Increasing disparities in air pollution health burdens in the United States

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Abstract

Ambient nitrogen dioxide (NO2) and fine particulate matter (PM2.5) pollution threaten public health in the United States (U.S.), and systemic racism has led to modern-day disparities in the distribution and associated health impacts of these pollutants. Many studies on environmental injustices related to ambient air pollution focus only on disparities in pollutant concentrations or provide only an assessment of pollution or health disparities at a snapshot in time. In this study we aim to document changing disparities in pollution-attributable health burdens over time and, for the first time, disparities in NO2-attributable health impacts across the entire U.S. We show that, despite overall decreases in the public health damages associated with NO2 and PM2.5, ethnoracial relative disparities in NO2-attributable pediatric asthma and PM2.5-attributable premature mortality in the U.S. have widened during the last decade. Racial disparities in PM2.5 attributable premature mortality and NO2-attributable pediatric asthma have increased by 19% and 16%, respectively, between 2010 and 2019. Similarly, ethnic disparities in PM2.5-attributable premature mortality have increased by 40% and NO2-attributable pediatric asthma by 10%. These widening trends in air pollution disparities are reversed when more stringent air quality standard levels are met for both pollutants. Our methods provide a semi-observational approach to tracking changes in disparities in air pollution and associated health burdens across the U.S.

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Abstract

Ambient nitrogen dioxide (NO_2) and fine particulate matter ($PM_{2.5}$) pollution threaten public health in the United States (U.S.), and systemic racism has led to modern-day disparities in the distribution and associated health impacts of these pollutants. Many studies on environmental injustices related to ambient air pollution focus only on disparities in pollutant concentrations or provide only an assessment of pollution or health disparities at a snapshot in time. In this study we aim to document changing disparities in pollution-attributable health burdens over time and, for the first time, disparities in NO_2-attributable health impacts across the entire U.S. We show that, despite overall decreases in the public health damages associated with NO_2 and $PM_{2.5}$, ethnoracial relative disparities in NO_2-attributable pediatric asthma and $PM_{2.5}$-attributable premature mortality in the U.S. have widened during the last decade. Racial disparities in $PM_{2.5}$-attributable premature mortality and NO_2-attributable pediatric asthma have increased by 19% and 16%, respectively, between 2010 and 2019. Similarly, ethnic disparities in $PM_{2.5}$-attributable premature mortality have increased by 40% and NO_2-attributable pediatric asthma by 10%. These widening trends in air pollution disparities are reversed when more stringent air quality standard levels are met for both pollutants. Our methods provide a semi-observational approach to tracking changes in disparities in air pollution and associated health burdens across the U.S.

1. Introduction

Ambient nitrogen dioxide (NO_2), a marker for the complex mixture of traffic-related pollution, and fine particulate matter ($PM_{2.5}$) pose pernicious threats to public health[1]. Exposure to $PM_{2.5}$ has a well-established association with premature death due to several specific causes[2–4], and recent studies have found moderate to high confidence linking NO_2 with new-onset pediatric asthma [5–9]. While levels of these pollutants have decreased in the United States (U.S.) following the passage of the Clean Air Act, its 1990 Amendments, and other regional measures[10], $PM_{2.5}$ and NO_2 continue to impact public health and lead to loss of human life. The economic value of these health effects is very high, with the value of statistical life estimated at nearly $10 million per statistical death in 2019 USD[11–13]. Systemic racism embedded within the fabric of urban planning and land use in the U.S. has led to modern-day disparities in exposure to these pollutants and their associated health impacts.

While studies consistently show that racialized and minoritized communities face higher levels of NO_2 and $PM_{2.5}$, recent work has led to different conclusions regarding whether relative $PM_{2.5}$ exposure disparities are narrowing, remaining constant, or widening[14–16]. Many previous studies have only focused on disparities in pollutant exposure, leaving a gap in understanding disparities in pollution-attributable health impacts. Despite the association of NO_2 with one of the most inequitably distributed diseases, pediatric asthma, no study has examined disparities in NO_2-attributable pediatric asthma across the entire U.S. and their changes over time. Thus, there is a need to understand the public health burdens associated with ambient $PM_{2.5}$ and NO_2 across the U.S. and track associated disparities with time, especially as commitments to address

environmental justice require concerted efforts to identify and map areas burdened by injustices and inequities[17].

Here we conduct a comprehensive assessment of disparities in public health burdens due to NO_2 and $PM_{2.5}$ across the fifty U.S. states, Washington, D.C., and Puerto Rico. Recently developed datasets, which fuse satellite data with physical models, enable us to resolve neighborhood-level differences in NO_2 and $PM_{2.5}$ and thereafter assess inequities in the health burdens from these pollutants using up-to-date demographic data and the latest epidemiological evidence linking exposure with health outcomes. The main contributions of our work are threefold. First, we compare and contrast injustices in NO_2- and $PM_{2.5}$-related health burdens. Second, we track how ethnoracial disparities in the health impacts attributable to these pollutants have changed over the last decade, a period of declining emissions from multiple polluting source sectors. Finally, we explore the degree to which more stringent NO_2 and $PM_{2.5}$ ambient air quality standards could reduce inequitable pollution-related health burdens for the most racialized and minoritized communities in the U.S.

2. Methods

Population and demographic data

The U.S. Census Bureau's American Community Survey (ACS) provides estimates of the population, age structure, and demographics within census tracts in the U.S.[18]. We used ACS 5-year estimates for the ~74000 census tracts in the fifty U.S. states, the District of Columbia, and Puerto Rico. Five-year estimates have a larger sample size and smaller margin of error than other ACS estimates with shorter timeframes. ACS' first 5-year estimates, based on data collected from 2005 to 2009, were released in 2010. Our analysis thus spans 2010 through 2019, and we updated the demographic data annually in our study. Tract-level ACS estimates from 2010-2019 correspond to tract boundaries from the 2010 decadal census, obtained from the U.S. Census Bureau's TIGER/Line geodatabase[19].

Pollutant concentrations

Surface-level NO_2 and $PM_{2.5}$ concentrations were derived from two existing global datasets that combine physical models with satellite retrievals to produce high-fidelity 0.01˚ x 0.01˚ (~1 km x 1 km) estimates of these deleterious pollutants[20,21]. We used annual average concentrations from 2010-2019, consistent with the years for which demographic data are available.

The 0.01˚ x 0.01˚ NO_2 dataset uses a land-use regression model from Larkin et al.[22] representing 2010-2012 concentrations and scales these concentrations to prior and subsequent years using NO_2 column densities from NASA's Ozone Monitoring Instrument satellite[21]. The 0.01˚ x 0.01˚ $PM_{2.5}$ dataset (V5.GL.02) fuses aerosol optical depth retrievals from several satellites with GEOS-Chem chemical transport model output[23] and thereafter calibrates estimates to ground-based $PM_{2.5}$ observations using Geographically Weighted Regression[24]. Text S1 further describes advantages to using these spatially complete datasets and details their performance compared with *in-situ* monitors.

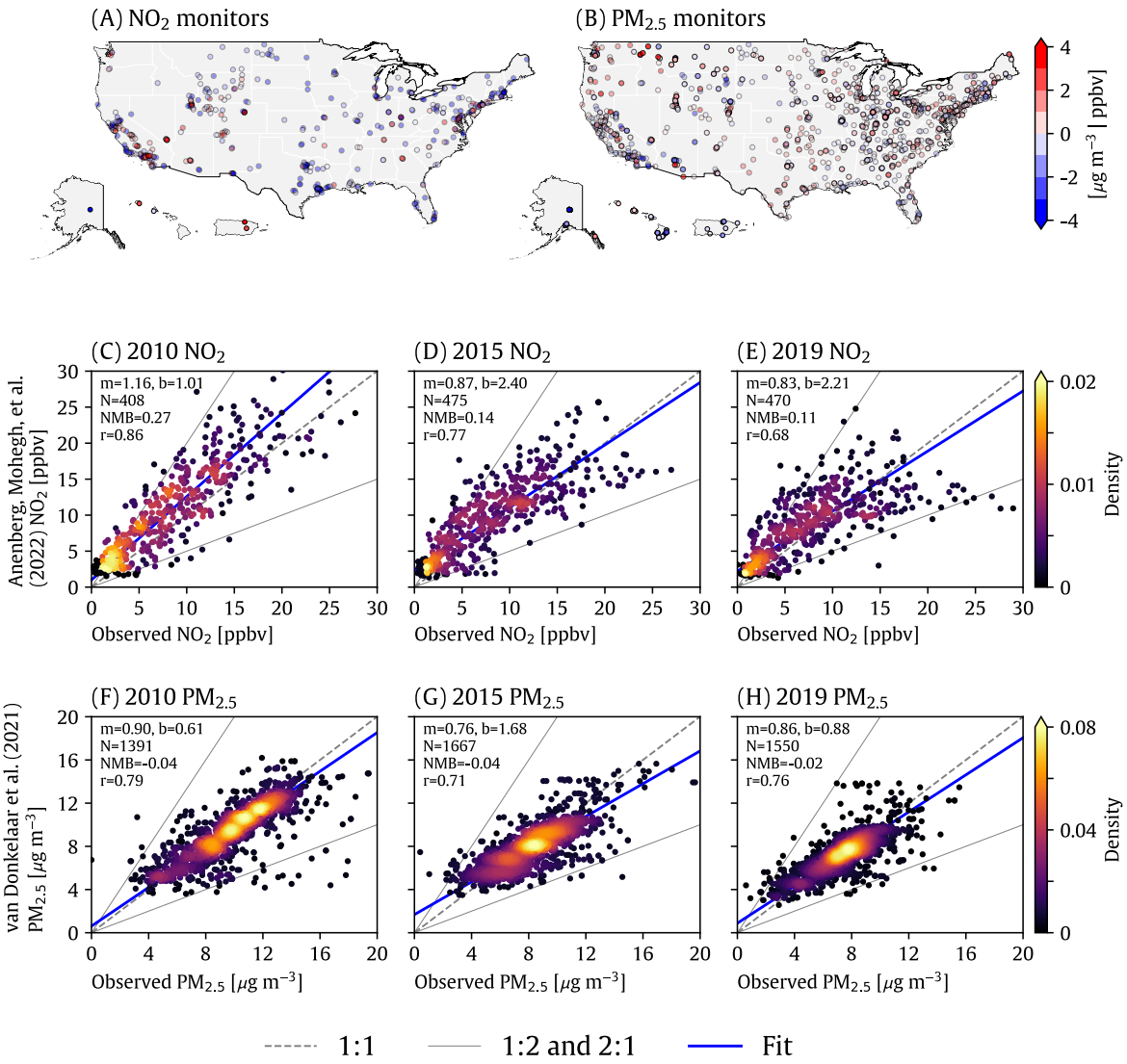

Figure 1. *Location of in-situ (A) NO_2 and (B) $PM_{2.5}$ monitors. Monitor locations represent the AQS network during 2019. Scatterplots are colored by density and show a comparison of (C)-(E) NO_2 and (F)-(H) $PM_{2.5}$ datasets against observations for 2010, 2015, and 2019. Dataset values represent census tract averages in the tract coincident with the AQS monitor. The reduced major axis linear regression is denoted by the blue lines in (C)-(E). Inset text in the scatterplots indicates the slope (m) and intercept (b) of the regression, the number of in-situ monitors (N), the normalized mean bias (NMB), and correlation coefficient (r). Monitors in (A)-(B) are colored by the difference between the observed and dataset values (< 0 corresponds to an overestimate by the datasets).*

Risk and rates

In this study we used cause-specific RR curves from the Global Burden of Disease (GBD) 2020 and mortality and incidence rates from GBD 2019. The GBD is an ongoing multinational research collaboration that assesses morbidity and premature mortality from a number of risk factors, including ambient air pollution. GBD estimates are updated annually, and recent GBD

releases have included several methodological updates that improved upon earlier estimates[25]. Rates from GBD 2020 were not yet available at the completion of this study.

