frac{(C_i - \alpha)}{10}}{1 + 0.06 \times \frac{C_i}{10}} \quad (3)$$

Where:

α = LUR model constant, assumed to represent regional background $PM_{2.5}$ concentrations

This health risk assessment approach, although the standard approach used in quantifying health impacts of $PM_{2.5}$ in ambient air, makes several important simplifying assumptions. First, the approach uses household location as a proxy for exposure. In the epidemiological studies that underlie the health impact function in equation 2, exposures at other locations, such as workplaces and schools, are averaged across large populations.[56] We assume exposures outside the household are similar in our application and thus use household exposure as a proxy for total exposure as well, which is common practice in other risk assessments of ambient air. [42, 50–54] Finally, we assume that 2010 death rates are applicable across all scenarios; the relationship between land use and transportation behavior remains stable across all scenarios; population density is uniform within TAZs; and $PM_{2.5}$ exposure concentrations are uniform within TAZs.

3. RESULTS

3.1 LUR Model

We calibrate the LUR model using the observed 2010 annual average $PM_{2.5}$ concentrations (where available) or predicted concentrations (where observed data are not available) shown in Table I. We test all unique combinations of buffer sizes and explanatory variables to maximize the adjusted R^2 of the model. Neither acres of industrial land use nor household density significantly predicted $PM_{2.5}$ concentration, regardless of buffer size; thus, they are not included in the final model. The final LUR model (adjusted R^2=0.80; root mean square error =0.61 μg/m^3; $F_{1,6}$=28.4, p=0.0018) for annual average $PM_{2.5}$ concentrations in the study area is:

$$PM_{2.5} = 8.69 + 0.0473 \times VKT \quad (4)$$

Where:

$PM_{2.5}$ = Predicted annual average $PM_{2.5}$ concentration, $\mu g/m^3$

VKT = Thousands of vehicle-kilometers (VKT) travelled during the AM peak within a 1,000 meter buffer of the center of the estimation cell, *thousands of VKT*

Both the model constant ($p<0.0001$; standard error = 0.445 μg/m^3) and the VKT parameter ($p=0.0011$; standard error = 8.89×10^{-3} μg/m^3 per thousand VKT) are statistically significant. Due to data limitations previously discussed, the model was calibrated using a small sample ($n=8$). Given the small sample size, we employ a standard leave-one-out cross validation (LOOCV) procedure for all monitoring stations to test the robustness of the model. The LOOCV shows that the model is quite robust despite limited availability of observational data (Table III), with a low root-mean-square error and high correlation between predicted and observed concentrations. Extrapolation beyond the range of observed independent variable values can be a concern for LUR models. However, in this study, VKT values outside the observed range (9.26–88.0) occurred for only 0.6%, 0.7%, and 0.3% of the study area in the base case, compact, and sprawl scenarios, respectively. The model is limited due to poor variation in industrial land-use around monitoring stations (Table II). While the model is unable to capture variation in $PM_{2.5}$ concentrations attributable to industrial or special (e.g., airport) land uses, this limitation is not necessarily relevant given the fundamental research aim of investigating how transportation behavior, air quality, and health impacts respond to changes in regional land-use patterns.

3.2 $PM_{2.5}$ Concentrations

In each grid cell, we use the TRM's traffic volume estimates for each roadway link to tabulate total VKT during 6:00–10:00 AM occurring within a 1 km buffer of the centroid of the cell and apply the LUR model to estimate annual average $PM_{2.5}$ concentrations (Figure 3). As expected based on the LUR model, the highest estimated annual average $PM_{2.5}$ concentrations are generally located near significant links in the transportation network: in the base case, the highest estimated concentration, 15.0 (9.61–41.3) μg/m^3, occurs at the intersection of two limited-access highways that provide regional mobility. While the 1 km buffer surrounding this grid cell comprises only 0.036% of the total study area, 0.62% of the predicted VKT occurs within the same buffer, illustrating how local impacts that may arise due to regional transportation patterns.

Estimated $PM_{2.5}$ concentrations in the compact development scenario are more spatially concentrated; however, the region-wide average estimated concentration is nearly identical to the base case (Table VI). Thus, while impacts are more localized, region-wide concentrations are relatively unchanged despite lower predicted transportation demand. The highest estimated concentration of $PM_{2.5}$ occurs in the same cell as in the base case; however, in the compact development scenario, the estimated concentration in this cell increases slightly to 15.2 (9.57–48.8) μg/m^3 from 15.0 μg/m^3. This result is intriguing in that potentially conflicting policy goals at the local and regional levels arise: while compact development may reduce VKT—and in turn may reduce vehicle emissions—air quality issues may become pronounced in specific locations.

Relative to the base case, the sprawl scenario reduces localized impacts; however, the region-wide average $PM_{2.5}$ concentration increases slightly (Table VI). The highest estimated concentration of $PM_{2.5}$ occurs in the same cell as in the base case and the compact development scenario; however, the maximum estimated concentration in the increased sprawl scenario decreases to 14.3 (9.30–39.4) μg/m^3 from 15.0 μg/m^3 in the base case and 15.2 μg/m^3 in the compact development scenario. Thus, while dispersing population across the study area reduces the highest predicted $PM_{2.5}$ concentrations, regional average $PM_{2.5}$ concentrations are slightly higher. While not estimated directly, vehicle emissions are an important source of $PM_{2.5}$; therefore, in conjunction with predicted increases in VKT, we can infer that the sprawl scenario increases aggregate vehicle emissions relative to the base case and compact development scenarios.

