Epidemiologic Reviews

Vol. 31, No. 1
DOI: 10.1093/epirev/mxp011
Advance Access publication October 21, 2009

Environmental Contributions to Disparities in Pregnancy Outcomes

Marie Lynn Miranda, Pamela Maxson, and Sharon Edwards

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

Correspondence to Dr. Marie Lynn Miranda, Nicholas School of the Environment, Duke University, A134-LSRC, Box 90328, Durham, NC 27708 (e-mail: mmiranda@duke.edu).

Table 1. Birth Outcomes Among US Singleton Livebirths in 1996 and 2006, by Maternal Race (4)

Outcome	1996		2006	
	No.	%	No.	%
Total births	3,784,805		4,121,930	
Preterm birth	364,356	9.63	454,583	11.03
Non-Hispanic White	183,652	8.02	214,935	9.68
Non-Hispanic Black	90,333	16.11	98,251	16.54
Hispanic	67,319	9.87	112,006	11.03
Very preterm birth	104,550	2.76	121,122	2.94
Non-Hispanic White	46,524	2.03	50,910	2.29
Non-Hispanic Black	33,442	5.96	33,221	5.59
Hispanic	18,442	2.70	29,611	2.92
Low birth weight	228,062	6.03	267,218	6.48
Non-Hispanic White	112,099	4.90	119,122	5.36
Non-Hispanic Black	64,656	11.53	70,308	11.84
Hispanic	36,404	5.34	58,725	5.78
Very low birth weight	41,045	1.08	46,961	1.14
Non-Hispanic White	17,858	0.78	18,818	0.85
Non-Hispanic Black	14,516	2.59	15,480	2.61
Hispanic	6,408	0.94	9,947	0.98

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).

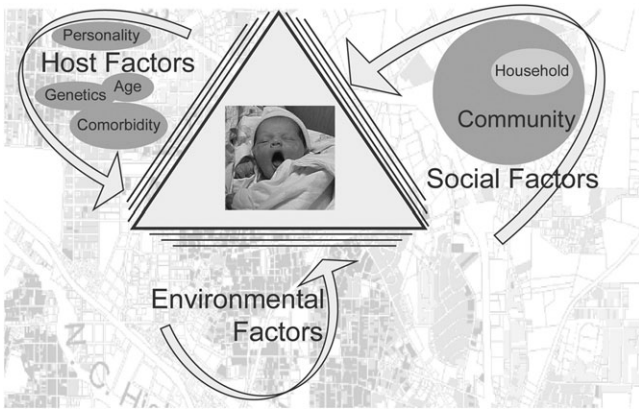

Figure 1. Forces shaping pregnancy outcomes.

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

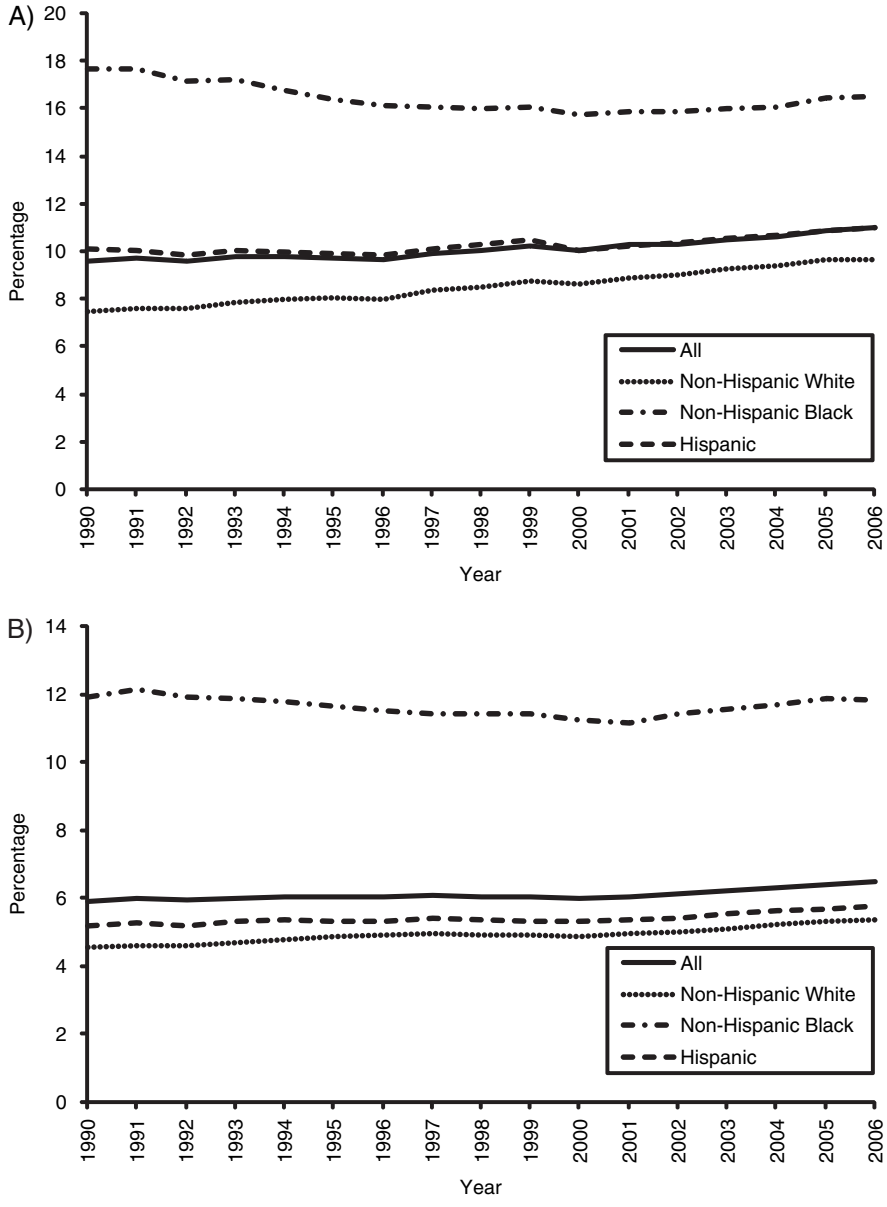

Figure 2. Rates of A) preterm birth and B) low birth weight among singleton livebirths, United States, 1990–2006 (31).

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

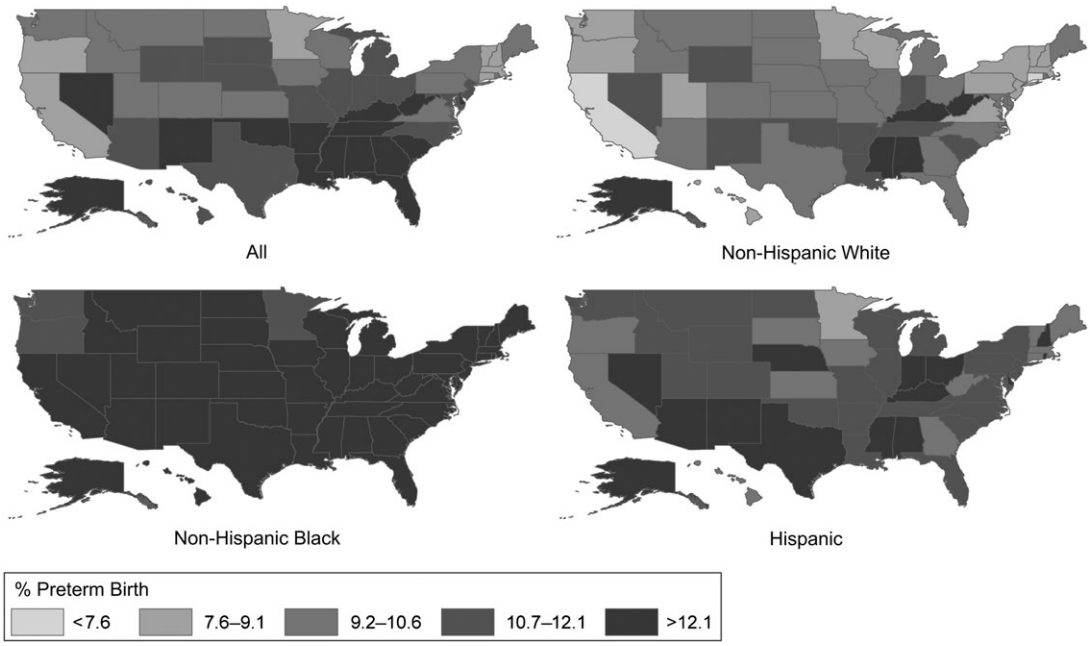

Figure 3. Rate of preterm birth among singleton livebirths, by maternal race, United States, 2006 (4). The *Healthy People 2010* target rate of preterm birth is 7.6% (30).

