Health Impacts Assessment of Ambient Air Pollution in Urban Areas: The Role of Socioeconomic Status and Transportation

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Received: date; Accepted: date; Published: date

Abstract: With recent rapid urbanization, sustainable development is required to prevent health risks associated with adverse environmental exposures from unplanned developments of cities (e.g., air pollution). Health impact assessment of air pollution across cities, and how these vary according to socioeconomic status and transportation trends, can help city planners and health sectors to make informed decisions to avoid or mitigate adverse exposures and impacts. In this study, we investigate the health impacts of air pollutants (PM$_{2.5}$ and NO$_2$) at the census tract level in Houston, Texas. 631 (95% CI: 366-809) premature deaths (adults 30 to 78 years old) were attributable to PM$_{2.5}$ in Houston, and 159 (95% CI: 0-609) premature deaths were attributable to NO$_2$, in 2010. Complying with the World Health Organization air quality guidelines (annual mean: 10 μg/m$^3$ for PM$_{2.5}$) can save 82 (95% CI: 42-95) lives in Houston. PM$_{2.5}$ was responsible for 7.3% all-cause premature deaths in Houston, in 2010, which is higher than the death rate caused by diabetes mellites, Alzheimer’s disease, and accidents in the US. Households with lower income had a higher risk of adverse exposure and attributable premature deaths. We also uncovered the relationship between health impacts from air pollution and the road traffic passing through census tracts.

Keywords: Burden of disease; Air pollution; Premature deaths; Attributable deaths; Road Traffic; Socioeconomic inequities

1. Introduction

About 55% of the world’s population lived in urban areas in 2018, while this percentage is expected to increase to 68% by 2050 [1]. To ensure that the benefits of urbanization are equally distributed and that the risks are mitigated, cities need to be sustainably developed urban areas to meet population needs—e.g., housing, transportation, health care, and other infrastructures. Unsustainable developments of cities are associated with health risks, including those from air pollution, climate change, unsafe drinking water, among others. According to a World Health Organization’s (WHO) study on more than 4300 cities worldwide, 80% of the urban population was living in urban areas that do not comply with WHO air quality guideline levels for Particulate Matter with a diameter equal or less than 2.5 micrometers (PM$_{2.5}$) [2]. It is shown that exposure to air pollution can cause serious health issues such as lung cancer [3]; respiratory diseases [4] including chronic obstructive pulmonary disease [5]; asthma [6]; and cardiovascular diseases [7] including stroke [5]. Conservative estimates from the World Bank attributed 4.2 million annual deaths worldwide to air pollution in 2016 [8]. Estimations showed that transportation-related air...
pollution, in particular, is responsible for one-fifth of deaths from air pollution in the United Kingdom, United States (US), and Germany [9].

While health impact assessment of air pollution at national, regional and global levels are useful, city governments, who are more agile, can only act within their jurisdictions and mitigate adverse exposures and impacts at the city level. In this context, we quantify and analyze the burden of disease attributable to air pollution, in the form of premature deaths, in an urban area. We analyze the spatial variation of estimated premature deaths across the city to investigate the possible role of transportation as well as population socioeconomics in the health impacts of air pollution. Mortalities attributable to air pollution in urban areas were quantified in a number of studies [10-15]. Although a study explored and showed that the exposure to air pollution and attributable burden of disease inversely correlates with population socioeconomic characteristics [14], the spatial variation of the air pollution health outcomes has not been discussed in terms of living in areas with higher level of road traffic. Such an analysis can be used to demonstrate the significance of road traffic in burden of disease analysis instead of running time and cost-intensive full-chain models.