Several limitations of the LUR model should be noted. We calibrated the model using 2010 data and did not consider meteorological data; thus, the model implicitly assumes 2010 meteorological conditions and can therefore only be used to estimate $PM_{2.5}$ concentrations in 2010. However, this limitation does not affect comparisons between scenarios. Additionally, given that LUR is sensitive to the differences in values across space rather than the magnitude of values, the spatial distribution of the modeled weekday morning peak traffic patterns sufficiently represents the spatial variation in traffic throughout the year. Despite these limitations, diagnostics of the LUR model suggest that it captures a large amount of observed variation in $PM_{2.5}$ concentrations given 2010 meteorological conditions.

In all three scenarios, portions of the study area exceed both the EPA standard for annual average $PM_{2.5}$ concentrations (12 μg/m^3) and the World Health Organization (WHO) air quality guidelines for annual average $PM_{2.5}$ concentrations (10 μg/m^3). [57, 58] The fine-grained spatial scale of our air quality predictions enables comparisons across scenarios of the number of people living in areas that exceed both the EPA and WHO standards for annual average $PM_{2.5}$ concentrations (Table IV). Relative to the base case scenario, compact development increases the number of people living in areas above the EPA and WHO thresholds by 59,962 and 236,913 persons, respectively, whereas the sprawl scenario decreases these numbers by 21,346 and 279,238 persons, respectively. Similarly, the compact development scenario increases the total area exceeding the EPA and WHO standards by 9.5 mi^2 and 13.6 mi^2, respectively. In contrast, the sprawl scenario decreases the total land area exceeding the EPA standard by 2.5 mi^2 but increases the area in exceeding the WHO standard by 20.3 mi^2 (6.7 mi^2 more than in the compact development scenario). Thus, while more compact development may result in higher numbers of people living in more polluted areas, more dispersed urban forms may *decrease* the spatial extent of relatively higher $PM_{2.5}$ (i.e., greater than 12 μg/m^3) concentrations and *increase* the spatial extent of moderate $PM_{2.5}$ concentrations (i.e., 10 to 12 μg/m^3).

3.2 Attributable Mortality

Figure 4 presents estimated death rates per 100,000 persons attributable to exposure to $PM_{2.5}$ above regional background for all scenarios. For the base case, the model estimates 47 (5–167) deaths in 2010 associated with $PM_{2.5}$ exposure (Table VI). In the compact development scenario, estimated premature mortality associated with $PM_{2.5}$ exposure increases to 65 (6–220). The compact development scenario increases $PM_{2.5}$ exposure despite a marginal reduction in regional $PM_{2.5}$ concentrations (Table IV); however, the point estimate of associated mortality is within the 95% confidence interval (CI) for the base case.

Compared to the base case, population density is relatively higher in areas with higher predicted VKT in the compact development scenario despite aggregate reductions in transportation demand (Table V). In the sprawl scenario, 31 (2–122) deaths are associated with $PM_{2.5}$ exposure—less than in the base case, but once again within the bounds of the 95% CI. It is counter-intuitive that sprawling development reduces the point estimate of mortality associated with exposure to $PM_{2.5}$ considering higher predicted regional VKT and higher predicted $PM_{2.5}$ concentrations over much of the study area compared to the base case scenarios (Table VI). However, dispersing population reduces household exposure enough to counteract the modest increase in the regional $PM_{2.5}$ concentrations. Table V shows that those living in areas with high traffic (TAZs in the highest decile of VKT; last row of the table) decrease substantially in the sprawl scenario compared to the base case. In contrast, in the compact development scenario, half of the population lives in areas with very high traffic.

Across scenarios, the areas with the highest predicted death rate associated with $PM_{2.5}$ generally share two characteristics: 1) close proximity major regional transportation corridors; and 2) high population density. Thus, while clustering population near transportation infrastructure may reduce transportation demand, our study indicates that this effect may not be powerful enough to counteract the negative health effects associated with concomitant increased exposure to $PM_{2.5}$. Furthermore, Figure 4 illustrates that regional mobility needs may result in highly localized $PM_{2.5}$ hotspots and, if development is clustered along or near the intersections major regional transportation links, highly localized health impacts. While this finding is intuitive, the tendency of highly localized health impacts to persist despite significant land-use change underscores the need to address transportation issues holistically when considering health outcomes.

Several limitations should be considered in interpreting these results. Because we consider transportation infrastructure, including public transit, fixed across all scenarios, it is likely that the compact development scenario over-predicts VKT. Compact development would likely be complemented by increased provision of local and express bus services and increased walkability, resulting in shifts to public transit and non-motorized transport (cycling and walking) that may not be captured by the TRM. However, this simplification likely results in minimal error given the conservative nature of the compact development scenario: while the scenario increases population density in urban cores, the magnitude of the increase is not necessarily large enough to trigger significant investment in public transit. Further, VKT in the sprawl scenario is likely underestimated. Many rural areas have few transportation links modeled by the TRM; thus, only a small portion of the redistributed rural population may live near a transportation network link with predicted use. Further, the TRM does not predict intra-zonal trips and trips on minor roads; thus, short-distance motorized and non-motorized trips are likely systematically under-predicted. While the low population density in the sprawl scenario likely results in a low number of short non-motorized trips, it also likely leads to a larger number of short motorized trips that are not captured by the TRM, resulting in unaccounted-for VKT from intra-zonal trips that may be larger in the sprawl scenario than in the other two scenarios. This effect is magnified in TAZs with large geographic extents, which are more heavily populated in the sprawl scenario. Additionally, this research does not consider many potential health benefits of compact development, such as increased physical activity from increased walking and cycling. While there is emerging interest in characterizing how increased physical activity and increased exposure to air pollutants interact to influence health outcomes in urban areas, we do not consider these potential interactions. Finally, while the efficacy of vehicle fleet characteristics in reducing transportation emissions and improving air quality has been demonstrated, [17–19] we do not consider changes in vehicle fleet characteristics. As the adoption of improved vehicle technology in the fleet increases over time, the negative health impacts associated compact development relative to other urban development patterns will likely be reduced. Overall, while this study supports recent findings that compactness may exacerbate health impacts, [17–18] these results should be interpreted in the context of studies that find positive air quality impacts from other interventions that may require population density above certain thresholds, such as increased investment in public transportation, and positive health impacts from increased physical activity that may be associated with walkable and transit-supportive urban forms. [59]

