Letter

All-Cause NO$_2$-Attributable Mortality Burden and Associated Racial and Ethnic Disparities in the United States

Sara F Camilleri,* Gaige Hunter Kerr, Susan C Anenberg, and Daniel E Horton

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ABSTRACT: Nitrogen dioxide (NO$_2$) is a regulated pollutant that is associated with numerous health impacts. Recent advances in epidemiology indicate high confidence linking NO$_2$ exposure with increased mortality, an association that recent studies suggest persists even at concentrations below regulatory thresholds. While large disparities in NO$_2$ exposure among population subgroups have been reported, U.S. NO$_2$-attributable mortality rates and their disparities remain unquantified. Here we provide the first estimate of NO$_2$-attributable all-cause mortality across the contiguous U.S. (CONUS) at the census tract-level. We leverage fine-scale, satellite-informed, land use regression model NO$_2$ concentrations and census tract-level baseline mortality data to characterize the associated disparities among different racial/ethnic subgroups. Across CONUS, we estimate that the NO$_2$-attributable all-cause mortality is ~170,850 (95% confidence interval: 43,970, 251,330) premature deaths yr^{-1} with large variability across census tracts and within individual cities. Additionally, we find that higher NO$_2$ concentrations and underlying susceptibilities for predominately Black communities lead to NO$_2$-attributable mortality rates that are ~47% higher compared to CONUS-wide average rates. Our results highlight the substantial U.S. NO$_2$ mortality burden, particularly in marginalized communities, and motivate adoption of more stringent standards to protect public health.

KEYWORDS: *Air pollution, NO$_2$ mortality, environmental justice, inequity, health impacts*
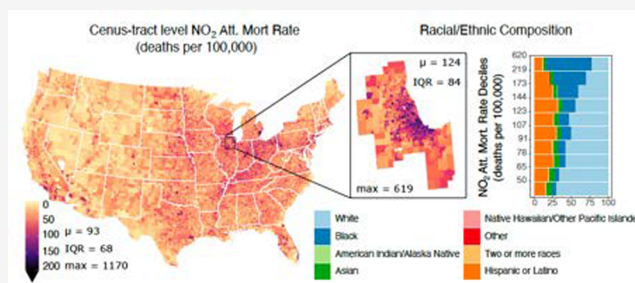
INTRODUCTION

Nitrogen dioxide (NO$_2$) is a health-harming gaseous pollutant that can lead to premature death.[1] In the U.S., vehicle emissions are the largest single contributor to ambient NO$_2$ concentrations.[2] Indeed, NO$_2$ concentrations are often used as a surrogate for the complex vehicle tailpipe emission mixture (e.g., HEI, 2022[1]). NO$_2$ is also a precursor of other health-damaging pollutants such as ozone (O$_3$) and contributes to fine particulate matter (PM$_{2.5}$).[3] Through successful air pollution regulations, NO$_2$ concentrations have declined,[4,5] but adverse health outcomes associated with NO$_2$ exposure persist for all air pollution levels, even those below regulatory limits[6−8] and typically impact minoritized populations the most.[9,10]

Assessing mortality risks attributable to NO$_2$ is an active and rapidly evolving field of research. Findings from the 2016 Integrated Science Assessment,[2] the scientific foundation for the current U.S. EPA National Ambient Air Quality Standards (NAAQS), found suggestive evidence linking long-term NO$_2$ exposure to all-cause mortality. New epidemiological evidence[11] confirms these impacts with moderate confidence which consequently led to the updated and more stringent World Health Organization NO$_2$ Air Quality Guidelines (AQGs).[12] Subsequently, higher confidence in these associations have been reported by the Health Effects Institute.[1]

