Environmental Contributions to Disparities in Pregnancy Outcomes

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Accepted for publication September 15, 2009.

One of the most persistent disparities in American health status is the pronounced difference in birth outcomes between non-Hispanic black and non-Hispanic white women. Poor pregnancy outcomes have a substantial impact on mortality, morbidity, and health care costs. Increasing evidence indicates that environmental exposures are associated with poor birth outcomes. This paper reviews the latest research on how environmental exposures affect pregnancy outcomes and then discusses how these exposures may be embedded within a context of significant social and host factor stress. The analysis suggests that environmental, social, and host factors are cumulatively stressing non-Hispanic black women and that this cumulative stress may be a cause of the persistent disparities in pregnancy outcomes.

environment; environmental pollution; health status disparities; infant, low birth weight; pregnancy outcome; premature birth; public health

INTRODUCTION

One of the most persistent disparities in American health status is the pronounced difference in birth outcomes between non-Hispanic black and non-Hispanic white women. Striking and persistent racial disparities exist in the rates of low birth weight (<2,500 g), very low birth weight (<1,500 g), preterm birth (<37 weeks of gestation), very preterm birth (<34 weeks of gestation), and infant mortality (livebirth with infant death before 12 months of age) (1–4) (Table 1). (Please note, to better assess disparities in pregnancy outcomes, unless otherwise stated, we restricted our analysis to women carrying singleton pregnancies. Unlike multifetal gestations, it is reasonable to have a public health expectation that singleton pregnancies can be carried to term and delivered at appropriate birth weights.) In 2005, while the overall leading cause of infant mortality in the United States was congenital anomalies, disorders related to short gestation and low birth weight were the leading cause for non-Hispanic black infants (5).

Poor pregnancy outcomes have a significant impact on mortality, morbidity, and health care costs. Preterm birth and low birth weight are leading causes of neonatal and infant mortality, as well as short-term and long-term morbidity (6, 7). Conditions associated with poor pregnancy outcomes include respiratory distress syndrome (8), variable heart rate (9), cerebral ventriculomegaly (10), cerebral palsy (11), mental retardation (12), blindness (13), deafness (12), learning disabilities (14, 15), behavioral disabilities (16), and motor impairment (17). Of similar importance is the impact of lower birth weight on increased risk of diabetes, obesity, cardiovascular disease, and other health problems in adulthood (18–20). In 2006, high rates of adverse outcomes meant that 454,583 infants were born preterm and that 267,218 infants were born low birth weight (4), creating a sizable population starting life with an increased risk of short-term and long-term health and developmental complications—a population disproportionately represented by children of color. Thus, understanding, and subsequently intervening to prevent, these adverse outcomes is of critical importance to the overall health of the nation.

In addition to the adverse health impact of poor pregnancy outcomes, economic costs are substantial. In 2006, the Institute of Medicine estimated that the annual cost of preterm birth in the United States was approximately $26 billion, or approximately $51,600 per preterm infant, with two-thirds of the costs related to medical care (6). These figures underestimate the true costs because minimal data exist on the costs of long-term disabilities specifically attributable to preterm birth. Many of these conditions impose significant financial costs on families and on the health care, public education, and social welfare systems.

Although it is widely agreed that maternal and fetal health and well-being are determined by multiple forces, surprisingly little is known about how those forces combine in certain subpopulations. For example, elevated physical
environmental exposures often occur in communities facing multiple social stressors such as deteriorating housing, inadequate access to health care, poor schools, high unemployment, high crime rates, and high poverty rates—all of which may compound the effects of physical environmental exposures. This phenomenon is especially severe for low-income and minority pregnant mothers, with significant health implications for the fetuses they carry. Although some reviews on the relation of environmental factors and pregnancy outcomes exist, none offer a framework for understanding environmental exposures within the larger context of social and host factors (21, 22). To garner an understanding of the current literature on environmental factors linked to pregnancy outcomes, we conducted a careful exploratory review using the PubMed/MEDLINE database (National Library of Medicine, Bethesda, Maryland). A search for articles with the 2 Medical Subject Headings (MeSH) terms “pregnancy” and “environmental exposure” returned 5,960 results (June 2009). We further explored the database and citations in the articles we found and then compiled the most relevant and timely articles in key environmental-emphasis areas: air quality, metals, water quality, pesticides, environmental tobacco smoke, and neighborhood environment. Finally, we supplemented the literature with primary data analysis that enabled us to consider the extent to which those persons subject to elevated environmental exposures concurrently face significant social and host factor stressors.