From a methodological standpoint, the previous burden of disease studies share a similar methodology for quantifying the air pollution health burden. Generally, the baseline exposure level is compared with either level of exposures recommended by health authorities or a no-exposure scenario, and the burden of disease for the health outcome of interest is quantified using Exposure-Response Functions (ERF) extracted from the literature [15]. The spatial resolution of air pollution burden of disease analysis in urban areas varied from census-tract level [15, 16] and lower super output area [14] to city-wide estimations [17]. The spatial level of the analysis is dependent on the availability of data. For example, Lelieveld et al. captured the contribution of air pollution to premature deaths for 100 km × 100 km spatial resolution [9]. Previous studies on the burden of disease attributable to air pollution also varied based on the data utilized for analyses. In simple terms, air pollution data is either collected from air quality monitoring [18] or generated by modeling [10, 13-15, 19]. Different studies used different pollutants in cities including PM$_{2.5}$ [14, 15], Nitrogen dioxide (NO$_x$) [14], and Ozone [9]. The air pollution health impacts assessment studies are mainly conducted in Europe. The health impacts attributable to air pollution are discussed in the US for specific diseases (chronic disease health impacts nationwide [20] and postneonatal infant mortality in the metropolitan area [21]) as well as all-cause premature deaths [22, 23]. An analysis of the health impacts of air pollution by Goodkind et al. showed that PM$_{2.5}$ was responsible for 107,000 premature deaths, in the US, in 2011 [22]. However, no study has focused on analyzing air pollution health impacts in US cities to the best of authors knowledge.

In our analysis, we ran a health impact assessment of two air pollutants (NO$_x$ and PM$_{2.5}$). Air pollution concentrations were estimated by Land Use Regression (LUR) model and a universal kriging framework. We quantified premature deaths attributable to these exposures using the standard burden of disease methodology, separately for each pollutant. The finer the spatial resolution of the analysis, the better the insight one can gain into health equity issues, contributors, and high-risk spots which can be effectively targeted by policies. We, therefore, ran our analysis at the census tract level to capture spatial variations at a fine scale. As a case study, we focused on the city of Houston, Texas; the fourth most populated city in the US. The methodology of this study is applicable to other cities, and the results can benefit both city planners and health professionals to detect high-risk spots in cities and plan interventions accordingly. Also, the results can raise public awareness of the health impacts associated with air pollution and its spatial distribution across cities and promote dialogue with policymakers and other stakeholders.
2. Materials and Methods

2.1. Study setting and definitions

The burden of disease attributable to air pollution was quantified in the city of Houston for the year 2010, the year which we had air pollution models for. The city of Houston is the largest city in Texas with 636.5 square miles (1646 square km) land area and 2,099,451 residents in 2010 [24]. The city is located in three US counties; Harris, Fort Bend, and Montgomery. The burden of disease analysis was conducted at the finest reasonable spatial resolution: the census tract level. The rationale behind analyzing the burden of disease at the census tract level is twofold. First, the mortality data was only available at the county level, and so approximations were required to assign baseline mortality rates to a finer spatial level. To minimize the error of these approximations, and yet investigate the spatial distribution of health outcomes, we chose to limit the spatial resolution of this study to the census tracts level. Second, to better analyze the road traffic spatial variations (discussed in subsequent sections), the census tract level was selected as the basis of the spatial burden of disease analyses. Consequently, 592 census tracts were included in this study, which was fully or partially located within the Houston city’s boundaries.

We quantified the health impacts in the form of attributable premature deaths. Premature death is defined as a measure of unfulfilled life expectancy [25], which is considered as the number of deaths before reaching the expected age of death in a population. The life expectancy in the US was 78.7 years old in 2010 [26]. The risk of mortality in association with NO₂ and PM₂.5 were sourced from meta-analysis studies for individuals older than 30 years old (details are provided in subsequent sections). Hereafter, the term premature deaths refers to the deaths of individuals aged 30 to 78 years old died in a natural way (as opposed to accidents and suicides).

2.2. Input data

The data used in this study were collected from multiple sources—namely, US census bureau, Centers of Disease Control and Prevention and Texas Department of Transportation—as described in the following sections.

