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Birth outcome racial disparities: a result of intersecting social and environmental factors

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Abstract

Adverse birth outcomes such as preterm birth, low birth weight and infant mortality continue to disproportionately affect black and poor infants in the United States. Improvements in healthcare quality and access have not eliminated these disparities. The objective of this review was to consider societal factors, including suboptimal education, income inequality, and residential segregation, that together lead to toxic environmental exposures and psychosocial stress. Many toxic chemicals, as well as psychosocial stress, contribute to the risk of adverse birth outcomes and black women often are more highly exposed than white women. The extent to which environmental exposures combine with stress and culminate in racial disparities in birth outcomes has not been quantified but is likely substantial. Primary prevention of adverse birth outcomes and elimination of disparities will require a societal approach to improve education quality, income equity, and neighborhoods.

Introduction

Twenty years ago, David and Collins published groundbreaking findings demonstrating that black women in Illinois who were born in the United States gave birth to substantially smaller infants than black women who immigrated from Africa to the United States.[1] Immigrant black women's birth outcomes were much more similar to white mothers than they were to their African American counterparts. This key observation largely debunked the hypothesis that racial disparities in birth outcomes result from genetic differences between

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races and ethnic groups.[2] There is something about being black in America over time, perhaps generations, that leads to worse birth outcomes for black infants. Over the following two decades, the search for modifiable factors that result in these disparities has been relentless, but disparities persist (Table 1). Black infants have a 50% higher risk of being born preterm (before 37 weeks of gestation), are almost twice as likely to be born low birth weight (LBW, less than 2,500 grams), and are more than twice as likely to die in the first year of life.[3–5] Fetal deaths are also more than twice as common among black women compared with white women.[6] Despite all of the effort and resources that have been expended to discover the reasons for birth outcome disparities, the mechanisms remain poorly understood.

While improvements in healthcare and healthcare access can improve health overall, tackling disparities has proven difficult. Massachusetts instituted universal health insurance in 2006, but disparities persist. In 2005, black infants were 40% more likely than white infants to be born preterm (12.4% versus 8.8%) and 64% more likely to be LBW (12.0% versus 7.3%).[7] In 2014, eight years after healthcare reform, black infants were still 18% more likely to be born preterm (10.0% versus 8.5%) and 48% more likely to be LBW (10.2% versus 6.9%).[8] The prevention of preterm birth relies on identifying high-risk pregnancies and targeting therapies such as progesterone or cerclage placement.[9] However, screening for women at high risk for preterm birth is not always universal, even when women receive prenatal care.[10] Further, even if optimal screening and use of medical interventions to prevent preterm birth were uniformly distributed, only a fraction of preterm deliveries would be averted. Data demonstrate that progesterone can reduce the risk of preterm birth in high-risk women, as defined by a prior preterm delivery or short cervical length, by 43%.[11] Assuming 20% of pregnancies were high risk, the overall reduction in preterm birth for the population would be just under 10%. A more comprehensive societal approach that extends beyond healthcare will be required to improve birth outcomes and eliminate their disparities.

In this review, we focus on social factors such as suboptimal education and poverty, in addition to environmental exposures to air pollution, metals such as lead, and recently identified toxins such as phthalates, that differ by race and ethnicity in the United States. Lastly, we discuss the impact of psychosocial stressors, including discrimination on birth outcomes. Each of these factors may increase the risk of adverse health outcomes for all adults and children in the United States, but also likely combine to lead to unequal beginnings for the youngest members of our society (Figure 1).

Education

In almost all epidemiologic studies, investigators adjust for socioeconomic position, so as to isolate the potentially causal relationship between other factors, such as smoking or diabetes, and the risk of an outcome, such a preterm birth. However, socioeconomic position itself is reproducibly associated with an increased risk of adverse birth outcomes. One of the primary contributors to socioeconomic position is education. The United States has a long history of racial segregation and inequality in schools that did not end with the 1954 *Brown v. Board of Education of Topeka* Supreme Court ruling outlawing segregation. In 2015, the National