However, the causality of these impacts is still uncertain. Given that NO$_2$ and other traffic-related air pollutants are often highly correlated in both space and time, it remains unclear whether NO$_2$ is a causal agent or simply an indicator of the complex traffic-related air pollution mixture.[1] Given its short atmospheric lifetime (~hours), NO$_2$ can exhibit tight spatial gradients with high concentrations close to emission sources such as heavily trafficked roads but gradually declining concentrations that fall to background levels at distances of ~700 m.[13] With more than 45 million people living within 300 feet of major roadways, of which a vast majority are people of color (POC),[14] NO$_2$ exposure does not impact all population subgroups equally. Indeed, despite nation-wide decreases in NO$_2$ concentrations in the past decade,[4] disparities in NO$_2$ exposure persist, with racial/ethnic population subgroups still carrying a disproportionate burden.[9,15]

Several studies have estimated the U.S. mortality burden associated with PM$_{2.5}$ and O$_3$; however, the U.S.-wide NO$_2$-
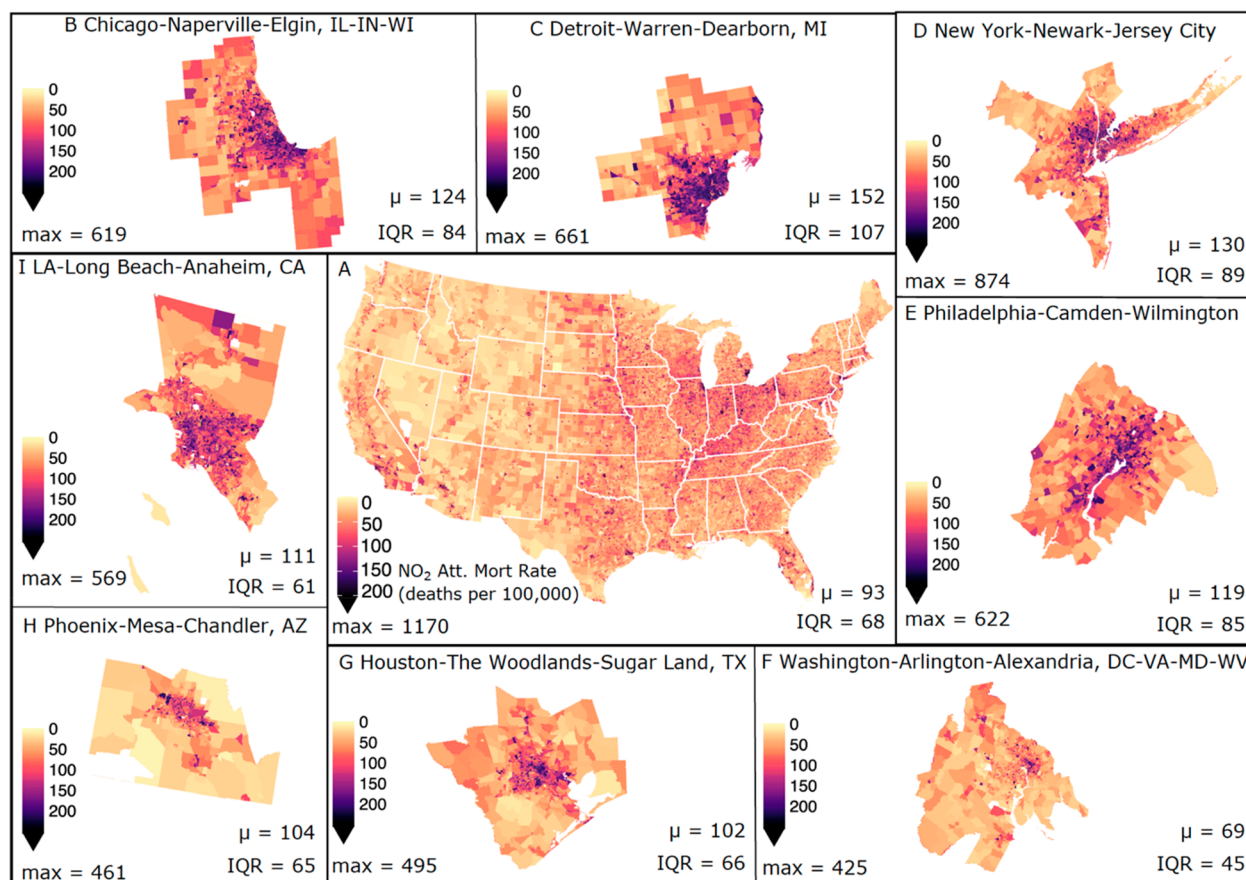
Figure 1. NO₂-attributable mortality rates. Spatial distribution of the estimated NO$_2$-attributable mortality rates expressed in deaths per 100,000 per year across (A) the Contiguous U.S. (CONUS), and a selection of the top 15 most populous MSAs: (B) Chicago-Naperville-Elgin, IL-IN-WI, (C) Detroit-Warren-Dearborn, MI, (D) New York-Newark-Jersey City, NY-NJ-PA, (E) Philadelphia-Camden-Wilmington, PA-NJ-DE-MD, (F) Washington-Arlington-Alexandria, DC-VA-MD-WV, (G) Houston-The Woodlands-Sugar Land, TX, (H) Phoenix-Mesa-Chandler, AZ, and (I) Los Angeles-Long Beach-Anaheim, CA, with MSA-averaged NO$_2$-attributable mortality rates (μ; deaths per 100,000 per year), IQRs, and maximum NO$_2$-attributable mortality rates (max; deaths per 100,000 per year), annotated at the bottom of each panel.

