Quantifying the Effect of Upstream Maternal Risk Factors on Racial/Ethnic Disparities in Neonatal Health across the United States from 2000-2019

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Abstract

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This study examined racial/ethnic disparities in newborn health and the extent to which differential exposure to three maternal risk factors – air pollution, cigarette smoking during pregnancy, and gestational hypertension – contributes to these disparities. Evidence presented here supports previous findings that racial/ethnic minorities have higher rates of low birthweight and preterm births. Specifically, infants born to NH Black women are substantially more likely to be born low birthweight and preterm. Additional evidence presented here also supports existing findings that infants born to NH Black women are more likely to die during the neonatal period than infants born to mothers of other races/ethnicities.

Introduction

Health outcomes in the United States are widely known to vary by race/ethnicity. In recent decades, addressing racial/ethnic disparities has been a key public health priority. One topic that has specifically been a focus is racial/ethnic disparities in the health of newborns. Previous studies have shown that babies of racial/ethnic minorities are more likely to experience poorer neonatal health outcomes than white babies (1). The magnitude of this disparity is most notable for Non-Hispanic Black babies, with existing evidence suggesting that compared to Non-Hispanic white babies, they are 1.5 times more likely to be born preterm, 2 times more likely to be born under 2500g (low birthweight), and 2.5 times more likely to die within the first year of life (1).

Until about the turn of the 21st century, many experts believed that racial/ethnic differences in newborn health were primarily driven by race/ethnicity-specific genetic factors (2). While many risk factors were understood to affect health outcomes, it was also thought that babies of certain racial/ethnic groups were naturally more likely to be born low-birthweight for instance (2). A landmark study in 1997 by Richard David and James Collins disrupted this way of thinking (3). They compared the birthweights of infants born to US-born Black women, African-immigrant Black women, and US-born white women. Strikingly, the birthweights of infants born to African-immigrant Black women were more similar to those of US-born white women than US-born Black women. This largely debunked the theory that birthweight and other newborn health indicators were rooted in race/ethnicity-specific genetics (2). Instead, it suggested that the unique structural factors impacting Black women in America predispose their babies to suboptimal newborn health (2).

In recent years, literature in the field has made progress in distinguishing that race/ethnicity itself is not a risk factor for increased rates of health outcomes (4, 5). Instead, racism intertwined in key social structures causes minorities to experience higher rates of many conditions. As research into social determinants of health has expanded, a wide array of factors have been investigated for the role they play in explaining racial/ethnic disparities in newborn health (5). None of these comprehensively explain the disparity, but they reflect the complicated web of interrelated factors that influence health (4). These risk factors can generally be categorized as environmental, behavioral, or clinical risk factors (5). Here, I have chosen to assess the impact that one environmental, one behavioral, and one clinical risk factor have on racial/ethnic disparities in newborn health.

Environmental risk factors encompass a wide array of variables that are largely out of the control of the individual. They reflect the characteristics of an individual's setting and community. In this analysis, the environmental risk factor of ambient air pollution is considered. It is believed that inhalation of particulate matter induces inflammation in the lungs of the mother, with pro-inflammatory proteins – and potentially pollutants themselves – ultimately being transported to the fetus (6). This is thought to be detrimental to fetal development, leading to negative neonatal health outcomes (6). Exposure to air pollution is well-documented to vary

by race/ethnicity and is intrinsically linked to a history of racial residential segregation in the United States (7, 8). Policies such as redlining – a discriminatory mortgage appraisal practice originated in the 1930s – created urban environments in which racial/ethnic minorities lived closest to major sources of pollution (7, 8). Although the 1968 Fair Housing Act aimed to combat this type of discrimination, the direct effects of these policies on air pollution exposure are still felt today (9). This makes ambient air pollution a critical environmental risk factor to consider when assessing racial/ethnic disparities in newborn health outcomes.

Behavioral risk factors include variables that an individual has more control over than environmental risk factors. These often include practices that individuals have the autonomy to alter for themselves. Here, maternal cigarette smoking is explored as a key behavioral risk factor affecting newborn health. Similar to air pollution, maternal cigarette use is thought to cause pro-inflammatory proteins and pollutants to be transported to the fetus (10). This is exacerbated by the unique effects of nicotine which decreases the flow of nutrients and oxygen to the fetus, further hampering fetal development and leading to negative neonatal health outcomes (10). Notably, maternal smoking rates among several racial/ethnic minorities, including non-Hispanic Black (NH Black), non-Hispanic Asian or Pacific Islander (NH API), and especially Hispanic women have been found to be lower than that of Non-Hispanic White (NH White) women (11). However, maternal smoking rates among non-Hispanic American Indian/Alaska Native (NH AIAN) mothers stand out as the highest (11). Concerningly, smoking rates among NH AIAN women have not decreased in line with other racial/ethnic groups in recent decades as cigarette smoking rates decline in general (11). Maternal cigarette use is therefore an important behavioral risk factor to investigate specifically for the improvement of indigenous health.

Clinical risk factors reflect the pre-existing health status of individuals which may make the development of subsequent health outcomes increasingly likely. Here, I will assess the clinical risk factor of gestational hypertension. High-blood pressure during pregnancy is thought to be detrimental to the development of blood vessels that deliver nutrients to the fetus, contributing towards intrauterine growth restriction and poor neonatal health outcomes (12). Rates of gestational hypertension have been found to be higher among NH Black women as compared to other racial/ethnic groups (13). It is thought that increased exposure to several environmental risk factors – including air pollution – may contribute to higher gestational hypertension rates in NH Black women (13). In addition to worse neonatal health outcomes, babies born to mothers experiencing gestational hypertension are also more likely to develop cardiovascular disease later in life (14). Gestational hypertension is a key clinical risk factor with long-term implications for the health of children, especially NH Black children.

In this study, I seek to assess the extent to which ambient air pollution, maternal cigarette smoking, and gestational hypertension affect racial/ethnic disparities in birthweight, gestational age, and neonatal mortality. Using near-census level data from the US National Vital Statistics System (NVSS) from 2000-2019, I will assess observed distributions of birthweight and gestational age (15). I will then incorporate several cohort studies that establish how much those three risk factors impact birthweight and gestational age (16, 17, 18). After developing race/ethnicity-state-year specific counterfactual distributions of birthweight and gestational age, I

will compare how these distributions compare to the observed distributions – and how the the expected attributable disease burden change. In this way, I hope to quantify the magnitude to which these risk factors explain well-established racial/ethnic disparities in neonatal health.

Methods

Main Data Source

This study will incorporate US National Vital Statistics System (NVSS) data from 2000-2019 for all 50 states and the District of Columbia (15). This data source includes almost 100% of births over this time period and is therefore a near-census instead of a survey. Only singleton births will be included and only births from mothers who are residents of the state where the birth occurred will be included. Births without race/ethnicity, birthweight, and gestational age data were also excluded although these represent an extremely small portion of births.

Race/Ethnicity Definitions

Race/ethnicity categorizations are based on the reported race/ethnicity of the mother – as race/ethnicity of the child was not always reported – and uses the US Office of Management and Budget (OMB) 1997 stratification of race/ethnicity (19). Race options included in some years of the NVSS data are more granular than the OMB 1997 categories, but the OMB 1997 groupings were utilized as several years of the NVSS data only reported race data at this higher level. Table 1 below shows how more granular race categorizations were mapped to the higher level OMB 1997 categories. Any individuals that indicated their ethnicity to be Hispanic were included in the Hispanic category instead of the indicated race group. The OMB 1997 race/ethnicity groupings used in this study are Non-Hispanic American Indian or Alaskan Native, Non-Hispanic Asian or Pacific Islander, Non-Hispanic Black, Non-Hispanic White, and Hispanic. From 2014-2019, a new 'more than one race' option was added to the NVSS data, but as this was not comparable over a longer time series and only included a small portion of the overall dataset, these rows were not included in the analysis.