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Assessment of Educational Progress produced a report to describe school racial composition in the United States and its association with academic achievement gaps.[12] The report stated that segregation persists: white students attend schools that average 9% black students, while black students most often attend schools that are 48% black. Both black and white students underperformed at schools with a higher density of black students compared to students at schools with a lower density of black students, but the achievement gap between black and white students was similar between schools. This finding suggests that where students attend school matters for achievement overall, but that, regardless of racial composition, schools are equally poor at eliminating achievement gaps. Further, four-year high school graduation rates are substantially lower for black students (73%) than white students (87%).[13] Fewer years of education are associated with increased risks of preterm birth, suboptimal fetal growth, still birth and infant mortality.[14] How lower levels of education may cause poor birth outcomes is not completely understood. Associations may be partially attributable to personal habits such as smoking, which is more common among women with lower education levels, but models including both smoking and education reveal independent associations of each of these factors with birth outcomes such as birth weight.[15] This suggests that low educational attainment may work through other pathways to lead to adverse birth outcomes. Additionally, equity in education is unlikely to eliminate disparities in birth outcomes. Schoendorf and colleagues compared mortality rates among infants of college educated black (n=42,230) and white (n=865,128) parents and found that even in this educated group, the mortality rate of black infants (10.2 per 100 live births) was twice that of white infants (5.4 per 100 live births).[16] They concluded that this disparity was attributable to differences in LBW (7% versus 3%) because mortality did not differ by race among infants who were not LBW. These findings suggest that addressing the many sources of inequality that lead to a higher risk of LBW (a composite outcome of preterm birth and growth restriction) will be required to eliminate racial disparities in birth outcomes.

Income

Another key driver of socioeconomic position is income, which is often determined by educational attainment.[17] Higher income is associated with improved birth outcomes. Parker and colleagues analyzed data from the 1988 National Maternal and Infant Health Survey, a nationally representative sample of over 6000 women.[18] Poor women were from households with incomes less than 100% of the federal poverty line, whereas affluent women were from households with incomes greater than 200% of the poverty line. In race-stratified analyses, the investigators found that for both black and white women, birth outcomes were worse among poor women compared with more affluent women. Specifically, poor white women were more likely to deliver LBW (6.5%) or small-for-gestational age (SGA) (10.6%) infants than affluent white women (3.6% and 7.4%, respectively). Poor black women were more likely to delivery LBW (12.1%) and preterm (12.2%) infants compared to wealthier black women (8.8% and 7.4% respectively). Of note, income is not the only driver of racial disparities as highlighted in this same study where poor white women had better birth outcomes than affluent black women for all three adverse outcomes (LBW: 6.5% versus 8.8%, SGA: 10.6% versus 15.6%, and preterm: 3.5% versus 7.4%, respectively).

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Income and race determine many aspects of life in the United States; one of the most important is where people live.[19] This relationship is further complicated by the fact that where one lives can, in turn, determine income potential due to employment opportunity and educational quality differences. The interactions among race, education, income, and neighborhood can lead to health disparities. As Williams and Collins argue, residential segregation leads to disparities in health through "pathogenic residential conditions" that disproportionately affect black families, such as proximity to abandoned buildings and more commercial and industrial facilities.[20] Housing quality itself also is more likely to be poor in highly segregated, non-white areas manifesting as excessive crowding, noise levels, and exposure to allergens such as dust mites and pollutants such as air particulates and lead. The extent to which environmental contamination of residential buildings affects disparities in birth outcomes remains incompletely understood.

Environmental exposures

In its report, *Preterm birth: causes, consequences, and prevention*, the Institute of Medicine summarized the data through 2006 on the contribution of environmental exposures to adverse birth outcomes.[21] The report concluded that the contribution of environmental pollutants to preterm birth was understudied, except in the case of lead and environmental tobacco smoke, both of which have been shown in multiple studies to increase the risk of preterm birth.

It has long been established that lead exposure increases preterm birth risk. Specifically, a systematic review by Andrews and colleagues demonstrated that women who delivered preterm had higher mean blood lead levels.[22] In other studies, placental lead was negatively associated with gestational age at birth and maternal blood lead was associated with lower birth weight.[23,24] These latter findings are consistent with cohort data demonstrating a higher risk of LBW among women with occupational lead exposure in Norway.[25] Our group recently found that this association was strongest for infants at the lower end of the birth weight-for-gestational-age spectrum, suggesting increased susceptibility to lead among fetuses that were already growing poorly.[26]

Despite the well-established associations between lead and adverse birth outcomes, only rarely do epidemiologic studies documenting disparities in preterm birth integrate environmental data. While lead levels generally have been declining throughout the United States, for each of the National Health and Nutrition Examination Surveys from 1999–2012 average levels have been consistently higher among non-Hispanic black compared with non-Hispanic white adults and children.[27] Given these persistent differences in lead levels, it is plausible that differential lead levels may contribute to disparities in birth outcomes.