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

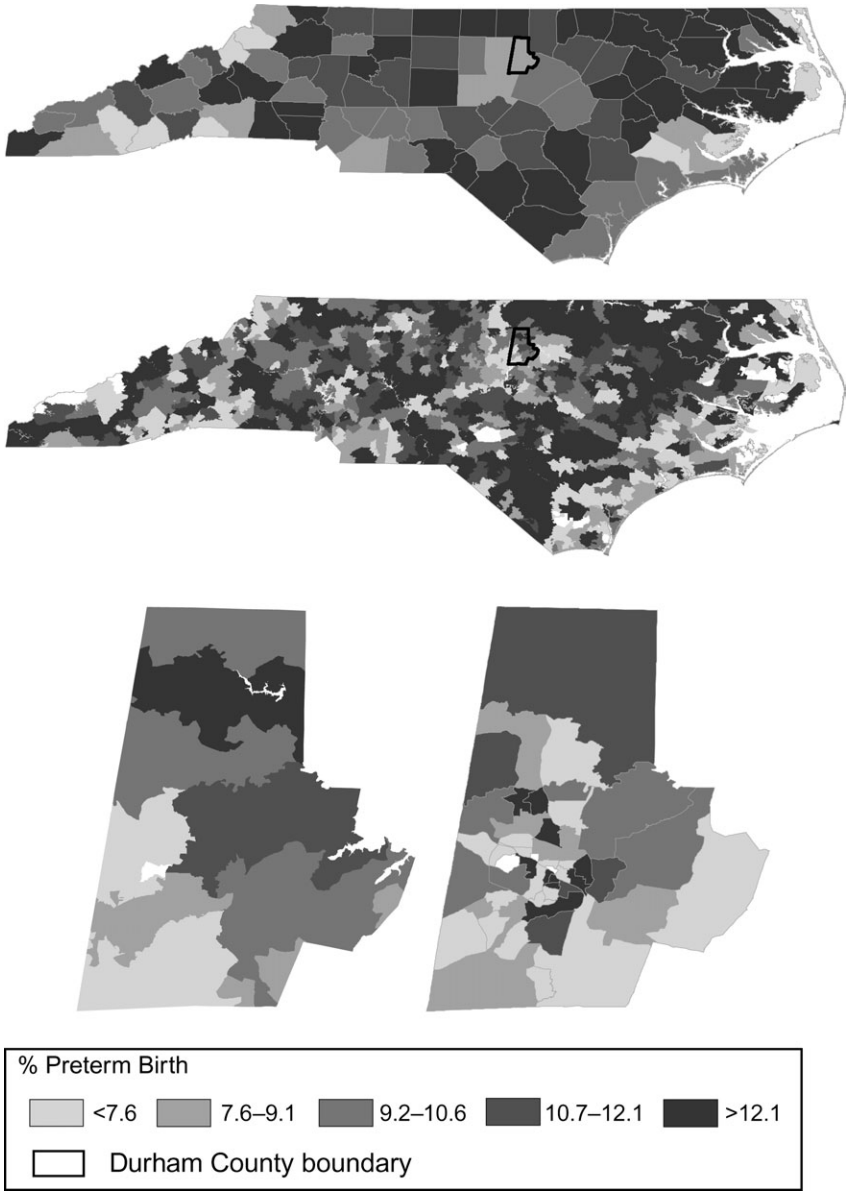

Figure 4. 2006 preterm birth rates among North Carolina and Durham County singleton livebirths, by county (top), zip code (middle and bottom left), and US Census tract (bottom right) (32). The *Healthy People 2010* target rate of preterm birth is 7.6% (30).

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

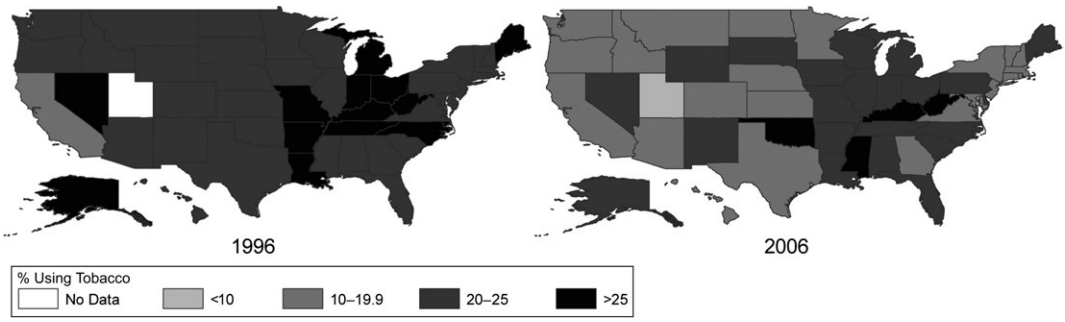

Figure 5. Tobacco use by US state, 1996 and 2006 (146).

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).

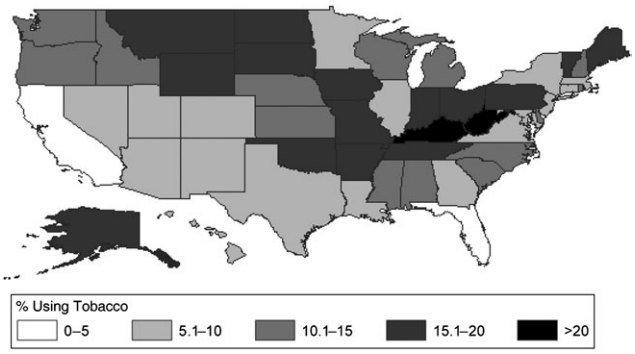

Figure 6. Self-reported tobacco use by pregnant women with US singleton livebirths, by state, 2004 (31).

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

adjusting for individual-level risk factors and census tract poverty (169). Birth weight is lower and preterm birth rates are higher in metropolitan statistical areas with moderate to high levels of isolation (170). Small-for-gestational-age births were most likely in neighborhoods with the lowest and highest 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