To understand the complex etiology of black birth outcomes relative to those for whites, Geronimus proposed the “weathering hypothesis” (23), which postulates that poor birth outcomes for African Americans are in part due to the cumulative and interactive effects of negative material and psychosocial stressors on the physical health and general well-being of black women (24–27). The weathering hypothesis argues that cumulative insults to the physical and emotional health of African-American women accelerate their biologic aging (as evidenced by earlier onset of chronic degenerative health problems such as hypertension, diabetes, and heart disease), compromising their ability to carry fetuses to term (23, 24, 28, 29).

Figure 1 depicts environmental, social, and host factors as 3 sides of an integrated triangle. Health disparities arise when the forces exerted by the triangle’s sides are asymmetric for different population groups. In this review, we use the heuristic presented in Figure 1 to consider how environmental exposures affect pregnancy outcomes and how these exposures may be embedded within a context of significant social and host factor stress. In this way, we attempt to expand Geronimus’s notion of cumulative stress (23) to include physical environmental exposures. We conducted literature searches of both the biomedical and social science fields using key terms associated with pregnancy outcomes “low birth weight”, “very low birth weight”, “preterm birth”, “very preterm birth”, “infant mortality”, as well as interacting each of those terms with the term “environmental exposures” for articles through 2008.

TRENDS IN ADVERSE PREGNANCY OUTCOMES

Nationally, the rate of preterm birth among singletons rose from 9.59% to 11.03% from 1990 to 2006. At the same time, the respective rates rose from 7.49% to 9.68% for non-Hispanic whites, increased from 10.10% to 11.03% for Hispanics, and decreased from 17.68% to 16.54% for non-Hispanic blacks (4). It is important to note that the rates for low birth weight and preterm birth in the entire US population, and when separated by race, are all well above the Healthy People 2010 targets (30). From 1990 to 2000, non-Hispanic black women experienced a decrease in the rates of preterm birth and low birth weight, resulting in a smaller disparity between the race groups (Figure 2A and 2B). Since 2000, however, rates for non-Hispanic blacks
have increased, as have the rates for both low birth weight and preterm birth for non-Hispanic white and Hispanic women. The disparities have remained similar because of all groups experiencing an increased risk (31).

Even within racial groups, geographic variation in adverse pregnancy outcomes is significant. Figure 3 shows the percentages of preterm births in 2006 among all singleton pregnancies to women and by major race/ethnicity groups (4). This figure illustrates the dramatic variations in the burden of preterm birth across the United States. When analyzing all preterm births, outcomes in the Southeast are worse than in other areas of the country. When considering outcomes by race, non-Hispanic black women have dramatically worse outcomes than non-Hispanic white and Hispanic women. Whereas documenting the pattern of geographic variation in preterm birth may provide important clues to understanding the etiology and developing effective interventions, the geographic variation itself also illustrates the degree to which the burden of disease is unequally distributed across the United States and across racial/ethnic groups.

As a further illustration, using North Carolina data (32; contact the authors for more information about this data set), Figure 4 demonstrates the geographic variability in preterm birth that becomes apparent at increasingly resolved geographic scales, from county rates to zip code rates and finally to US Census tract rates for one county in the state (in

Figure 2. Rates of A) preterm birth and B) low birth weight among singleton livebirths, United States, 1990–2006 (31).
this case, Durham County). Note that the values in the boxed key for this figure are the same as those for Figure 3, making the additional insight from more resolved geographic scale clearer. A rich area of current investigation revolves around how the geographic pattern of poor birth outcomes may correlate with environmental exposures (V. Berrocal, Duke University, unpublished manuscript) (33–35).