Lead is not the only environmental exposure that may contribute to adverse birth outcomes. Air pollution and its components have been shown to increase the risks of lower birth weight and shorter gestation. Stieb and colleagues performed a meta-analysis and systematic review of ambient air pollution exposure in pregnancy and birth outcomes.[28] The most consistent findings were negative associations of carbon monoxide, nitrogen dioxide and particulate matter with birth weight. The most consistent estimates of increases in the risk of preterm

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birth were from third-trimester air pollution exposure. The authors concluded that the increased certainty regarding third-trimester exposures and preterm birth could be either biological or a result of less heterogeneity among studies focused on the third trimester. Ghosh and colleagues also reviewed the literature and concluded that fetal sex affected the association between air pollution and preterm birth, with exposed male fetuses at higher risk of preterm birth compared to exposed female fetuses.[29] In sum, it is indisputable that air pollution is bad for human health and increases the risk of adverse birth outcomes. However, air pollution, like income and education, is not uniformly distributed across society.

Black adults and children experience higher levels of air pollution exposure than their white counterparts.[30,31] Air pollution results from many sources, including traffic and industrial emissions from fossil fuel combustion, that lead to particulate and gaseous emissions that can harm human health. Predominantly black neighborhoods experience higher levels of exposure due to urban concentrations of populations with close residential proximity to traffic and industrial pollution.[32] The extent to which differential exposure to air pollution contributes to disparities in birth outcomes has not been fully explored.

Other environmental contaminants can increase the risk of adverse birth outcomes. One class of chemicals that has garnered recent interest is phthalates. Phthalates are a class of ubiquitous man-made chemicals that are used to manufacture plastics, building materials such as flooring, adhesives, fast food packaging, and personal care products. [33–36] Phthalates are just one of several contaminants that can act as endocrine disruptors. In the United States, nearly all pregnant women have detectable levels of phthalate metabolites in urine samples.[37,38] Although phthalate metabolites have short half-lives, humans are constantly exposed to varying levels of phthalates from personal care product use, indoor environments, and dietary patterns.[39–41] Phthalate metabolite levels have been shown to be associated with preterm birth, specifically spontaneous preterm birth.[42,43]

As with lead and air pollution, phthalates are not uniformly distributed throughout society. In a review on the contribution of endocrine-disrupting chemicals and their contribution to racial disparities in reproductive outcomes, James-Todd and colleagues highlight differences in exposure to these chemicals by race.[44] Specifically, non-Hispanic black women have higher levels of the low molecular weight phthalates that come from personal care products, adhesives and some medications than non-Hispanic white women.[44–46] Personal care product use often is socially programmed, and one study found that vaginal douching differences might account for differences by race in levels of one particular phthalate metabolite (di-ethyl phthalate).[47] This finding is interesting because douching may be associated with increased risks of preterm birth and low birth weight.[48–51] Whether sources of phthalate exposure or different phthalate metabolite levels resulting from these sources are associated with excess preterm birth risk among black women has not been established. However, it is certainly plausible that differences in the built environment (i.e. flooring), personal care product use and fast food consumption could all lead to differences in phthalate levels by race.

Not all chemical exposures lead to worse birth outcomes and not all chemical exposures are higher in black women compared with white women. Perfluoroalkyl substances and

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organochlorines, which are chemicals used in the manufacturing of textiles and industrial farming, respectively, can pollute water and contaminate food supplies.[52,53] While there is some evidence that these chemicals negatively affect fetal growth, not all data support this claim. In a recent study in Scandinavia, infants born to Swedish, but not Norwegian, women with higher exposure to perfluorooctanoate, polycholirnated biphenyl (PCB) 153 and hexacholorbenzene were more likely to be born SGA.[54] Further, according to the Centers for Disease Control and Prevention National Health and Nutrition Examination Study biomonitoring data, black women have slightly lower levels of all three of these chemicals.[27] With respect to PCBs and preterm birth, there have been multiple negative studies suggesting that PCBs do not increase preterm birth risk in the United States.[21] It is notable that these environmental chemicals are not associated with an increased risk of adverse birth outcomes nor are they associated with race. Identifying which chemicals both increase the risk of adverse birth outcomes and disproportionately affect black women may allow for interventions that could reduce disparities.