Table 1: *Mapping NVSS racial groups to OMB 1997 racial categories.*

OMB 1997 Race Categories	Race Categories Utilized in NVSS Data
Non-Hispanic White	White
Non-Hispanic Black	Black
Non-Hispanic Asian or Pacific Islander	Japanese, Chinese, Hawaiian, Filipino, Asian Indian, Samoan, Vietnamese, Korean, Guamanian, Other Asian or Pacific Islander, Other Asian, Other Pacific Islander
Non-Hispanic American Indian or Alaska Native	American Indian

Data Processing and Crosswalking

Birthweight values were not adjusted in data processing, as these were measured and reported in a consistent fashion. Gestational age values were adjusted during data processing as they were reported using two distinct approaches – an obstetrician's estimate (OE) and an assessment based on a woman's last menstrual period (LMP). Systematic biases were identified between the two approaches, with LMP estimates underestimating gestational age in pre-term births and overestimating gestational age in post-term births. To identify the appropriate correction factors, I fit a meta-regression—Bayesian, regularized, trimmed (MR-BRT) model to predict the log-adjusted ratio of LMP-estimated gestational age to OE-estimated gestational age across the domain of observed LMP gestational age values (20). A cubic spline was fit using knots placed at 20, 22, 28, 32, 47, and 42 weeks of LMP-estimated gestational age with a random effect on the year of the birth. The results of this model were used to standardize LMP gestational age values to what we would expect a corresponding OE value to be.

Figure 1: *MR-BRT model for the log-adjusted ratio of LMP-estimated gestational age to OE-estimated gestational age across the domain of LMP-estimated age.*

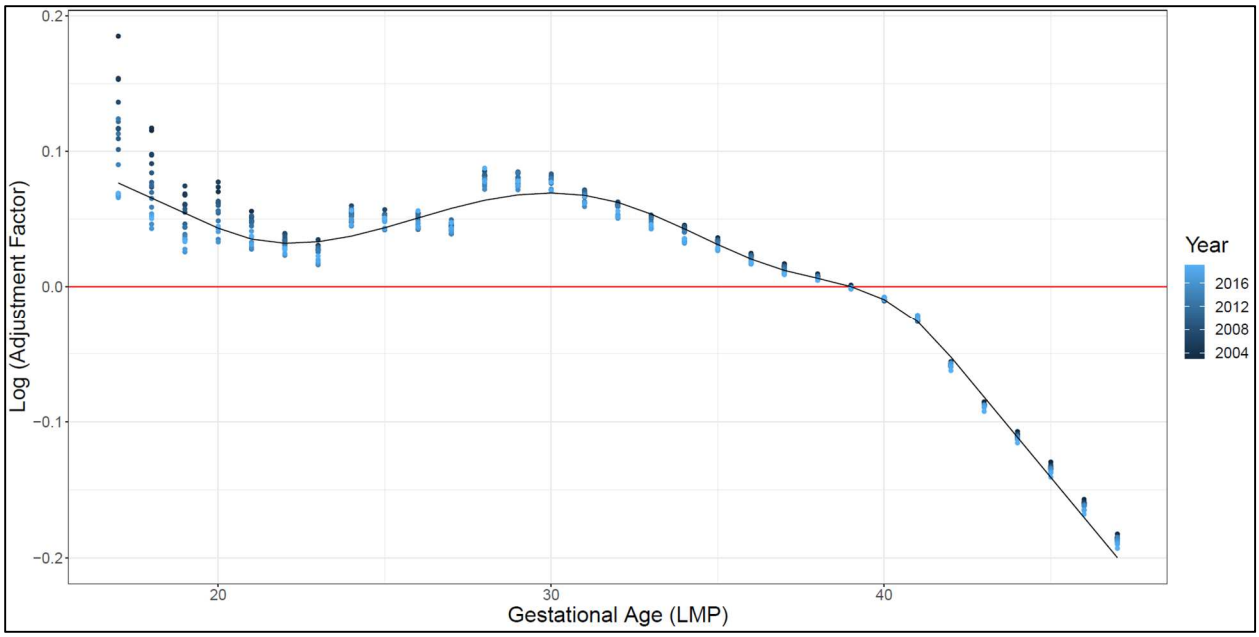


Table 2: *LMP Gestational ages and corresponding post-OE-standardized gestational age.*

GA (Last Menstrual Period) *weeks*	**GA (Obstetric Estimate)** *weeks*
17	18.35
18	19.21
19	20.06
20	20.88
21	21.75
22	22.72
23	23.77
24	24.91
25	26.11
26	27.35
27	28.61
28	29.84
29	31.03
30	32.15
31	33.16
32	34.05
33	34.81
34	35.48
35	36.10
36	36.74
37	37.45
38	38.23
39	39.00
40	39.62
41	39.94
42	39.85
43	39.62
44	39.36
45	39.09
46	38.80
47	38.49

Developing Counterfactuals

Three upstream risk factors were chosen that are known to have effects on birthweight and gestational age. One environmental risk factor (air pollution), one behavioral risk factor (cigarette smoking during pregnancy), and one clinical risk factor (gestational hypertension) were chosen to reflect the wide variety of upstream risk factors that impact birthweight and gestational age. NVSS includes individual level data on cigarette smoking during pregnancy and

gestational hypertension. Shifts were applied to the birthweights and gestational ages of babies born to mothers who experienced these exposures. Race/ethnicity-county-specific air pollution estimates were incorporated, using the county in which the birth occurred. These estimates were developed using granular race/ethnicity-specific populations from within census tracts to assess within-county variation in air pollution exposure.

For each risk factor, a meta-analysis or cohort study was identified that established the effect of the risk factor on birthweight and gestational age. The meta-analysis for air pollution includes a detailed dose-response relationship that allows for high levels of granularity in our analysis (16). The cohort-study for cigarette smoking includes a less granular dose-response relationship, distinguishing two levels of cigarette use (17). The cohort study for gestational hypertension only includes one exposure group, defining these individuals as pregnant women who experienced diastolic blood pressure equal to or above 90 mm Hg for the first time after 20 weeks of pregnancy (18). The tables below reflect the mean 'shifts' added to birthweights and gestational ages of women who experienced an exposure. For each applied shift, 100 draws of the shift were calculated to generate 100 counterfactual birthweights and gestational ages to propagate uncertainty.