2.2.1. Population, socioeconomic and geographic data

Population and socioeconomic data were collected from the US Census Bureau for 2010 at the census tract level along with the census tracts’ geographic characteristics. We stratified the burden of disease by household economy using the median household income at the census tract level, as sourced from the US Census Bureau. The average of households’ median income in the city of Houston, in 2010, was $52,857 dollars (see Table 1 for a descriptive statistic of median income and Figure 4d for its spatial distribution across the city). City of Houston geographical limits was sourced from the city’s open data portal which was used to identify the census tracts within the city’s boundaries (retrieved from https://cohgis-mycity.opendata.arcgis.com/datasets/houston-city-limit).

2.2.2. Mortality data

The baseline mortality data for Texas in the year 2010 was sourced from the Centers for Disease Control and Prevention (CDC) (https://wonder.cdc.gov/mcd.html). The mortality data was available both in the form of the number of deaths and crude mortality rate¹ at the county level with 95% Confidence Interval (CI). For quantifying the premature deaths attributable to air pollution, the number of all-cause natural deaths for people aged 30 to 78 years old was used in this study. Given that the city of Houston is located in three counties: Harris, Fort Bend, and Montgomery, the mortality data for these three counties was collected. We distributed the number of mortality cases

¹ Crude mortality rate is the total number of deaths to residents in a county divided by the total population for the county (for a calendar year) and multiplied by 100,000.
(available at the county level) across census tracts proportionally based on their population size. In 2010, a total number of 8,667 all-cause premature deaths (natural deaths excluding accidental mortalities) were reported in the city of Houston (30 to 78 years old). The summary statistics of the mortality data at the census tract level are reported in Table 1.

2.2.3. Air pollution data

NO\textsubscript{2} and PM\textsubscript{2.5} concentrations data were sourced from a previously published and validated LUR model, also employed in our previous study [27]. We used the annual average concentrations in μg/m\textsuperscript{3} for the years 2010 at the centroid of each census tract. The data is originally estimated at the centroid of the census blocks. We estimated the weighted average of concentrations at census blocks. The weights were assigned based on the census block area. The NO\textsubscript{2} concentrations were converted from ppb to μg/m\textsuperscript{3} through multiplying by 1.88 [28]. Table 1 provides a detailed summary of pollutant concentrations across the city. Spatial distribution of air pollutants is shown in Figure 1. In following we briefly discuss the models used for predicting NO\textsubscript{2} and PM\textsubscript{2.5} concentrations.

The NO\textsubscript{2} estimates were obtained from a LUR model, developed by, and described in detail in [29]. In brief, the model uses satellite data and Environmental Protection Agency (EPA) air quality monitor readings of NO\textsubscript{2} concentrations alongside several covariates (for example, impervious surfaces, elevation, major roads, residential roads, and distance to the coast) to estimate NO\textsubscript{2} concentrations. The model also incorporates temporal scaling by estimating average monthly monitor readings for 11 consecutive years. The final model used has a relatively high predictive power at unmeasured locations which was tested using a hold-out cross-validation with good model performance ($R^2 = 0.82$); which is comparable with other continental-scale NO\textsubscript{2} LUR models [30-32].

Annual average concentrations of PM\textsubscript{2.5} were estimated using data from 17 years (1999–2015). The data were derived from regulatory monitors and estimates were constructed in a universal kriging framework [33]. Partial least squares were estimated for model performance from hundreds of geographic variables, including land use, population, and satellite-derived estimates of land use and air pollution. Hold-out cross-validation (CV) indicated good model performance (10-fold CV- $R^2$, 0.85). Annual PM\textsubscript{2.5} concentrations were predicted at the census tracts centroids (with the similar procedure described above).
2.2.4. Road traffic

In this study, the annual averaged daily Vehicle Mile Traveled per Area (VMTA) was used to investigate the relation between road traffic and air pollution health burden in the city at the census tract level. VMTA represents the density of total Vehicle Mile Traveled (VMT) at a census tract. The VMT was calculated by aggregating the multiplication of the road segment length and Annual Daily Traffic (ADT) passing through a road segment located within a census tract. Then, the VMT was divided by the census tract area to represent the density of road traffic. Equation 1 shows the VMTA calculation for each census tract:
In addition to the roads located within the census tract, the possible impacts of roads near the census tract boundary were taken into account. According to WHO, PM$_{2.5}$ and NO$_2$ concentrations decrease to background concentrations within 100–150 m (328–492 feet) of a roadway [34]. To this end, we included the VMT passing though the roads in 492 feet distance of the census tract boundary to estimate the VMTA of the census tract.