Physiologic mechanisms by which environmental exposures lead to adverse birth outcomes are not completely described, but exposures such as lead, air pollution and phthalates can result in oxidative stress and/or inflammation,[55–59] which are implicated in preterm birth and LBW.[60–63] More recently, placental mitochondrial DNA content, which can be diminished by oxidative stress, has been shown to be lower in the settings of both higher exposure to air pollution and lower birth weights.[64] While biologically plausible, definitively determining whether environmental factors cause adverse birth outcomes is difficult. Randomized trials of exposure, the gold standard to determine causal relationships, are unethical when an exposure is known to be harmful. However, randomized controlled trials to reduce indoor air pollution by replacing open fire cooking with stoves have been performed in developing countries to determine whether respiratory outcomes are improved. Thompson and colleagues performed subgroup analysis among pregnant participants of one such study in Guatemala.[65] Women who were randomized to the stove with a chimney had infants who were an average of 89 (95% CI −27, 204) grams heavier than infants born to mothers who cooked using open fires. While the result did not reach statistical significance, studies such as these will help to determine causal relationships between environmental exposures and birth outcomes.

Psychosocial stressors

While not a toxic environmental exposure, per se, psychosocial stress can result from where people live. Racial disparities in birth outcomes may be due, in part, to excess exposure to stress.[66,67] Exposure to certain stressors, specifically violence[68–70] and discrimination[71,72] in pregnancy, are associated with the risk of preterm birth. Further, responses to stressors, including depression and pregnancy-related anxiety, predict preterm birth.[73,74] While stress in pregnancy does not affect only minority women, black women report more stress than white women, which can lead to adverse birth outcomes. [75,76] For example, black women are vastly more likely to experience racial discrimination that white women. Rankin and colleagues conducted a case-control study of black women in the U.S. and found that women who delivered preterm had over twice the odds of high exposure to interpersonal racial discrimination in public settings in the prior year (odds ratio: 2.5, 95% CI: 1.2–5.2).[72]

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This is consistent with a prior finding from this group that women who delivered very low birth weight infants (<1,500 g) were more likely to report high levels of lifetime exposure to interpersonal discrimination, an association that persisted after adjustment for other sociodemographic factors.[75]

Not surprisingly, stress may result directly from the environment. For example, natural disasters,[77] terrorism,[78] noise[79] and even perceptions of air pollution[80] may affect psychosocial stress. Furthermore, psychosocial stress may increase physiologic susceptibility to environmental chemicals and exposures. Cory-Slechta and colleagues performed experiments in a rodent model using restraints as a stressor and maternal lead exposure.[81] They analyzed corticosterone and neurotransmitter levels in dams and offspring. They found that maternal corticosterone increased in response to lead alone and stress alone. Further, they found that stress in combination with maternal lead exposure among female offspring increased dopamine concentrations in the frontal cortex, whereas neither stress alone nor lead alone did so. There also is recent evidence in humans that stress and lead interact during human pregnancy, although the direction of the interaction was not that of potentiation. Tamayo Y Ortiz and colleagues found that the negative association between maternal lead exposure and developmental scores in offspring at two years of age was more pronounced among women who reported fewer negative life events.[82] While this would potentially suggest a buffering effect of stress against the negative effects of lead, the potential biologic interaction of the two exposures to affect offspring outcomes warrants further study. Additionally, there is evidence of prenatal air pollution and psychosocial stress interactions resulting in worse outcomes. For example, Cowell and colleagues demonstrated that among boys prenatal exposure to black carbon, a component of air pollution, and maternal stress were associated with lower Attention Concentration Index scores, whereas there was no main effect of either stress or black carbon alone.[83]

Whether psychosocial stress and environmental factors interact to affect birth outcomes is not well studied, although there are some data to suggest that lower socioeconomic position may increase susceptibility to air pollution with respect to preterm birth and LBW risk.[84] For example, Ponce and colleagues found that traffic-related air pollution, as measured by distance-weighted traffic density, disproportionally increased the risk for preterm birth in neighborhoods of low socioeconomic position in the winter in Los Angeles County, California in the mid-1990s.[85] However, the extent to which psychosocial stress reflects, predicts, or interacts with environmental exposures and social position to result in adverse birth outcomes and disparities remains unknown.