attributable mortality burden has not yet been assessed. Given recent epidemiological findings of increased confidence in the links between NO$_2$ and mortality risks, there is a need to understand these impacts on a national scale. In a global study, Song et al.[16] recently estimated the U.S. NO$_2$-attributable mortality burden but only for highly urbanized sites. Previous works have shown large disparities in NO$_2$ exposure,[4,9,10,15] but none have looked at the associated disparities in NO$_2$ mortality burdens. Disparities in NO$_2$-attributable pediatric asthma burdens have been quantified; however, such studies are limited by the resolution of available baseline asthma rates which are typically only available at coarser-than-tract resolutions.[4] In this work, we fill these research gaps by estimating contiguous U.S. (CONUS)-wide NO$_2$-attributable health impacts at the census tract-level, enabling us to determine neighborhood-scale equity implications. To provide this health-critical estimate, we leverage high-resolution modeled NO$_2$ concentrations (~1 km), the latest evidence linking long-term NO$_2$ exposure to mortality and tract-level mortality rates.

■ METHODS AND MATERIALS

NO₂ Concentrations. Surface NO$_2$ concentrations at ~1 km horizontal resolution are derived from global data sets that incorporate a land use regression model (LURM) and satellite

data.[5] The underlying LURM represents mean 2010−2012 NO$_2$ concentrations (Larkin et al.[17]), which were scaled to subsequent years using NO$_2$ column densities from satellite data sets.[5] The annual normalized mean bias for this data set, against in situ observations in 2015, was 0.14 ppbv with a correlation coefficient of 0.77 (additional comparisons in Kerr et al.[4]). We use concentration data from 2015, averaged to underlying tracts and consistent with available all-cause mortality baseline mortality rates. Tract boundaries are obtained from the U.S. Census Bureau and correspond to the 2010 decadal census.[18] This highly spatially resolved NO$_2$ data set is appropriate given the heterogeneity of NO$_2$, particularly in urban areas, i.e., areas that are often burdened by high NO$_2$ concentrations, and allows us to determine the NO$_2$-related mortality burden and associated disparities at equity-relevant neighborhood scales which would otherwise be underestimated at coarser resolutions.[19]