The ArcGIS spatial tools were used to find the roads, its length and the ADT within and outside the census tracts’ boundaries. Specifically, we identified the roads located within a 492 feet distance of the census tract boundary using ArcGIS and included in VMTA calculations. Road network and ADT data were sourced from the Texas roadway inventory data by Texas Department of Transportation (https://www.txdot.gov/inside-txdot/division/transportation-planning/roadway-inventory.html). The data was not available for 2010, and therefore, the ADT data from 2011 was used. We assume that the road traffic spatial distribution across census tracts is consistent between 2010 and 2011. A summary of road traffic statistic is presented in Table 1. Figure 4c illustrates the spatial distribution of road traffic across Houston at the census tract level.

Table 1. A summary of input data statistics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sample Size</th>
<th>Min</th>
<th>Median</th>
<th>Mean</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>All-cause Premature deaths (persons) (# of Census Tracts)</td>
<td>592</td>
<td>1</td>
<td>14</td>
<td>15</td>
<td>61</td>
</tr>
<tr>
<td>NO$_2$ Concentration (µg/m$^3$)</td>
<td>592</td>
<td>7.47</td>
<td>19.38</td>
<td>19.52</td>
<td>34.09</td>
</tr>
<tr>
<td>PM$_{2.5}$ Concentration (µg/m$^3$)</td>
<td>592</td>
<td>6.80</td>
<td>11.61</td>
<td>11.41</td>
<td>13.30</td>
</tr>
<tr>
<td>VMTA (veh.mi/mi$^2$)</td>
<td>592</td>
<td>0.72</td>
<td>99.27</td>
<td>163.04</td>
<td>1077.54</td>
</tr>
<tr>
<td>Median Household Income (dollar)</td>
<td>592</td>
<td>9,926</td>
<td>43,352</td>
<td>52,857</td>
<td>214,861</td>
</tr>
</tbody>
</table>

2.3. Burden of disease model

We used a standard burden of disease estimation framework previously developed in the literature [15]. This framework is employed to estimate the premature deaths attributable to air pollution. In brief, the inputs to the burden of disease model included the NO$_2$ and PM$_{2.5}$ concentrations, as well as the baseline all-cause deaths rate in the studied region. Next, the Relative Risk ($RR_{diff}$) of all-cause deaths in association with the difference between current concentration levels and the counterfactual exposure level were estimated. Equation 2 presents the $RR_{diff}$ calculations for a linear ERF.

$$RR_{diff} = RR \times \frac{(E_{current} - E_{counterfactual \ exposure})}{RR_{unit}}$$

where $RR$ is the relative risk extracted from epidemiology studies, $E_{current}$ represents the current concentration level, $E_{counterfactual \ exposure}$ represents counterfactual exposure level, and $RR_{unit}$ is the exposure unit of $RR$ obtained from ERFs. Then, the Population Attributable Fraction (PAF) was calculated using Equation 3. The PAF represents the ratio of premature deaths attributable to air pollutants to all-cause deaths for the difference between current concentration levels and the counterfactual exposure level.

$$PAF = \frac{RR_{diff} - 1}{RR_{diff}}$$

Finally, the attributable deaths were estimated using the mortality rate and population counts for people aged 30 to 78 years old, and the estimated PAF (using Equation 4). The employed burden of disease estimation framework is presented in Figure 2. This procedure was used for estimating the premature mortality across the 592 census tracts.

$$Attributable \ Mortality = PAF \times Mortality \ rate \times Population \ counts$$
2.4. Exposure-response functions