RR curves measuring the association of long-term $PM_{2.5}$ exposure with premature death and NO_2 exposure with new cases of pediatric asthma were estimated from systematic reviews and meta-regression based on a Bayesian, Regularized, Trimmed approach (Figure S1)[25]. RR curves for NO_2-attributable pediatric asthma are applied to the population aged 0 to 18. We included $PM_{2.5}$-attributable premature mortality for six different endpoints in our study: chronic obstructive pulmonary disease (hereafter "COPD"), ischemic heart disease; ischemic and intracerebral hemorrhagic stroke ("stroke"); lung, tracheal, and bronchial cancer ("lung cancer"); lower respiratory infection; and type 2 diabetes. RR curves for lower respiratory infection were applied to the entire population, while the other premature mortality endpoints were applied to the population aged 25 years and older.

The uncertainty interval for the RR estimates of pediatric asthma conferred by NO_2 exposure spans 1 due to between-study heterogeneity unexplained by study design (Figure S1G). Despite the uncertainty interval spanning 1, the association was deemed strong enough for inclusion in the GBD, and the mean relationship indicates increasing risk of new-onset pediatric asthma with NO_2[26]. Additionally, a recent report from the Health Effects Institute (HEI) classified the association of new-onset pediatric asthma with traffic-related air pollution as having medium to high confidence, and the Health Effects Institute's NO_2-pediatric asthma RR estimate had an uncertainty interval that only marginally spanned 1[9]. We chose not to use statistical significance as the sole determining factor for inclusion in our study because this reliance for the convenience of statistical properties may neglect historically-excluded groups[9]. By using only the health endpoints included in the GBD 2020 Study, we are being conservative; the HEI traffic-related air pollution also found that children's acute lower respiratory infection, adult-onset asthma, and mortality (all cause, circulatory, and ischemic heart disease) all had significant associations with NO_2 with overall high confidence.

The theoretical minimum risk exposure levels (TMREL) for $PM_{2.5}$ and NO_2, the level below which we assume no increased risk of $PM_{2.5}$-attributable premature mortality or NO_2-attributable pediatric asthma, is modeled by the GBD as uniform distributions bounded by the minimum and fifth percentiles of exposure distributions from ambient air pollution cohort studies with the lowest study-specific exposure distributions[25]. We treated the midpoints of these distributions (i.e., 4.15 $\mu g\ m^{-3}$ for $PM_{2.5}$, 5.37 ppbv for NO_2) as our TMRELs.

We obtained death rates per 100,000 population for COPD, ischemic heart disease, stroke, lung cancer, lower respiratory infection, and type 2 diabetes and incidence rates per 100,000 pediatric population for asthma from the GBD 2019 study for each year and state in our analysis (Figure S2). For each endpoint, the rates vary by 5-year age groups (e.g., <5, 5-9, 10-14, etc.). Death and incidence rates are generally higher in the Southeastern and Eastern U.S. for most endpoints, while some endpoints such as COPD have less consistent spatial heterogeneity and substantially vary, even among bordering states (Figure S2).

Methods

To facilitate comparison of pollutant concentrations with the populations they impact, we averaged the NO_2 and $PM_{2.5}$ datasets to underlying census tracts in the U.S. (Figure S3). The median area of all (urban) census tracts is 5.2 km^2 (3.7 km^2) and supports this averaging approach. There are, however, 5.2% of tracts too small in area to contain coincident grid cells. Following Kerr et al.[27] we used inverse distance weighting to interpolate pollutant concentrations to the centroid of these small tracts. We found good agreement between tract-averaged NO_2 or $PM_{2.5}$ and *in-situ* observations from the Environmental Protection Agency's (EPA) Air Quality System (AQS) network of administrative and regulatory (not low-cost) monitors, supporting their ability to capture spatiotemporal pollution variability across the U.S. (Text S1, Figure 1).

We conducted several scenarios where NO_2 and $PM_{2.5}$ reach target concentrations to assess how meeting these targets will reduce the associated health burdens and potentially advance environmental justice. Targets represent the National Ambient Air Quality Standards (NAAQS) established by the EPA and the World Health Organization (WHO) interim targets (ITs) and air quality guidelines (AQGs), updated in 2021[28]. If NO_2 or $PM_{2.5}$ concentrations in a particular tract were larger than a target level, we assigned the concentration to the target value.

We calculated the population attributable fraction (PAF), that is, the fraction of the burden of disease that might be attributable to $PM_{2.5}$ or NO_2 exposure, for our endpoints of interest. For a given pollutant concentration X in tract t, the PAF was calculated as

$$PAF(X_t) = \begin{cases} \frac{RR(X_t)-1}{RR(X_t)} - \frac{RR(TMREL)-1}{RR(TMREL)}, \text{for } X_t \geq TMREL \\ 0, \text{for } X_t < TMREL. \end{cases} \quad [1]$$

The PAF was then used to calculate the total NO_2-attributable pediatric asthma burden or $PM_{2.5}$-attributable premature mortality burden in each tract as

$$Burden_t = pop_t \times PAF_t \times k_s. \quad [2]$$

Here, *pop* corresponds to the susceptible population in each tract *t*; *k* to baseline incidence and deaths rates from the GBD; and *s* to state, the highest level of availability granularity from the GBD. We present both cause-specific $PM_{2.5}$-attributable premature deaths from these six endpoints and their sum in our analysis.

Uncertainty in pollution-attributable health burdens was primarily characterized using the 95% uncertainty interval of RR estimates. Other terms in the health impact function (Equation 2) also have associated uncertainty. Achakulwisut et al.[13] investigated the uncertainty in underlying disease incidence rates, finding this source of uncertainty to be the least influential in estimating health burdens. Given the form of Equation 2, we expect any uncertainties in death and incidence rates would linearly scale our results and likely not substantially affect relative differences across demographic groups or overall trends. Although the satellite data and physical models used to estimate NO_2 and $PM_{2.5}$ contain appreciable uncertainties, our comparison of these datasets against *in-situ* observations highlights their fidelity (Text S1, Figure 1).

We assessed $PM_{2.5}$, NO_2, and the associated health burdens in individual census tracts but aggregate our results to a national level and individual metropolitan statistical areas (MSAs). MSAs have at least one urbanized area of 50,000 or more residents[29]. A majority of the U.S. population (89%) lived in one of the 389 MSAs in 2019. We refer to MSAs by their colloquial names (e.g., Los Angeles-Long Beach-Anaheim, CA MSA = Los Angeles).

The U.S. Census Bureau treats race and ethnicity as separate, distinct identities. In addition to respondents' race(s), respondents self-identify as "Hispanic or Latino" or "Not Hispanic or Latino." Following this distinction, we characterized environmental injustices stemming from $PM_{2.5}$, NO_2, and the associated health burdens for both racial and ethnic groups using two complementary methods:

(1) **Top and bottom deciles of population subgroups**. Census tracts were designated as the "most white" and "least white" or "most Hispanic" and "least Hispanic" using the top and bottom 10 percentile (decile) of the white or Hispanic population distribution. This approach allows us to understand pollution-attributable health burdens in the most minoritized communities of the U.S. and contrast with the burdens experienced by majority communities and has been previously used in the literature[16,27,30]. Population subgroups do not include only tracts in certain states or geographic regions but, when defined across the entire U.S., do reflect urban-rural population differences to a certain degree. For example, the 7330 census tracts that comprise the most white and least white classifications in the U.S. include tracts from 52 and 49 states, territories, or districts and are 60% and 90% urban, respectively.

(2) **Population-weighted.** Population-weighted metrics were calculated with the following:

$$X_g = \frac{\sum_{t=i}^{N} pop_{t,g} \times X_t}{\sum_{t=i}^{N} pop_{t,g}}, \quad [3]$$

where X represents a pollutant or disease rate, pop represents the population, g represents a population subgroup, and t represents a census tract.

The population age structure varies between top and bottom decile subgroups (Figure S4). Presenting NO_2-attributable pediatric asthma crude rates or $PM_{2.5}$-attributable premature mortality crude rates does not account for these different age distributions (Text S2). Whenever rates are presented for the top and bottom deciles of population subgroups, they represent age-standardized rates directly adjusted to data from the entire U.S. population corresponding to the same year. Age standardization was conducted by multiplying each five-year age-specific rate by the fraction of five-year age group population to the entire U.S. population. We also explored the impact that omitting age-standardization has on our results (Text S2, Figure S5).

We tested whether differences in distributions of pollutants and associated disease burdens significantly vary across different ethnic and racial groups with the non-parametric Kolomogorov-Smirnov (K-S) test. The significance of trends in pollutants, burdens, and disparities was assessed with least-squares regression. If the *p*-values associated with the K-S test statistic or regression fell below 0.05, we classified the difference between distributions or trends as statistically significant.

Costs associated with $PM_{2.5}$-attributable premature deaths were estimated with the EPA's value of statistical life used for valuing mortality risk changes ($7.4 million in 2006 USD or $9.4 million in 2019 USD)[31]. This value represents the marginal rate of substitution between money and small changes in the risk of death. The body of literature on the economic burden of pediatric asthma is limited, but a 2018 study synthesized publications reporting on healthcare

costs and healthcare utilization for pediatric asthma and found average annual costs per child ranged from $3076 to $13612 in 2015 USD[32]. We used the midpoint of these values, adjusted for inflation to 2019 USD, as our estimate ($8,473).

We provide supplementary data files with information on tract-level NO_2 and $PM_{2.5}$ concentrations and attributable health burdens and rates for 2019 to make data used in this study accessible for stakeholders and enable scientific transparency and reproducibility. These files and their contents are described in Text S3.

3. Results

As cause-specific premature mortality rates and $PM_{2.5}$ have declined (Text S1, Figures 2A-B, S2), total $PM_{2.5}$-attributable deaths across the fifty U.S. states, Washington, D.C., and Puerto Rico have decreased by 28.5% from 69000 (48500—87000) in 2010 to 49400 (34500—62600) in 2019 (Figure 2A). New cases of NO_2-attributable pediatric asthma have declined by an even larger percentage, 39.8%, from 191000 (-282900—407900) in 2010 to 114900 (-158600—259400) in 2019 (Figure 2B) even with positive trends in pediatric asthma incidence in all states besides Puerto Rico. This wide uncertainty interval in estimated NO_2-attributable pediatric asthma cases stems from between-study heterogeneity (Section 2). The monetary value attributed to mortality risk (value of a statistical life) for premature deaths due to $PM_{2.5}$ and the estimated direct costs of NO_2-attributable pediatric asthma during 2019 translate to $466 billion in 2019 USD, roughly 2.2% of the 2019 U.S. gross domestic product. These total costs were dominated by $PM_{2.5}$-attributable mortality, which accounted for $464 billion, or 99.7%, of the estimated costs.