CHARACTERIZING DISPARITIES

The relative differences in adverse birth outcomes have remained fairly constant over the past few decades, narrowing only slightly because of the increase in white multifetal gestations (36). Numerous studies have found that socioeconomic status and income inequality are correlated with birth outcomes (37–40). A variety of other social factors have been linked to poor birth outcomes, including maternal education (41–45), marital status (46), pregnancy intention (47), and teenage pregnancy (48). In addition, host factors such as maternal obesity (49, 50), maternal comorbidities (M. L. Miranda, Duke University, unpublished manuscript) (51), and genetic vulnerabilities (52–69) have each been linked to poor pregnancy outcomes. Here, we review the environmental factors that may contribute to disparities in poor birth outcomes. Given that non-Hispanic black women tend to be more systematically exposed than non-Hispanic white women to adverse environmental conditions (70–74), the physical environment likely interacts with adverse social environments (75, 76), as well as host factors, to contribute to the observed poorer birth outcomes for non-Hispanic black women.

Air quality

Air quality throughout the gestational period, as well as during specific windows of vulnerability, has been shown to influence the risk of low birth weight, preterm birth, fetal growth restriction, and fetal and infant death (33, 77–92). Minority populations, who are already at risk of adverse birth outcomes, are additionally more likely to be exposed to and experience the effects of poor air quality (2, 77, 93, 94). In comparison to white children, a substantially higher percentage of African-American children aged 0–5 years were found to live in poor households located in relatively close proximity to one or more industrial sources of air pollution (95). Furthermore, certain subpopulations may be even more susceptible to air pollution—those whose general health status is compromised or those with social disadvantages that translate into increased environmental or occupational exposure to toxins, adverse behaviors (poor diet, alcohol consumption, and smoking), and lack of adequate access to health care and preventive health measures (96).

Several components of air pollution have been associated with adverse birth outcomes. Ritz et al. (97), in their Southern California sample, found an increased risk of preterm birth with increasing levels of carbon monoxide and particulate matter less than 2.5 μm in aerodynamic diameter during pregnancy. Also in California, Huynh et al. (98) found that the top 2 quartiles of exposure to particulate matter less than 2.5 μm in aerodynamic diameter induced a modest effect of preterm birth following adjustment for maternal age, race/ethnicity, education, marital status, and parity, with the quartile with the highest measurements of

![Figure 3. Rate of preterm birth among singleton livebirths, by maternal race, United States, 2006 (4). The Healthy People 2010 target rate of preterm birth is 7.6% (30).](image-url)
particulate matter less than 2.5 μm in aerodynamic diameter having the most effect on preterm birth.

Timing of exposure during pregnancy has been associated with outcomes as well. Increased exposure to sulfur dioxide and particulate matter less than 10 μm in aerodynamic diameter during the last 6 weeks of pregnancy has been associated with an increased risk of preterm delivery (99). Conversely, an increased odds ratio was observed for low birth weight with maternal exposure to sulfur dioxide during the first month of pregnancy and an increased risk of preterm birth with exposure to sulfur dioxide and carbon monoxide during the last month of pregnancy. An increased risk of fetal growth restriction has also been shown to be associated with maternal exposure to sulfur dioxide, nitrogen dioxide, and carbon monoxide during the first month of pregnancy (87).