We extracted ERF for NO$_2$ and PM$_{2.5}$ from two meta-analysis studies. A meta-analysis including data from 22 cohort studies with a total of 367,251 participants was used for the ERF of NO$_2$ and premature mortality [35]. Based on the proposed ERFs which associated the natural deaths with NO$_2$, the RR of deaths per 10 μg/m$^3$ NO$_2$ is estimated as 1.02 (95% CI: 0.99-1.04) for individuals older than 30 years. The proposed ERFs were not statistically deviated from linear functions and therefore, we used the linear ERFs in this study. The RR was adjusted for sex, calendar time, smoking status, smoking intensity, smoking duration, environmental tobacco smoke, fruit intake, vegetable intake, alcohol consumption, body-mass index (BMI), educational level, occupational class, employment status, marital status, and area-level socioeconomic status [35]. Note that the 0.99 RR implies that no adverse health impact is associated with 10 μg/m$^3$ decrease in NO$_2$.

The ERF for PM$_{2.5}$ was extracted from a meta-analysis by WHO [36]. This meta-analysis was performed on 14 studies and resulted in RR for different regions. For the US, using a linear ERF, the overall RR of natural deaths associated with PM$_{2.5}$ was estimated as 1.07 (95% CI: 1.02-1.12) per 10 μg/m$^3$ for individuals 20 years and older. The RR was not adjusted for the impact of NO$_2$ and as such, we estimated the burden from both pollutants separately and emphasize that these should not be added up [36].

2.5. Counterfactual scenario

We estimated the premature deaths attributable to air pollution for two counterfactual scenarios:

1. Zero-exposure of the population to air pollution,
2. Air pollution concentration complying WHO air quality guideline values.

For the first scenario, the current concentrations, as estimated from the air pollution models, were compared to zero-concentration to demonstrate the health impacts of ambient air pollution in the city. In the second scenario, the current concentrations were compared to the WHO air quality guideline values. WHO recommends NO$_2$ does not exceed 40 μg/m$^3$ annual mean and PM$_{2.5}$ does not exceed 10 μg/m$^3$ annual mean [28]. The RR of mortality in association to NO$_2$ and PM$_{2.5}$ was rescaled for the difference between the current concentration levels and the counterfactual scenarios as shown in Equation 3.


2.6. Sensitivity analysis

Uncertainties are inherited in variables incorporated in burden of disease assessment studies, mainly arising from the uncertainty in the baseline health data and the selected ERFs, among others. To explore the range of uncertainties from the variables included in our analysis, including the baseline mortality, the ERFs, and the conversion of the noise metrics, we run two uncertainty analysis. First, we estimated the most conservative and most extreme burden of disease scenarios using the combinations of the lower and upper 95% CI for each of the variables above. These two scenarios are reported in parentheses after the estimated values of premature deaths attributable to air pollution. Second, examined the effect of each variables’ uncertainty in premature deaths estimations. To this end, we reran the analysis for each variable individually. In this context, the burden of disease for the upper and lower 95% CI of each variable was estimated.

3. Results

3.1. Premature deaths attributable to air pollution

Table 2 summarizes the estimated premature deaths attributable to NO\(_2\) and PM\(_{2.5}\) in the city of Houston, in 2010. 631 premature deaths for the age group between 30 to 78 years old were attributable to PM\(_{2.5}\). Considering most conservative and the most extreme burden of disease scenarios, the number of premature deaths attributable to PM\(_{2.5}\) varied from 366 to 809 deaths. Similarly, 159 (95% CI: 0-609) premature deaths were attributable to NO\(_2\). Exceeding WHO guidelines recommendations resulted in 82 (95% CI: 42-95) preventable premature deaths attributable to PM\(_{2.5}\). Figure 3 illustrates the range of the percentage of premature deaths attributable to air pollution (zero-exposure scenario) to all-cause deaths and its distribution at the census tract level. The spatial distribution of premature deaths attributable to PM\(_{2.5}\) and NO\(_2\) (zero-exposure scenario) is shown in Figure 4a and 4b, in the form of a percentage of all-cause premature deaths. The percentage of premature deaths attributable to NO\(_2\) was higher in the census tracts located in the Central Business District (CBD). Also, a spatial similarity is observed between the distribution of VMTA and air pollutants’ health impacts (Figure 4).

