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Racial disparities in preterm birth in the US; a biosensor of physical and social environmental exposures

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Abstract

Race is a social construct that involves a person's self-assigned, and externally-perceived, group membership. Group membership can determine much about Americans' lives and health. Complex health disorders such as cardiovascular disease, asthma, and obesity disproportionately affect Non-Hispanic black Americans. An *individual's* risk of any of these disorders encompasses both genetic predisposition and environmental stimuli. We propose that environmental stressors may be large contributors to differences in preterm birth rates in the United States between *racial groups*. Environmental exposures differ by race due to ongoing residential, educational and economic racial segregation as well as discrimination. Characterizing and mitigating environmental factors that contribute to differential preterm risk could identify women at risk, prevent some preterm births, and reduce perinatal health disparities.

Keywords

Race; pregnancy; preterm birth; disparities; environment

Introduction

The infant mortality rate in the United States exceeds that of most other developed nations, ranking 26th among Organisation for Economic Co-operation and Development countries.[1] Non-Hispanic black infants in the United States die more than twice as often as non-

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Hispanic white infants (11.4 versus 4.9 per 1,000 live births).[2] This disparity reflects disparities in preterm birth (PTB) rates, since two-thirds of infant mortality occurs in preterm infants.[3] The PTB rate is 52 percent higher for black (13.8%) than white (9.0%) women. Efforts to reduce PTB and its disparities have failed (Figure 1). We propose that racial disparities in PTB are a cumulative biosensor of exposures that vary by race, arising from longstanding inequities.

PTB disparities are not due to genetic sequence variation between racial groups

While some monogenic diseases track (incompletely) with race, such as sickle cell anemia and cystic fibrosis, the vast majority of health conditions cannot be mapped to genetic variation between racial groups. Most human genetic variation is found within ancestral groups with only 5–10% of gene frequencies differing between ancestral groups.[4] Nonetheless, different frequencies of single nucleotide polymorphisms (SNPs) by race have led some investigators to search for genetic differences that cause racial disparities in PTB. However, SNPs explain an exceedingly small portion of PTB risk, and are often not replicated.[5,6] Some strong evidence supports that disparities in birth outcomes are largely attributable to environmental, as opposed to genetic variation. One example is the phenomenon of erosion of immigrant health over generations. Birth weight (BWT) distributions of infants born to African-born black women and US-born white women nearly overlap, whereas infants born to US-born black women were substantially smaller.[7] Indeed in a study of 27 states' births in 2008, foreign-born black women had significantly lower odds of PTB than US-born black women even after adjustment for sociodemographic, health behavioral and medical risk factors (adj. OR 0.727; CI 0.726–0.727).[8] Others have used twin and kinship studies to analyze the genetic versus the environmental contributions to PTB. These find that the pooled genetic contribution (<35%) is dwarfed by non-genetic or broadly-defined environmental influences, particularly when self-identified African Americans are compared to self-identified European Americans.[9,10] Further, PTB is a heterogeneous phenotype comprised of distinct pathophysiologic pathways. Spontaneous PTB results from premature cervical remodeling and/or myometrial contractility, whereas medically-indicated PTB occurs when providers elect to deliver a fetus due to maternal or fetal conditions such as preeclampsia or intrauterine growth restriction. Black women are at higher risk of both spontaneous and medically-indicated PTB,[11] and it is unlikely that a set of genetic sequences that track with race would lead to such different phenotypes. Given the small contribution of genetics to PTB, coupled with the very small differences in genetic sequences between racial groups in the US, broadly-defined environmental factors must be responsible for racial disparities in PTB.