3.3 Sensitivity Analysis

Because the variables used to predict relationships linking urban form, transportation behavior, $PM_{2.5}$ concentrations, and health are uncertain, our results also are uncertain. To consider the effects of uncertainty in key model variables on the results of this research, we perform a sensitivity analysis, focusing on four sources of uncertainty: 1) the LUR model constant (representing the regional background $PM_{2.5}$ concentration); 2) the LUR model parameter (which predicts how VKT changes affect $PM_{2.5}$ concentration); 3) estimated VKT; and 4) the relative risk of premature mortality for 10 μg/m^3 increases in $PM_{2.5}$ concentration. We repeat our analysis while varying one of these model input at a time, using the upper and lower bounds of the 95% CI for each variable: 8.24 μg/m^3–9.13 μg/m^3 for the LUR model constant; 0.0385–0.0562 μg/m^3 per thousand VKT for the LUR model parameter; the upper and lower bounds of VKT in each TAZ accounting for uncertainty and variability (see Appendix 1); and 1.02–1.11 for the relative risk per 10 μg/m^3 in $PM_{2.5}$ concentration.

Figure 5 shows that the health risk estimates are most sensitive to uncertainty in the relative risk parameter. In addition, the estimates are sensitive to assumptions about VKT in each TAZ. Nonetheless, the differences among scenarios— the fundamental research question being explored—are stable at both the low and high ends of the 95% CIs for these variables. By contrast, the LUR model constant and VKT parameter in the LUR model have comparatively small effects on the risk estimates. In sum, the sensitivity shows that (1) the relative risk function is the greatest source of uncertainty in estimating mortality attributable to $PM_{2.5}$ and (2) the differences among scenarios are generally consistent when random model input variables range across their 95% CIs, although the magnitude of these differences changes in some instances. This latter finding strengthens the conclusion that compact development may exacerbate health impacts if pursued without other strategies to reduce automobile dependency.

4. DISCUSSION

Compact development alone may not be an effective strategy for reducing the health impacts of exposure to $PM_{2.5}$ in urban areas, even though it may reduce regional VKT. If households are clustered near significant transportation corridors to achieve compact urban forms, an increasing proportion of the population may be exposed to high concentrations of $PM_{2.5}$ unless complementary incentives to reduce trip length, encourage use of public and non-motorized transit, and/or increase the adoption of lower emitting vehicle technologies are provided. Thus, other policies should be considered, alone or in conjunction with compact development, to reduce the health impacts of transportation— a conclusion consistent with studies finding that road pricing strategies may reduce exposure to pollutants in ambient air while land development controls may increase exposure and that an urban sprawl land development scenario may decrease exposure to pollutants in for individuals who move to the urban periphery but increase exposure for individuals who remain in urbanized areas, compared to a base case scenario [18, 9] Our findings also support recent literature suggesting that neighborhood-scale air quality is an important risk factor for a variety of negative health outcomes and that holistic policy approaches are critical in improving health in urban areas.[60] While not considered here, if compactness is associated with spatial homogeneity of income or races, there is potential for disproportionate impacts on specific populations, raising equity and environmental justice concerns. [61]

While our findings support decentralization as a means of reducing air quality-related public health impacts in a limited sense, this result must be strongly qualified. Given existing urban forms, the relatively slow nature of land use change, and existing large-scale transportation infrastructure that supports private automobile mobility, simply avoiding development in areas with locally high $PM_{2.5}$ concentrations due to established regional mobility patterns is not a feasible policy option. Further, decentralized development may also decrease opportunities for physical activity, increase emissions of pollutants that exacerbate global climate change, and diminish ecosystem services with indirect human health benefits via increased land consumption—all outcomes that likely have negative impacts on human health at both local and global scales.

In a broader sense, an intriguing finding is the potential for counterintuitive outcomes when considering human health. The complex relationships that link urban form, transportation behavior, air quality, and public health merit continued research to better inform policymakers with the goal of improving public health in urban areas. Considering the rising use of HIA in the United States, our findings support the integration of quantitative methods into HIA practice to untangle complex relationships and elucidate tradeoffs that occur in real-world decision-making environments. A second thought-provoking finding is the critical role that large transportation infrastructure investments may play in influencing the health impacts of transportation in urban areas. Despite significant land-use change across scenarios, we find the location of and degree to which $PM_{2.5}$ hotspots are above regional average concentrations remarkably consistent. The consistency of $PM_{2.5}$ hotspots despite significant changes in regional land use is likely attributable to the hierarchal nature of transportation systems, which tend to have limited redundancy and funnel large proportions of regional traffic through specific network links. Thus, existing transportation infrastructure may play a significant role in characterizing the relationships linking land use, transportation behavior, air quality, and public health—and ultimately, constrain the ability of certain policy instruments to positively affect public health outcomes. The potential of transportation investments to influence how land-use, transportation behavior, and air quality interact to health outcomes supports the integration of quantitative HIA into long-range scenario-planning efforts to test the sensitivity of health outcomes to future land use visions in the context of future transportation infrastructure investments.