Table 2. The number of premature deaths attributable to air pollution in Houston

<table>
<thead>
<tr>
<th>Counterfactual scenario</th>
<th>Premature deaths cases (95% CI)</th>
<th>Air Pollutant</th>
<th>Counterfactual Concentration (μg/m(^3))</th>
<th>Adjusted RR associated with 10 μg/m(^3) increase (95% CI)</th>
<th>Attributable premature deaths (95% CI)</th>
<th>% of attributable premature deaths to all-cause deaths (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zero-exposure scenario</td>
<td>8,667 (8,499-8834)</td>
<td>PM(_{2.5})</td>
<td>0</td>
<td>1.07 (1.02-1.12)</td>
<td>631 (366-809)</td>
<td>7.3% (4.3%-9.2%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>NO(_2)</td>
<td>0</td>
<td>1.01 (0.99-1.04)</td>
<td>159 (0-609)</td>
<td>1.8% (0.0%-6.9%)</td>
</tr>
<tr>
<td>Complying with WHO guidelines</td>
<td>8,667 (8,499-8834)</td>
<td>PM(_{2.5})</td>
<td>10</td>
<td>1.07 (1.02-1.12)</td>
<td>82 (42-95)</td>
<td>0.9% (0.5%-1.1%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>NO(_2)</td>
<td>40</td>
<td>1.01 (0.99-1.04)</td>
<td>0 (0-0)</td>
<td>0.0% (0.0%-0.0%)</td>
</tr>
</tbody>
</table>

\(^1\)NO\(_2\) concentration across the city was lower than the WHO guideline recommendation.
Figure 3. Range and distribution of estimated premature deaths attributable to PM$_{2.5}$ and NO$_2$ as a percentage of all-cause deaths.
Figure 4. Spatial variation of (a) percentage of premature deaths attributable to PM$_{2.5}$ to all-cause death, (b) percentage of premature deaths attributable to NO$_2$ to all-cause death across the city of Houston at the census tract level, in 2010.
3.2 Premature deaths attributable to air pollution by household income and road traffic

The relation between median household income at each census tract and the premature deaths from air pollution was further explored. The comparison revealed an inverse correlation between the median household income at the census tract level and the ratio of premature deaths attributable to air pollution from all-cause premature deaths (the average lines in Figure 5a). In other words, it is expected that the ratio of premature deaths attributable to PM$_{2.5}$ and NO$_2$ reduces (by 10% of the estimated ratio (Figure 5a)) with an increase in household income from $20,000 to $75,000.

Figure 4 (continue). Spatial variation of (c) vehicle mile traveled per area, and (d) median household income across the city of Houston at the census tract level, in 2010.
According to Figure 3, the ratio of premature deaths attributable to PM$_{2.5}$ and NO$_2$ at the census tract level can vary from 0.0% to 3.3% and 0.0% to 8.6%, respectively. After a closer look at the spatial distribution of the ratio of premature deaths across the city, a relationship between VMTA and ratio of premature deaths attributable to air pollutants was detected (illustrated in Figure 5b). This relationship is consistent with the similarity between the spatial variation of the premature deaths attributable to air pollutants and VMTA indicated in Figure 4. The relation between the ratio of premature deaths and VMTA is stronger for the estimated deaths from NO$_2$ comparing to PM$_{2.5}$ (R-squared of the fitted curve for NO$_2$ is 0.52 versus 0.23 for PM$_{2.5}$).

![Figure 5](image_url)

(a)

![Figure 5](image_url)

(b)

**Figure 5.** Variation of the percentage of premature deaths attributable to air pollution to all-cause deaths by (a) median household income, and (b) road traffic

### 3.3. Sensitivity analysis

The most conservative estimation of premature mortality attributable to NO$_2$ resulted in zero deaths which imply no association between NO$_2$ and premature deaths. The most extreme
estimation resulted in 609 deaths (reported in Table 1). The most conservative and most extreme estimations for premature mortality attributable to PM$_{2.5}$ resulted in 366 and 809 deaths.