Environmental factors must play a role in black-white disparities in PTB

We propose a framework wherein micro- and macro-environmental factors shape the likelihood of a healthy, term delivery (Figure 2). Micro-environmental factors include behavioral factors where an element of individual choice can be made, such as smoking and diet. Macro-environmental factors include social and physical environmental exposures such

as neighborhood violence, air and water pollution, heavy metals, and other exposures that are not directly due to an individual's decisions, but are present in the environment. Both micro- and macro-environmental exposures can affect an *individual* woman's physiology, and alter the likelihood of delivering preterm. We will argue that the macro-environment plays a significant and under-appreciated role in *population*-level disparities.

Micro-environmental incompletely explain black-white differences in PTB

To implicate an exposure as a contributing factor to black-white disparities, it must be more highly prevalent among black women. Smoking is one of the strongest risk factors for PTB.[12] Differences in smoking rates do not explain differences in PTB risk. Only 5.6% of Non-Hispanic black women report smoking in pregnancy, compared with 10.1% of Non-Hispanic white women.[13] White women who smoke have similar PTB rates (12.9%) to black women who do not smoke (13.5%), suggesting that while smoking increased individuals' risk of PTB, differential PTB rates between races is not due to smoking.

Diets are culturally patterned practices that can differ by race/ethnicity.[14] No single dietary practice has been implicated in PTB. A key micronutrient during pregnancy hypothesized to be involved in racial disparities in health is vitamin D. Unlike smoking, vitamin D deficiency is more common among black women; lighter pigmented skin is more efficient at synthesizing vitamin D from sunlight.[15] While some observational studies have shown associations between vitamin D deficiency and PTB,[16] intervention trials have not. In a trial in South Carolina, 257 women were randomized to three different doses of vitamin D supplementation and while 25(OH)D levels increased according to dose, there was no difference in PTB.[17] In a recent, large trial of 1164 women randomized to high doses of monthly vitamin D vs. placebo in Bangladesh, there were no differences in birth outcomes.[18] These results suggest that supplementing vitamin D for black women is unlikely to mitigate disparities in PTB.

Iron deficiency and anemia, which are more common among African Americans,[19,20] has likewise been associated with PTB in observational studies,[21] but randomized controlled trials of iron supplementation have been disappointing.[22] In a recent study, over 15,000 women were randomized to one of three interventions: folic acid alone, folic acid with iron, and multiple micronutrients.[23] While the last group had a significant decrease in spontaneous PTB, there was no difference in the folic acid with iron group compared with the folic acid only group. Simply treating iron deficiency is unlikely to mitigate racial disparities in PTB.

Macro-environmental factors that may help to explain racial disparities in PTB

A large body of evidence has connected neighborhoods to health.[24] This is particularly relevant due to residential racial segregation and environmental inequity between black and white neighborhoods.[25,26] The extent to which segregation and disproportionate exposure to toxic environments among black families contributes to racial disparities in PTB is unknown. Yet, evidence is mounting to connect physical and social environmental exposures

to PTB risk. Most implicated exposures are concentrated in black neighborhoods, and black women living in racially-segregated black neighborhoods have higher risk of PTB than black women living in integrated neighborhoods.[27]

Physical environmental exposures

Several physical environmental exposures have well-documented associations with PTB risk and disproportionately affect black families. Air pollution exposure increases the risk of PTB[28,29] and black Americans are more highly exposed due to residential proximity to industrial facilities,[30] traffic,[31] and other sources. One recent study of over 37,000 births in China demonstrated that as interquartile range of short-term exposure to gases (SO_2, and NO_2) and particulate matter (<2.5 ($PM_{2.5}$) and <10 (PM_{10}) microns in diameter) increased, the odds of PTB were 3.7–6.5 times higher.[32] Lead exposures also disproportionately affect black families.[33] According to the Environmental Protection Agency's biomonitoring program, regardless of poverty level, black children (1.1 μg/dL) have higher median lead levels than white children (0.8 μg/dL) and are twice as likely to have elevated levels (>5 ug/dL).[34,35] Lead has been shown to be associated with increased risk of PTB.[36] A recent case-control study (n=102 preterm cases and 306 term controls) also in China found that infants in the highest tertile of lead levels, versus the lowest, had higher odds of being born preterm (adj. OR 2.96, CI: 1.49–5.87).[37]