Conclusions

Recognizing that experiences of being black in America vary, certain truths require an examination of societal exposures that contribute to injustice. One of those truths is that black women in the United States have a much higher risk of adverse birth outcomes compared with white women. While some individual choices may mitigate or potentiate these risks, exposures that are not modifiable on an individual level persist. The cycle of residential segregation, educational disadvantage, income inequality and the resulting environmental exposure to unclean air and water must be interrupted to make significant

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progress in eliminating disparities in birth outcomes. Incremental improvements in healthcare and access to healthcare will not do this. Primary prevention of adverse birth outcomes and their disparities requires improving schools and neighborhoods, achieving income equality and enacting environmental reform. Until these changes occur, the births of small and preterm black infants will continue to embody the legacy of longstanding racial discrimination.

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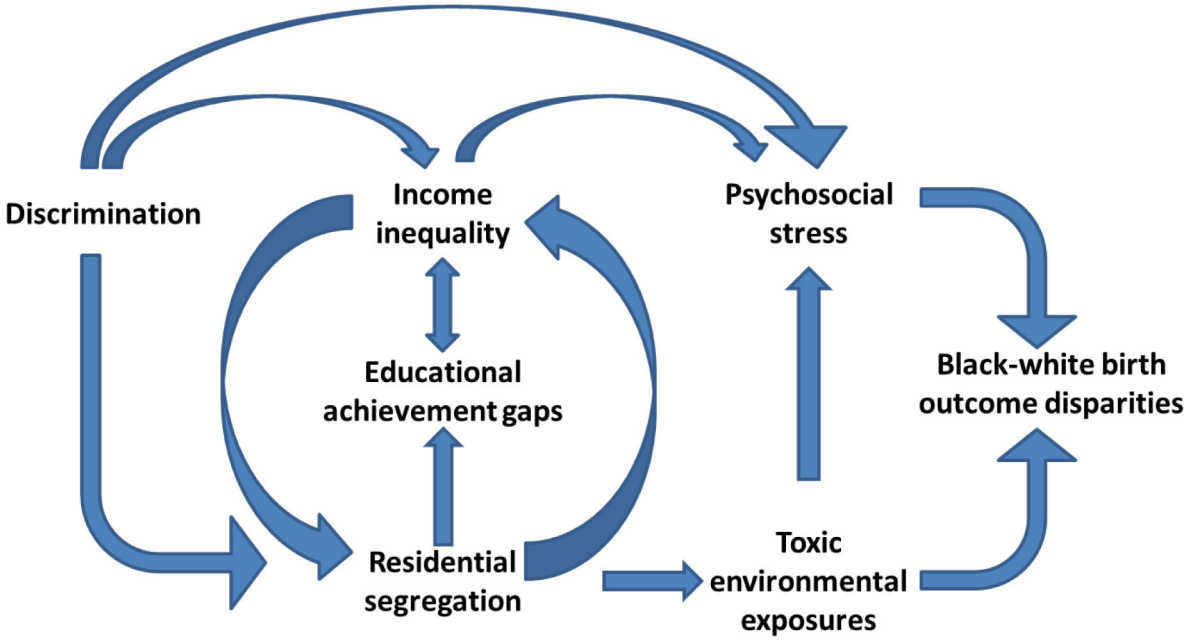

Figure 1.

Conceptual model of societal factors in the United States that lead to psychosocial stress, toxic environmental exposures, and ultimately contribute to racial disparities in birth outcomes.

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Table 1

Black-white disparities in birth outcomes in the United States[4–6]

	Black	White	Black/White disparity
Outcome	**%**	**%**	**RR**
Preterm birth (< 37 weeks)	13.0	8.9	1.46
Very preterm (< 34 weeks)	4.6	2.4	1.92
Low birth weight (< 2,500 grams)	12.8	7.0	1.83
Very low birth weight (< 1,500 grams)	2.8	1.1	2.55
	n/1,000	**n/1,000**	**RR**
Infant mortality (< 1 year)	11.05	4.93	2.24
Neonatal mortality (< 28 days)	7.32	3.37	2.17
Fetal death (> 20 weeks)	10.3	4.88	2.11