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Appendix 1. Characterization of Uncertainty and Variability in VKT estimates

To consider potential sources of uncertainty and variability in the VKT estimates that underlie our LUR model, we consider: 1) VKT uncertainty given uncertain trip generation within the TRM; and 2) variability in VKT within groups of similar TAZs.

To account for uncertainty within the TRM, we assume that the uncertainty in the TRM output is characterized by a normal distribution having a mean equal to the TRM output and a standard deviation equal to the mean multiplied by the coefficient of variation (CV) derived by Zhang et al. in a study characterizing the uncertainty in model outputs from a four-step transportation demand model using Monte Carlo simulation to account for uncertain model inputs (Figure A1).[26]

To account for variability, we first remove all TAZs with zero population and then divide the remaining TAZs into quintiles by population density for each scenario. We then calculate the CV for each quintile and the zero-population group for each scenario (Table A1). We assume that the variability in VKT within each TAZ is characterized by a lognormal distribution with a mean equal to the mean predicted by the TRM (adjusted for uncertainty) and a standard deviation equal to the mean multiplied by the CV calculated for each group of TAZs.

We combine the VKT distributions described above with other model parameters, assuming distributions as appropriate, in Analytica (Lumina Decision Systems, Los Gatos, CA) to estimate health risks (Figure A1; Table A2). All distributions are truncated as appropriate to avoid spurious values in the tails of the distribution (e.g., VKT is truncated to avoid values less than zero). We use the VKT distributions obtained from above, combining both uncertainty and variability, to estimate $PM_{2.5}$ in each TAZ using our calibrated LUR model (Equation 4). We assume both the model constant and the VKT parameter are normally distributed. We then subtract background $PM_{2.5}$ concentrations from these estimates and apply Equation 3, assuming that the RR function is normally distributed.

Table A1

Summary of TAZs by Quintile of Population Density

		Base Case		Compact Development		Sprawl	
		Mean VKT	C.V.	Mean VKT	C.V.	Mean VKT	C.V.
Population Density Quintile	1	37,940	0.53	51,760	0.47	26,002	0.68
	2	30,310	0.70	35,944	0.56	33,328	0.61
	3	22,767	0.99	31,723	0.68	27,763	0.80
	4	14,592	1.38	22,886	0.99	17,612	1.12
	5	12,431	1.60	20,280	1.29	14,918	1.28
Zero Pop. TAZs		53,496	0.50	19,164	1.45	50,528	0.50
ALL TAZs		25,439	0.95	27,237	0.99	25,545	0.87

Table A2

Analytica Model Variables

Variable Name	Variable Type	Specification
TAZ	Index	Range: 1 – 2,579
Mean VKT (VKT)	Discrete; indexed by TAZ	TRM outputs (data not shown)
C.V. from Uncertainty (CV_U)	Discrete	0.4245[26]
S.D. from Uncertainty (SD_U)	Expression	$CV_U \times VKT$
VKT with Uncertainty (VKT_U)	Normal; indexed by TAZ	Mean: VKT S.D.: SD_U
C.V. from Variability (CV_V)	Discrete	See Table A1
S.D. from Variability (SD_V)	Expression	$CV_V \times VKT_U$
VKT (VKT_{U+V})	Lognormal; indexed by TAZ	Mean: VKT_U S.D.: CV_V
Beta0 (β_0)	Normal	Mean: 8.689 S.D.: 0.555
Beta1 (β_1)	Normal	Mean: 0.0474 S.D.: 0.011
PM	Expression; indexed by TAZ	$\beta_0 + \beta_1 \times VKT_{U+V}$
PM Above	Expression; indexed by TAZ	PM – β_0
Relative Risk Function	Normal	Mean: 1.06[56] S.D.: 0.023
Relative Risk (RR)	Expression; indexed by TAZ	1 + ((PM_Above/10)*(RR-1))
Attributable Fraction (AF)	Expression; indexed by TAZ	(RR-1)/(RR)
Baseline Death Rate (DR) *(per 100,000 persons)*	Discrete; indexed by TAZ	2010 Death Rates by County[55] (data not shown)
Population (Pop)	Discrete; indexed by TAZ	TRM input data or scenario specifications (data not shown)
Mortality (Mort)	Expression; indexed by TAZ	AF*Pop*DR/100,000
PM Death Rate (DR_{PM}) *(per 100,000 persons)*	Expression; indexed by TAZ	Mort/Pop*100,000

Variable Name	Variable Type	Specification
Sum of Mortality	Expression	Sum(Mort,TAZ)