Population and Demographic Data. We use census tract age-stratified population data and demographic information from the American Community Survey (ACS).[20] Specifically, we obtain 5-year estimates from 2015 to 2019 which incorporate a larger sample size, thus reducing the margin of error compared to data sets covering shorter time periods. For each tract, the population data represent each 5-yr age group ranging from 30 to 85+ years. Using the racial/
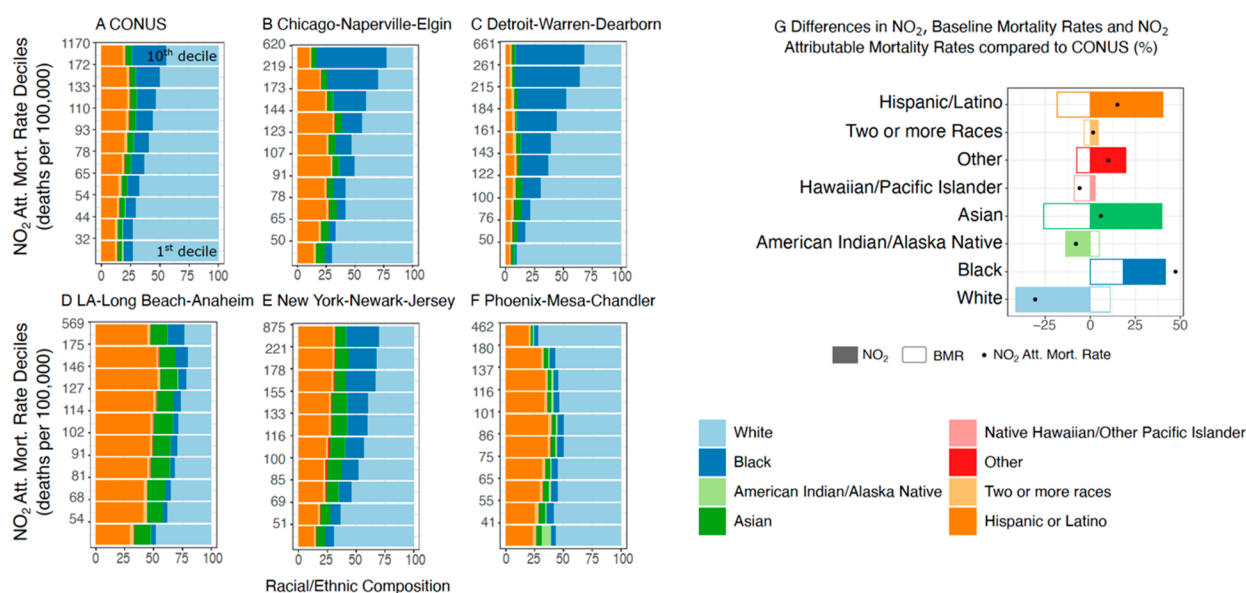
Figure 2. Racial and Ethnic Disparities. The racial and ethnic composition (in %) of the population for deciles of NO_2-attributable mortality rates for (A) the contiguous U.S. and (B−F) across selected MSAs (in deaths per 100,000 per year). (G) Comparison of NO_2 concentrations, Baseline Mortality Rates (BMRs), and estimated NO_2-attributable mortality rates for the upper 10th percentile of each population subgroup relative to CONUS-wide averages (in %). All race groups, except Hispanic or Latino, include only those identifying as non-Hispanic.

ethnic characterizations from the ACS demographic data, population subgroups include non-Hispanic: White, Black, American Indian or Alaska Native, Asian, Native Hawaiian/Other Pacific Islander, people identifying with another race or with two or more races, and the Hispanic/Latino subgroup. We conduct our analysis at the census tract-level for CONUS but also present estimates for a selection of the top 15 most populous Metropolitan Statistical Areas (MSAs) using the U.S. TIGER/Line spatial boundaries from the U.S. Census Bureau.[21]

Health and Equity Impacts. To estimate the NO_2-attributable mortality for each census tract within the U.S. we use an epidemiologically derived log−linear concentration−response function consistent with previous literature and described in eq 1[5]

$$\text{Mort}_{CT} = \text{BMR}_{CT} \times \text{POP}_{CT} \times (1 - \exp^{(-\beta x_{CT})}) \quad (1)$$

where Mort_{CT} represents the estimated attributable mortality at the census tract level expressed as a function of the tract baseline all-cause mortality rate (BMR_{CT}) and the population (POP_{CT}) for each 5-year age group together with the bracketed term representing the Attributable Fraction (AF); the fraction of the underlying all-cause mortality attributable to NO_2 with β representing the concentration−response coefficient and x_{CT} the NO_2 concentrations.