$PM_{2.5}$-attributable mortality rates were generally highest in metropolitan statistical areas (MSAs) in the Ohio River Valley and Gulf Coast (Figure 2C). MSAs with the ten highest rates (Birmingham, AL; Mobile, AL; Gulfport, MS; Evansville, IN; Daphne, AL; Punta Gorda, FL; Mansfield, OH; Weirton, WV; Hot Springs, AR; and Kokomo, IN) are generally located in these two regions, which contain heavy manufacturing and petrochemical industries. The $PM_{2.5}$-attributable premature mortality rate averaged over these ten metropolitan areas was 42.1 deaths per 100,000, nearly double the rate averaged over all MSAs (22.4 deaths per 100,000). These increased rates stem from the high population-weighted $PM_{2.5}$ concentrations in these MSAs (8.25 $\mu g\ m^{-3}$ versus the MSA average of 6.96 $\mu g\ m^{-3}$) and elevated underlying mortality in states containing these MSAs (Figure S2).

NO_2-attributable pediatric asthma rates in MSAs have more spatial heterogeneity than $PM_{2.5}$-attributable rates, and even relatively isolated MSAs can experience higher-than-average rates (Figure 2D). Among the most salient features in Figure 2D are the large NO_2-attributable asthma rates in the Permian Basin. The NO_2-attributable asthma rate averaged over the five largest MSAs in the Permian Basin (El Paso, TX; Lubbock, TX; Amarillo, TX; Midland, TX; and Odessa, TX) was 252.8 new cases of asthma per 100,000 children. This rate is nearly four times higher than the rate averaged over all MSAs (93.4 cases per 100,000 children) and even slightly greater than the rate in nearby Dallas-Fort Worth, TX (248.2 cases per 100,000 children). Oil and gas production in the Permian Basin has been linked to elevated levels of NO_2, methane, and volatile organic compounds[33,34] and increased pediatric asthma hospitalizations[35].

Despite long-term decreases of $PM_{2.5}$ and NO_2, the least white and most Hispanic communities still faced significantly higher concentrations of $PM_{2.5}$ and NO_2 than the most white and least Hispanic communities in 2019 (Figure 3). These disparities, which are characterized using the top and bottom deciles of population subgroups, were substantially larger for NO_2 than $PM_{2.5}$ and for different racial subgroups than ethnic subgroups.

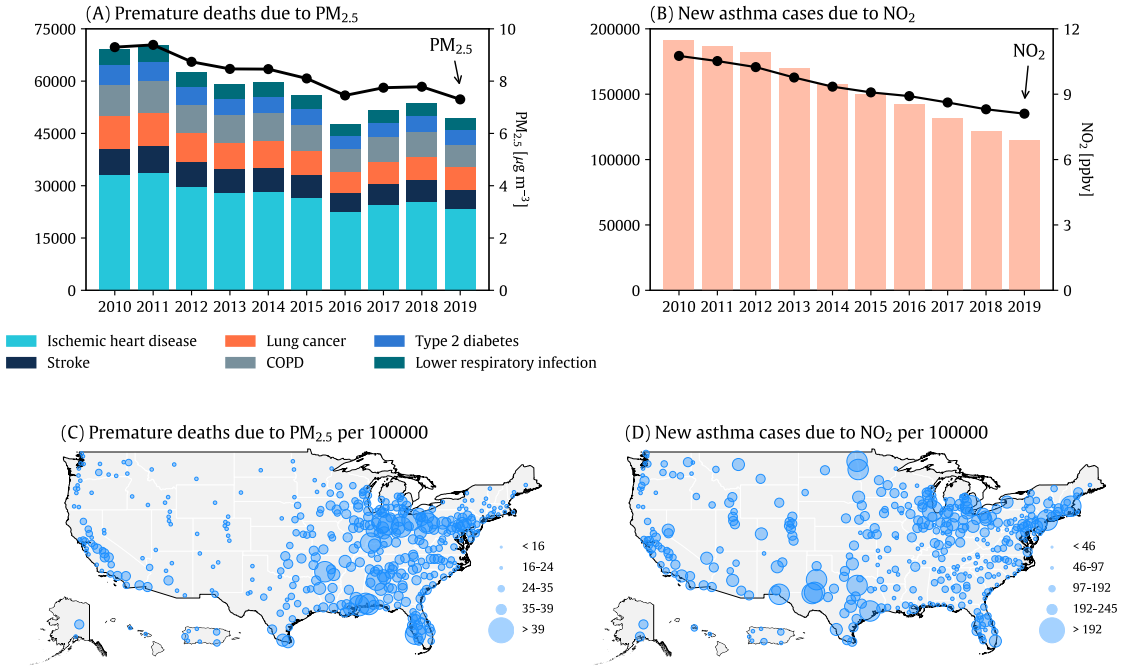

Figure 2. *Annual (A) $PM_{2.5}$ and $PM_{2.5}$-attributable mortality and (B) NO_2 and NO_2-attributable pediatric asthma in all fifty U.S. states, Washington D.C., and Puerto Rico. Quantities in (A) and (B) represent population-weighted pollutant concentrations for pollutants and sums for health burdens. Scatter points in (C)-(D) represent MSAs, and their size is proportional to the (C) $PM_{2.5}$-attributable deaths in 2019 per 100,000 population and (D) new cases of NO_2-attributable pediatric asthma in 2019 per 100,000 pediatric population. Rates in (C)-(D) and are discretized into five categories: <30th, 30-60th, 60-90th, 90-95th, and >95th percentiles of MSA rates. Alaska, Hawaii, and Puerto are not to scale.*

The least white communities in the U.S. experienced higher rates of cause-specific premature mortality attributable to $PM_{2.5}$ from all endpoints compared to the most white communities in 2019, and relative disparities have a range of 1.25—1.41, depending on the specific endpoint considered (Figures 3A). Ethnic relative disparities exhibit a wider range (0.89—1.29; Figure 3B). Opposite to expectations, we found that the least Hispanic communities in the U.S. experienced slightly higher $PM_{2.5}$-attributable premature mortality rates from COPD and lung cancer than the most Hispanic communities (Figure 3B).

Ethnoracial disparities in NO_2-attributable pediatric asthma are striking (Figure 3). NO_2-attributable asthma rates in the least white and most Hispanic communities of the U.S. were higher than rates in the most white and least Hispanic communities by a factor of 7.5 and 3.2 in 2019, respectively. In 28.8% of MSAs, all census tracts designated as most white had zero cases of NO_2-attributable pediatric asthma (i.e., NO_2 concentrations fell below the TMREL). This lack of NO_2-attributable asthma cases in the least white tracts only occurred in 2.3% of MSAs.

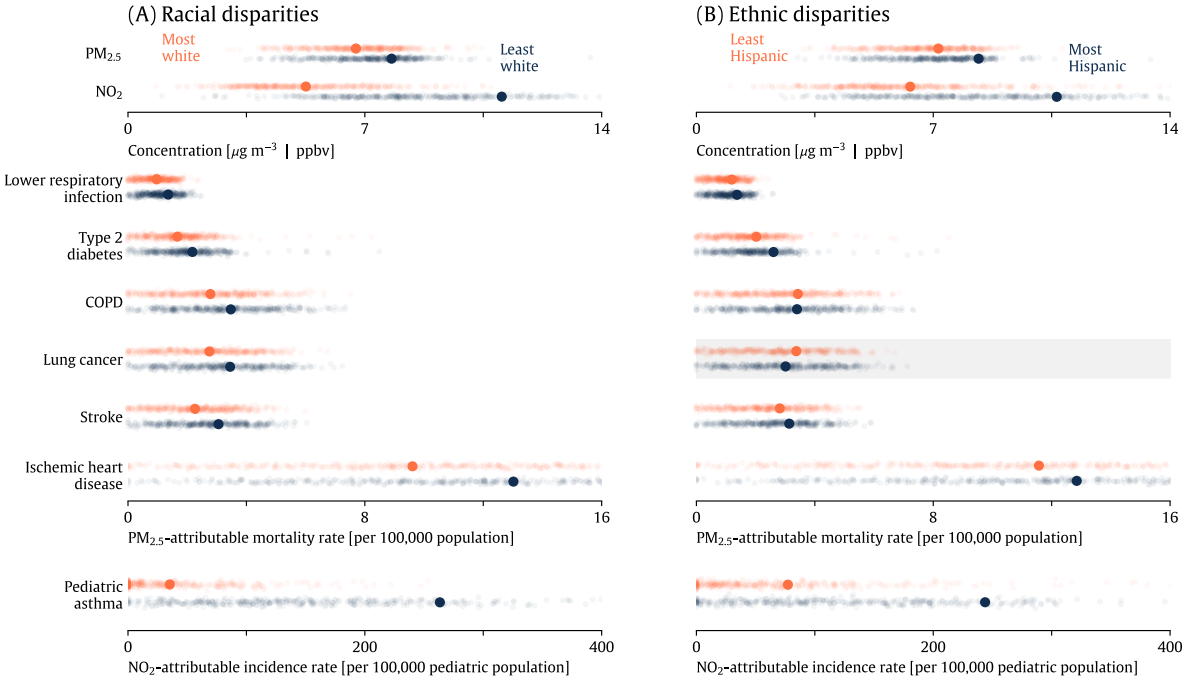

Figure 3. *Ethnoracial disparities in pollutant concentrations and associated pollution-attributable health burdens calculated for 2019 using the top and bottom deciles of population subgroups. Large scatter points correspond to concentrations or burdens for population subgroups calculated with all census tracts, while smaller jittered points correspond to these quantities in individual metropolitan statistical areas (MSAs) of the U.S. Rows shaded in gray indicate that the difference between the MSA distributions is not significant ($p > 0.05$).*

While NO_2-attributable pediatric asthma and $PM_{2.5}$-attributable premature mortality rates have decreased across the U.S. over the last decade, the magnitude of these decreases has not been uniform (Figure 4). Decreases in majority white and non-Hispanic communities outpaced decreases in majority non-white and Hispanic communities. As a result, relative racial disparities in NO_2-attributable pediatric asthma have increased from a factor of 6.3 difference between most and least white communities in 2010 to a factor of 7.5 difference in 2019 (19% increase; Figure 4A). Similarly, relative racial disparities in $PM_{2.5}$-attributable premature mortality grew by 16%, ethnic disparities in $PM_{2.5}$-attributable premature mortality by 40%, and ethnic disparities in NO_2-attributable pediatric asthma by 10%.

At the beginning of the decade, the most Hispanic communities in the U.S. faced *lower* $PM_{2.5}$-attributable death rates (Figure 4D), similar to our findings for some cause-specific endpoints in Figure 3B. However, ethnic subgroup most burdened with respect to $PM_{2.5}$-attributable

premature mortality reversed around 2015 (Figure 4D). By 2019, the most Hispanic communities had 8% higher $PM_{2.5}$-attributable premature mortality rates than the least Hispanic communities. While ethnoracial relative disparities in $PM_{2.5}$-attributable mortality are generally around 1 (equality between subgroups) and therefore small compared to disparities in NO_2-attributable pediatric asthma rates, their increasingly inequitable distribution is noteworthy. If trends during the 2010s are an indication of the future, we expect that these disparities will continue to grow.

Examining trends and disparities in $PM_{2.5}$ and NO_2 concentrations can shed light on the drivers of the widening disparities. We found that disparities in $PM_{2.5}$ concentrations are widening (Figure S6A-B), supported also by recent work from Jbaily et al.[16]. Relative disparities in NO_2 concentrations have narrowed (Figure S6C-D), indicating that the increase in NO_2-attributable pediatric asthma disparities is driven by changing underlying incidence rates across subgroups. Although these rates only vary by state (Figure S2S-U), a larger number of tracts belonging to a particular decile population subgroup located in a state in which pediatric asthma incidence has exhibited a greater increase relative to other states would lead to this finding.