Phthalates have also been shown to increase the risk of PTB.[38] These ubiquitous chemicals are used as plasticizers, and are components of personal care products, fast food packaging, and building materials (e.g., flooring). Phthalate metabolite levels can vary by race/ethnicity due micro-environmental [39] as well as macro-environmental exposures.[40] In a prospective, nested case-control study (n=130 cases, 352 controls) in Boston, women who delivered preterm had higher geometric means of several phthalate metabolites and higher odds of spontaneous PTB (ORs = 1.22–1.67 per ln-unit increase). While evidence that physical environmental exposures contribute to PTB risk is accumulating, it is not yet clear what proportion of racial disparities in PTB is attributable to differences in the physical environment.

Social human environmental exposures

Social environments (human interactions) also strongly affect PTB risk. Exposure to racism and discrimination is associated with increased risk of PTB.[41,42] In a case-control study of 312 black women (104 cases and 208 controls) in Chicago, women who reported high levels of exposure to lifetime interpersonal racism were more likely to have delivered a very low BWT (<1,500g) infant than an infant >2,500g (adj. OR 2.6, CI: 1.2–5.3).[41]

Violent crime, a severe chronic urban stressor, is often concentrated in lower-income and minority neighborhoods,[43] and has been shown to affect birth outcomes. A study in North Carolina found that among over 30,000 live births, neighborhood exposure to violent crime was associated with PTB;[44] notably, the range of violent crime counts near black women was higher than for white women with so little overlap, that race-specific tertiles were necessary for analysis. Recently, investigators used a natural experiment to perform a population-based longitudinal analysis of BWT in Mexico before and after the rise of the

Mexican Drug War. Increase in violent crime was associated with lower BWT after adjustment for maternal and community confounding variables.[45] Like physical environmental exposures, the extent to which differential social exposures by race may explain racial disparities in PTB is unknown.

Interaction of physical and social environments

In 1992, Geronimus proposed an analytic framework to explain why black women had increased risk of adverse perinatal outcomes.[46] She postulated that black-white infant mortality differential among infants born to older (versus younger) women was due to the cumulative effects of socioeconomic disadvantage. One potential mechanism transmitting socioeconomic disadvantage to racial disparities is through increasing susceptibility to physical exposures through chronic stress and related physiologic impacts on immune and metabolic function (allostatic load).[47] Indeed there is evidence that social and physical environments interact to result in differential birth outcomes.[48] Ponce et al analyzed birth certificate data for 37,347 deliveries in California, and found that air pollution was associated with odds of PTB only among women in the lowest socioeconomic stratum.[49] A recent analysis of 53,843 mother-infant pairs in California, used an exposure index to include air, land, and water pollution sources.[50] Women in the highest quintile of exposure had twice the odds of PTB versus women in the lowest quintile, and, dichotomizing the index, only women in the low-socioeconomic stratum had elevated odds of PTB with higher pollution exposures.[50] These data suggest that socioeconomic disadvantage changes susceptibility to physical environments.

Social and physical inequity is not limited to residential life. Employment opportunities vary by race in the United States resulting in differential physical occupational exposures[51] and higher levels of job strain defined as high-demand and low-control in the workplace,[52] among black workers.[53] In a case-control (n=1,242 preterm cases, 4,413 term controls) study in Quebec, Croteau and colleagues found that women with high levels of job strain including physically demanding postures and lack of control at work had higher odds of PTB.[54] Further in a study of black and white women in North Carolina, job strain was associated with higher odds of PTB, specifically among black women (OR 1.8, 95% CI 1.1–3.1) compared with white women (OR 1.0, 95% CI: 0.5–2.0).[55] Taken together, given that race often tracks with socioeconomic position that determines where women live and work, interactions of physical and social environments likely explain much of the racial disparity in PTB.