In addition to ambient air concentrations of pollutants, areas of locally poor air quality associated with mobile sources may impact pregnancy outcomes. A Taiwan study found that mothers living within 500 m of a freeway were significantly more likely to deliver preterm than mothers living 500–1,500 m from the freeway (100). In Los Angeles County, California, researchers determined a 10%–20% increase in low birth weight and preterm birth risk for women living near high-traffic roads (100, 101). Importantly, many more minorities (60% of Hispanics and 50%
of non-Hispanic blacks, compared with 33% of non-Hispanic whites) live in areas failing to meet 2 or more of the national ambient air quality standards (102).

**Metals**

Exposure to metals increases the risk of adverse birth outcomes (69, 103, 104). The effects of the heavy metals cadmium and manganese, the exposure routes of which include cigarette smoke and food consumption, have been poorly studied, but rodent studies indicate the occurrence of birth deformities and fetal growth restriction (103). Several human studies have shown a significant correlation between cadmium and decreased birth weight (105, 106). Arsenic and lead have been shown to increase incidence of low birth weight and preterm birth (105, 107–110). Exposure to high concentrations of arsenic has also been associated with a 6-fold increase in stillbirth after adjusting for potential confounders (111). Non-Hispanic black women experience greater exposure to environmental lead over the life course (112–116), which may aggravate their risk of both hypertension and poor birth outcomes (110, 117, 118). Furthermore, long-term child outcomes for prenatal lead exposure include cognitive effects at 12 and 24 months of age (119).

**Water quality**

Water quality may be associated with low birth weight, fetal growth restriction, and risk of spontaneous abortion and stillbirth. Recent evidence has suggested that chlorination by-products such as trihalomethanes from water disinfection may increase the risk of low birth weight and stillbirth (120, 121). Water contamination caused by waste disposal increases the risk of low birth weight. Rodenbeck et al. (122) found that the odds ratio for very low birth weight compared with normal birth weight was 3.3 for maternal exposure to trichloroethylene via contaminated drinking water from waste-disposal practices. Proximity to landfill sites is associated with a slight statistically significant increased risk of neural tube defects, low birth weight, and very low birth weight, with adjusted risks of 1.05, 1.03, and 1.05, respectively (123). Herbicide-contaminated drinking water in Iowa has been associated with fetal growth restriction for births occurring between 1984 and 1990 (124). In addition, there are incidents and general evidence of racial (and class) inequities in exposure to contaminants in water, as well as proximity to potential water contamination sources (125–127).

**Pesticide use**

The use of pesticides is ubiquitous, and many women continue to use pesticides during pregnancy (128). Prenatal pesticide exposure has been associated with adverse birth outcomes. Increased levels of dichlorodiphenyldichloroethane have been associated with lower birth weight and smaller head circumference (129). Whyatt et al. (130) found a significant inverse relation between organophosphates in umbilical cord plasma and birth weight and length, particularly among those newborns with the highest exposure. Birth weight was lower among those with the highest combined cord plasma chlorpyrifos and diazinon exposure levels. High levels of exposure to polycyclic organic matter increased the odds of small-for-gestational-age births (131).

Proximity to agricultural areas has also been associated with adverse birth outcomes. Increases in the risk of neural tube defect have been associated with maternal residence within 1,000 m of agricultural applications of benomyl, methyl carbamate or organophosphorus pesticides, or pesticides listed as endocrine disruptors, cholinesterase inhibitors, or developmental toxins (132). Schreinemachers (133) found that in rural, agricultural counties, where wheat acreage occupies a larger percentage of the land and where frequency of use of chlorophenoxy herbicides is higher, anomalies of the circulatory/respiratory and musculoskeletal/integumental system significantly increased.

**Environmental tobacco smoke**

Tobacco smoke can contain as many as 4,000 different chemicals (134). Exposure to environmental tobacco smoke may begin in utero and continue throughout development. One in 5 American children less than 7 years of age lives in a home in which someone, usually a parent, smokes regularly (135). Despite lower general exposure to environmental tobacco smoke compared with non-Hispanic white children, non-Hispanic black children have higher rates of tobacco-related illnesses and levels of cotinine, the major nicotine metabolite (136). Inner-city children have a greater likelihood of environmental tobacco smoke exposure, with 70%–80% of them reported to have levels of cotinine indicative of environmental tobacco smoke exposure (137).