Results of the uncertainty analysis of lower and upper 95th CI of each variable is depicted in Figure 6. The uncertainty in the RRs had the largest role in the results’ uncertainty where the estimated attributable premature mortality to NO$_2$ and PM$_{2.5}$ could be changed by up to 276.1% and 41.0%, respectively. The uncertainties in the mortality rates resulted in up to 2.1% deviation in the estimated attributable premature deaths. Overall, a higher level of uncertainty was associated with premature mortality attributable to NO$_2$ compared to PM$_{2.5}$.

![Figure 6. Uncertainty analysis of the variables](image_url)

### 4. Discussion

#### 4.1 Key findings

This study sheds light on the health burden attributable to air pollution in the city of Houston, Texas, in the form of premature deaths. The results showed that, in 2010, 631 (95% CI: 366-809) premature deaths were attributable to PM$_{2.5}$ and 159 (95% CI: 0-609) premature deaths were attributable to NO$_2$. The estimated number of premature deaths from PM$_{2.5}$ and NO$_2$ can be translated into 7.3% and 1.8% of all-cause premature deaths in the city, respectively. The ratio of premature deaths attributable to PM$_{2.5}$ is higher than the death rate caused by diabetes mellitus, Alzheimer’s disease, and accidents in the US (2.8%, 3.4%, and 4.9%, respectively, in 2010 according to [26]), while the ratio of premature deaths from NO$_2$ is comparable with the death rate from suicide as well as influenza and pneumonia (1.6% and 2.0% in 2010 according to [26]). Complying with the WHO air quality guideline recommended values for PM$_{2.5}$ concentration (10 µg/m$^3$) can save 82 (95% CI: 42-05) lives in Houston.

We found that the burden of premature deaths from air pollution was higher in the census tracts located in the CBD. Also, the similarity of the road traffic spatial variation and ratio of premature deaths attributable to air pollutants was demonstrated. We showed an inverse correlation between the median household income and the ratio of premature deaths attributable to air pollution. The ratio of premature deaths attributable to air pollution decreases by 10% when the household’s median income increased from $20,000 to $75,000. Also, a positive relation between road traffic and premature deaths attributable to air pollution was shown: the more vehicles passing through a squared mile of a census tract, the higher risk of deaths from air pollution is expected for the residents of that census tract. The stronger relationship between the ratio of premature deaths attributable to NO$_2$ and VMTA compared to the ratio of premature deaths attributable to PM$_{2.5}$ and VMTA is in an agreement with the fact the road traffic is responsible for a significant portion of
NO₂ (68% of NO₂ in Beijing, China [37] and 40% of NO₂ in Jamshedpur, India [38] and 56% of NO₂ in European cities [39] while in the US, traffic contributed to 38% of all nitrogen oxide (NOx) emissions [40]). A higher level of uncertainty was observed in NO₂ estimation which is in line with the weak association between premature deaths and NO₂ in epidemiology studies. This implies that the evidence on premature mortality from PM₂.₅ is more reliable compared to NO₂, given the stronger association between PM₂.₅ and mortality and also the stronger case for biological plausibility.

### 4.2. Strengths and limitations

In this study, we estimated the premature deaths attributable to air pollutant at census tracts which enables us to better investigate the spatial distribution of the attributable deaths. The concentrations are predicted in a lower resolution (census block) and then converted to a higher resolution (census tract). We also investigated the premature deaths attributable to NO₂ and PM₂.₅ by the level of road traffic (VMTA) passing through the census tracts and in the catchment area around the census tract where we expect traffic to be most influential. This approach is introduced as an alternative of full-chain models to represent the traffic-related air pollution role on public health, with considerably less effort. We compared the reliability of the premature deaths estimation attributable to NO₂ and PM₂.₅.