Table 3: *Expected mean birthweight and gestational age increases for a range of ambient air pollution exposures.*

Air Pollution Exposure *($\mu g/m^3$)*	**Birthweight Shift** *g*	**Gestational Age Shift** *weeks*
< 4.2	0	0
5	1.08	0.04
6	2.44	0.09
7	3.77	0.14
8	5.10	0.19
9	6.41	0.24
10	7.71	0.29
11	9.00	0.34
12	10.29	0.39
13	11.55	0.44
14	12.81	0.48
15	14.06	0.53
16	15.29	0.58
17	16.52	0.62
18	17.73	0.67
19	18.94	0.71
20	20.13	0.75

Table 4: *Expected mean birthweight and gestational age increases for two levels of cigarette smoking during pregnancy.*

Maternal Cigarette Use *cigarettes/day*	**Birthweight Decrease** *g*	**Gestational Age Decrease** *weeks*
None	0	0
1-10	117	0.72
11+	351	1.01

Table 5: *Expected mean birthweight and gestational age increases for infants born to mothers experiencing gestational hypertension.*

Gestational Hypertension	**Birthweight Decrease** *g*	**Gestational Age Decrease** *weeks*
No	0	0
Yes	549	1.40

There was a limited number of race/ethnicity-state-years where data on maternal cigarette use and gestational hypertension had high missingness. In these few cases, instead of applying shifts to individuals experiencing the exposures of interest, the entire distribution itself was shifted. One hundred draws of counterfactual shifts for race/ethnicity-state-years with high missingness were estimated using linear imputation from race/ethnicity-state-years that had low missingness. Time trends in previously calculated mean shifts for that race/ethnicity-state were therefore leveraged to impute distribution shifts for years with lower data coverage of these variables.

Joint Distribution and Relative Risks

Observed birthweight and gestational age values – along with estimated counterfactual values – were grouped into joint bins of birthweight and gestational age to align with existing relative risk estimates. Relative risk estimates for all-cause mortality derived as a part of the Global Burden of Diseases, Injuries, and Risk Factors Study 2020 were utilized in subsequent calculations of population attributable fractions (21). These relative risks have been calculated for bins of 500g of birthweight by 2 weeks of gestational age for death among children aged 0-6 days and 7-27 days. Relative risks for each bin are shown in the figures below separately for both age groups.

Figure 2: *Relative risks for all-cause early neonatal (0-6 days) mortality by birthweight and gestational age.*

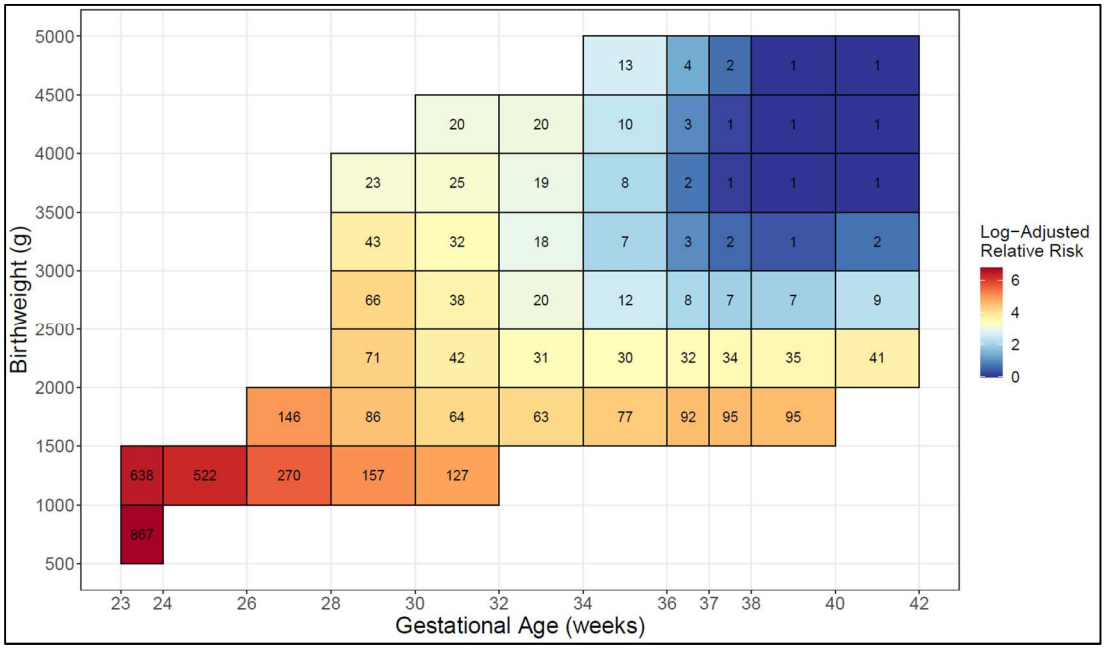
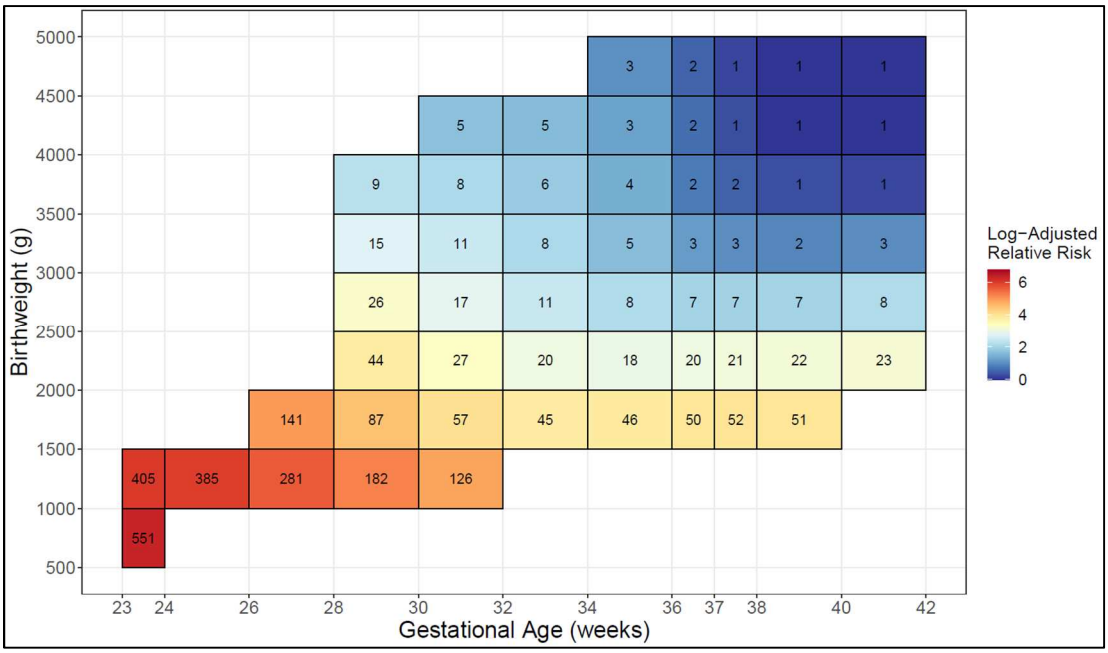


Figure 3: *Relative risks for all-cause late neonatal (7-28 days) mortality by birthweight and gestational age.*



Aging Birth Cohorts

Observed and counterfactual joint birthweight and gestational age distributions were aged through the neonatal period. To accurately reflect the disease burden attributable to birthweight and gestational age, prevalence in each joint birthweight and gestational bin must be adjusted to reflect deaths among children in each bin as children age. In this way, birth prevalence of the most high-risk birthweight and gestational age bins decreases from the early neonatal to late neonatal period, as these babies are most likely to die and therefore be removed from the total population. State-specific neonatal mortality information from the Global Burden of Diseases, Injuries, and Risk Factors Study 2020 was used to estimate how birth prevalences translated to prevalences in the early neonatal and late neonatal period (21).