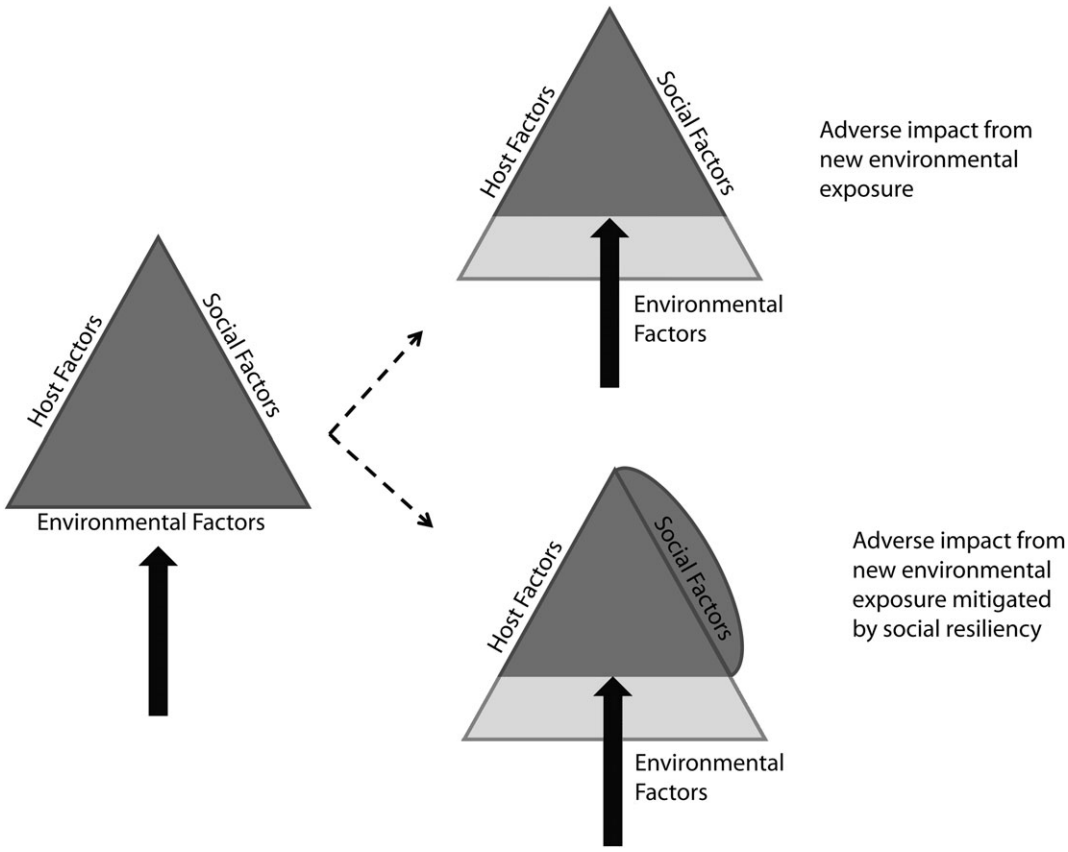

Figure 7. Conceptual representation of social/environmental interactions.

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).

Table 2. Distribution (%) of Select Risk Factors for Disparities in Pregnancy Outcomes, by Maternal Race and Housing Status

Risk Factor	High Risk				Low Risk	Data Source (Reference No.)
Environmental Factors						
Air quality grade—ozone	F	D	C	B	A	County grades from the American Lung Association's *State of the Air 2009* (207); 2000 US Census (208)
Non-Hispanic White	82.9	3.1	8.4	3.1	2.6	
Non-Hispanic Black	89.5	3.3	5.1	1.2	0.9	
Air quality grade—daily $PM_{2.5}$ exposure	F	D	C	B	A	County grades from the American Lung Association's *State of the Air 2009* (207); 2000 US Census (208)
Non-Hispanic White	38.3	13.3	27.3	13.1	7.9	
Non-Hispanic Black	52.7	14.3	21.5	9.3	2.2	
Tenure status	Renter				Owner	American Housing Survey 2007 (209)
Non-Hispanic White	24.4				75.6	
Non-Hispanic Black	52.9				47.1	
Primary source of water safe to drink	No				Yes	American Housing Survey 2007 (209)
Owner-occupied	6.5				93.5	
Renter-occupied	11.6				88.4	
Opinion of the neighborhood	Worst				Best	American Housing Survey 2007 (209)
Owner-occupied	0.9	1.7	9.5	41.0	46.9	
Renter-occupied	2.4	4.3	16.9	42.0	34.5	
Serious crime in the neighborhood in the last 12 months	Yes				No	American Housing Survey 2007 (209)
Owner-occupied	13.2				86.8	
Renter-occupied	21.6				78.4	
Bars on windows of buildings within 300 feet[a]	Yes				No	American Housing Survey 2007 (209)
Owner-occupied	6.8				93.2	
Renter-occupied	16.9				83.1	
Street in need of repair	Yes				No	American Housing Survey 2007 (209)
Owner-occupied	36.7				63.3	
Renter-occupied	44.5				55.5	

Table continues

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 be renter-occupants, 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% vs. 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).

Table 2. Continued

Risk Factor	High Risk						Low Risk	Data Source (Reference No.)
Trash, litter, or junk on the street or properties within 300 feet	Yes						No	American Housing Survey 2007 (209)
Owner-occupied	6.5						93.5	
Renter-occupied	14.5						85.5	
				Host Factors				
Overall health status	Poor		Fair	Good	Very good		Excellent	Females aged 20–44 years in the Current Population Survey 2007 (210)
Non-Hispanic White	1.6		4.9	18.8	36.8		37.9	
Non-Hispanic Black	2.5		7.9	27.1	35.5		27.0	
Hypertension	Yes						No	Females aged 20–44 years in NHANES 2003–2006 (211)
Non-Hispanic White	5.5						94.5	
Non-Hispanic Black	15.3						84.7	
Overweight/obesity	Obese			Overweight			Healthy	Females aged ≥20 years in NHANES 2003–2006 (211)
Non-Hispanic White	32.2			27.2			38.2	
Non-Hispanic Black	53.2			26.5			19.2	
				Social Factors				
Marital status	Not married						Married	National Vital Statistics, 2006 (CDC) (4)
Non-Hispanic White	26.6						73.4	
Non-Hispanic Black	70.7						29.3	
Educational attainment	No high school		Some high school	High school	Some college		College	National Vital Statistics, 2006 (CDC) (4)
Non-Hispanic White	1.5		10.0	26.4	26.8		35.3	
Non-Hispanic Black	2.2		21.5	36.4	26.9		13.0	
Income-to-poverty ratio	<50%	50%–<75%	75%–<100%	100%–<125%	125%–<150%	150%–<175%	≥175%	Females aged 20–44 years in the Current Population Survey 2007 (210)
Non-Hispanic White	5	3	3	3	4	4	78.7	
Non-Hispanic Black	12.8	7.2	6.0	6.1	5.1	5.2	57.5	

Abbreviations: CDC, Centers for Disease Control and Prevention; NHANES, National Health and Nutrition Examination Survey; $PM_{2.5}$, particulate matter <2.5 μm in aerodynamic diameter.

[a] One foot = 0.3 m.

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, which will need to jointly address the multiple components shaping the lives of women during the preconception, prenatal, and postnatal periods.

ACKNOWLEDGMENTS

Authors affiliation: Nicholas School of the Environment, Duke University, Durham, North Carolina.

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

Conflict of interest: none declared.