Prenatal and childhood exposure to secondhand tobacco smoke is known to increase risk of outcomes such as low birth weight (138, 139), birth defects (140), sudden infant death syndrome (141–143), and asthma and respiratory illnesses (135). Combined prenatal exposure to environmental tobacco smoke and polycyclic aromatic hydrocarbons at levels currently found in several major US cities has been associated with decreased birth weight and head circumference (144). In addition, exposure to polycyclic aromatic hydrocarbons is likely to contribute to the occurrence of small-for-gestational-age as well as preterm birth among African Americans (145). Refer to Figure 5 for rates of tobacco use across the United States (146).

Smoking during pregnancy may be linked to home-environment conditions (147, 148) or to physical abuse (138). The combination of physical abuse, smoking, and alcohol consumption/illicit drug use is significantly related to birth weight (138). In the United States in 2006, women reporting tobacco use during pregnancy delivered preterm at a rate of 14.0% compared with 11.0% among women not reporting tobacco use. Low infant birth weight is also significantly higher among smokers, with rates of 11.0% among those using tobacco and 6.1% among those not using tobacco (4).

Rates of self-reported tobacco use during pregnancy declined in all race groups from 1996 to 2006. There remains racial disparity in maternal smoking during pregnancy nationally, with non-Hispanic white women more likely than...
non-Hispanic black women to smoke, who in turn are more likely than Hispanic women to smoke (4). Refer to Figure 6 for self-reported tobacco use among pregnant women across the United States.

**Neighborhood environment**

Elements of the built environment can also influence maternal health (149) through direct exposure and by limiting the effectiveness of traditional medical care and outreach strategies (150). Neighborhoods with higher concentrations of non-Hispanic blacks and Hispanics have higher levels of physical environmental contaminants than economically comparable neighborhoods with higher concentrations of non-Hispanic whites (151). Research has shown that anxiety, depression, and psychological distress more generally increase with number of housing problems (152, 153). Higher neighborhood problem scores have been associated with poor self-rated health, psychological distress, and impaired physical function, independent of age, sex, neighborhood socioeconomic status, individual deprivation, and social capital (154). The built environment can also restrict residents’ physical activity (155, 156). Non-Hispanic blacks disproportionately live in low socioeconomic status neighborhoods suffering from problems such as dilapidated buildings, lack of space for exercise, and lack of public services, which in turn negatively affect health (157–160).

Neighborhood economic conditions can influence preterm birth through differences in access to health care, quality and type of food available in grocery stores, amount of green space, number of safe places for exercise, and amount of environmental pollutants (161, 162). Neighborhood disadvantage is often quantified by using neighborhood-level poverty rate or income level. Areas of concentrated poverty, typically defined as neighborhoods with poverty rates higher than 20%, are associated with diminished quality of the neighborhood’s social and physical environment, high rates of neighborhood turnover and mobility, crime, social disorder, and attenuation of both individual socioeconomic attainment and upward mobility (76). Mothers from neighborhoods with lower median household incomes have been found to be at greater risk of adverse birth outcomes (163).

Living in tracts with high unemployment, low educational levels, poor housing, a low proportion of managerial or professional occupations, and high poverty levels increases the odds of preterm birth for non-Hispanic whites. Interestingly, effects were still significant, but smaller, for non-Hispanic blacks. Tract-level low educational levels, high unemployment, low-level occupations, and high poverty rates increased the odds of preterm birth for non-Hispanic blacks (164, 165). Masi et al. (166) found that tract economic disadvantage was associated with significantly lower birth weight for all maternal racial/ethnic groups.