Population Attributable Fractions

Population attributable fractions (PAFs) were calculated for the early neonatal and late neonatal age groups using the following formula, where *x* is a bin of joint birthweight and gestational age, *RR(x)* is the age-specific relative risk for all-cause mortality among kids born in bin *x*, *P(x)* is the age-specific prevalence of kids born in bin *x*, and *u* is the highest exposure bin.

$$PAF = \frac{(\sum_{x=1}^{u} RR(x) * P(x)) - 1}{\sum_{x=1}^{u} RR(x) * P(x)}$$

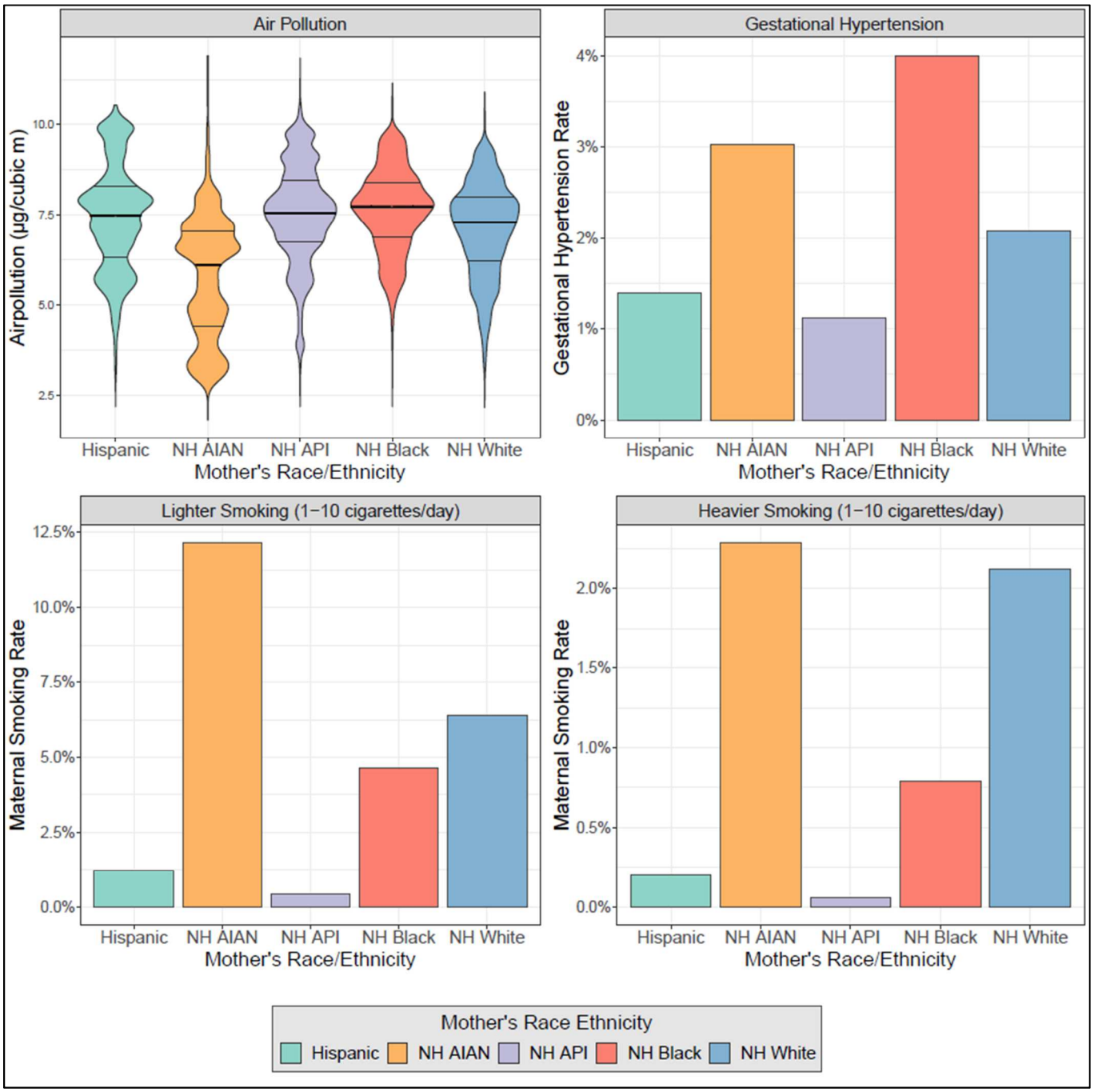
PAFs reflecting the entire neonatal period (0-28 days) were later aggregated using age-group-specific PAFs for the early and late neonatal periods and the total number of neonatal deaths in each age group. This aggregate PAF reflects the proportion of neonatal death that can be attributed to low birthweight and gestational age. PAFs were calculated for observed distributions as well as counterfactual distributions after shifts were applied.

Results

Exposure to upstream maternal risk factors varied substantially by race/ethnicity. Figure 4 below summarizes nationwide exposure to the selected environmental risk factor (ambient air pollution), behavioral risk factor (maternal cigarette smoking), and clinical risk factor (gestational hypertension) in 2019. The median air pollution exposure value is lowest for NH AIAN mothers at 6.18 $\mu g/m^3$ and NH White mothers at 7.28 $\mu g/m^3$. Median air pollution exposures were higher for Hispanic, NH API, and NH Black mothers, respectively at 7.57 $\mu g/m^3$, 7.61 $\mu g/m^3$, and 7.72 $\mu g/m^3$. Maternal smoking rates were highest among NH AIAN mothers, with 12.2% smoking 1-10 cigarettes per day and 2.3% smoking 11+ cigarettes per day. Although NH White mothers had only a slightly higher light smoking rate than NH Black mothers (6.4% compared to 4.6%), NH White mothers were much more likely to smoke 11+ cigarettes per day (2.1% compared to 0.8%). Smoking rates among Hispanic and NH API mothers were markedly lower than the other racial/ethnic groups with only 1.4% and 0.5% of mothers in these