Improving education alone will not solve disparities

One way to improve socioeconomic position is through education. However, education alone does not eliminate birth outcome disparities. In 1992, Schoendorf demonstrated that racial disparities in infant mortality existed even among infants born to college-educated women and that the disparity was entirely explained by differential risk of LBW (7% and 3% among black and white infants, respectively).[56] This phenomenon persists today. Disparities widen as education levels rise, as shown in Figure 3, which displays PTB rates by education level in 2016 in the US.[13] These data suggest that education alone does not mitigate exposures that differ by race over a lifetime. Whether racial disparities among well-educated women

are due to earlier life exposures (or even exposure to prior generations that could be passed along through epigenetic phenomena) or due to ongoing exposure to racism or other environmental stressors, which are not eliminated with education, remains unknown.

The study complexity needed to address racial disparities in PTB

Tackling racial disparities in PTB will require rigorously quantifying the relative contribution of adverse environmental factors that are disproportionately concentrated in black communities. One way this could be studied is by analyzing interracial couples with one black parent and one white parent. A recent population-based study of over 1.6 million live-births in California demonstrated that couples where one parent is black and the other is white have lower risk of spontaneous PTB than when both parents are black; the risk reduction was greater when the woman was white (adjusted OR: 0.7, 95% CI: 0.7–0.8) than when the man was white (adjusted OR: 0.9, 95% CI: 0.8–1.0).[57] While the risk reduction may be due to unmeasured socioeconomic position differences between couples who have interracial marriages compared to black couples, understanding which exposures differ among these families may shed light on environmental causes of PTB. Investigation into how the environment may shape racial disparities in preterm birth requires interdisciplinary collaboration between perinatal and environmental scientists, sociologists and other social scientists, biostatisticians, epidemiologists, and public health professionals. Identifying priorities for environmental exposure reduction is a critical step to motivate policy efforts to reduce racial disparities in PTB.

The importance of phenotyping PTBs

Due to the small impact of any individual environmental exposure on PTB, it is critical to clarify which subtype of PTB is affected, and to have large study populations. Otherwise associations can be missed (Type II error). Theoretically, if a toxic chemical affects the risk of preterm labor (one presentation of spontaneous PTB) with a relative risk of 1.4, but not preterm preeclampsia (one presentation of medically-indicated PTB) with a relative risk of 0.9, the overall relative risk of PTB from the chemical might be 1.2 with a confidence interval that crosses 1. In this case, the chemical might be deemed falsely benign with respect to PTB. Phenotyping PTBs is resource-intensive, but will be required for detecting PTB subtype-specific environmental effects.

Consideration of mixtures

Because many physical and social environmental exposures co-locate, considering the effects of mixtures is also extremely important given the small impacts of single exposures. For example, a single elevated phthalate may not alone cause spontaneous PTB, but combined with air pollution, lead, and violence, the phthalate could be the last necessary stressor to trigger premature myometrial contractility or cervical remodeling necessary for spontaneous PTB.

Getting closer to causation

Given the many variables that potentially confound an environmental exposure’s effect on PTB, demonstrating that a toxic chemical affects the risk of a specific PTB phenotype

among black women within an educational or income stratum would help to identify true targets. Better causal inference modeling, including mediation may also facilitate a better understanding as to the relative contributions of multiple exposures.[58] Multilevel modeling can also clarify the relative contribution of neighborhood factors versus individual-level factors.[59,60] Additionally, collaborating with translational scientists and toxicologists to identify biologic pathways by which toxic chemicals could lead to a specific PTB phenotype in animal models or in-vitro can help to clarify whether as association is more likely to be truly causal.