In a recent study, women who lived in more disadvantaged neighborhoods had greater stress levels, reported less internal locus-of-control and emotional support, and were more likely to smoke, drink alcohol, use hard drugs, and have later or no prenatal care and inadequate weight gain. Strongest associations with neighborhood risk were observed for smoking, hard-drug use, and net weight gain during pregnancy (167). Conversely, neighborhoods with high levels of residential stability may be conducive to strong ties among residents, lower levels of perceived stress, and more positive health outcomes. Residential stability, then, may be protective of mental and physical health (151) and has been associated with an increase in birth weight (168).

Neighborhood racial composition has been related to birth outcomes. The risk of low birth weight increases with degree of residential segregation at the census tract level after
discussing immigrant populations, representing isolation and segregation, respectively (171).

**Discussion**

Health disparities arise through differences in levels of exposure, combinations of exposures, and response to exposures originating from all 3 sides of the triangle shown in Figure 1. Considering how all 3 sides operate simultaneously is critical to disentangling the complex etiology of poor pregnancy outcomes. Social stressors that have been linked to poor maternal health may co-occur with, or even cause, disparities in environmental exposures. For example, the effects of institutionalized racism can segregate African Americans into neighborhoods that are more polluted, have higher rates of unemployment and crime, and have lower levels of resources (172). At the same time, host factors, such as presence of maternal complications and personality traits related to the stress response, may amplify or mitigate the effect of the environmental exposure in some individuals within the community.

Within the framework of Figure 1, the area within the triangle represents the “space” that has been carved out for any particular maternal-child pair to prosper. The area of the triangle is larger for women with few social and environmental stressors and more protective host factors and, of course, is smaller for those who experience social or environmental stress or who have host factor vulnerabilities. Imagine a new environmental exposure that affects a particular individual or group of individuals (Figure 7, left). The force of that exposure will reduce the space/area of the triangle available to that individual/group (Figure 7, top right). If, however, the individual or group has protective social factors in place, then the resiliency created by the positive social factors may mitigate in whole or in part against the adverse environmental exposures. This resiliency essentially increases the space available, although not necessarily equal to the area that was available prior to the exposure (note the bowing out of the social-factors side of the triangle (Figure 7, bottom right)).

Poverty status can act as an indicator of a whole suite of risk factors for poor pregnancy outcomes, as well as a measure of potential resiliency to other stressors, especially so on the social-factors side of the triangle in Figure 1. Mothers living in poverty have higher rates of clinical depression, are more likely to suffer from chronic stress, and experience more negative life events than do nonpoor mothers (173, 174). They are nearly 3 times as likely to suffer from domestic violence and have more negative health behaviors, such as smoking and inactivity (148, 175). They are also more likely to be unmarried and solely responsible for raising their children (176). Furthermore, they may lack adequate social networks, which can act as a buffer to the detrimental effects of stress and depression (46). Living in neighborhoods that have high crime or unemployment rates or are violent, are overcrowded, or have high median rents is associated with low birth weight and very low birth weight (42, 177, 178). In 2007, dramatic disparity existed in poverty rates by race group: 24.5% of non-Hispanic blacks and 21.5% of Hispanics were living in poverty compared with 8.2% of non-Hispanic whites. There is also geographic disparity in poverty rates. The poverty rate in 2007 was 14.2% in the South, 11.4% in the Northeast, 11.1% in the Midwest, and 12% in the West (179).

We argue that psychosocial stressors shape the effects of environmental exposures. Recent animal studies indicate that enriched environments may reverse some of the long-term deficits in learning associated with lead exposure. In this study, lead-exposed rats were randomly assigned to “isolation” cages or “environmental enrichment” cages after exposure. Results indicate that lead-induced spatial learning deficits may be reversed by stimulating postexposure environments—with obvious implications for treatment of childhood lead intoxication (180). In addition, a feedback loop may exist in that the psychosocial stress induced by symptomatic and asymptomatic behaviors and participation in a treatment regime may affect the attributes of the child’s environment that might otherwise serve a protective role. For example, children exposed to lead place heavy burdens on caretakers and may overwhelm the personal, professional, and financial resources available to parents, significantly affecting their ability to provide a supportive home environment (K. Joyner, United Parents Against Lead, personal communication, 1999).