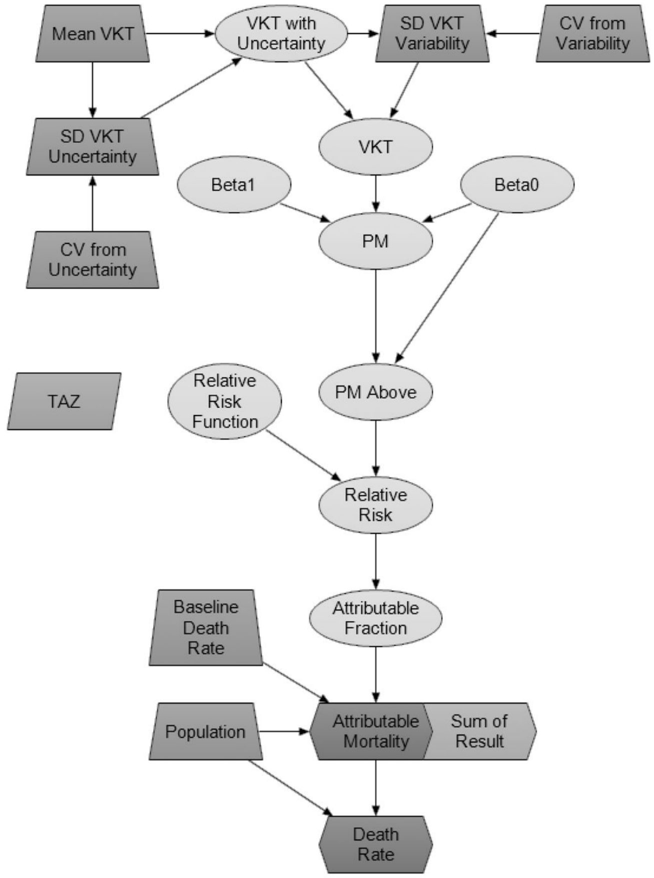

Figure A1.
Analytica Model Schematic

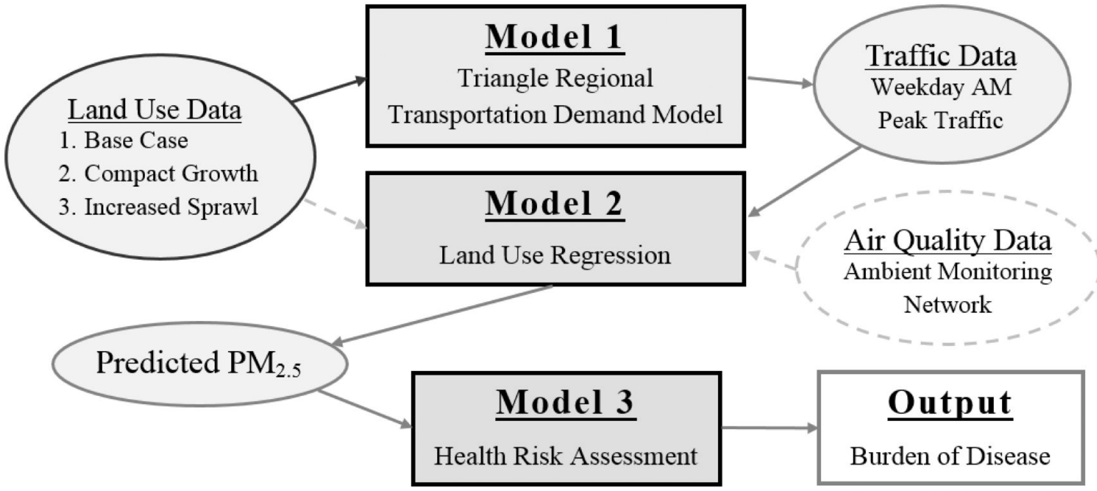

Figure 1.

The modeling approach employed in this research links a transportation demand model (Model 1) to a LUR model of $PM_{2.5}$ concentrations (Model 2) and a health risk assessment model (Model 3) to investigate of the effects of urban form on traffic, air quality, and health. Dashed lines indicate data used only for calibration of the LUR model.