Realizing policy change

It is important to focus on social and environmental determinants of health in order to reduce disparities, as opposed to simply improving medical care for all which can sometimes widen black-white gaps.[61] Once the targets that are likely both causative agents for PTB and disproportionately concentrated in black communities are identified, policy changes could reduce disparities. The built environment can affect physical activity through walkability and recreational resources. Local food environments can affect diet quality. Together these factors can affect likelihood of diabetes and hypertension[24] which can be associated with the risk of medically-indicated PTB.[62] Additionally, clean-up efforts can improve birth outcomes. Recently, a study of over 57,000 births within 20 miles of coal power plants that were retired between 2001 and 2011 demonstrated significant reductions in PTB after the closures with the largest impact among black women.[63] Ultimately, exposure reduction of multiple implicated exposures will be required to narrow the racial gap in PTB that has persisted for decades.

Conclusion

Longstanding racial disparities in PTB are likely largely due to social and physical exposures that vary by race due to enduring inequity in the United States. The next frontier in tackling perinatal health disparities will require studying and rectifying the injustices of unequal environments. Only then will black families have an equal chance of healthy, full term births.

Abbreviations:

PTB	preterm birth
BWT	birth weight
CI	confidence interval

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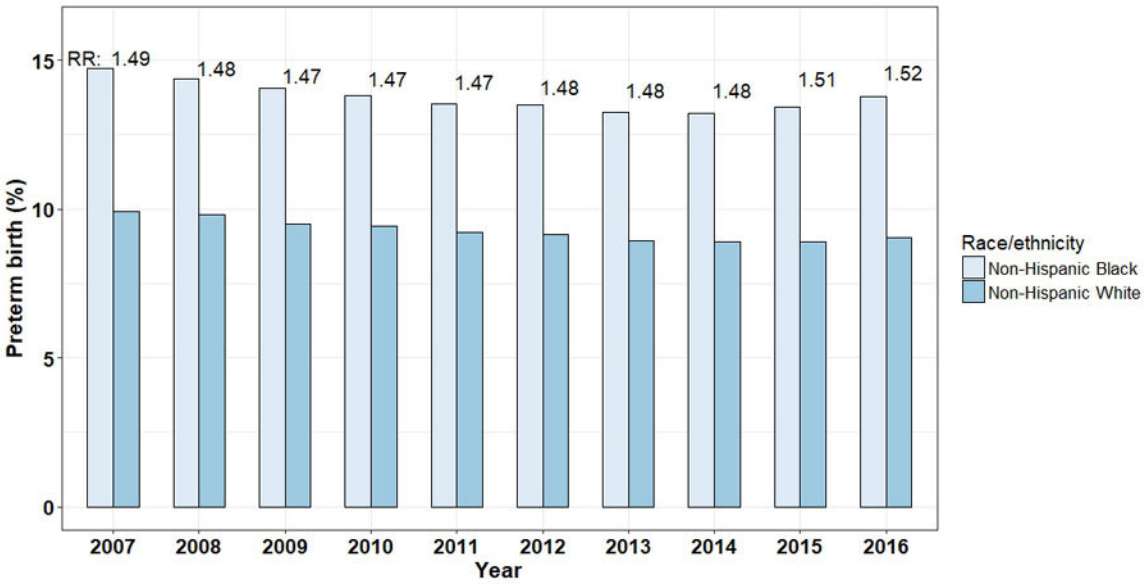

Figure 1.

Black-white disparities in preterm birth over 10 years in the US[64,65]; RR=relative risk