Census tract baseline mortality incidence rates for each 5-year age group were obtained from Industrial Economic (IEc 2010−2015) and derived from USALEEP abridged life tables with modifications for broader use in national health benefits analyses.[22] We derived β from a relative risk (RR) of 1.04 (95% confidence interval (CI) 1.01−1.06)[1] per 10 μg m^{-3} from the recent Health Effect Institute (HEI) systematic review and meta-analysis[1] converted to ppb equivalent (1 ppb NO_2 = 1.88 μg m^{-3}) in line with previous studies.[23] Evidence for NO_2-related health impact thresholds is limited; therefore, here we use the full NO_2 concentration range. Presented health

impacts (Figure 1) hold for ages 30+ with uncertainties reflecting the RR 95% CI.

To assess the tract-level NO_2-attributable mortality disparities across the U.S., we analyze the racial/ethnic composition within each NO_2-attributable mortality decile (Figure 2A−F) and compare BMRs, NO_2 concentrations, and NO_2-attributable mortality rates for the 10th decile (>90th percentile) of each racial/ethnic population subgroup to CONUS-level average rates (Figure 2G).

■ RESULTS AND DISCUSSION

Health Impacts. Across CONUS we estimate that NO_2 concentrations are linked to ~170,850 (CI: 43,970, 251,330) premature deaths yr^{-1} or ~90 deaths per 100,000 people yr^{-1} (henceforth "deaths"; Figure 1A). Reflective of large spatial heterogeneity in NO_2 concentrations across CONUS, we note higher NO_2-attributable mortality rates for the Midwest and Northeast with a maximum burden of 1170 deaths found within Monroe County, NY (Rochester, NY MSA). Averaging NO_2-attributable mortality rates across each of the 380 MSAs within CONUS, we find the highest rate of 152 deaths in the Detroit-Warren-Dearborn MSA (Figure 1C) and lowest (28 deaths) in the Hinesville, GA MSA. For the Detroit MSA, averaged NO_2-attributable mortality rates are 1.6 times the CONUS average, largely driven by NO_2 concentrations 1.4 times the CONUS levels. A similar pattern is found in the Chicago MSA where rates are 1.3 times the CONUS mean due to elevated NO_2. The Youngstown-Warren-Boardman, PA MSA also has higher than average mortality rates (1.6 times); however, it has NO_2 concentrations similar to the CONUS average, but with an average BMR 1.4 times CONUS levels. To isolate the singular impact of NO_2 exposure on estimated mortality burdens, we estimate the AF (eq 1) and find that across CONUS ~7% of the underlying baseline mortality is attributable to NO_2, with a maximum of ~21% in New York City. We note that NO_2-attributable mortality rates within urban cores are even higher than MSA averaged rates, as NO_2

concentrations increase within heavily trafficked urban areas. For example, the NO_2 mortality rate for the City of Chicago is 1.7 times the CONUS MSA mean level as NO_2 concentrations within the city are almost double.

Within MSAs, we note large tract-level variability in the NO_2-related mortality rates. For example, rates for tracts within the Detroit and New York-Newark-Jersey City MSAs reach a maximum of ~660 and ~870 deaths with an interquartile range (IQR) of 107 and 89 deaths, respectively. The large variability in estimated NO_2-related mortality rates among tracts within an MSA reflects the wide range in both NO_2 concentrations (1.8−22.8 ppb) and BMRs (180−5560 deaths per 100,000), highlighting the importance of spatially resolved air pollution and health data and the potential for estimates conducted at coarser resolutions to underestimate the true health impact.