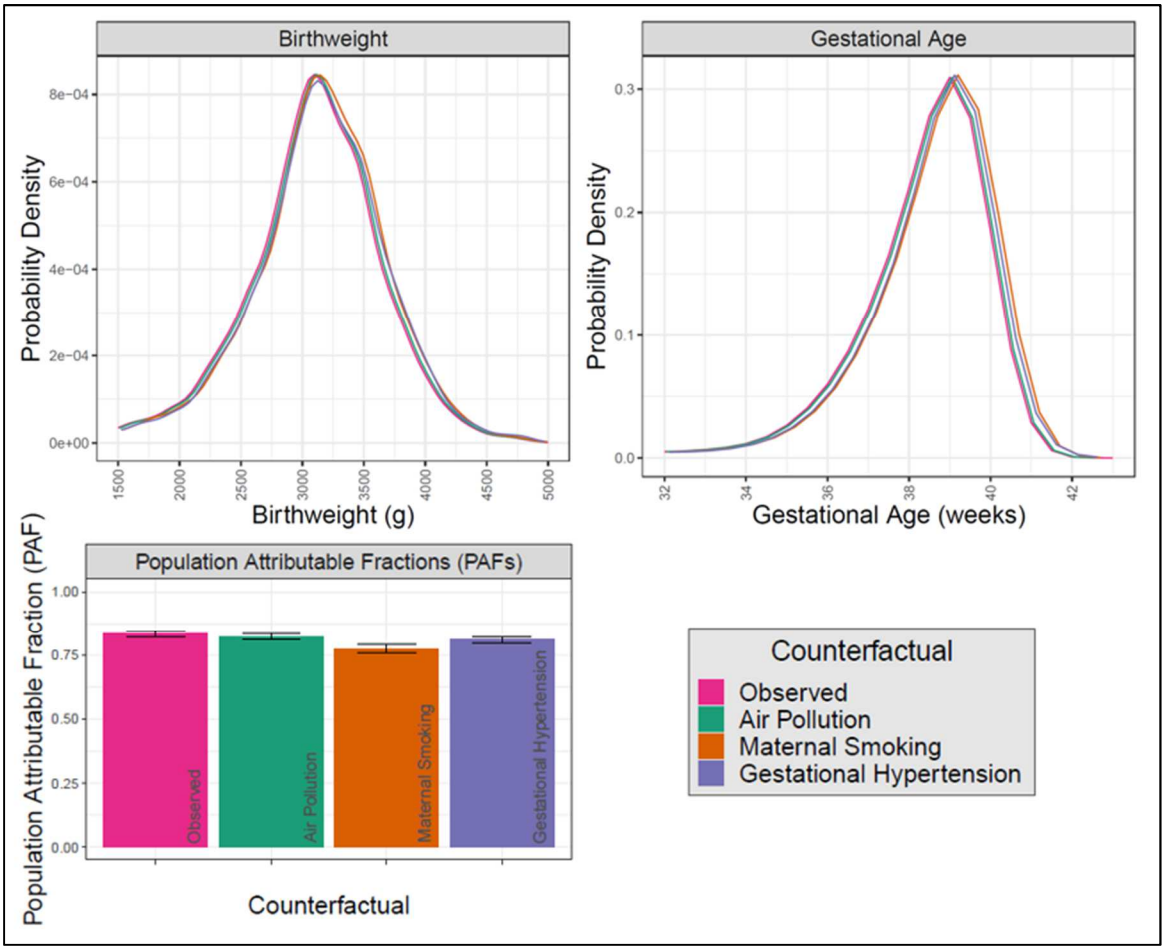
racial/ethnic groups smoking cigarettes at all during pregnancy. Gestational hypertension rates were highest among NH Black mothers at 4.0%, which was almost double the rate of NH White mothers at 2.1%. NH AIAN mothers also had higher rates of gestational hypertension at 3.0%, with Hispanic and NH API women having the lowest rates.

Figure 4: *Exposure to upstream maternal risk factors in 2019 by race/ethnicity.*



To illustrate the process by which observed birthweights and gestational ages were adjusted to create counterfactual distributions and counterfactual PAFs, results from the representative example of infants born to NH Black women in Wisconsin in 2019 have been shown in detail in Figure 5. Birthweights and gestational ages of infants born to mothers who experienced the three exposures were altered, generally shifting the distributions to the right. Both the rate of the exposure and the magnitude of the applied shifts influence the degree to which the counterfactual distribution differs from the observed distribution. Birthweights and gestational ages further to the right may fall into bins with lower relative risks – shown in figures 2 and 3. In this way, counterfactual PAFs may be lower than observed PAFs. In the case of PAFs among infants born to NH Black women in Wisconsin in 2019, the PAF is significantly lower in the maternal smoking and gestational hypertension counterfactuals. This suggests that the attributable burden of low birthweight and short gestational age would decrease if the effects of maternal smoking and gestational hypertension were removed. For this demographic-year, air pollution was not found to have a significant impact on the attributable burden of low birthweight and gestational age.

Figure 5: *Observed and counterfactual birthweight distributions, gestational age distributions, and population attributable fractions for infants born to NH White women in Washington in 2019.*



This process was repeated for all race/ethnicity-state-years. As in the previous example of infants born to NH Black women in Wisconsin, counterfactual preterm/low birthweight rates that remove the effects of smoking and gestational hypertension tend to be significantly lower than the observed rates. Conversely, preterm/low birthweight rates that remove the effect of air pollution often no statistically significant decrease compared to observed rates. Figures 6 and 7 show the observed and counterfactual rates of low birthweight and preterm birth for each race/ethnicity-state with more than 100 births in 2019. Across almost all states, rates of low birthweight range from 2.2% among babies born to NH AIAN mothers in Wyoming to 16.2% among babies born to NH Black mothers in Mississippi. Rates of preterm birth range from 7.4% among babies born to Hispanic women in West Virginia to 22.3% among babies born to NH Black women in Louisiana.

Figure 6: *Low birthweight rate by race/ethnicity-state-counterfactual in 2019. Only demographics with more than 100 births are included.*