REFERENCES

1. Singh GK, Yu SM. Infant mortality in the United States: trends, differentials, and projections, 1950 through 2010. *Am J Public Health*. 1995;85(7):957–964.
2. Johnston RB Jr, Williams MA, Hogue CJ, et al. Overview: new perspectives on the stubborn challenge of preterm birth. *Paediatr Perinat Epidemiol*. 2001;15(suppl 2):3–6.
3. Wise PH, Kotelchuck M, Wilson ML, et al. Racial and socioeconomic disparities in childhood mortality in Boston. *N Engl J Med*. 1985;313(6):360–366.
4. National Center for Health Statistics. Natality Public Use Files 1990–2006. Hyattsville, MD: Centers for Disease Control and Prevention, US Department of Health and Human Services. (http://www.cdc.gov/nchs/data_access/Vitalstatsonline.htm). (Accessed January 6, 2009).
5. Mathews TJ, MacDorman MF. Infant mortality statistics from the 2005 period linked birth/infant death data set. *Natl Vital Stat Rep*. 2008;57(2):1–32.
6. Behrman RE, Butler AS. *Preterm Birth—Causes, Consequences and Prevention*. Washington, DC: National Academies Press; 2007.
7. Hack M, Klein NK, Taylor HG. Long-term developmental outcomes of low birth weight infants. *Future Child*. 1995; 5(1):176–196.
8. Lemons JA, Bauer CR, Oh W, et al. Very low birth weight outcomes of the National Institute of Child Health and Human Development Neonatal Research Network, January 1995 through December 1996. NICHD Neonatal Research Network [electronic article]. *Pediatrics*. 2001;107(1):E1.
9. Doussard-Roosevelt JA, Porges SW, Scanlon JW, et al. Vagal regulation of heart rate in the prediction of developmental outcome for very low birth weight preterm infants. *Child Dev.* 1997;68(2):173–186.
10. Ment LR, Vohr B, Allan W, et al. The etiology and outcome of cerebral ventriculomegaly at term in very low birth weight preterm infants. *Pediatrics*. 1999;104(2 pt 1):243–248.
11. Kuban KC, Leviton A. Cerebral palsy. *N Engl J Med.* 1994; 330(3):188–195.
12. Lorenz JM, Wooliever DE, Jetton JR, et al. A quantitative review of mortality and developmental disability in extremely premature newborns. *Arch Pediatr Adolesc Med*. 1998;152(5):425–435.
13. Crofts BJ, King R, Johnson A. The contribution of low birth weight to severe vision loss in a geographically defined population. *Br J Ophthalmol*. 1998;82(1):9–13.
14. Saigal S, Hoult LA, Streiner DL, et al. School difficulties at adolescence in a regional cohort of children who were extremely low birth weight. *Pediatrics*. 2000;105(2):325–331.
15. Resnick MB, Gueorguieva RV, Carter RL, et al. The impact of low birth weight, perinatal conditions, and sociodemographic factors on educational outcome in kindergarten [electronic article]. *Pediatrics*. 1999;104(6):e74.
16. Ross G, Lipper EG, Auld PA. Social competence and behavior problems in premature children at school age. *Pediatrics*. 1990;86(3):391–397.
17. Avchen RN, Scott KG, Mason CA. Birth weight and school-age disabilities: a population-based study. *Am J Epidemiol*. 2001;154(10):895–901.
18. Osmond C, Barker DJ, Winter PD, et al. Early growth and death from cardiovascular disease in women. *BMJ*. 1993; 307(6918):1519–1524.
19. Barker DJ, Martyn CN, Osmond C, et al. Growth in utero and serum cholesterol concentrations in adult life [comment]. *BMJ*. 1993;307(6918):1524–1527.
20. Weiss JL, Malone FD, Emig D, et al. Obesity, obstetric complications and cesarean delivery rate—a population-based screening study. *Am J Obstet Gynecol*. 2004;190(4): 1091–1097.
21. Weck RL, Paulose T, Flaws JA. Impact of environmental factors and poverty on pregnancy outcomes. *Clin Obstet Gynecol*. 2008;51(2):349–359.
22. Stillerman KP, Mattison DR, Giudice LC, et al. Environmental exposures and adverse pregnancy outcomes: a review of the science. *Reprod Sci.* 2008;15(7):631–650.
23. Geronimus AT. The weathering hypothesis and the health of African-American women and infants: evidence and speculations. *Ethn Dis*. 1992;2(3):207–221.
24. Geronimus AT. Black/white differences in the relationship of maternal age to birthweight: a population-based test of the weathering hypothesis. *Soc Sci Med.* 1996;42(4):589–597.
25. Geronimus AT. Understanding and eliminating racial inequalities in women's health in the United States: the role of the weathering conceptual framework. *J Am Med Womens Assoc.* 2001;56(4):133–136, 149–150.
26. Geronimus AT, Bound J, Waidmann TA, et al. Inequality in life expectancy, functional status, and active life expectancy across selected black and white populations in the United States. *Demography.* 2001;38(2):227–251.
27. Geronimus AT, Bound J, Waidmann TA, et al. Excess mortality among blacks and whites in the United States. *N Engl J Med*. 1996;335(21):1552–1558.
28. Rich-Edwards JW, Buka SL, Brennan RT, et al. Diverging associations of maternal age with low birthweight for black and white mothers. *Int J Epidemiol*. 2003;32(1):83–90.
29. Rauh VA, Andrews HF, Garfinkel RS. The contribution of maternal age to racial disparities in birthweight: a multilevel perspective. *Am J Public Health*. 2001;91(11): 1815–1824.
30. US Department of Health and Human Services. *Healthy People 2010: Understanding and Improving Health*. 2nd ed. Washington, DC: US Government Printing Office; 2000.
31. Centers for Disease Control and Prevention, National Center for Health Statistics. VitalStats. (http://www.cdc.gov/nchs/vitalstats.htm). (Accessed April 6, 2009).
32. North Carolina State Center for Health Statistics. Detailed Birth Record. Confidential Patient Record Database, 1990–2007. Raleigh, NC: State Center for Health Statistics.
33. Gray S, Edwards S, Miranda M. Assessing exposure metrics for air pollution and birthweight models in North Carolina. *J Expo Sci Environ Epidemiol*. In press.
34. Tassone E, Miranda ML, Gelfand AE. Disaggregated spatial modeling for areal unit categorical data. *J R Stat Soc Ser (C)*. In press.