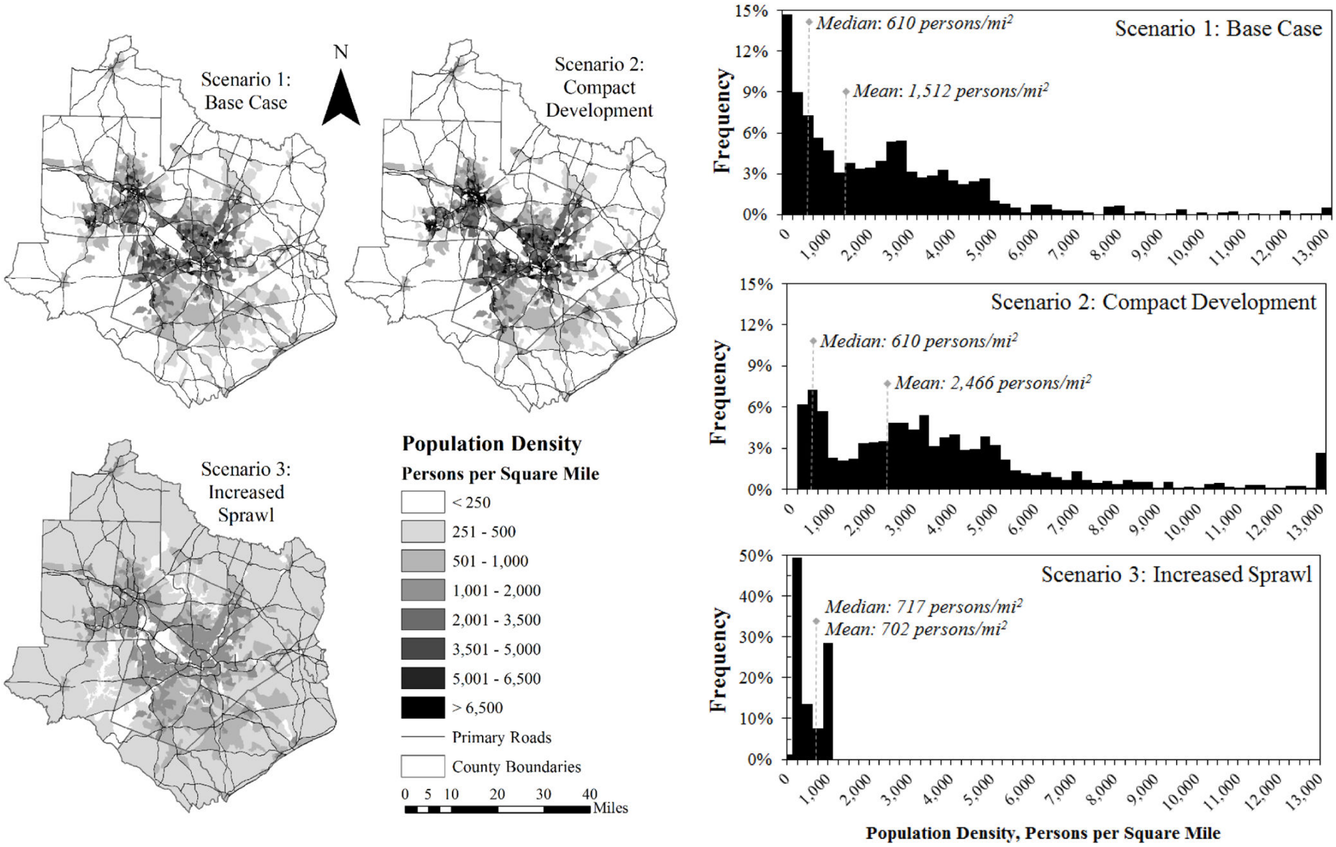

Figure 2.

Population spatial distributions (*left*) and histograms of population density in TAZs (*right*) for the three scenarios. The compact development scenario moves households from low-density areas to higher density areas, while the sprawl scenario does the opposite.