Disparities in NO_2-Attributable Mortality. Across CONUS, NO_2-related mortality burdens disproportionately impact communities of color. Specifically, tracts with the largest estimated NO_2-attributable mortality rates (10th decile) are 29% Black, 5% Asian, 18% Hispanic or Latino, and 45% White, while the racial/ethnic makeup of the total population is 12% Black, 5% Asian, 18% Hispanic or Latino, and 61% White (Figure 2A).

For individual MSAs, the burden on communities of color is amplified. For example, in the Chicago and Detroit MSAs ~60% of the population in tracts with the highest NO_2-related mortality rates is Black (Figure 2B,C), a subgroup that comprises less than ~21% of the total population for these MSAs. Similarly, in the New York MSA, the Black and Hispanic/Latino subgroups represent 16% and 24% of the total MSA population; however, each represents ~30% of the population where high NO_2-attributable mortality rates occur (Figure 2E). Reflecting a higher portion of Hispanic/Latino communities in the Los Angeles-Long Beach-Anaheim, MSA (45%), this subgroup constitutes the majority of NO_2-attributable mortality deciles (>41%), except the first decile, which is 48% White. In the Phoenix-Mesa-Chandler MSA, the White population comprises the majority of all NO_2-attributable mortality deciles (>50%, Figure 2F), reflecting a predominately White MSA (77%).

Estimated NO_2 health impacts do not depend solely on pollutant concentrations but also on underlying susceptibilities of exposed populations.[24] In Figure 2G we assess the two driving factors that determine NO_2-attributable mortality rates for the top 10% of each population subgroup relative to CONUS-wide averages: (1) NO_2 concentrations and (2) BMRs. NO_2-attributable mortality rates in predominately White or American Indian/Alaska Native tracts are lower than CONUS average rates (−31% and −8%; Figure 2G), largely driven by lower NO_2 concentrations. Native Hawaiian/ Other Pacific Islanders also experience lower NO_2 mortality burdens compared to CONUS average rates (−6%); however, lower rates are driven by lower BMRs. Conversely, the NO_2 mortality burden is 6% and 15% higher than average in communities that are predominately Asian or Hispanic/Latino stemming from higher NO_2 exposure and lower BMRs. Tracts within which populations are predominately Black have both higher NO_2 exposures as well as higher underlying susceptibilities, leading to disproportionately large NO_2-attributable mortality rates (+47%) compared to other subgroups.

■ DISCUSSION

In this work, we present a tract-level CONUS-wide estimate of NO_2-attributable mortality and identify large variability in NO_2 mortality burdens across tracts. Additionally, we show disparities in NO_2-attributable mortality rates across CONUS and highlight the disproportionate burden on communities of color, driven by higher-than-average NO_2 exposure and higher underlying susceptibilities.

Our CONUS-wide estimate of approximately 171,000 annual premature deaths from NO_2 exposure for ages 30+ is broadly consistent with a recent estimate by Song et al.[16] who estimate premature NO_2 mortality in 13,169 urban areas globally. For U.S. urban centers, Song et al. estimate ~38,000 premature deaths per year. However, our estimate is CONUS-wide and uses a different RR with no concentration threshold together with tract-level all-cause mortality rates, as opposed to national-level;[25] as such, a direct comparison is not possible. Evidence suggesting an increase in mortality risks associated with long-term exposure to NO_2, even at low concentration levels, is continuously emerging;[6−8] thus, here we opt to quantify the NO_2-related premature mortality impacts at all exposure levels. We note that the NO_2 exposures used to determine the RR used here[1] are generally well-below the EPA's annual NAAQS of 53 ppb, suggesting large public health risks persist, even at levels below regulatory limits. While all CONUS tracts exhibit NO_2 concentrations below the NAAQS, ~77% exceed the more stringent World Health Organization (WHO) AQG of 10 μg m^{-3}. We estimate that if NO_2 concentrations are reduced to the WHO AQG in all tracts exceeding it, ~42% of NO_2-attributable CONUS deaths would be prevented (~71,980 avoided deaths) but disparities within individual MSAs would persist (Figure S1).