35. Berrocal VJ, Gelfand AE, Holland DM. A spatio-temporal downscaler for output from numerical models. *J Agric Biol Environ Stat*. In press.
36. Branum AM, Schoendorf KC. Changing patterns of low birthweight and preterm birth in the United States, 1981–98. *Paediatr Perinat Epidemiol*. 2002;16(1):8–15.
37. Lynch JW, Everson SA, Kaplan GA, et al. Does low socioeconomic status potentiate the effects of heightened cardiovascular responses to stress on the progression of carotid atherosclerosis? *Am J Public Health*. 1998;88(3): 389–394.
38. Lynch J, Smith GD, Hillemeier M, et al. Income inequality, the psychosocial environment, and health: comparisons of wealthy nations. *Lancet*. 2001;358(9277):194–200.
39. Lynch J, Smith GD, Harper S, et al. Is income inequality a determinant of population health? Part 1. A systematic review. *Milbank Q*. 2004;82(1):5–99.
40. Finch BK. Early origins of the gradient: the relationship between socioeconomic status and infant mortality in the United States. *Demography.* 2003;40(4):675–699.
41. Cogswell ME, Yip R. The influence of fetal and maternal factors on the distribution of birthweight. *Semin Perinatol*. 1995;19(3):222–240.
42. Roberts EM. Neighborhood social environments and the distribution of low birthweight in Chicago. *Am J Public Health*. 1997;87(4):597–603.
43. Luo ZC, Wilkins R, Kramer MS. Effect of neighbourhood income and maternal education on birth outcomes: a population-based study. *CMAJ*. 2006;174(10):1415–1421.
44. McGrady GA, Sung JF, Rowley DL, et al. Preterm delivery and low birth weight among first-born infants of black and white college graduates. *Am J Epidemiol*. 1992;136(3): 266–276.
45. Schoendorf KC, Hogue CJ, Kleinman JC, et al. Mortality among infants of black as compared with white college-educated parents. *N Engl J Med*. 1992;326(23):1522–1526.
46. Simons RL, Lorenz FO, Wu CI, et al. Social network and marital support as mediators and moderators of the impact of stress and depression on parental behavior. *Dev Psychol*. 1993;29(2):368–381.
47. Williams L, Morrow B, Shulman H, et al. *2002 PRAMS Surveillance Report: Unintended Pregnancy and Contraceptive Use*. Atlanta, GA: Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention; 2006.
48. Hamilton BE, Martin JA, Ventura SJ. Births: preliminary data for 2007. *Natl Vital Stat Rep*. 2006;55(11):1–18.
49. Leddy MA, Power ML, Schulkin J. The impact of maternal obesity on maternal and fetal health. *Rev Obstet Gynecol*. 2008;1(4):170–178.
50. Watkins ML, Rasmussen SA, Honein MA, et al. Maternal obesity and risk for birth defects. *Pediatrics*. 2003;111(5 pt 2): 1152–1158.
51. Xiong X, Saunders LD, Wang FL, et al. Gestational diabetes mellitus: prevalence, risk factors, maternal and infant outcomes. *Int J Gynaecol Obstet*. 2001;75(3):221–228.
52. Porter TF, Fraser AM, Hunter CY, et al. The risk of preterm birth across generations. *Obstet Gynecol*. 1997;90(1):63–67.
53. Treloar SA, Macones GA, Mitchell LE, et al. Genetic influences on premature parturition in an Australian twin sample. *Twin Res.* 2000;3(2):80–82.
54. Engel SA, Erichsen HC, Savitz DA, et al. Risk of spontaneous preterm birth is associated with common proinflammatory cytokine polymorphisms. *Epidemiology.* 2005;16(4): 469–477.
55. Engel SA, Olshan AF, Savitz DA, et al. Risk of small-for-gestational age is associated with common anti-inflammatory cytokine polymorphisms. *Epidemiology.* 2005;16(4): 478–486.
56. Annells MF, Hart PH, Mullighan CG, et al. Interleukins-1, -4, -6, -10, tumor necrosis factor, transforming growth factor-beta, FAS, and mannose-binding protein C gene polymorphisms in Australian women: risk of preterm birth. *Am J Obstet Gynecol*. 2004;191(6):2056–2067.
57. Amory JH, Adams KM, Lin MT, et al. Adverse outcomes after preterm labor are associated with tumor necrosis factor-alpha polymorphism -863, but not -308, in mother-infant pairs. *Am J Obstet Gynecol*. 2004;191(4):1362–1367.
58. Roberts AK, Monzon-Bordonaba F, Van Deerlin PG, et al. Association of polymorphism within the promoter of the tumor necrosis factor alpha gene with increased risk of preterm premature rupture of the fetal membranes. *Am J Obstet Gynecol*. 1999;180(5):1297–1302.
59. Zhang XQ, Varner M, Dizon-Townson D, et al. A molecular variant of angiotensinogen is associated with idiopathic intrauterine growth restriction. *Obstet Gynecol*. 2003;101(2): 237–242.
60. Kajantie E, Rautanen A, Kere J, et al. The effects of the ACE gene insertion/deletion polymorphism on glucose tolerance and insulin secretion in elderly people are modified by birth weight. *J Clin Endocrinol Metab.* 2004;89(11):5738–5741.
61. Doh K, Sziller I, Vardhana S, et al. Beta2-adrenergic receptor gene polymorphisms and pregnancy outcome. *J Perinat Med.* 2004;32(5):413–417.
62. Bray MS, Krushkal J, Li L, et al. Positional genomic analysis identifies the β2-adrenergic receptor gene as a susceptibility locus for human hypertension. *Circulation*. 2000;101(25): 2877–2882.
63. Ward K, Hata A, Jeunemaitre X, et al. A molecular variant of angiotensinogen associated with preeclampsia. *Nat Genet.* 1993;4(1):59–61.
64. Arngrímsson R, Purandare S, Connor M, et al. Angiotensinogen: a candidate gene involved in preeclampsia? *Nat Genet.* 1993;4(2):114–115.
65. Wang X, Zuckerman B, Pearson C, et al. Maternal cigarette smoking, metabolic gene polymorphism, and infant birth weight. *JAMA*. 2002;287(2):195–202.
66. Infante-Rivard C. Drinking water contaminants, gene polymorphisms, and fetal growth. *Environ Health Perspect*. 2004; 112(11):1213–1216.
67. Lee DK, Fu K, Liang L, et al. Transgenic mouse blastocysts that overexpress metallothionein-I resist cadmium toxicity in vitro. *Mol Reprod Dev.* 1996;43(2):158–166.
68. Yang K, Julan L, Rubio F, et al. Cadmium reduces 11 beta-hydroxysteroid dehydrogenase type 2 activity and expression in human placental trophoblast cells [electronic article]. *Am J Physiol Endocrinol Metab*. 2005;290(1): E135–E142.
69. Wang X, Chen D, Niu T, et al. Genetic susceptibility to benzene and shortened gestation: evidence of gene-environment interaction. *Am J Epidemiol*. 2000;152(8):693–700.
70. Brown P. Race, class, and environmental health: a review and systematization of the literature. *Environ Res.* 1995;69(1): 15–30.
71. Lopez R. Segregation and black/white differences in exposure to air toxics in 1990. *Environ Health Perspect*. 2002; 110(suppl 2):289–295.
72. Stretesky PB. The distribution of air lead levels across U.S. counties: implications for the production of racial inequality. *Sociol Spectr.* 2003;23(1):91–118.