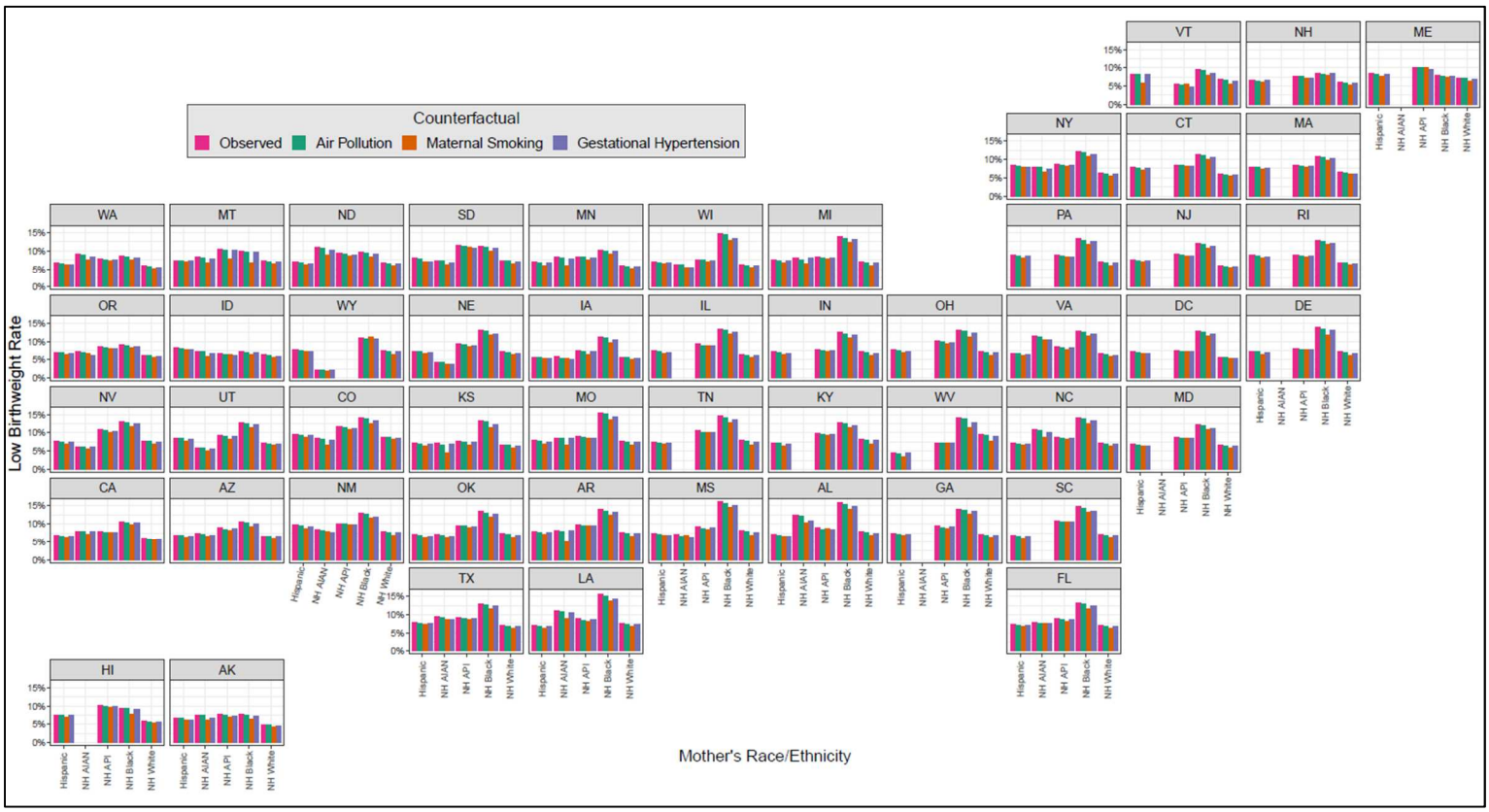
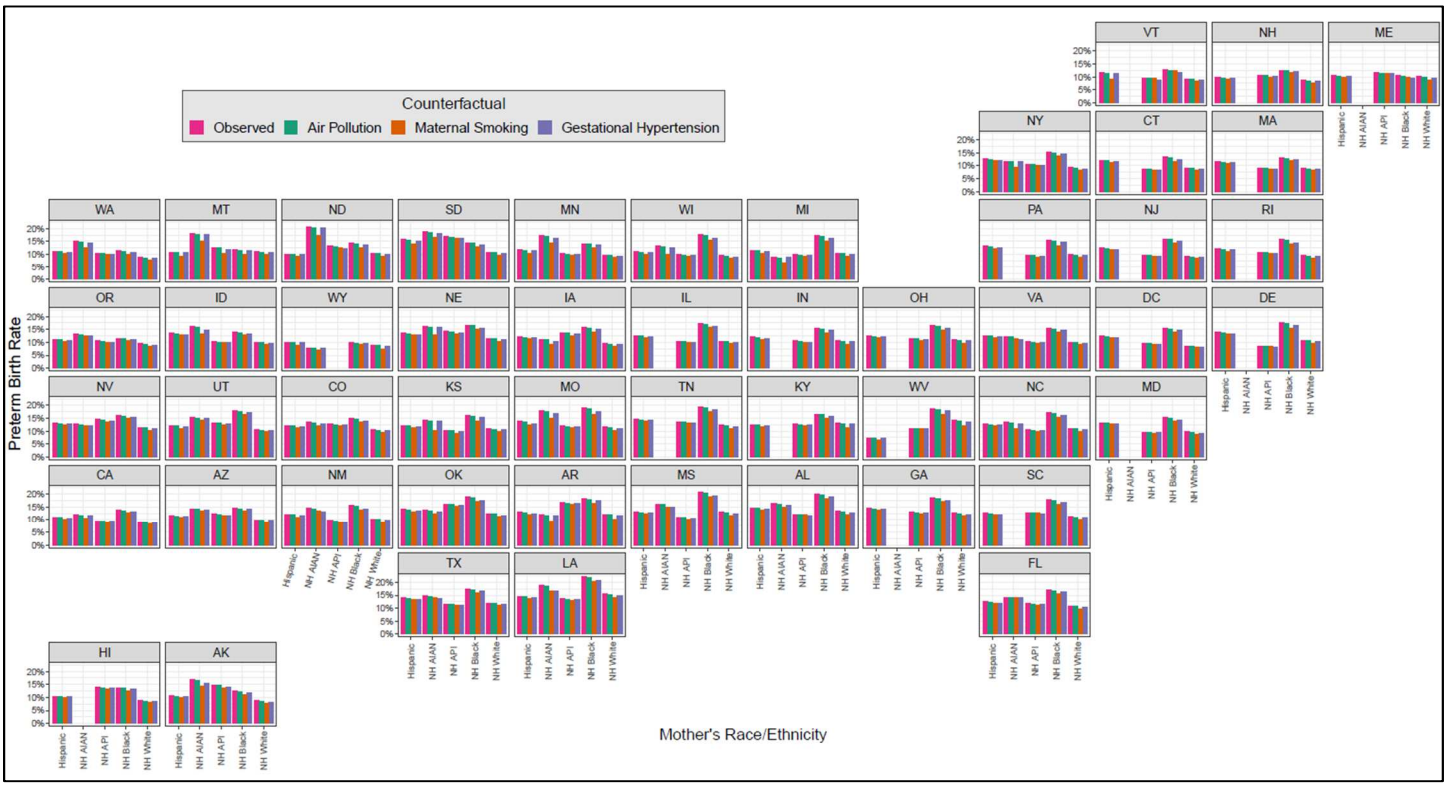
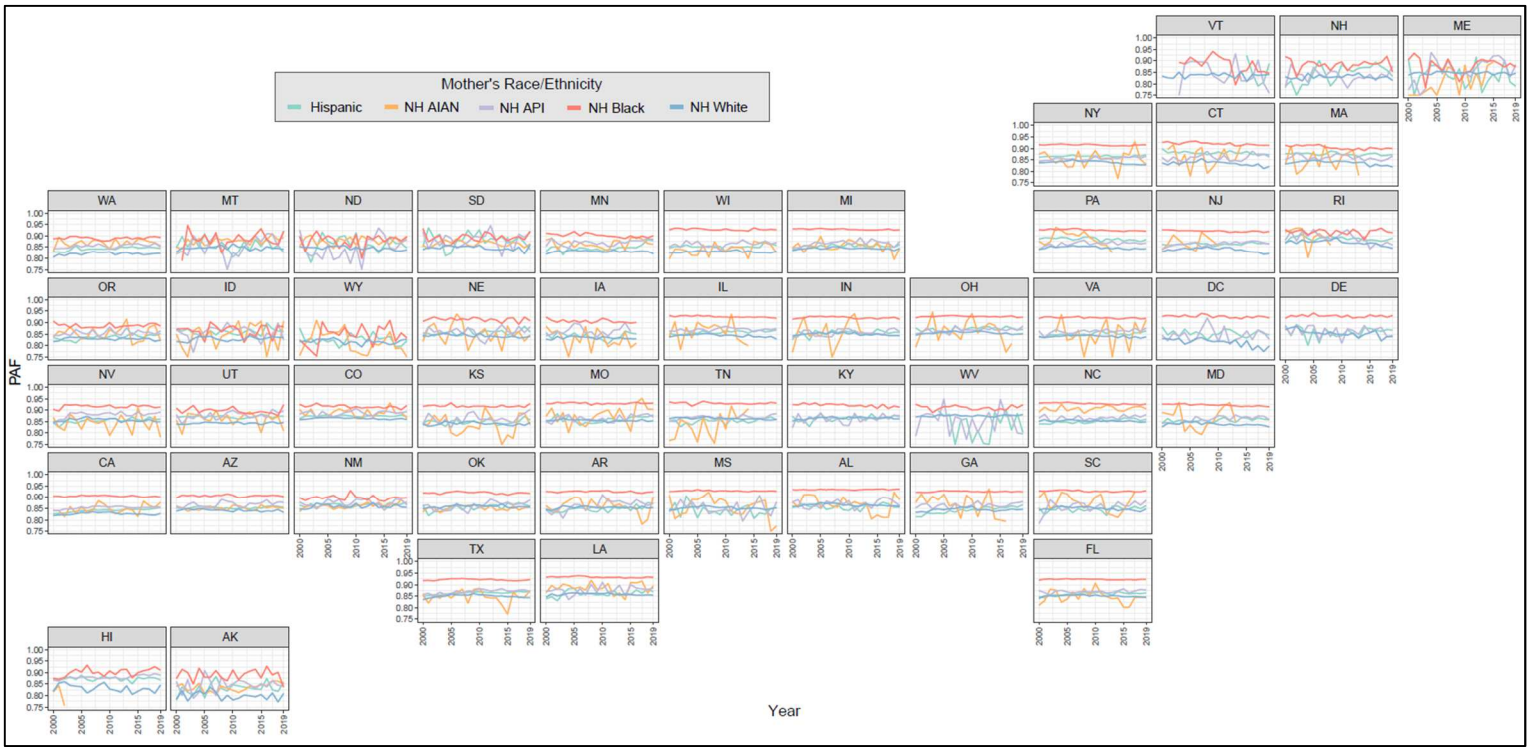