This study has certain limitations. We focused on two major air pollutants (NO₂ and PM₂.₅), among others, and so the estimated health impacts of air pollution could be underestimated. We assumed the air pollutants’ concentrations does not spatially vary within a census tract which implies that all population living in a census tract are exposed to the same average concentration level. To assign the mortality data (available at the county level) to a census tract, we assumed that the crude mortality rate is constant across the census tract located within a county. Based on that, the mortality cases were distributed between the census tract according to their population. The extracted ERFs were estimated for adults older than 30 years, and so the potential deaths in the younger population were not quantified. Our approach may, therefore, result in underestimating the health impacts of air pollution in Houston. It is also important to note that the health impacts of PM₂.₅ and NO₂ cannot be added up because of the overlap in the biological pathways to impact human health. Although we showed the relationship between road traffic and deaths attributable to air pollution, no conclusion can be drawn on the contribution of the traffic-related air pollution to public health as we did not use an approach that allows source apportionment. Although the LUR predicts air pollution with fairly high accuracy, it considers all sources of air pollution and we could not parse out the exact contribution of traffic from other sources in the exposure and associated burden of disease. Because of the limitations in availability of the ADT data, the data from 2011 is used in this study. In this context, we assumed the spatial variation of traffic did not change from 2010 to 2011.

### 4.3. Policy and research recommendations

Four groups of strategy have been discussed in the literature to improve the air quality and consequently reduce the adverse health impacts, namely, regulations on air quality control, road traffic-related emission control interventions, energy generation related emission control interventions and greenhouse gas emission control interventions [41]. Among others, a more significant increase in air quality and its subsequent health benefits was observed after traffic-related interventions [41]. Supporting policies to manage the travel demand in more vulnerable spots is an effective traffic-related intervention, namely, improving public transport, improving infrastructure for active transportation, parking control, road pricing and prohibiting car traffic [42]. The results of this study underscore the necessity of Health Impact Assessments (HIA) of transportation-related projects and designs. HIA can contribute to cost-benefit assessment of the project and help the city to make more informed decisions. The socioeconomic status of populations is known as a factor that affects the vulnerability to the adverse health impacts of air pollution [43]. To protect this subpopulation, required settings need to be developed as air quality standards.
The contribution of the traffic-related air pollutants can be captured in future studies with a full-chain analysis comprising transportation modeling, emission estimation and dispersion modeling and assessing the health impacts of traffic-related air pollution through burden of disease analyses. Similar studies can be conducted to evaluate the impacts of new technologies (e.g., electric and hybrid vehicle with lesser emission rate) on public health within cities. Also, a comparison air pollution health impacts with other health risk factors in cities can indicate the significance of the health risk factors and help decision-makers to prioritize the required course of action to promote public health and ensure sustainable developments of cities.

5. Summary and conclusion

Quantifying the health impacts of air pollution may support decision-makers, urban planners, and health practitioners to make more informed decisions to avoid or mitigate adverse health impacts and ensure that the most vulnerable populations are not the most impacted. In this study, we quantified the health impacts of air pollution in the form of premature deaths, at the census tract level in the city of Houston, Texas. We investigated the role of transportation and socioeconomic status in air pollution burden of disease. In result, 7.3% and 1.8% of all-cause deaths in Houston were attributable to PM$_{2.5}$ and NO$_2$ in 2010. We showed that about 1% of the premature deaths attributable to air pollutants can be prevented by complying with the WHO air quality guideline value for PM$_{2.5}$ concentrations. Deaths attributable to air pollution were higher in the city’s CBD, and in a census tract with a higher level of exposure to road traffic, especially in the case of NO$_2$ attributable deaths. Also, we found that premature deaths attributable to air pollution were higher in households with lower income. The findings of this study underline the importance of assessing the health burden of air pollution and its spatial distribution across cities to support sustainable development.


**Funding:** This research was made possible by funding from the Texas A&M Transportation Institute’s (TTI) Center for Advancing Research in Transportation Emissions, Energy, and Health (CARTEEH), a U.S. Department of Transportation’s University Transportation Center with the grant number (69A3551747128).

**Acknowledgments:** We would like to thank Matthew Bechle and Professor Julian Marshall at the University of Washington for providing the 2010 air pollution exposure estimates.

**Conflicts of Interest:** The authors declare no conflict of interest. The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, or in the decision to publish the results.
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