73. Stretesky P, Lynch M. Environmental justice and the predictions of distance to accidental chemical releases in Hillsborough County, Florida. *Soc Sci Q*. 1999;80(4):830–846.
74. Wolff MS, Britton JA, Wilson VP. Environmental risk factors for breast cancer among African-American women. *Cancer*. 2003;97(1 suppl):289–310.
75. Adler NE, Marmot M, McEwen BS, et al. *Socioeconomic Status and Health in Industrial Nations: Social, Psychological, and Biological Pathways*. Vol 896. New York, NY: The New York Academy of Sciences; 1999.
76. Do DP, Finch K, Basurto-Davila R, et al. Does place explain racial health disparities? Quantifying the contribution of residential context to the black/white health gap in the United States. *Soc Sci Med*. 2008;67(8):1258–1268.
77. Matthews TJ, Menacker F, MacDorman MF. Infant mortality statistics from the 2002 period: linked birth/infant death data set. *Natl Vital Stat Rep*. 2004;53(10):1–30.
78. Auten RL Jr, Mason SN, Tanaka DT, et al. Anti-neutrophil chemokine preserves alveolar development in hyperoxia-exposed newborn rats. *Am J Physiol Lung Cell Mol Physiol*. 2001;281(2):L336–L344.
79. Bobak M. Outdoor air pollution, low birth weight, and prematurity. *Environ Health Perspect*. 2000;108(2):173–176.
80. Bobak M, Leon DA. Air pollution and infant mortality in the Czech Republic, 1986–88. *Lancet*. 1992;340(8826): 1010–1014.
81. Gouveia N, Bremner SA, Novaes HM. Association between ambient air pollution and birth weight in São Paulo, Brazil. *J Epidemiol Community Health*. 2004;58(1):11–17.
82. Maisonet M, Bush TJ, Correa A, et al. Relation between ambient air pollution and low birth weight in the Northeastern United States. *Environ Health Perspect*. 2001;109(suppl 3): 351–356.
83. Loomis D, Castillejos M, Gold DR, et al. Air pollution and infant mortality in Mexico City. *Epidemiology*. 1999;10(2): 118–123.
84. Maroziene L, Grazuleviciene R. Maternal exposure to low-level air pollution and pregnancy outcomes: a population-based study. *Environ Health*. 2002;1(1):6.
85. Pereira LA, Loomis D, Conceição GM, et al. Association between air pollution and intrauterine mortality in São Paulo, Brazil. *Environ Health Perspect*. 1998;106(6):325–329.
86. Ritz B, Yu F, Chapa G, et al. Effect of air pollution on preterm birth among children born in Southern California between 1989 and 1993. *Epidemiology*. 2000;11(5):502–511.
87. Liu S, Krewski D, Shi Y, et al. Association between gaseous ambient air pollutants and adverse pregnancy outcomes in Vancouver, Canada. *Environ Health Perspect*. 2003;111(14): 1773–1778.
88. Lee BE, Ha EH, Park HS, et al. Exposure to air pollution during different gestational phases contributes to risks of low birth weight. *Hum Reprod*. 2003;18(3):638–643.
89. Lin CM, Li CY, Yang GY, et al. Association between maternal exposure to elevated ambient sulfur dioxide during pregnancy and term low birth weight. *Environ Res*. 2004; 96(1):41–50.
90. Wang X, Ding H, Ryan L, et al. Association between air pollution and low birth weight: a community-based study. *Environ Health Perspect*. 1997;105(5):514–520.
91. Ritz B, Yu F. The effect of ambient carbon monoxide on low birth weight among children born in Southern California between 1989 and 1993. *Environ Health Perspect*. 1999; 107(1):17–25.
92. Yang CY, Tseng YT, Chang CC. Effects of air pollution on birth weight among children born between 1995 and 1997 in Kaohsiung, Taiwan. *J Toxicol Environ Health A*. 2003;66(9): 807–816.
93. Perera F, Hemminki K, Jedrychowski W, et al. In utero DNA damage from environmental pollution is associated with somatic gene mutation in newborns. *Cancer Epidemiol Biomarkers Prev*. 2002;11(10 pt 1):1134–1137.
94. Jedrychowski W, Bendkowska I, Flak E, et al. Estimated risk for altered fetal growth resulting from exposure to fine particles during pregnancy: an epidemiologic prospective cohort study in Poland. *Environ Health Perspect*. 2004;112(14): 1398–1402.
95. Perlin SA, Wong D, Sexton K. Residential proximity to industrial sources of air pollution: interrelationships among race, poverty, and age. *J Air Waste Manag Assoc*. 2001; 51(3):406–421.
96. Ritz B, Wilhelm M. Ambient air pollution and adverse birth outcomes: methodologic issues in an emerging field. *Basic Clin Pharmacol Toxicol*. 2008;102(2):182–190.
97. Ritz B, Wilhelm M, Hoggatt KJ, et al. Ambient air pollution and preterm birth in the environment and pregnancy outcomes study at the University of California, Los Angeles. *Am J Epidemiol*. 2007;166(9):1045–1052.
98. Huynh M, Woodruff TJ, Parker JD, et al. Relationships between air pollution and preterm birth in California. *Paediatr Perinat Epidemiol*. 2006;20(6):454–461.
99. Sagiv SK, Mendola P, Loomis D, et al. A time-series analysis of air pollution and preterm birth in Pennsylvania, 1997–2001. *Environ Health Perspect*. 2005;113(5): 602–606.
100. Yang CY, Chang CC, Chuang HY, et al. Evidence for increased risks of preterm delivery in a population residing near a freeway in Taiwan. *Arch Environ Health*. 2003;58(10): 649–654.
101. Wilhelm M, Ritz B. Residential proximity to traffic and adverse birth outcomes in Los Angeles county, California, 1994–1996. *Environ Health Perspect*. 2003;111(2):207–216.
102. Metzer R, Delgado JL, Herrell R. Environmental health and Hispanic children. *Environ Health Perspect*. 1995;103(suppl 6): 25–32.
103. Sharara FI, Seifer DB, Flaws JA. Environmental toxicants and female reproduction. *Fertil Steril*. 1998;70(4):613–622.
104. Savitz DA, Whelan EA, Kleckner RC. Effect of parents' occupational exposures on risk of stillbirth, preterm delivery, and small-for-gestational-age infants. *Am J Epidemiol*. 1989; 129(6):1201–1218.
105. Gerhard I, Monga B, Waldbrenner A, et al. Heavy metals and fertility. *J Toxicol Environ Health A*. 1998;54(8):593–611.
106. Ronco AM, Arguello G, Muñoz L, et al. Metals content in placentas from moderate cigarette consumers: correlation with newborn birth weight. *Biometals*. 2005; 18(3):233–241.
107. Hopenhayn C, Ferreccio C, Browning SR, et al. Arsenic exposure from drinking water and birth weight. *Epidemiology*. 2003;14(5):593–602.
108. Ahmad SA, Sayed MH, Barua S, et al. Arsenic in drinking water and pregnancy outcomes. *Environ Health Perspect*. 2001;109(6):629–631.
109. Ostrea EM, Morales V, Ngoumgna E, et al. Prevalence of fetal exposure to environmental toxins as determined by meconium analysis. *Neurotoxicology*. 2002;23(3):329–339.
110. Gonzáles-Cossío T, Peterson KE, Sanin LH, et al. Decrease in birth weight in relation to maternal bone-lead burden. *Pediatrics*. 1997;100(5):856–862.
111. von Ehrenstein OS, Guha Mazumder DN, Hira-Smith M, et al. Pregnancy outcomes, infant mortality, and arsenic in

drinking water in West Bengal, India. *Am J Epidemiol*. 2006; 163(7):662–669.
112. Brody DJ, Pirkle JL, Kramer RA, et al. Blood lead levels in the US population. Phase 1 of the Third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991). *JAMA*. 1994;272(4):277–283.
113. Geronimus AT, Hillemeier MM. Patterns of blood lead levels in US black and white women of childbearing age. *Ethn Dis*. 1992;2(3):222–231.
114. Pirkle JL, Kaufmann RB, Brody DJ, et al. Exposure of the U.S. population to lead, 1991–1994. *Environ Health Perspect*. 1998;106(11):745–750.
115. Lanphear BP, Weitzman M, Eberly S. Racial differences in urban children's environmental exposures to lead. *Am J Public Health*. 1996;86(10):1460–1463.
116. Hertz-Picciotto I, Schramm M, Watt-Morse M, et al. Patterns and determinants of blood lead during pregnancy. *Am J Epidemiol*. 2000;152(9):829–837.
117. Vupputuri S, He J, Muntner P, et al. Blood lead level is associated with elevated blood pressure in blacks. *Hypertension*. 2003;41(3):463–468.
118. Sorel JE, Heiss G, Tyroler HA, et al. Black-white differences in blood pressure among participants in NHANES II: the contribution of blood lead. *Epidemiology.* 1991;2(5):348–352.
119. Téllez-Rojo MM, Hernández-Avila M, González-Cossío T, et al. Impact of breastfeeding on the mobilization of lead from bone. *Am J Epidemiol*. 2002;155(5):420–428.
120. Nieuwenhuijsen MJ, Toledano MB, Eaton NE, et al. Chlorination disinfection byproducts in water and their association with adverse reproductive outcomes: a review. *Occup Environ Med*. 2000;57(2):73–85.
121. King WD, Dodds L, Allen AC. Relation between stillbirth and specific chlorination by-products in public water supplies. *Environ Health Perspect*. 2000;108(9):883–886.
122. Rodenbeck SE, Sanderson LM, Rene A. Maternal exposure to trichloroethylene in drinking water and birth-weight outcomes. *Arch Environ Health*. 2000;55(3):188–194.
123. Elliott P, Briggs D, Morris S, et al. Risk of adverse birth outcomes in populations living near landfill sites. *BMJ*. 2001;323(7309):363–368.
124. Munger R, Isacson P, Hu S, et al. Intrauterine growth retardation in Iowa communities with herbicide-contaminated drinking water supplies. *Environ Health Perspect*. 1997; 105(3):308–314.
125. Wilson SM, Howell F, Wing S, et al. Environmental injustice and the Mississippi hog industry. *Environ Health Perspect*. 2002;110(suppl 2):195–201.
126. Faber DR, Krieg EJ. Unequal exposure to ecological hazards: environmental injustices in the Commonwealth of Massachusetts. *Environ Health Perspect*. 2002;110(suppl 2): 277–288.
127. Calderon RL, Johnson CC Jr, Craun GF, et al. Health risks from contaminated water: do class and race matter? *Toxicol Ind Health*. 1993;9(5):879–900.
128. Whyatt RM, Garfinkel R, Hoepner LA, et al. Within- and between-home variability in indoor-air insecticide levels during pregnancy among an inner-city cohort from New York City. *Environ Health Perspect*. 2007;115(3):383–389.
129. Wolff MS, Engel S, Berkowitz G, et al. Prenatal pesticide and PCB exposures and birth outcomes. *Pediatr Res.* 2007;61(2): 243–250.
130. Whyatt RM, Camann D, Perera FP, et al. Biomarkers in assessing residential insecticide exposures during pregnancy and effects on fetal growth. *Toxicol Appl Pharmacol*. 2005; 206(2):246–254.
131. Vassilev ZP, Robson MG, Klotz JB. Outdoor exposure to airborne polycyclic organic matter and adverse reproductive outcomes: a pilot study. *Am J Ind Med*. 2001;40(3):255–262.
132. Rull RP, Ritz B, Shaw GM. Neural tube defects and maternal residential proximity to agricultural pesticide applications. *Am J Epidemiol*. 2006;163(8):743–753.
133. Schreinemachers DM. Birth malformations and other adverse perinatal outcomes in four U.S. wheat-producing states. *Environ Health Perspect*. 2003;111(9):1259–1264.
134. Etzel RA, Balk SJ. *Pediatric Environmental Health*. 2nd ed. Elk Grove Village, IL: American Academy of Pediatrics; 2003.
135. US Environmental Protection Agency. America's Children and the Environment: Measures of Contaminants, Body Burdens, and Illnesses. EPA 240-R-03–001. Washington, DC: United States Environmental Protection Agency, Office of Children's Health Protection; 2003. (http://www.epa.gov/envirohealth/children). (Accessed April 6, 2009).
136. Wilson SE, Kahn RS, Khoury J, et al. Racial differences in exposure to environmental tobacco smoke among children. *Environ Health Perspect*. 2005;113(3):362–367.
137. Weaver VM, Davoli CT, Murphy SE, et al. Environmental tobacco smoke exposure in inner-city children. *Cancer Epidemiol Biomarkers Prev.* 1996;5(2):135–137.
138. McFarlane J, Parker B, Soeken K. Physical abuse, smoking, and substance use during pregnancy: prevalence, interrelationships, and effects on birth weight. *J Obstet Gynecol Neonatal Nurs*. 1996;25(4):313–320.
139. Goel P, Radotra A, Singh I, et al. Effects of passive smoking on outcome in pregnancy. *J Postgrad Med*. 2004;50(1):12–16.
140. Buescher PA. Smoking in pregnancy in North Carolina. Maternal characteristics and trends, 1988–1994. *N C Med J*. 1997;58(5):356–360.
141. Schoendorf KC, Kiely JL. Relationship of sudden infant death syndrome to maternal smoking during and after pregnancy. *Pediatrics*. 1992;90(6):905–908.
142. Mitchell EA, Ford RP, Stewart AW, et al. Smoking and the sudden infant death syndrome. *Pediatrics*. 1993;91(5): 893–896.
143. Klonoff-Cohen HS, Edelstein SL, Lefkowitz ES, et al. The effect of passive smoking and tobacco exposure through breast milk on sudden infant death syndrome. *JAMA*. 1995; 273(10):795–798.
144. Perera FP, Rauh V, Whyatt RM, et al. Molecular evidence of an interaction between prenatal environmental exposures and birth outcomes in a multiethnic population. *Environ Health Perspect*. 2004;112(5):626–630.
145. Choi H, Rauh V, Garfinkel R, et al. Prenatal exposure to airborne polycyclic aromatic hydrocarbons and risk of intrauterine growth restriction. *Environ Health Perspect*. 2008; 116(5):658–665.
146. Centers for Disease Control and Prevention. Behavioral Risk Factor Surveillance System Survey Data 1996 and 2006. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention. (http://apps.nccd.cdc.gov/brfss/). (Accessed April 6, 2009).
147. Malek SH, Hopkins DP, Molloy M, et al. The public health challenge of youth smoking in North Carolina. Putting what we know into practice. N C Med J. 2002;63(3):153–161.
148. Adler NE, Boyce T, Chesney MA, et al. Socioeconomic status and health. The challenge of the gradient. *Am Psychol*. 1994;49(1):15–24.
149. Weich S, Burton E, Blanchard M, et al. Measuring the built environment: validity of a site survey instrument for use in urban settings. *Health Place*. 2001;7(4):283–292.