Figure 7: *Preterm birth rate by race/ethnicity-state-counterfactual in 2019. Only demographics with more than 100 births are included.*



Racial/ethnic differences in birthweight and gestational age distributions translate to racial/ethnic differences in population attributable fractions. Shown below in Figure 8, PAFs for infants born to NH Black mothers are consistently the highest among all racial/ethnic groups. Infants born to NH White mothers and NH API mothers tended to have the lowest PAFs. Although racial disparities in PAFs are consistent across states, the magnitude of these disparities varies. For example, even though infants born to NH Black mothers in Southwestern states like Colorado and New Mexico have the highest PAFs of any other racial/ethnic group, the disparity is not as extreme as it is in Midwestern states like Wisconsin and Michigan. In general, PAF values for low birthweight and short gestational age are high, reflecting the extent to which these risk factors influence neonatal mortality. For some state-years, PAFs for infants born to NH Black women can approach 95%, suggesting that 95% of neonatal mortality can be attributed to birthweight and gestational age.

Figure 8: *Population attributable fractions for low birthweight and short gestational age by race/ethnicity-state from 2000-2019.*



Counterfactual PAFs were compared to observed PAFs to estimate the extent to which three maternal risk factors explain neonatal disease burden. The magnitude of expected decreases in PAF were largest for maternal smoking and gestational hypertension. Removing the effect of air pollution was never found to reduce a PAF by more than 1%, and in most cases the PAF reduction was not statistically significant. In contrast, when the effect of maternal smoking was removed, PAFs often decreased significantly. For infants born to NH White and NH AIAN mothers – who had the highest smoking rates during pregnancy – the PAFs often decreased by 2% or more. PAF reductions for other racial/ethnic groups were commonly not significant. When the effect of gestational hypertension was removed, PAFs also decreased significantly, but no racial/ethnic category consistently achieved a greater reduction than others. In general, smoking and gestational hypertension appear to contribute marginally to the total observed PAF for low birthweight and short gestational age, but not substantially enough to explain existing racial disparities on their own.

Figure 9: *Expected decrease in PAFs if mothers were not exposed to air pollution by race/ethnicity-state from 2000-2019.*

Figure 10: *Expected decrease in PAFs if mothers did not smoke cigarettes during pregnancy by race/ethnicity-state from 2000-2019.*

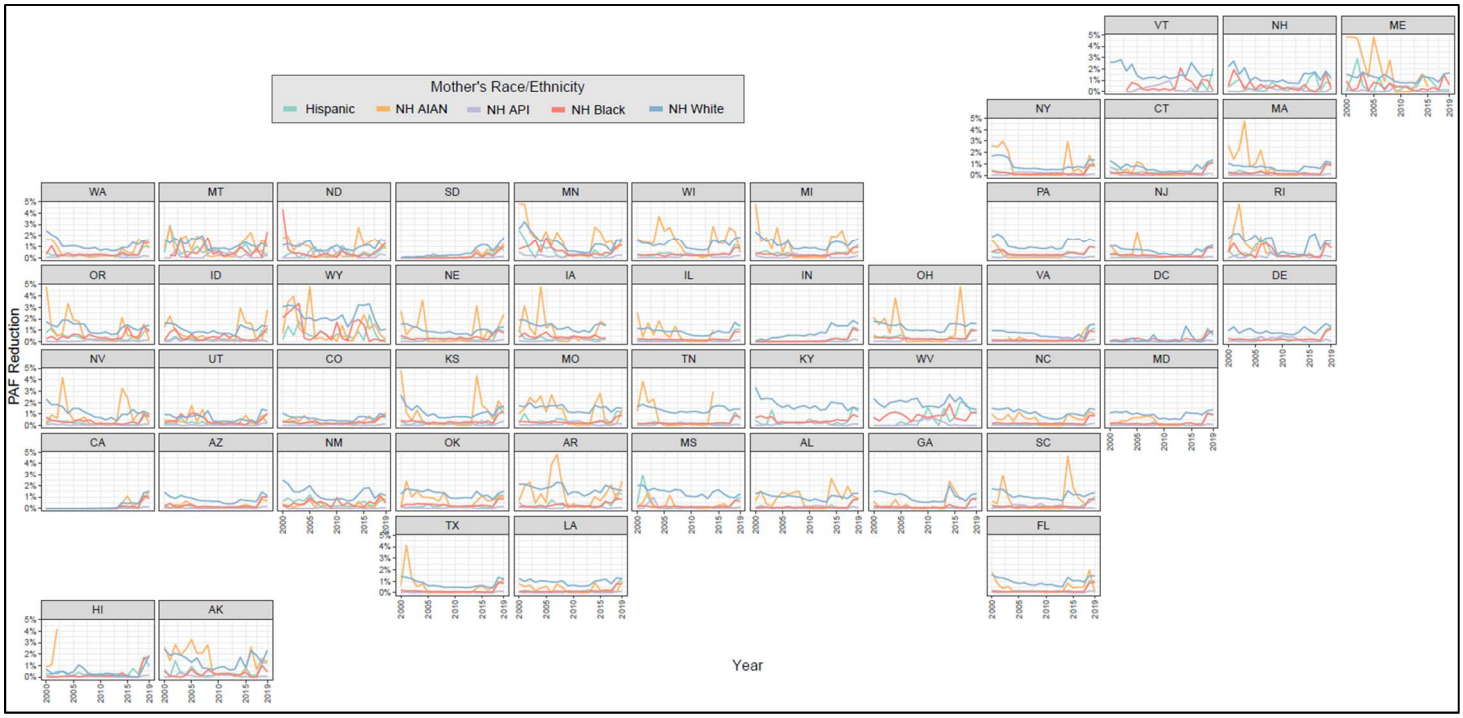
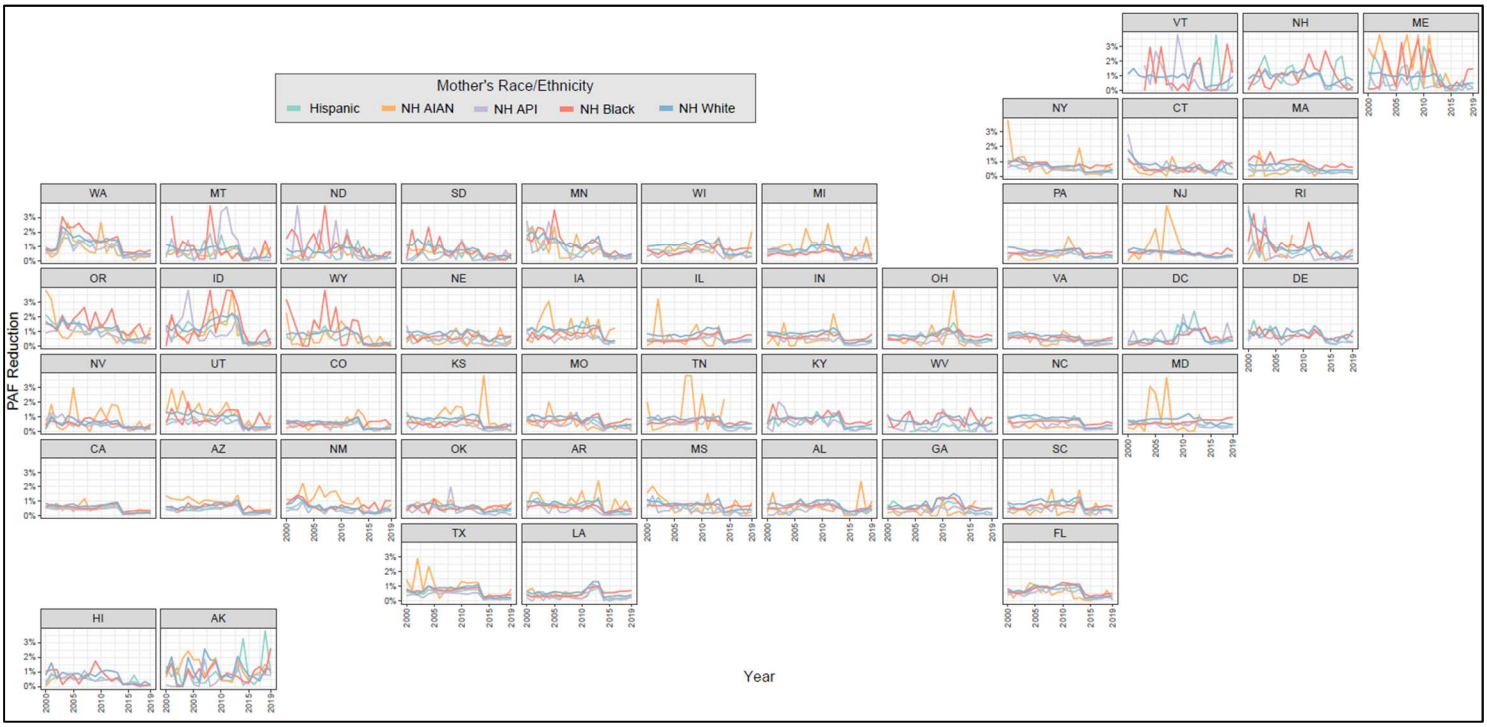