In another example, one of the key contaminants of concern in tobacco smoke is cadmium (181). Even after controlling for potential confounders, cadmium has been negatively correlated with children’s psychometric test scores. Cadmium has an even greater negative effect than lead on verbal IQ scores (182). Importantly, diets often associated with those in lower socioeconomic brackets—namely, those high in fat or low in calcium, protein, or iron—promote absorption of cadmium in the body (183). Although these examples relate more to child development, it is plausible that similar phenomena may be playing out with respect to pregnancy outcomes.

Maternal stress—defined by any physical or psychological challenge that threatens normal homeostasis—plays an important role in the maternal and fetal immune or inflammatory response. (So a stressor on the social-factors side of the triangle can induce or coengage a stress on the host-factor side of the triangle, in the form of an immune or inflammatory response.) For example, bacterial vaginosis, a microbial genital tract infection, is a known risk factor for adverse birth outcomes and is clearly associated with, and possibly the cause of, a heightened maternal and fetal inflammatory response. High levels of chronic stress have been associated with bacterial vaginosis in pregnant women (162, 184, 185). Low socioeconomic status, young, or unmarried women are more likely to develop bacterial vaginosis, after controlling for known risk factors such as income, education, and history of sexually transmitted disease (186). Ten percent of women with bacterial vaginosis experience adverse pregnancy outcomes such as...
spontaneous preterm delivery, premature rupture of membranes, and amniotic fluid infection (186).

Inflammation in gestational tissues more generally is a major risk factor for adverse birth outcomes. Proinflammatory cytokines stimulate the synthesis and release of 1) maternal prostaglandins and metalloproteases; 2) fetal inflammatory cytokines, cortisol, and dehydroepiandrosterone sulfate; and 3) placental corticotrophin-releasing hormone (187–190). These effects may be one of the pathways whereby stress can lead to adverse birth outcomes (191). A major contribution of maternal “immune stress” to adverse fetal outcomes is likely stimulation of production and secretion of corticotrophin-releasing hormone, which then has multiple effects that promote preterm birth and retard fetal growth.

Evidence from both animal models and human epidemiologic research underscores the important role that prenatal stress plays in a broad range of fetal developmental outcomes (192). Environmental exposures may be some of the “physical challenges” that contribute to maternal immune stress given that several environmental contaminants including heavy metals, air pollution, and environmental tobacco smoke have been linked to alterations in the host inflammatory response (193–201). In addition, the stressors faced by African-American women as a marginalized group may directly compromise their physiologic functioning (172, 202–204) as well as encourage unhealthy behaviors as a coping mechanism for stress (148, 205, 206).

To explore the extent to which non-Hispanic black women tend to cluster more on the higher-risk end of environmental, social, and host factor stressors, we constructed Table 2. For each category of stressor (environmental, social, and host), we include a series of risk factors and show how non-Hispanic blacks and non-Hispanic whites are distributed across the risk spectrum for each factor.

In terms of environmental stressors, we begin with air quality, including both ozone and particulate matter. These air quality grades are taken from the American Lung Association’s State of the Air 2009 report (207). A greater proportion of non-Hispanic black women living within the American Lung Association study area were exposed to high ozone levels (89.5% compared with 82.9% for non-Hispanic whites) (207, 208). This differential is not especially great, likely resulting from the fact that, according to the guidelines developed by the American Lung Association, most American communities that are part of the air quality monitoring network are exposed to unhealthy levels of ozone. The contrast for particulate matter is much more dramatic. Of non-Hispanic blacks in the study area, 52.7% lived in communities receiving a grade of “F” from the American Lung Association, compared with 38.3% of non-Hispanic whites (207, 208).
Unlike for air quality, especially good national data are not available on quality of drinking water or the built environment that are broken out by race subgroups. We can indirectly assess these measures, however, by noting that non-Hispanic blacks are much more likely to be renter-occupants (52.9%) as opposed to owner-occupants, compared with non-Hispanic whites (24.4%). In turn, in terms of water quality, 11.6% of renter-occupants do not have a safe primary source of drinking water compared with 6.5% of owner-occupants (209). Because non-Hispanic blacks are much more likely to live in renter-occupied housing, we can reasonably conclude that non-Hispanic blacks are also much less likely to have a safe primary source of water to drink.