Our use of state-level cause-specific mortality rates and pediatric asthma incidence rates could impact results since these rates have been shown to vary on neighborhood scales and are often higher in minoritized and marginalized communities[36,37]. We hypothesize that incorporating rates at a higher spatial resolution would likely accentuate the disparities uncovered in this study. To test this hypothesis, we considered a recently developed dataset estimating tract-level all-cause mortality rates in 2015 (Text S2). Since these rates represent all-cause mortality rather than the six specific causes we examined in this study, we combined these tract-level rates with all-cause mortality RR estimates from Turner et al.[38], which were also recently used in an EPA review of the NAAQS for $PM_{2.5}$. Consistent with our hypothesis, we find that using tract-level mortality rates leads to even higher burdens placed on the least white and most Hispanic communities of the U.S. (Text S2, Figure S5). The analysis in Figure S5 also suggests that similar conclusions regarding disparities and the most exposed population subgroup are found using cause-specific RR estimates from the GBD or the all-cause RR estimates from Turner et al.[38]. While we do not have pediatric asthma incidence rates in census tract to conduct a similar sensitivity analysis for NO_2-attribuable pediatric asthma, we believe that such data would also lead to even greater disparities.

We have also considered relative disparities calculated with population-weighted concentrations and disease rates to complement disparities calculated with the top and bottom deciles of population subgroups. The sign of disparities in population-weighted pollution concentrations, pediatric asthma, and premature death rates due to pollution in 2019 (Figure S7) is consistent with results from Figure 3, although the magnitude of these population-weighted disparities was slightly smaller than disparities estimated using the top and bottom deciles approach. Trends in population-weighted relative disparities are more mixed than the trends calculated with top and bottom deciles (Figure S8). We find that racial relative disparities in population-weighted pollution-attributable asthma and premature mortality have non-significant positive trends, ethnic premature mortality disparities a significant positive trend, and ethnic asthma disparities a significant negative trend (Figure S8). While we rely on contemporary statistical methods to quantify whether results are "significant," reliance on these methods can be problematic and may

neglect historically excluded groups[39]. We believe that any disproportionate impacts related to pollution, regardless of significance, warrants further research and commensurate action.

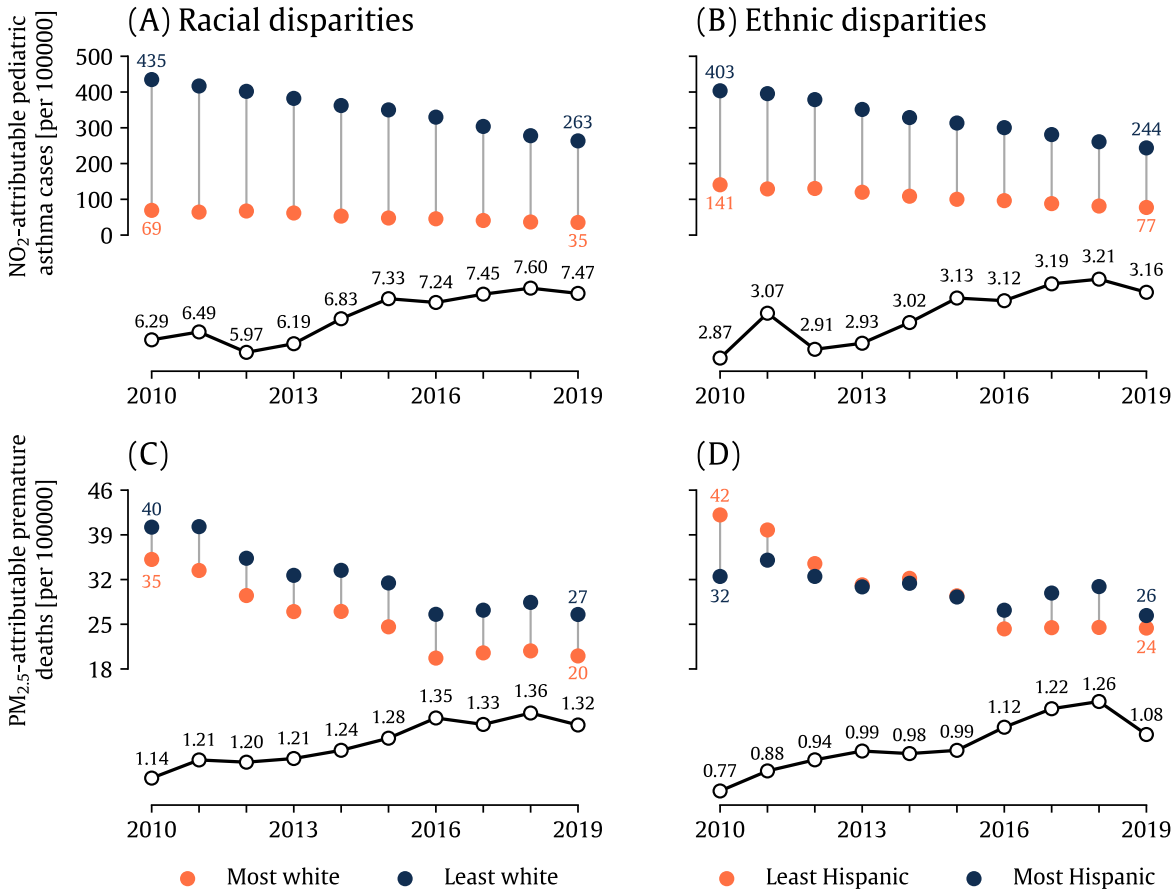

Figure 4. *Trends in (A-B) NO_2-attributable pediatric asthma and (C-D) $PM_{2.5}$-attributable premature mortality rates for the most and least white and Hispanic tracts in the U.S. Black time series and corresponding text beneath each panel indicate the relative disparities, defined as the ratio of the rate for the bottom decile population subgroup (least white, most Hispanic) to the rate for top decile (most white, least Hispanic). A value of 1 for relative disparities implies that pollution-attributable burdens are equally shared across subgroups. For reference, rates for the first and last years of the analysis are indicated alongside the scatter points.*

The NAAQS do not adequately protect the public from the adverse effects of $PM_{2.5}$ and NO_2 based on our own assessment of the health burdens that occur when the NAAQS were attained in the vast majority of tracts (Figure 5) as well as numerous toxicological and clinical studies that highlight health effects of these pollutants at levels below the current NAAQS[40,41]. The current annual $PM_{2.5}$ NAAQS of 12 μg m^{-3}, last revised in 2012, was met in all but 486 (0.7%) of census tracts in 2019, and the highest 2019 NO_2 concentration in all census tracts of the U.S. (28.3 ppbv) was about half the annual NO_2 NAAQS of 53 ppbv, which has not been revised since 1971. Yet, Figure 2A-B highlights the major public health damages associated with these pollutants.

Enacting and attaining more stringent $PM_{2.5}$ and NO_2 standards could reduce pollution-attributable health burdens, with potentially outsized benefits for communities of color (Figure 5). As an example, we consider how a $PM_{2.5}$ standard of 8 $\mu g\ m^{-3}$ could advance environmental justice. This level is the lower end of the range recommended by EPA's Clean Air Scientific Advisory Committee in March 2022. If a new $PM_{2.5}$ standard of 8 $\mu g\ m^{-3}$ was adopted and met in all tracts where this level is not currently met, the decrease in $PM_{2.5}$-attribuable premature mortality rates in the least white communities of the U.S. would be roughly four times larger than the decrease in the most white communities (Figure 5). Similarly, if the WHO interim target-3 (IT-3) was met, total pediatric NO_2-attributable asthma burdens would drop by 20%, but the least white communities in the U.S. would experience a fivefold greater reduction in pediatric asthma rates than in the most white communities (-32.6% versus -6.3%).

Reducing 2019 NO_2 and $PM_{2.5}$ to the stringent WHO AQGs in all tracts where these guidelines are not met would lead to a 73.2% reduction in $PM_{2.5}$-attributable mortality and eliminate NO_2-attributable pediatric asthma (all concentrations would be below our assumed TMREL). Attaining the AQGs also eliminates the current patterns of injustice by which communities of color experience greater pollution-attributable health burdens.

4. Discussion

Our study documents the substantial impact of air pollution on human health from 2010 through 2019, exploring how communities of color shoulder a disproportionate share of this burden. Results paint a mixed picture of progress: despite overall decreases in NO_2 and $PM_{2.5}$ and associated health impacts during the 2010s, significant ethnoracial disparities in the health impacts attributable to these pollutants remain. We found that relative disparities in NO_2-attributable pediatric asthma are several times larger than relative disparities in $PM_{2.5}$-attributable premature mortality, and relative disparities in $PM_{2.5}$ concentrations and pollution-attributable health impacts from $PM_{2.5}$ and NO_2 are widening.

Our finding that disparities in $PM_{2.5}$ and associated health burdens are growing is an important and alarming conclusion of this study and complements recent work by Jbaily et al.[16], who highlighted increasing $PM_{2.5}$ disparities among racial and ethnic groups but did not examine associated health impacts. One potential explanation for the widening $PM_{2.5}$ disparities could be the declining importance of the power generation sector[42]. The largest benefits of power plant closures have accrued to the white population[30]. As the role of the power generation sector on $PM_{2.5}$ decreased, light-duty and heavy-duty vehicles have become an increasingly important source of primary $PM_{2.5}$. Our previous work has shown the collocation of marginalized and minoritized neighborhoods with the roadways used by these vehicles[27].

Our assessment of the economic costs caused by $PM_{2.5}$ and NO_2 agrees well with a global economic assessment conducted by Yin et al.[43], who found that $PM_{2.5}$-attributable economic costs amount to 2.7% GDP in the U.S. Furthermore, premature mortality and pediatric asthma burdens documented in this study generally align with other recent studies[12,21,42,44,45]. We note, however, that our estimates are lower. One key reason for this discrepancy is that our TMRELs are higher (i.e., more conservative) than those in other studies, which assume, for example, there is no level below which $PM_{2.5}$ would not increase the risk of death[12,37]. Our TMRELs, derived

from the latest GBD, represent uncertainty about the lowest level of exposure associated with increased mortality or morbidity given the exposure distribution[46]. A growing number of studies specifically analyzing health effects of pollutants at low concentrations[47] will continue to increase the community's understanding of low-level health effects.