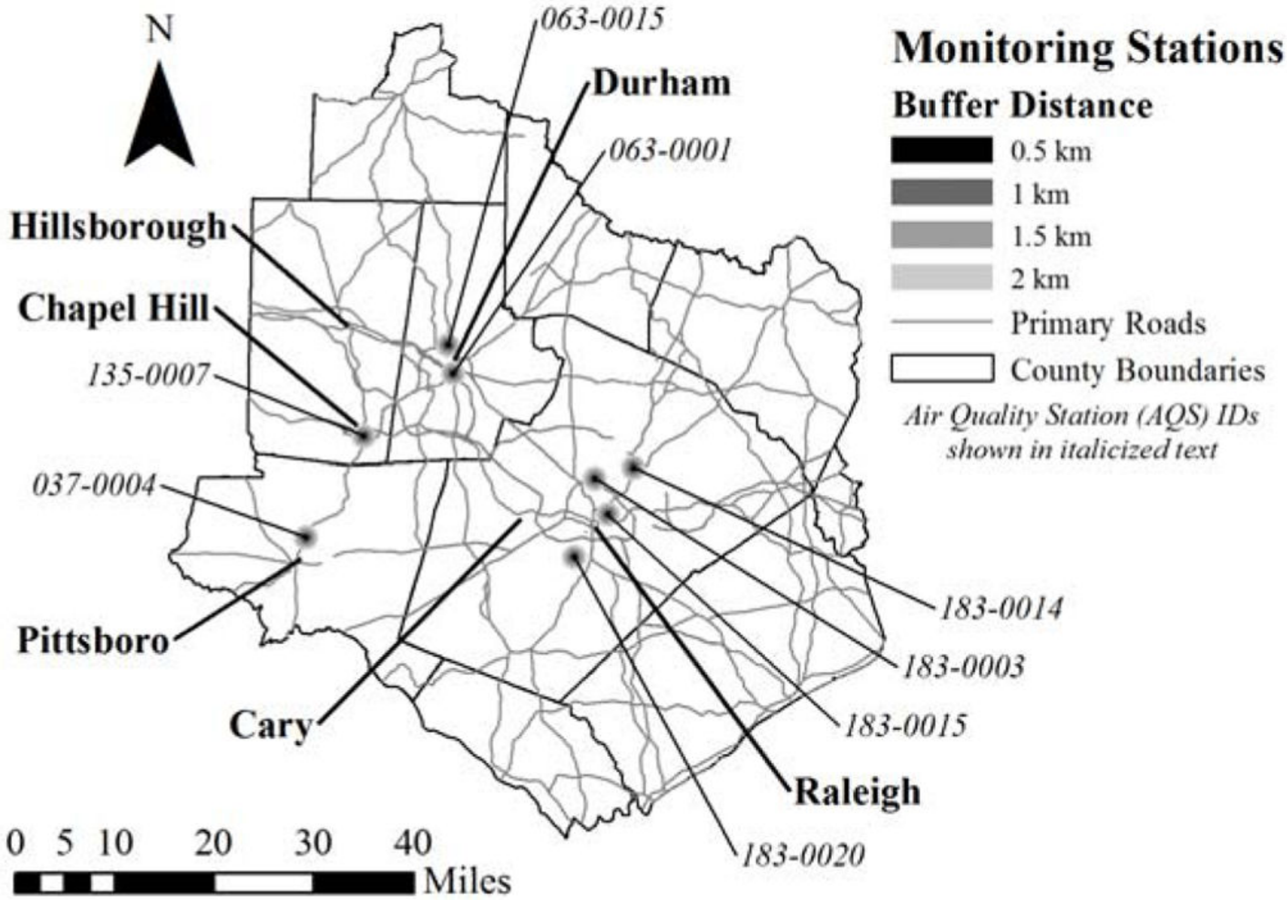

Figure 3.
Monitoring station locations

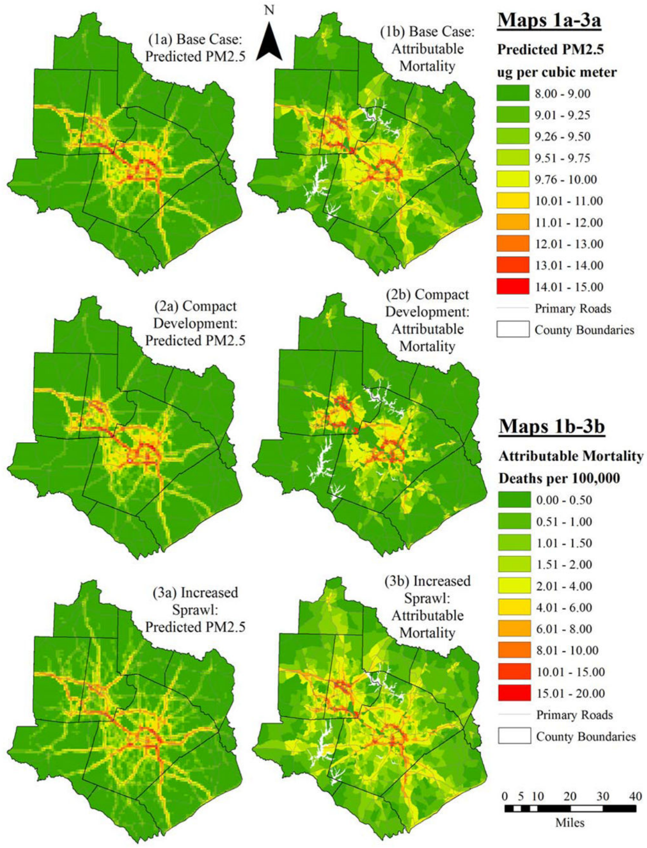

Figure 4.

Annual average $PM_{2.5}$ concentrations and attributable mortality for each scenario.

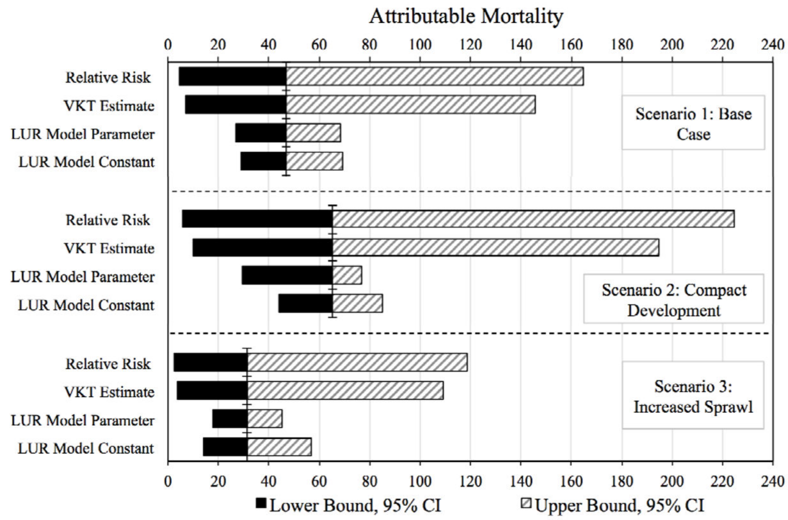

Figure 5.

Relative effect of varying each model component from its lower to upper 95% CI value on estimated mortality for each scenario

Table I

Regression models and $PM_{2.5}$:PM_{10} ratios for estimating $PM_{2.5}$ concentrations in cases where data are missing

Air Quality Station ID	Most Recent Year	*n (years of data)*	Model Form: $PM_{2.5} = \alpha + \beta \times$years after 1990						Average $PM_{2.5}$:PM_{10} Ratio
			α *($\mu g/m^3$)*	β *($\mu g/m^3$ yrs)*	F-statistic	Adj. R^2	2010 Value *($\mu g/m^3$)* Predicted	2010 Value *($\mu g/m^3$)* Observed	
183-0015	2003	4	20.26^{*}	-0.567^{*}	$F_{1,2} = 54.7^{*}$	0.95	8.9	*n/a*	*n/a*[a]
183-0003	2000	11	16.98^{**}	-0.181^{Ψ}	$F_{1,9} = 3.1^{\Psi}$	0.18	13.4	*n/a*	*n/a*[b]
135-0007	2008	10	16.59^{**}	-0.244^{**}	$F_{1,8} = 21.9^{**}$	0.70	11.7	*n/a*	*n/a*[a]
063-0001	2007	9	18.30^{**}	-0.320^{**}	$F_{1,7} = 23.4^{**}$	0.74	11.9	*n/a*	0.684
183-0014	2010	12	20.50^{**}	-0.520^{**}	$F_{1,10} = 70.9^{**}$	0.86	10.2	10.2	0.668
183-0020	2010	3	*n/a (too few observations;* $F_{1,1} = 0.48$, ns*)*				*n/a*	10.0	*n/a*[a]
037-0004	2010	12	17.47^{**}	-0.396^{**}	$F_{1,10} = 29.1^{**}$	0.77	9.5	8.9	*n/a*[a]
063-0015	2010	4	24.33^{*}	-0.728^{Ψ}	$F_{1,2} = 6.8^{\Psi}$	0.64	10.1	10.2	0.634