150. Barry K, Britt DW. Outreach: targeting high-risk women through community partnerships. *Womens Health Issues*. 2002;12(2):66–78.
151. Schulz AJ, Zenk SN, Israel BA, et al. Do neighborhood economic characteristics, racial composition, and residential stability predict perceptions of stress associated with the physical and social environment? Findings from a multilevel analysis in Detroit. *J Urban Health*. 2008;85(5):642–661.
152. Wilkinson D. *Poor Housing and Ill Health: A Summary of Research Evidence*. Edinburgh, United Kingdom: The Scottish Office Central Research Unit, Housing Research Branch; 1999:1–15.
153. Evans GW, Wells NM, Chan HY, et al. Housing quality and mental health. *J Consult Clin Psychol*. 2000;68(3):526–530.
154. Steptoe A, Feldman PJ. Neighborhood problems as sources of chronic stress: development of a measure of neighborhood problems, and associations with socioeconomic status and health. *Ann Behav Med*. 2001;23(3):177–185.
155. Saelens BE, Sallis JF, Black JB, et al. Neighborhood-based differences in physical activity: an environment scale evaluation. *Am J Public Health*. 2003;93(9):1552–1558.
156. Saelens BE, Sallis JF, Frank LD. Environmental correlates of walking and cycling: findings from the transportation, urban design, and planning literatures. *Ann Behav Med*. 2003;25(2): 80–91.
157. Acevedo-Garcia D, Lochner KA. Residential segregation and health. In: Kawachi I, Berkman L, eds. *Neighborhoods and Health*. New York, NY: Oxford University Press; 2003: 265–287.
158. Bullard RD. *Dumping in Dixie: Race, Class, and Environmental Quality.* 3rd ed. Boulder, CO: Westview Press; 2000.
159. Williams DR, Collins C. Racial residential segregation: a fundamental cause of racial disparities in health. *Public Health Rep*. 2001;116(5):404–416.
160. Corburn J. Confronting the challenges in reconnecting urban planning and public health. *Am J Public Health*. 2004;94(4): 541–546.
161. Diez Roux AV. Residential environments and cardiovascular risk. *J Urban Health*. 2003;80(4):569–589.
162. Reagan PB, Salsberry PJ. Race and ethnic differences in determinants of preterm birth in the USA: broadening the social context. *Soc Sci Med*. 2004;60(10):2217–2228.
163. Zeka A, Melly SJ, Schwartz J. The effects of socioeconomic status and indices of physical environment on reduced birth weight and preterm births in Eastern Massachusetts. *Environ Health*. 2008;7:60.
164. Messer LC, Vinikoor LC, Laraia BA, et al. Socioeconomic domains and associations with preterm birth. *Soc Sci Med*. 2008;67(8):1247–1257.
165. Krieger N, Chen J, Waterman P, et al. Choosing area based socioeconomic measures to monitor social inequalities in low birth weight and childhood lead poisoning: the Public Health Disparities Geocoding Project (US). *J Epidemiol Community Health*. 2003;57(3):186–199.
166. Masi CM, Hawkley LC, Piotrowski ZH, et al. Neighborhood economic disadvantage, violent crime, group density, and pregnancy outcomes in a diverse, urban population. *Soc Sci Med*. 2007;65(12):2440–2457.
167. Schempf A, Strobino D, O'Campo P. Neighborhood effects on birthweight: an exploration of psychosocial and behavioral pathways in Baltimore, 1995–1996. *Soc Sci Med*. 2009; 68(1):100–110.
168. Shiono PH, Rauh VA, Park M, et al. Ethnic differences in birthweight: the role of lifestyle and other factors. *Am J Public Health*. 1997;87(5):787–793.
169. Grady SC. Racial disparities in low birthweight and the contribution of residential segregation: a multilevel analysis. *Soc Sci Med*. 2006;63(12):3013–3029.
170. Bell JF, Zimmerman FJ, Almgren GR, et al. Birth outcomes among urban African-American women: a multilevel analysis of the role of racial residential segregation. *Soc Sci Med*. 2006;63(12):3030–3045.
171. Auger N, Daniel M, Platt RW, et al. Association between perceived security of the neighbourhood and small-for-gestational-age birth. *Paediatr Perinat Epidemiol*. 2008; 22(5):467–477.
172. Hogue CJ, Hoffman S, Hatch MC. Stress and preterm delivery: a conceptual framework. *Paediatr Perinat Epidemiol*. 2001;15(suppl 2):30–40.
173. Dix T. The affective organization of parenting: adaptive and maladaptive processes. *Psychol Bull.* 1991;110(1): 3–25.
174. Downey G, Coyne JC. Children of depressed parents: an integrative review. *Psychol Bull.* 1990;108(1):50–76.
175. Tjaden P, Thoennes N. *Prevalence, Incidences, and Consequences of Violence Against Women: Findings From the National Violence Against Women Survey.* Washington, DC: National Institute of Justice and Centers for Disease and Prevention; 1998.
176. White L, Rogers SJ. Economic circumstances and family outcomes: a review of the 1990s. *J Marriage Fam*. 2000;62: 1035–1051.
177. Sims M, Rainge Y. Urban poverty and infant-health disparities among African Americans and whites in Milwaukee. *J Natl Med Assoc.* 2002;94(6):472–479.
178. Collins JW Jr, David RJ. Urban violence and African-American pregnancy outcome: an ecologic study. *Ethn Dis*. 1997;7(3):184–190.
179. DeNavas-Walt C, Proctor BD, Smith JC. *Income, Poverty, and Health Insurance Coverage in the United States: 2007*. Washington, DC: US Census Bureau; 2008. (Current Population Reports, P60-235).
180. Guilarte TR, Toscano CD, McGlothan JL, et al. Environmental enrichment reverses cognitive and molecular deficits induced by developmental lead exposure. *Ann Neurol*. 2003;53(1):50–56.
181. Staessen JA, Nawrot T, Hond ED, et al. Renal function, cytogenetic measurements, and sexual development in adolescents in relation to environmental pollutants: a feasibility study of biomarkers. *Lancet*. 2001;357(9269): 1660–1669.
182. Thatcher RW, Lester ML, McAlaster R, et al. Effects of low levels of cadmium and lead on cognitive functioning in children. *Arch Environ Health*. 1982;37(3):159–166.
183. Agency for Toxic Substances and Disease Registry. *ToxFAQs for Cadmium*. Atlanta, GA: Agency for Toxic Substances and Disease Registry; 2008. (CAS#: 7440-43-9).
184. Culhane JF, Rauh V, McCollum KF, et al. Maternal stress is associated with bacterial vaginosis in human pregnancy. *Matern Child Health J*. 2001;5(2):127–134.
185. Nansel TR, Riggs MA, Yu KF, et al. The association of psychosocial stress and bacterial vaginosis in a longitudinal cohort. *Am J Obstet Gynecol*. 2006;194(2):381–386.
186. Ness RB, Hillier S, Richter HE, et al. Can known risk factors explain racial differences in the occurrence of bacterial vaginosis? *J Natl Med Assoc.* 2003;95(3):201–212.
187. Petraglia F, Sutton S, Vale W. Neurotransmitters and peptides modulate the release of immunoreactive corticotropin-releasing factor from cultured human placental cells. *Am J Obstet Gynecol*. 1989;160(1):247–251.