Figure 11: *Expected decrease in PAFs if mothers did develop gestational hypertension by race/ethnicity-state from 2000-2019.*



Discussion

This study examined racial/ethnic disparities in newborn health and the extent to which differential exposure to three maternal risk factors – air pollution, cigarette smoking during pregnancy, and gestational hypertension – contributes to these disparities. Evidence presented here supports previous findings that racial/ethnic minorities have higher rates of low birthweight and preterm births. Specifically, infants born to NH Black women are substantially more likely to be born low birthweight and preterm. Additional evidence presented here also supports existing findings that infants born to NH Black women are more likely to die during the neonatal period than infants born to mothers of other races/ethnicities.

A strength of this analysis is that it goes beyond assessing differences in low birthweight and preterm birth rates to examine changes in entire birthweight and gestational age distributions. It further considers the consequences of these changes for attributable burden. Racial/ethnic disparities are perhaps most apparent in PAF space, as slight differences in birthweight and gestational age distributions can have notable implications for PAFs considering the high magnitude of relative risks for extremely low birthweight and preterm babies.

Population attributable fractions were often significantly reduced in the counterfactuals that removed the effect of maternal smoking and gestational hypertension. PAFs for air pollution were rarely significantly lowered. Notably, even in cases where the maternal smoking and gestational hypertension counterfactual PAFs were significantly reduced, the magnitude of this reduction was only about 2%. This indicates that none of these three risk factors adequately explains a sizable portion of the health burden attributable to low birthweight or short gestational age at the population level. It must be clearly delineated that these results reflect the health consequences at the population level, as the health impact of these risk factors on individuals remain potent –as reflected in Table 3, Table 4, and Table 5 (16, 17, 18). Considering the relatively high birthweight and gestational age shifts associated with maternal smoking and gestational hypertension, these risk factors are particularly important.

This study has several limitations which hindered the analysis from being as comprehensive as possible. Firstly, only three risk factors were considered. Other environmental risk factors (such as lead exposure), behavioral risk factors (such as alcohol use), and clinical risk factors (such as diabetes) are known to contribute to low birthweight/preterm birth and it is possible these play a bigger role (22, 23, 24). It is also likely that certain infants are born to mothers who experience several of these exposures in tandem, perhaps worsening the extent to which they are born low birthweight or preterm.

Second, the quality of data used to inform counterfactual distributions could have been strengthened. The counterfactual shifts used to estimate the impact of cigarette smoking and gestational hypertension are based on single cohort studies instead of a meta-analysis (17, 18). Ideally, these shifts would consider a wide breadth of cohort studies, allowing for more granularity of the dose-response relationship especially with cigarette smoking. Importantly, the cigarette smoking shifts were applied to women who self-reported smoking cigarettes during pregnancy. This is known to be an underestimate of the true smoking rate due to stigma associated with the practice of smoking during pregnancy (25).

Third, relative risks utilized in PAF calculation could be more specific. The relative risk functions used in this analysis are not continuous, and are instead calculated for 500 gram by 2 week bins of birthweight and gestational age (21). Utilized counterfactual shifts are often less than these increments, meaning that many births likely still fell in the same risk bin even with the applied shifts (16, 17, 18). Continuous shifts would help capture additionally incurred risk for births that stay within the same 500 gram by 2 week bin. Further, relative risks derived from the Global Burden of Diseases, Injuries, and Risk Factors Study 2020 do not vary by race/ethnicity (21). This is not reflective of reality, but race/ethnicity specific relative risks are not available to our knowledge. Many factors – including medical racism – have been linked to higher mortality rates among infants born to NH Black women (26, 27). Unfortunately, mortality information used to age the birth cohort forward to the early and late neonatal periods were not available for each specific racial/ethnic group.

Lastly, it is crucial to acknowledge that OMB 1997 categorizations of race/ethnicity are not necessarily comprehensive and mask within-group heterogeneity (19). Perhaps the most striking example of this is in the case of Asian and Pacific Islanders, which are grouped together

in the OMB 1997 categorization of race/ethnicity. In 2019, the rate of preterm birth among babies born to Asian (Chinese, Filipino, Indian, Korean, Vietnamese, Other Asian) mothers was 10.1% compared to 16.0% among babies born to Pacific Islander (Guamanian, Hawaiian, Samoan, Other Pacific Islander) mothers. This underscores that these racial/ethnic groupings are not perfect and can potentially obscure meaningful disparities.

Racial disparities in health have historically been presented in the literature without identifying specific drivers of those disparities. Often they are prescribed to nebulous factors such as education or income, which may indirectly influence health. In this way, many studies simply identify racial disparities without mechanistically or clinically explaining them. Historically, genetics have even been blamed when mechanisms weren't presented. Recently, studies have begun to consider differential racial/ethnic exposure to risk factors with well understood mechanisms for impacting health. This approach yields findings that are more actionable for policymakers who seek to address racial/ethnic disparities in health.

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