** $p < .01$

* $p < .05$

Ψ $p < .15$

$PM_{2.5}$:PM_{10} Ratio, All Stations: 0.673

[1] Imputed using time series data

[2] Imputed using $PM_{2.5}$:PM_{10} ratio

[a] Station does not track PM_{10}

[b] Station does not track $PM_{2.5}$

Table II

Summary of all explanatory variables tested

Air Quality Station ID	Weekday AM Peak VKT *(Thousands of VKT)*				Populating Density *(Persons/mi²)*				Industrial Land-use *(Acres)*				Elevation *(m)*
	Buffer size *(km)*				Buffer size *(km)*				Buffer size *(km)*				
	0.5	**1**	**1.5**	**2**	**0.5**	**1**	**1.5**	**2**	**0.5**	**1**	**1.5**	**2**	
183-0015	1.91	**16.8**	77.7	164	5,080	4,480	4,060	3,480	0	0	0	20	127
183-0003	16.2	**88.0**	145	220	2,850	2,650	2,540	2,530	0	0	0	0	125
183-0014	8.91	**48.3**	109	175	4,280	3,670	3,120	2,750	0	15	36	63	100
183-0020	3.98	**8.89**	14.0	36.6	309	309	310	749	0	0	30	38	120
135-0007	8.66	**47.2**	96.4	146	3,860	4,900	5,180	4,400	0	10	13	15	145
037-0004	0.10	**9.26**	18.8	26.0	147	161	170	173	0	0	0	6	121
063-0001	21.0	**77.5**	145	207	4,360	4,080	3,880	4,150	4	17	34	42	147
063-0015	13.6	**36.0**	104	166	2,120	2,360	2,380	2,530	6	82	86	86	118
Mean	*10.4*	***41.5***	*88.7*	*143*	*2,880*	*2,830*	*2,710*	*2,600*	*1.3*	*16*	*25*	*34*	*125*
Stnd. Dev.	*6.64*	***28.0***	*47.0*	*68.0*	*1,750*	*1,700*	*1,650*	*1,400*	*2.2*	*26*	*27*	*28*	*14*

NOTE: **Bold** values indicate explanatory variable values used in the final LUR model.

Table III

Results of LOOCV for Each Monitoring Station

Air Quality Station ID *(Station Removed)*	**Model Constant *(μg/m³)***	**VKT parameter *(μg/m³ VKT)***	**Predicted $PM_{2.5}$ *(μg/m³)***	**Actual *(Imputed)* $PM_{2.5}$ *(μg/m³)***	**Square Error *(μg²/m⁶)***
37-183-0015	8.90	0.0445	9.7	*8.9*[a]	0.5
37-183-0003	8.89	0.0392	12.3	*13.4*[a,b]	1.2
37-183-0014	8.76	0.0483	11.1	10.2	0.8
37-183-0020	8.25	0.0541	8.7	10.0	1.7
37-135-0007	8.62	0.0466	10.8	*11.7*[a]	0.7
37-037-0004	8.79	0.0458	9.2	8.9	0.1
37-063-0001	8.61	0.0516	12.6	*11.9*[a]	0.6
37-063-0015	8.72	0.0472	10.4	10.2	0.1

[a] Time series imputed value

[b] $PM_{2.5}$:PM_{10} imputed value

Root MSE **(μg/m³)**: 0.84

Correlation of Predicted to Observed: 0.82 *($p < .05$)*

Table IV

Area and population exposed to $PM_{2.5}$ concentrations above EPA and WHO standards

	EPA standard (12 μg/m^3)		WHO standard (10 μg/m^3)	
	Area Exceeding Standard, mi^2 *(%)*	**Population Exposed *(%)***	**Area Exceeding Standard, mi^2 *(%)***	**Population Exposed *(%)***
Base Case	24.4 *(0.7%)*	35,500 *(2.2%)*	236 *(7.0%)*	407,000 *(25.6%)*
Compact Development	33.9 *(1.0%)*	95,400 *(6.0%)*	249 *(7.4%)*	644,000 *(40.5%)*
Sprawl	21.9 *(0.7%)*	14,100 *(0.9%)*	256 *(7.6%)*	191,000 *(12.0%)*

Table V

VKT, Population Density and Percent of Total Population by VKT Decile

VKT Decile	Mean VKT *(thousands)*			Mean Population Density *(persons/mi^2)*			Percent of Total Population *(%)*		
	Base	**Compact**	**Sprawl**	**Base**	**Compact**	**Sprawl**	**Base**	**Compact**	**Sprawl**
1	0.0	0.0	0.0	121	48	276	2.6%	1.1%	6.2%
2	0.2	0.0	0	118	40	302	2.6%	0.9%	6.8%
3	0.5	0.1	1.2	138	40	332	3.0%	0.9%	7.5%
4	1.0	0.3	2.2	170	52	359	3.7%	1.2%	8.1%
5	1.5	0.7	3.3	197	87	376	4.2%	1.9%	8.5%
6	2.3	1.3	4.9	271	138	419	5.9%	3.1%	9.5%
7	3.8	2.6	7.0	376	307	460	8.1%	6.9%	10.4%
8	6.7	5.9	10.4	496	471	540	10.7%	10.5%	12.2%
9	14.1	13.6	17.3	1,057	1,060	640	22.8%	23.7%	14.5%
10	42.8	44.4	44.5	1,687	2,232	714	36.4%	49.9%	16.2%

Table VI

Vehicle miles travelled, annual average $PM_{2.5}$ concentrations, and attributable mortality by development scenario (mean and 95% CI)

Outcome → ↓ Scenario	VKT	$PM_{2.5}$ Concentration		Attributable Mortality	
	Total (*thousands*)	Mean (*$\mu g/m^3$*)	Maximum (*$\mu g/m^3$*)	Total (*persons*)	Max. Rate (*per 100k persons*)
Scenario 1: Base Case	**21,300 (2,600–80,200)**	**9.0 (7.8–10.7)**	**15.0 (9.6–41.3)**	**47 (5–160)**	**17 (2.1–85)**
Scenario 2: Compact Development	20,200 (2,700–76,800)	9.0 (7.8–10.7)	15.2 (9.6–48.8)	65 (6–220)	20 (2.8–90)
Relative to Base Case	−5.2%	−0.2%	+1.7%	+39.1%	+21.0%
Scenario 3: Increased Sprawl	26,700 (2,600–96,700)	9.1 (7.9–10.9)	14.3 (9.3–39.4)	31 (2–120)	17 (1.7–68)
Relative to Base Case	+25.2%	+1.0%	−4.4%	−33.1%	+0.82%