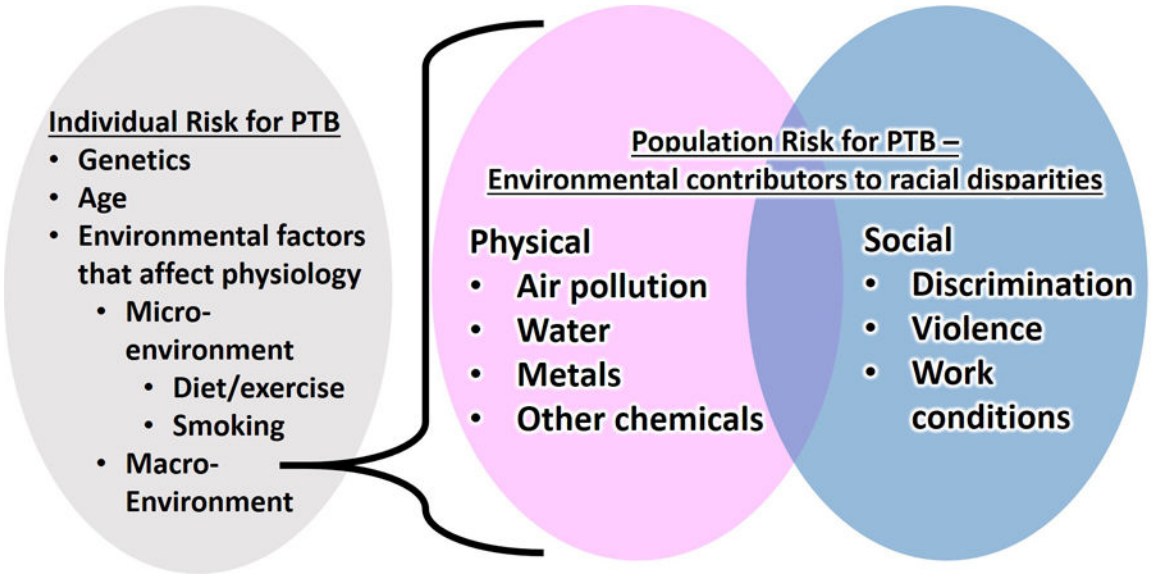

Figure 2.
Expanded framework for environmental exposures that shape racial disparities in preterm birth (PTB)

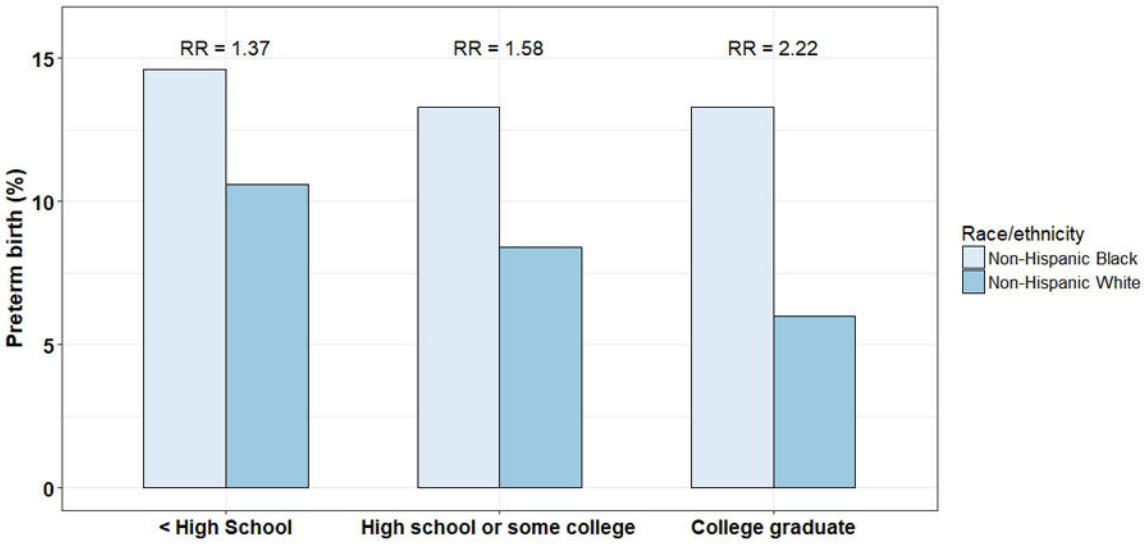

Figure 3.

Black-white disparities in preterm birth according to maternal education in the US. Data from CDC Wonder, singleton births from 2016;[13] RR=relative risk