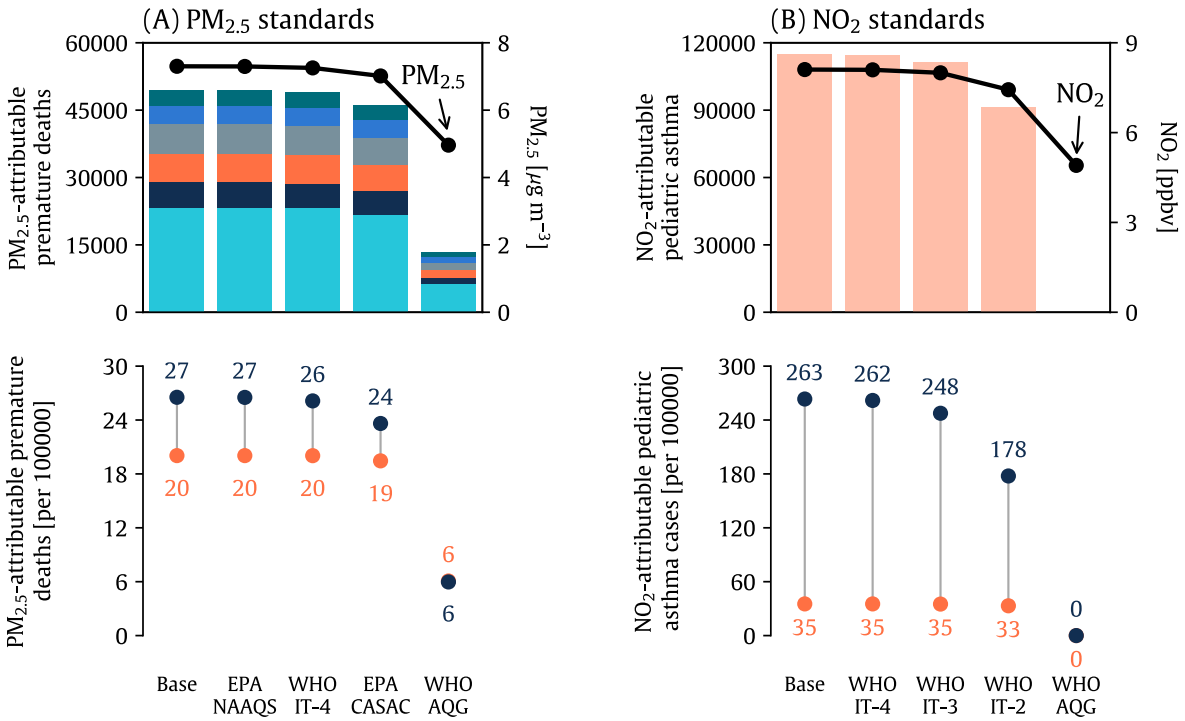

Figure 5. *Air quality, health, and environmental justice benefits achieved by attaining (A) $PM_{2.5}$ and (B) NO_2 standards in tracts where pollutant concentrations exceeded these standards in 2019. $PM_{2.5}$ standards include the Environmental Protection Agency (EPA) National Ambient Air Quality Standard (NAAQS) of 12 μg m^{-3}; the World Health Organization (WHO) Interim Target 4 (IT-4) of 10 μg m^{-3}; the lower bound of the recommended range (8-10 μg m^{-3}) recommended by the Clean Air Scientific Advisory Committee (CASAC) in their March 2022 letter to the EPA Administrator; and the WHO Air Quality Guidelines (AQG) of 5 μg m^{-3}. NO_2 standards include the WHO IT-1 of ~21.3 ppb (assuming an ambient pressure of 1013.25 hPa and temperature of 298.15 K); the WHO IT-2 of ~16 ppb; the WHO IT-3 of ~10.6 ppb; and the WHO AQG of ~5.3 ppb. Interpretation of the top panels, which show (A) total $PM_{2.5}$-attributable premature deaths and population-weighted $PM_{2.5}$ concentrations and (B) total NO_2-attributable pediatric asthma cases, follows Figure 2A-B. Interpretation of the bottom panels, showing disparities in pollution-attributable health burdens under the various standards for the most and least white racial population subgroups, follows Figure 4A and C.*

Examining the impacts of higher resolution death rates suggests that our use of state-level rates may underestimate ethnoracial relative disparities (Text S2; Figure S5). Our choice of RR estimates could also impact the magnitude of disparities. We relied on uniform RR estimates applied to the entire population in this study, but risk may differ among different demographic groups due to social determinants of health or biological differences. Akinbami et al.[48] found that children belonging to ethnoracial minority groups had as high or higher relative risk for asthma

diagnoses than non-Hispanic white children, and Spiller et al.[49] showed uniform RR estimates, rather than race-ethnicity specific estimates, underestimated pollution-related health impacts for minority communities. However, the literature on this topic is not consistent: Alexeeff et al.[50] did not find a difference in the association between exposure to $PM_{2.5}$ and COPD by race or ethnicity, and Parker et al.[51] similarly found that the association between $PM_{2.5}$ and heart disease mortality was not statistically different for non-Hispanic white adults versus Black or Hispanic adults. Since the literature remains inconsistent on this topic, it is unknown how race- and ethnicity-specific RR estimates would impact the findings uncovered herein. Future work might leverage race- and ethnicity-specific RR estimates, such as those developed by Di et al.[52] from Medicaid-eligible persons in the U.S.

Measures of socioeconomic status such as educational attainment and income have often been used in environmental justice studies. Here, we have chosen to focus on race and ethnicity. Tessum et al.[53] demonstrated that people of color at *every* income level face disproportionate $PM_{2.5}$ exposure, and ethnoracial disparities are not a proxy for socioeconomic disparities. Policies to reduce pollution burdens based strictly on socioeconomic status may not do so equitably[54], thus buttressing our focus on ethnoracial patterns of injustice.

Systems and practices that introduce and perpetuate systemic racism and discrimination are responsible for these disparities[55]. Marginalized and minoritized communities are disproportionately exposed to virtually all major emissions sectors; traffic (particularly heavy-duty diesel traffic), industry, and construction have been pointed out as the most important in explaining $PM_{2.5}$ and NO_2 disparities [28,53,56]. Disparities in pollution and associated health impacts have been linked to "redlining," a practice beginning in the 1930s by which financial services were denied to residents in certain urban areas based on their race or ethnicity[57–59]. While this discriminatory practice officially ended in 1968, its numerous effects on present-day zoning practices and the placement of highways and industries in racialized and minoritized neighborhoods have been documented[58,60,61].

Minoritized, racialized, and marginalized communities in the U.S. persistently experience disproportionate air pollution-attributable disease burdens. Ethnoracial health disparities due to NO_2-attributable pediatric asthma are substantially larger than those from $PM_{2.5}$-attributable premature mortality, but relative disparities for both these health outcomes in the most versus least minoritized communities of the U.S. have widened in the past decade. Alternative ways of defining disparities (e.g., population-weighted, most versus least burdened) indicate that the exact sign and significance of trends can be somewhat metric-specific. Regardless of which metric is used, though, recent trends in relative disparities in the U.S. *have clearly not matched* the obvious macro-level reductions in ambient NO_2 and $PM_{2.5}$ pollution due to the Clean Air Act and related measures.

Increasing the stringency of the NAAQS for $PM_{2.5}$ and NO_2 to be in alignment with the 2021 WHO AQGs could have outsized benefits for marginalized and minoritized communities. Codification and attainment of these AQGs would effectively eliminate current patterns of injustice and broadly reduce pollution-attributable health burdens across the nation. Accomplishing sufficient pollution remediation will require reductions from almost every emission sector given their disproportionate impacts on marginalized and minoritized

communities. Recent efforts to reduce emissions from transportation (e.g., plug-in electric vehicle tax credits, EPA's proposed heavy-duty engine and vehicle standards) and rethink land use (e.g., Department of Transportation's Reconnecting Communities Pilot Program) are steps in the right direction and urgently needed. While the investments needed to develop new control technologies and implement other mitigation measures are not trivial, the potential economic benefits of such investments due to improved public health would outweigh them.

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Supporting Information for "Increasing disparities in air pollution health burdens in the United States"

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Text S1: Surface-level pollution datasets and their trends

Conducting our analysis with recently developed 0.01˚ x 0.01º (~1 km x 1 km) pollutant datasets transformed to census tract averages is a major strength of our study and allows us to capture the heterogeneities and microenvironments that characterize air quality exposure, especially in urban areas. Paolella et al.[1] showed that the spatial resolution of $PM_{2.5}$ data impacts concentration disparities, and Mohegh et al.[2] investigated how the spatial resolution of NO_2 data affects pediatric asthma burdens. When taken together, these studies suggest that using pollution datasets with a spatial resolution coarser than ~1 km^2 leads to smaller estimated health impacts or disparities. Thus, our use of ~1 km^2 pollutant datasets likely provides a more accurate characterization of disparities and associated health burdens than coarser datasets could afford. $PM_{2.5}$ and NO_2 data at a finer resolution than 1 km^2 have been shown to lead to greater health burdens[3], but we are not aware of a nationwide dataset that would provide sub-km^2 data for both these pollutants.

These datasets provide complete spatial coverage of NO_2 and $PM_{2.5}$, unlike the sparse coverage available from *in-situ* monitors (Figure 1A-B), enabling us to characterize the health effects associated with these pollutants on environmental justice-relevant scales. Although the performance of these two datasets against observations has been documented in the literature[4,5], we provide such an analysis tailored to our time period and domain in Figure 1. The comparison of observed NO_2 and $PM_{2.5}$ concentrations derived from these datasets yields a high degree of confidence in their ability to capture spatiotemporal variability in surface-level NO_2 and $PM_{2.5}$ (Figure 1). We note a high dataset bias for NO_2 relative to observations, a low dataset bias for $PM_{2.5}$, and that the correlation of the datasets with observations slightly decreases over our measuring period. The decreasing correlation could reflect a growing number of monitors sited adjacent to roadways as part of the EPA near-road monitoring network initiated in 2010. We do not expect that the $PM_{2.5}$ and NO_2 datasets would resolve the incremental NO_2 or primary $PM_{2.5}$ impacts from traffic[6].

Between 2010 and 2019, nationwide-averaged population-weighted $PM_{2.5}$ decreased by 21.5% at a rate of -0.2 $\mu g\ m^{-3}\ yr^{-1}$ (p<0.01), and population-weighted NO_2 decreased by 24.6% at a rate of -0.3 $ppbv\ yr^{-1}$

(p<0.01) (Figure 2A-B). The nationwide-averaged trends shown in Figure 2A-B mask some regional heterogeneities. We observed positive $PM_{2.5}$ trends in Montana, Nevada, Oregon, Puerto Rico, and Washington using state-averaged population-weighted concentrations; all positive $PM_{2.5}$ trends were not significant except for Puerto Rico. State-averaged population-weighted NO_2 in Maine, Montana, New Hampshire, North Dakota, South Dakota, and Vermont also exhibited positive trends, with significant trends in Maine, North Dakota, and South Dakota. Many states with positive trends are located in the Western U.S., which has been challenged by air quality impacts related to increasing wildfire activity in recent years[7,8]. The growing impact of wildfires on air quality could also explain the non-monotonic decrease of nationwide-averaged $PM_{2.5}$ during the measuring period and the slight increase from 2016-2018 (Figure 2A).

Another notable feature in Figure 2D is the large NO_2-attributable pediatric asthma rates in Fargo, ND and Grand Forks, ND-MN. Several studies have documented the impact of agriculture on NO_x emissions in the Great Plains and Upper Midwest, particularly during the summer months[9,10]. Our NO_2 estimates derive from a land use regression model representative of 2010-2012 concentrations scaled to more recent years by satellite retrievals[5]. Persistent snow and cloud cover in the Great Plains and Upper Midwest during the winter months lead to fewer retrievals during this season, and thus NO_2 estimates may be more representative of summer NO_2 levels. The dearth of *in-situ* monitors in this region preclude us from commenting on the performance of the NO_2 dataset (Figure 1A).

Text S2: Sensitivity of results to higher-resolution incidence rates and age standardization

Our analysis does not consider sub-state or racial variations in underlying incidence and mortality rates. State-level rates represent the highest level of granularity currently available from the GBD, and annual incidence rates at finer resolutions such as in counties or census tracts may contain missing data to protect confidentiality and privacy.