The pollution-attributable morality burden for the U.S. is more commonly estimated for $PM_{2.5}$,[4,26−29] as links between long-term exposure to $PM_{2.5}$ and adverse health outcomes are long-established. Estimates for U.S.-wide $PM_{2.5}$-related mortality burdens range from ~50,000 to ~131,000 premature deaths[4,26−29] depending on the year and methodology of the study. Our NO_2 mortality estimate is higher compared to existing $PM_{2.5}$-related literature; however, a direct comparison is nontrivial. Discrepancies in estimates can be linked to use of coarser health data which can underestimate health impacts by around 15%,[30] choice of RR, and the use of thresholds below which no health impacts are incurred. Transportation is one of the largest contributors to NO_2, which leads to peak concentrations along highways and major road networks with levels exceeding the WHO AQGs within cities and urban areas.[5] A large portion of people living close to these hotspots are POC, who also generally experience higher-than-average susceptibilities. We therefore hypothesize that steeper NO_2 concentration gradients within highly susceptible urban populations is also a likely driver of our higher NO_2 mortality estimate.[4] Additionally, the relationship between NO_2 and all-cause mortality is unadjusted for confounding pollutants such as $PM_{2.5}$, and therefore our estimated NO_2 mortality burden is not independent of $PM_{2.5}$ influences. LURM-derived NO_2 concentrations at ~1 km scales also have uncertainties; e.g., NO_2 estimates are based on empirical relationships rather than physics-backed models and rely on roadway information with no data on transportation sources (e.g., vehicle types or traffic counts[17]) which can influence NO_2 spatial variability.[30]

Our finding highlighting the unjust NO_2 mortality burden on POC complements previous exposure-focused studies suggesting higher NO_2 and $PM_{2.5}$ concentrations for marginalized communities.[9,15,31−33] Studies looking at the associated health disparities are scarce, especially for NO_2. A recent U.S. study found that in the 2010s, disparities in NO_2-attributable pediatric asthma increased despite decreases in overall pollutant concentrations.[4] The disparities in NO_2-related mortality presented in this work are estimated using the same RR for all population subgroups and do not account for varying RR values across different races. For $PM_{2.5}$, Spiller et al.[29] show that not using race-specific RR could underestimate $PM_{2.5}$ health impacts especially among older Black Americans and therefore lead to lower estimated disparities in health outcomes. For NO_2 mortality, race-specific RRs have been quantified;[34] however, these were restricted to ages 65+. Despite the lack of 30+ race-specific RRs, our conclusions using tract-level baseline mortality data are similar to those of Spiller et al.[29] for $PM_{2.5}$; i.e., we find higher attributable mortality rates for minoritized populations, especially Black populations. Nonetheless, it is unclear how the NO_2 race-specific RRs for ages 30+ would influence our results.

While several studies have confirmed the unjust burden of NO_2 exposure on marginalized communities,[4,9,10,15] none have quantified the CONUS all-cause NO_2-attributable mortality burden and associated disparities. Our use of census tract pollutant and health data enables us to capture wider and more representative variability in underlying population subgroup susceptibilities and associated disparities in NO_2 mortality burdens, a result which would otherwise be underrepresented if using county-, state-, or national-level data.[4] Our results indicate that policies aimed at reducing NO_x emissions such as those pertaining to the transportation sector[35,36] could reduce long-standing environmental injustices and motivate more stringent air quality standards.

ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge at https://pubs.acs.org/doi/10.1021/acs.estlett.3c00500.

Disparities in NO_2-attributable mortality rates following reductions in NO_2 concentrations to the WHO Air Quality Guidelines (PDF)

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Notes

The authors declare no competing financial interest.

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