Table 2 shows similar patterns regarding measures of the built environment. Those living in renter-occupied housing units are more likely to rate their neighborhoods as being on the bottom end of the scale (6.7% compared with 2.6% for owner-occupied units). Renters are also more likely to report that a serious crime has occurred in the neighborhood in the last 12 months (21.6% compared with 13.2% for owner-occupied units). Renter-occupied housing units are also more likely to have bars on the windows of buildings within 300 feet (90 m) (16.9% compared with 6.8%); be on a street in need of repair (44.5% vs. 36.7%); or have trash, litter, or junk on the street or properties within 300 feet (14.5% vs. 6.5%) (209). Again, because non-Hispanic blacks are much more likely to live in renter-occupied housing, we can reasonably conclude that non-Hispanic blacks are also more likely to be dealing with a depauperate built environment.

In addition to documenting differences in environmental exposures, Table 2 also provides data on racial differences in host factors. Non-Hispanic black women aged 20–44 years are more likely to rate their overall health status as poor or fair (10.4% compared with 6.5% for non-Hispanic white women) and are more likely to have hypertension (15.3% compared with 5.5% for non-Hispanic white women) (210).
Non-Hispanic black women aged 20 years or older are also more likely to be overweight/obese (53.2%/26.5% vs. 32.2%/27.2% for non-Hispanic white women) (211). In the social-factors category, non-Hispanic black women giving birth are more likely to be unmarried (70.7% vs. 26.6% for non-Hispanic white women) and are more likely to have less than a high school education (23.7% vs. 11.5% for non-Hispanic white women) (31). Although data on pregnant women specifically are not available, non-Hispanic black women generally are more likely to have a low income-to-poverty ratio (12.8% at <50% of the poverty line vs. 5.0% for non-Hispanic white women) (210).

Table 2 demonstrates that, for the multiple factors that affect pregnancy outcomes, non-Hispanic blacks cluster on the high-risk end of the spectrum. This finding is consistent with Geronimus’s notion of cumulative stress (23) as described in the weathering hypothesis, where cumulative stress is now defined to include physical environmental exposures. In terms of the rubric presented in Figure 1, environmental exposures/stressors are compressing the “space” available for maternal-child pairs to prosper—and these exposures are likely embedded within a setting in which both social and host factors are less likely to serve as mitigating forces. So, the resiliency to environmental exposures that can be created by positive social and host factors is more likely to be absent for non-Hispanic blacks compared with non-Hispanic whites.

Despite extensive public policy efforts to maximize access to prenatal care, significant racial disparities in pregnancy outcomes persist. The systematic and consistent disproportionate exposure of non-Hispanic blacks to both environmental and social stressors, especially within the context of greater presentation of overweight/obesity and comorbidities on the host-factor side, may account for the persistent race-based disparities. We argue that research that carefully examines the joint effects of social and environmental stressors—conducted at the individual level so that we truly know who is experiencing multiple stressors—holds potential for revealing the complex etiology that likely drives disparities in pregnancy outcomes. Such understanding is critical to the development of successful intervention programs aimed at narrowing the health disparities in pregnancy outcomes.
pregnancy outcomes, which will need to jointly address the multiple components shaping the lives of women during the preconception, prenatal, and postnatal periods.

ACKNOWLEDGMENTS

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This research was supported by funding from the US Environmental Protection Agency (RD-83329301-0).

Conflict of interest: none declared.

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