188. Falkenberg ER, Davis RO, DuBard M, et al. Effects of maternal infections on fetal adrenal steroid production. *Endocr Res.* 1999;25(3-4):239–249.
189. Gomez R, Romero R, Edwin SS, et al. Pathogenesis of preterm labor and preterm premature rupture of membranes associated with intraamniotic infection. *Infect Dis Clin North Am.* 1997;11(1):135–176.
190. Romero R, Gomez R, Ghezzi F, et al. A fetal systemic inflammatory response is followed by the spontaneous onset of preterm parturition. *Am J Obstet Gynecol*. 1998;179(1):186–193.
191. Glynn LM, Wadhwa PD, Dunkel-Schetter C, et al. When stress happens matters: effects of earthquake timing on stress responsivity in pregnancy. *Am J Obstet Gynecol*. 2001; 184(4):637–642.
192. Wadhwa PD, Glynn L, Hobel CJ, et al. Behavioral perinatology: biobehavioral processes in human fetal development. *Regul Pept*. 2002;108(2-3):149–157.
193. Krocova Z, Macela A, Kroca M, et al. The immunomodulatory effect(s) of lead and cadmium on the cells of immune system in vitro. *Toxicol In Vitro*. 2000;14(1):33–40.
194. Kayama F, Yoshida T, Elwell MR, et al. Cadmium-induced renal damage and proinflammatory cytokines: possible role of IL-6 in tubular epithelial cell regeneration. *Toxicol Appl Pharmacol*. 1995;134(1):26–34.
195. Chen CJ, Liao SL. Oxidative stress involved in astrocytic alterations induced by manganese. *Exp Neurol*. 2002;175(1): 216–225.
196. Steerenberg PA, Zonnenberg JA, Dormans JA, et al. Diesel exhaust particles induced release of interleukin 6 and 8 by (primed) human bronchial epithelial cells (BEAS 2B) in vitro. *Exp Lung Res.* 1998;24(1):85–100.
197. O'Neill M, Veves A, Zanobetti A, et al. Air pollution and cardiovascular health among subjects with diabetes in Greater Boston: a preliminary analysis: ISEE-590. *Epidemiology.* 2003;14(5):S116–S117.
198. Takizawa H, Abe S, Ohtoshi T, et al. Diesel exhaust particles up-regulate expression of intercellular adhesion molecule-1 (ICAM-1) in human bronchial epithelial cells. *Clin Exp Immunol*. 2000;120(2):356–362.
199. Gentile D, Howe-Adams J, Trecki J, et al. Association between environmental tobacco smoke and diminished dendritic cell interleukin 10 production during infancy. *Ann Allergy Asthma Immunol*. 2004;92(4):433–437.
200. Cozen W, Diaz-Sanchez D, James Gauderman W, et al. Th1 and Th2 cytokines and IgE levels in identical twins with varying levels of cigarette consumption. *J Clin Immunol*. 2004;24(6):617–622.
201. Noakes PS, Holt PG, Prescott SL. Maternal smoking in pregnancy alters neonatal cytokine responses. *Allergy.* 2003; 58(10):1053–1058.
202. Race/ethnicity, gender, socioeconomic status—research exploring their effects on child health: a subject review. *Pediatrics*. 2000;105(6):1349–1351.
203. Collins JW Jr, David RJ, Symons R, et al. Low-income African-American mothers' perception of exposure to racial discrimination and infant birth weight. *Epidemiology.* 2000; 11(3):337–339.
204. Mustillo S, Krieger N, Gunderson EP, et al. Self-reported experiences of racial discrimination and Black-White differences in preterm and low-birthweight deliveries: the CARDIA Study. *Am J Public Health*. 2004;94(12): 2125–2131.
205. Berkman L, Breslow L, Wingard D. Health practices and mortality risk. In: Berkman L, Breslow L, eds. *Health and Ways of Living: The Alameda County Study.* New York, NY: Oxford University Press; 1983:61–112.
206. Repetti RL, Taylor SE, Seeman TE. Risky families: family social environments and the mental and physical health of offspring. *Psychol Bull.* 2002;128(2):330–366.
207. American Lung Association. *State of the Air 2009*. Washington, DC: American Lung Association; 2009.
208. Bureau of the Census, US Department of Commerce. Census 2000 Summary File 3. Washington, DC: Bureau of the Census; 2003. (http://www2.census.gov/census_2000/datasets/Summary_File_3/). (Accessed February 24, 2007).
209. Bureau of the Census, US Department of Commerce. Housing and Household Economic Statistics Division. American Housing Survey 2007, National Data. 2008. (http://www.census.gov/hhes/www/housing/ahs/nationaldata.html). (Accessed May 29, 2009).
210. Bureau of the Census and Bureau of Labor Statistics, US Department of Commerce. Current Population Survey, Annual Social and Economic Supplement. Females, Aged 20–44. 2007. (http://www.census.gov/cps/). (Accessed May 29, 2009).
211. National Center for Health Statistics. *Health, United States, 2008 With Chartbook*. Hyattsville, MD: Centers for Disease Control and Prevention, US Department of Health and Human Services; 2009.