Underlying incidence and mortality rates have been shown to vary on neighborhood scales with higher values in areas with lower socioeconomic status and a higher percentage of minorities[11,12]. Spiller et al.[13] showed that considering race- and ethnicity-specific rates does not significantly affect the total number of deaths but distributed the deaths differently among demographic groups in a national-scale analysis of $PM_{2.5}$-attributable mortality in the U.S. Recently, the EPA's BenMAP-CE software used to estimate air pollution-related health impacts has included estimates of census tract all-cause mortality rates using life tables from the U.S. Small-Area Life Expectancy Estimates Project (USALEEP)[14]. These rates are based on death records over the period 2010-2015.

We use these tract-level incidence rates and investigate how they affect ethnoracial disparities. Since these rates represent all-cause mortality (rather than cause-specific mortality investigated in the main text), we combine these higher resolution incidence rates with all-cause RR estimates from Turner et al.[15] of 1.06 per 10 $\mu g\ m^{-3}$ annual average $PM_{2.5}$, which was used in the most recent $PM_{2.5}$ Regulatory Impact Analysis from the EPA [16]. We did not apply a TMREL when calculating all-cause premature mortality with the Turner et al.[15] RR estimates. Therefore, do not expect the total number of $PM_{2.5}$-attributable premature deaths and ethnoracial absolute disparities to match the results in the main text; however, we hypothesize that examining the *relative* disparities using these different methods will allow us to test whether our results are robust to different incidence rates and RR estimates.

In this sensitivity analysis we calculate national-level ethnoracial relative disparities for the following cases:

1) Turner et al.[15] RR estimates with state-level all-cause mortality rates. Burdens and rates are calculated for the population aged 30 and older for each five-year age group (30-34, 35-39, etc.) and thereafter standardized to account for differences in the age structure across population subgroups.
2) Same as 2 but no age standardization is applied.
3) Turner et al.[15] RR estimates with tract-level all-cause mortality rates. Tract-level rates from Raich et al.[14] are available for ten-year age groups (25-34, 35-44, etc.) so we apply the RR estimates to the population aged 25 and older in ten-year age groups and standardize for different age structures. We acknowledge that disparities calculated with these methods (for population aged 25 and older) are not directly comparable with the disparities from 1-2 (for population aged 30 and older); however, we expect differences to be minimal.
4) Same as 3 but no age standardization is applied.

Based on the period represented by USALEEP tract-level rates, all results for this sensitivity test represent 2015 values, and the age structure is standardized to the full U.S. population for that year.

Age-standardized disparities calculated as the sum of cause-specific mortality rates (from the main text) and all-cause mortality rates calculated with Turner et al.[15] RR estimates and state-level underlying rates from the GBD are similar in magnitude (Figure S5). This result suggests our key conclusions are robust to different methods for calculating $PM_{2.5}$-attributable premature mortality.

As expected, when state-level rates are replaced by tract-level rates, the magnitude of disparities grows (Figure S5). The racial relative disparities calculated with Turner et al.[15] RR estimates and state-level rates is 1.12, which increases to 1.46 when calculated using tract-level rates (Figure S5A). The year in which we conduct this sensitivity test, 2015, is the year in which $PM_{2.5}$-attributable mortality rates were nearly at parity for the most and least Hispanic subgroups using the methods described in the main text (Figure 4). When ethnic disparities are determined with different RR estimates and underlying incidence rates, we reach slightly different conclusions regarding the most exposed population subgroup. Still, disparities are slight regardless (~5% difference in rates between subgroups; Figure S5B).

Lastly, exploring the impact of age standardization highlights how differences in the population age structure can influence results. When no age standardization is applied, the least Hispanic population subgroup consistently emerges as the most exposed ethnic subgroup, regardless of the choice of RR estimates or underlying rates (Figure S5B). Both the most white and least Hispanic subgroups have a larger shares of their population that reach older ages (Figure S4). Underlying incidence rates for the elderly are considerably higher than rates for younger age groups (not shown). For example, the nationwide average of state-level rates of death from ischemic heart disease for the population aged 85 and greater is nearly 2,000 times higher than for the population aged 25-29 (3314 per 100,000 versus 2 per 100,000). Without age standardization even a relatively small difference in the elderly population between population subgroups could skew results.

Based on the results of this sensitivity analysis, incorporating higher resolution rates is unlikely to materially change our key conclusion that ethnoracial minorities in the U.S. face disproportionately

higher rates of premature mortality attributable to $PM_{2.5}$. Future studies that include higher resolution estimates of underlying rates or rates stratified by race and ethnicity could see disparities accentuated as we showed in Figure S5. While we have not explicitly investigated how higher resolution incidence rates of pediatric asthma impact our results, it is likely that these higher resolution rates could also lead to even starker disparities.

Text S3: Tract-level pollution and health burdens geographical information system data

We provide census tract-averaged $PM_{2.5}$ and NO_2 concentrations and total pollution-attributable health burdens for each endpoint examined in our study (pediatric asthma, chronic obstructive pulmonary disease, ischemic heart disease, stroke, lung cancer, lower respiratory infection, type 2 diabetes) as well as the corresponding crude rates for each endpoint. These data are provided as supplementary data files in shapefile format for easy integration in geographic information system (GIS) software. Index of feature geometry (.shx) and attribute information (.dbf) files are included alongside the feature geometry (.shp) file.

Census tract boundaries and select metadata (GEOID, latitude, longitude; Table S1) are taken from the 2010 Census TIGER/Line shapefiles[17]. Table S1 lists the fields included in the shapefiles and a short description of each field. Note that there are a small number of census tracts missing NO_2 and $PM_{2.5}$ concentrations (77 and 234, respectively) and have a value of NaN. These missing tract-averaged concentrations represent tracts whose underlying grid cells in the native datasets had NaN values and are generally found near bodies of water. NaN values for pollutant concentrations propagate to the associated disease burdens and rates.

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Field	Description
NO2	Annual average NO_2 concentration, units of ppbv
PM25	Annual average $PM_{2.5}$ concentration, units of $\mu g\ m^{-3}$
BURDENASTH	Total new NO_2-attributable pediatric asthma cases
BURDENCOPD	Total $PM_{2.5}$-attributable premature deaths from COPD
BURDENIHD	Total $PM_{2.5}$-attributable premature deaths from ischemic heart disease
BURDENSTR	Total $PM_{2.5}$-attributable premature deaths from stroke
BURDENLC	Total $PM_{2.5}$-attributable premature deaths from lung cancer
BURDENLRI	Total $PM_{2.5}$-attributable premature deaths from lower respiratory infection
BURDENT2D	Total $PM_{2.5}$-attributable premature deaths from type 2 diabetes
RATEASTH	NO_2-attributable pediatric asthma rates per 100,000 population aged 18 years or less
RATECOPD	$PM_{2.5}$-attributable death rates from COPD per 100,000 population aged 25 years and greater
RATEIHD	$PM_{2.5}$-attributable death rates from ischemic heart disease per 100,000 population aged 25 years and greater
RATESTR	$PM_{2.5}$-attributable death rates from stroke per 100,000 population aged 25 years and greater
RATELC	$PM_{2.5}$-attributable death rates from lung cancer per 100,000 population aged 25 years and greater
RATELRI	$PM_{2.5}$-attributable death rates from lower respiratory infection per 100,000 population
RATET2D	$PM_{2.5}$-attributable death rates from type 2 diabetes per 100,000 population aged 25 years and greater
GEOID	Unique census tract identifier; the GEOID is an 11 digit concentration of the state Federal Information Processing System (FIPS) codes (2 digits), county FIPS code (3 digits), and census tract code (6 digits).
INTPTLAT	Latitude of census tract centroid
INTPTLON	Longitude of census tract centroid

Table S1. *Fields included in supplementary data shapefile, representing concentrations, burdens, and rates in 2019. Rows in gray represent fields taken directly from the native TIGER/Line shapefiles[17] and can be used for mapping as well as matching pollutant concentrations and attributable health burdens with census data.*

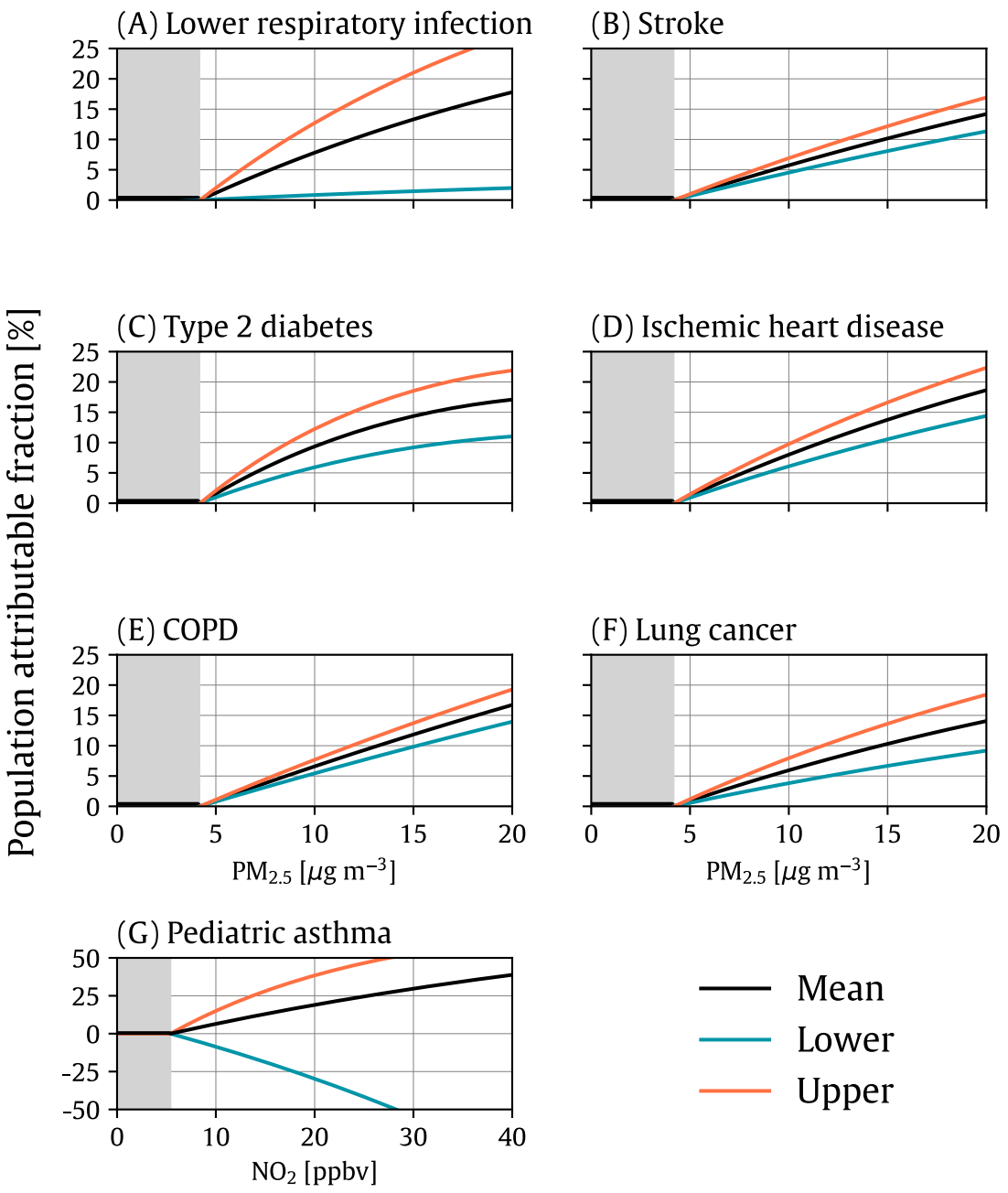

Figure S1. *For the endpoints of interest, curves indicate the proportion of their incidence attributable to $PM_{2.5}$ and NO_2 exposure, generated with RR estimates from the GBD. The abscissa has been truncated to the nearest multiple of 10 that corresponds to the maximum tract-averaged $PM_{2.5}$ or NO_2 concentrations in the U.S. during 2010-2019. The grey regions denote the concentrations of $PM_{2.5}$ and NO_2 equal to or less than the counterfactual scenario of theoretical minimum risk exposure used in this study.*

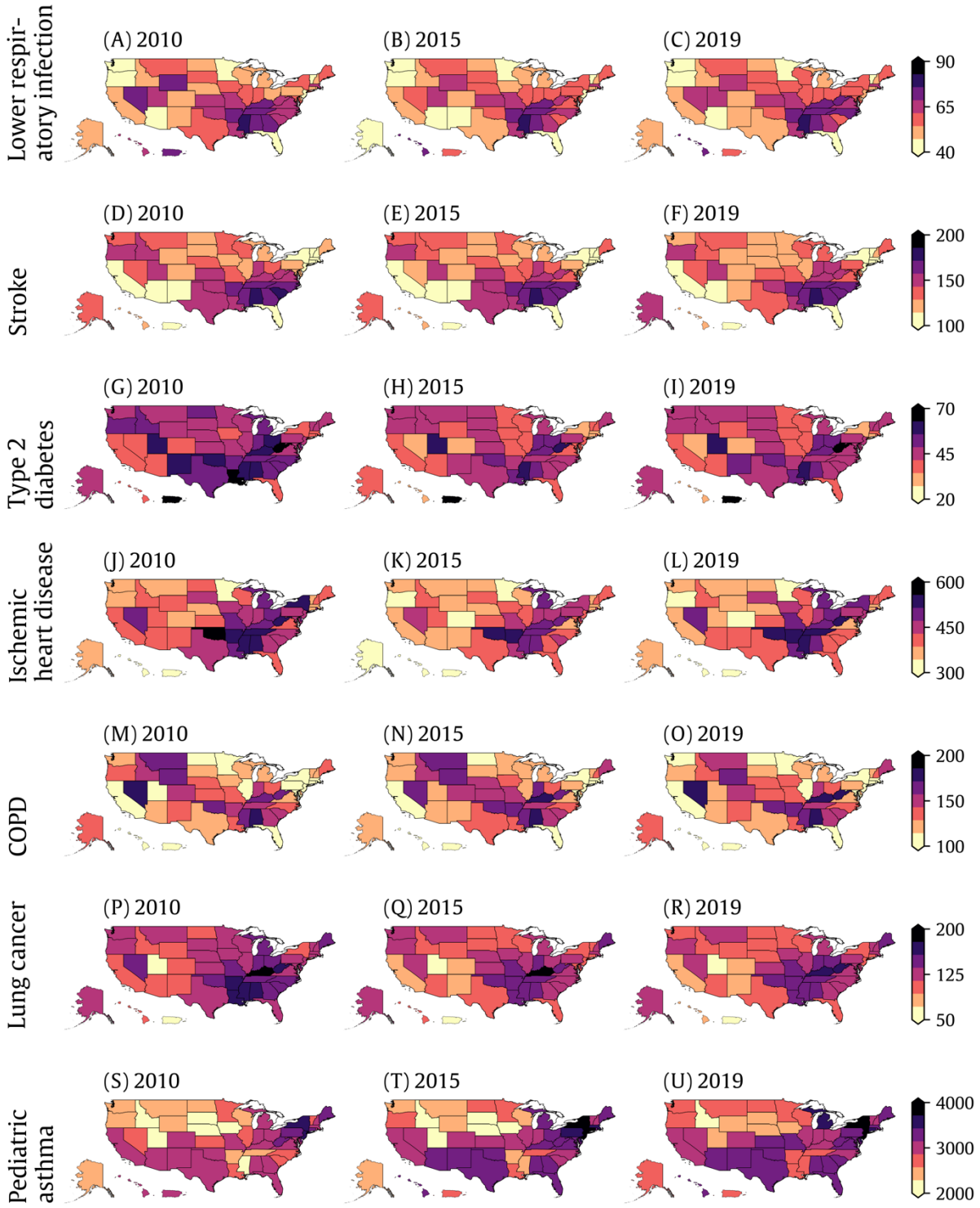

Figure S2. *Underlying premature mortality and incidence rates from the GBD for 2010, 2015, and 2019. Rates vary for each five-year age, and rates shown in this figure represent an average over these groups.*

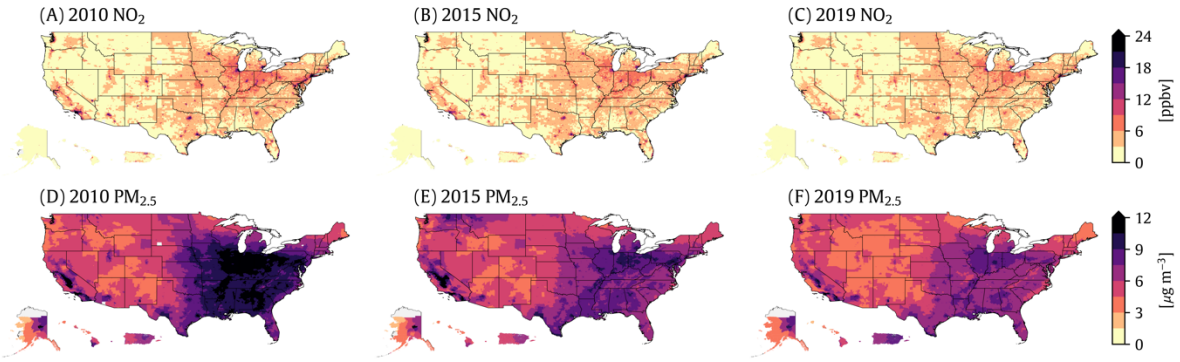

Figure S3. *Census tract-averaged surface level NO_2 and $PM_{2.5}$ concentrations in 2010, 2015, and 2019. These time periods were chosen to reflect the beginning, middle, and end years of this study. Tracts colored in gray either lie outside the coverage of the datasets or represent unorganized territories without tracts.*

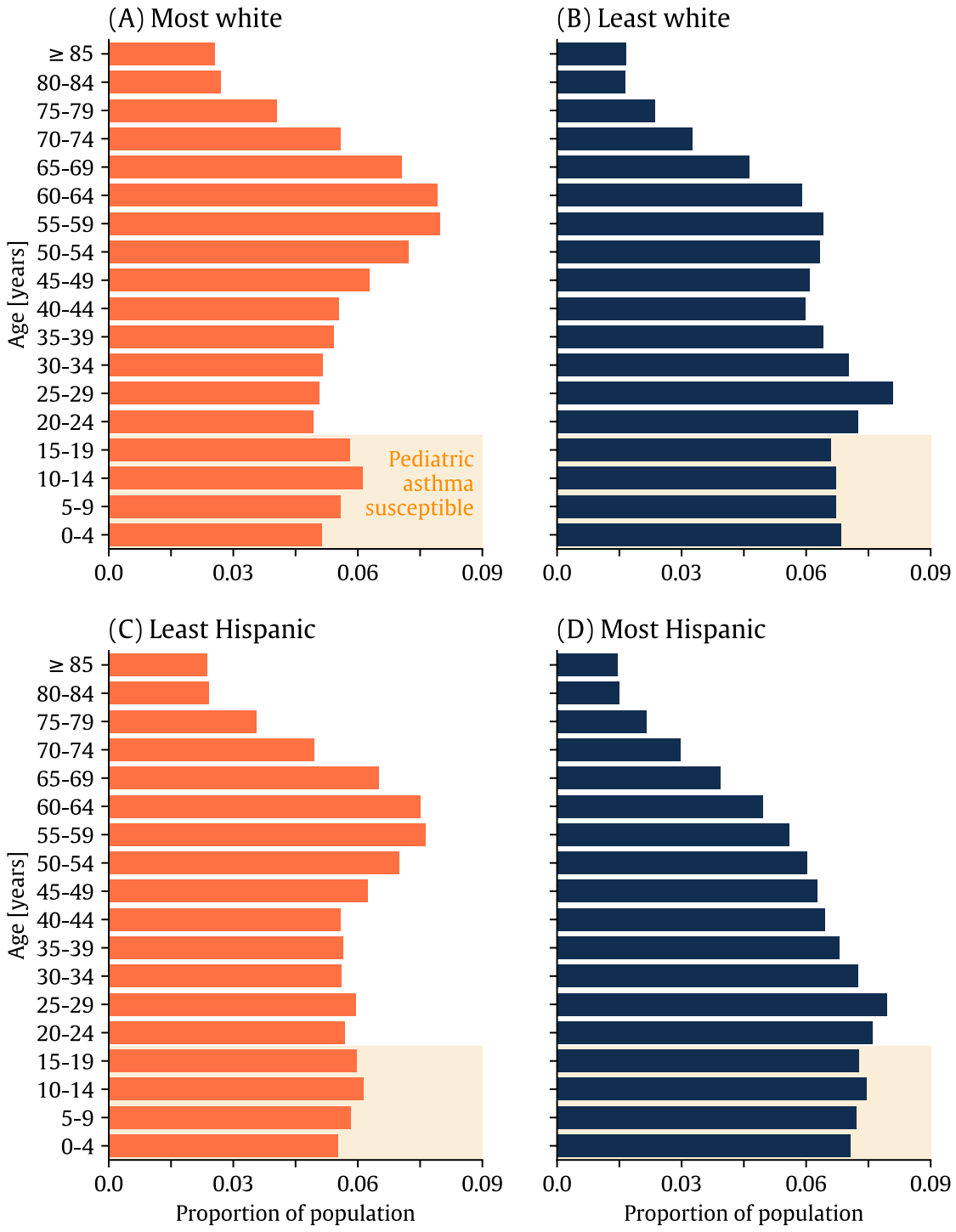

Figure S4. *Differences in population age structure for the top and bottom deciles of population subgroups in 2019. The shaded region in plots denotes the age groups which are included in our calculation for NO_2-attributable pediatric asthma.*

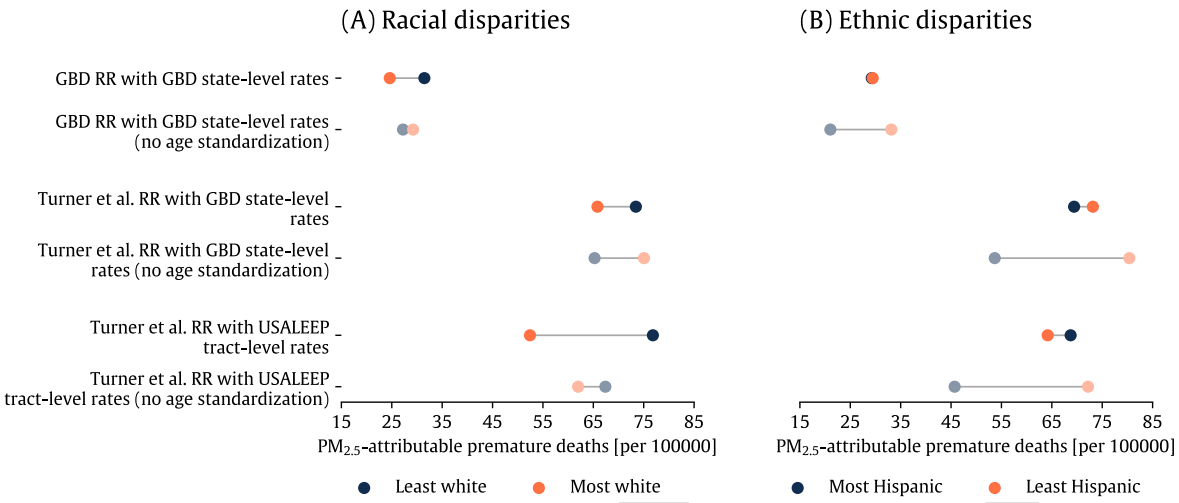

Figure S5. *Disparities in $PM_{2.5}$-attributable premature mortality rates for (A) racial and (B) ethnic extreme deciles of population subgroups in the U.S. during 2015 using different RR estimates and underlying incidence rates. Lighter colors signify rates calculated without the age standardization applied throughout the main text.*

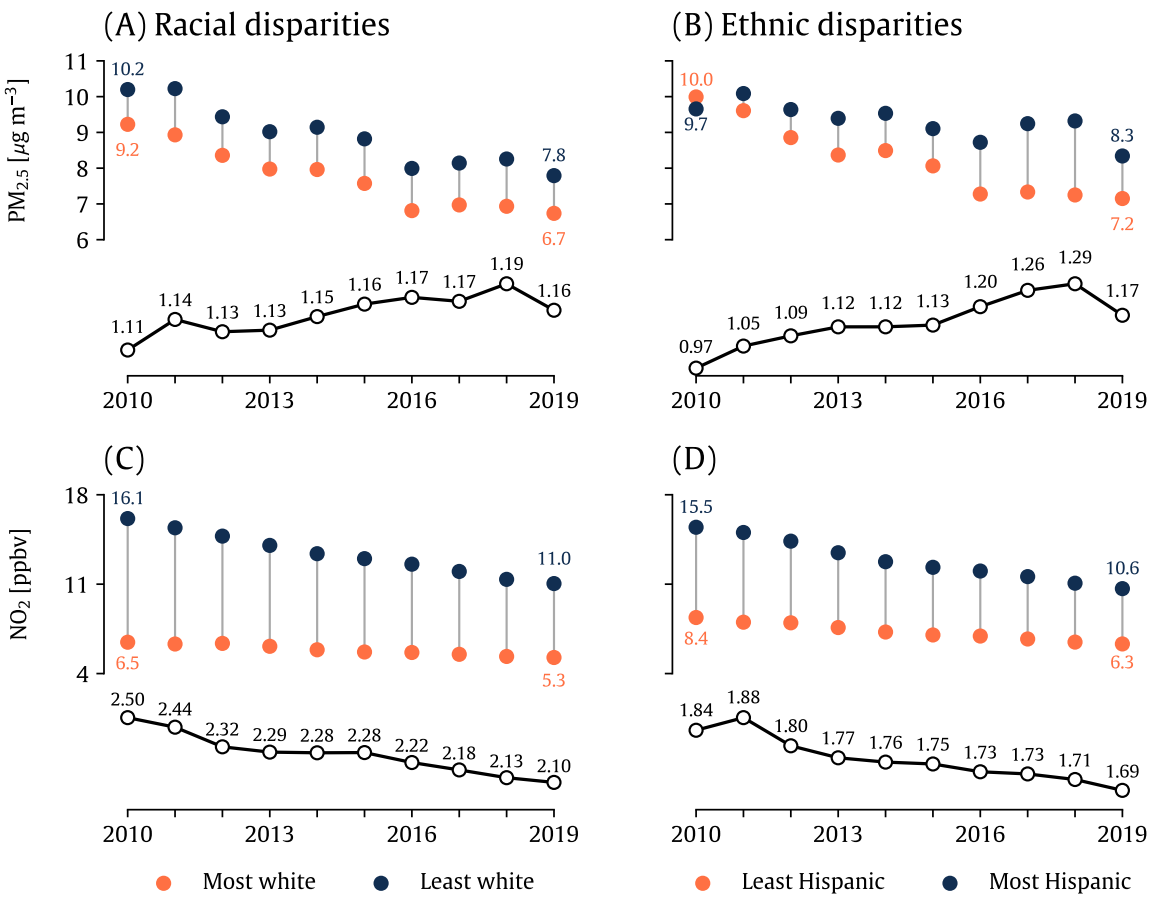

Figure S6. *Interpretation follows Figure 4 in the main text, but subplots show trends in concentrations of (A-B) $PM_{2.5}$ and (C-D) NO_2 for different (A,C) racial and (B,D) ethnic population subgroups, defined using the top and bottom deciles approach.*

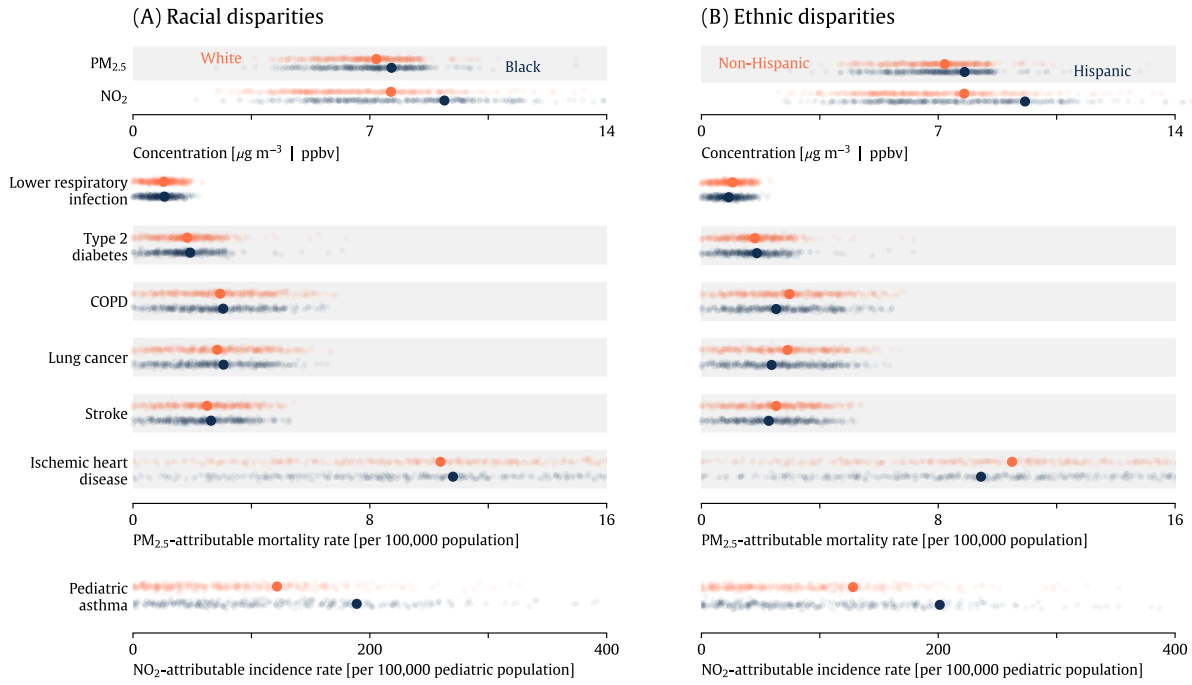

Figure S7. *Same as Figure 3 in the main text but concentrations or rates are formed with population-weighted categories rather than population subgroups.*

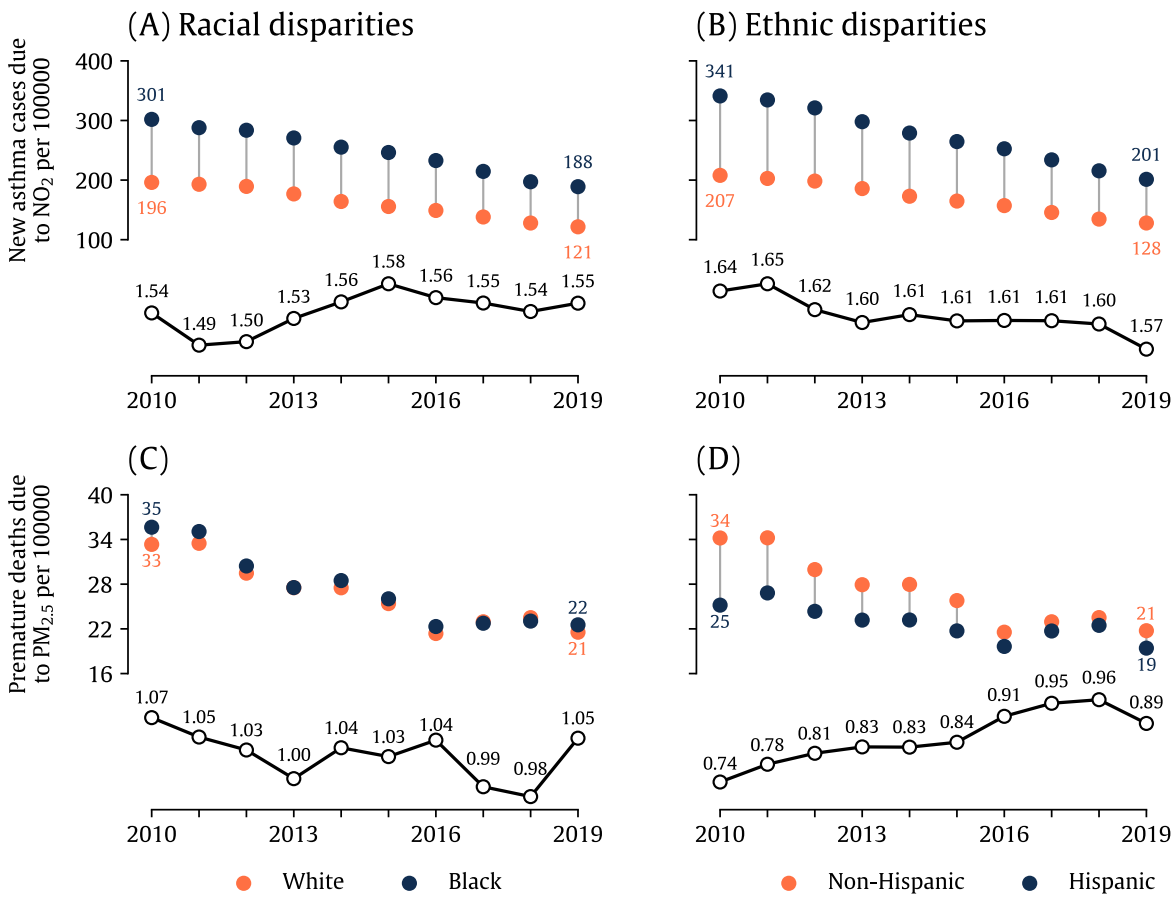

Figure S8. *Same as Figure 4 in the main text but rates and disparities are calculated using population-weighting.*