University of Nebraska - Lincoln
DigitalCommons@University of Nebraska - Lincoln

Sociology Department, Faculty Publications | Sociology, Department of

1-1-2013

The Transgenerational Consequences of Discrimination on African-American Health Outcomes

Bridget J. Goosby
University of Nebraska-Lincoln, bgoosby2@unl.edu

Chelsea Heidbrink
University of Nebraska-Lincoln, cheidbrink2@unl.edu

Follow this and additional works at: http://digitalcommons.unl.edu/sociologyfacpub

Goosby, Bridget J. and Heidbrink, Chelsea, "The Transgenerational Consequences of Discrimination on African-American Health Outcomes" (2013). *Sociology Department, Faculty Publications.* Paper 230.
http://digitalcommons.unl.edu/sociologyfacpub/230

This Article is brought to you for free and open access by the Sociology, Department of at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in Sociology Department, Faculty Publications by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.

Published in *Sociology Compass* 7:8 (August 2013), pp. 630–643; doi: 10.1111/soc4.12054

Copyright © 2013 John Wiley & Sons Ltd. Used by permission.

Submitted October 22, 2012; revised February 15, 2013; accepted February 17, 2013; published online August 7, 2013.

The Transgenerational Consequences of Discrimination on African-American Health Outcomes

Bridget J. Goosby and Chelsea Heidbrink

Department of Sociology, University of Nebraska Lincoln

Corresponding author – Bridget J. Goosby, Department of Sociology, University of Nebraska–Lincoln, 742 Oldfather Hall, Lincoln, NE 68588, USA; email bgoosby2@unl.edu

Abstract

Disparities in African American health remain pervasive and persist transgenerationally. There is a growing consensus that both structural and interpersonal racial discrimination are key mechanisms affecting African American health. The Biopsychosocial Model of Racism as a Stressor posits that the persistent stress of experiencing discrimination take a physical toll on the health of African Americans and is ultimately manifested in the onset of illness. However, the degree to which the health consequences of racism and discrimination can be passed down from one generation to the next is an important avenue of exploration. In this review, we discuss and link literature across disciplines demonstrating the harmful impact of racism on African American physical health and the health of their offspring.

Racial differences in health outcomes in the United States are widespread and stark. Though there have been important technological advances contributing to the increase in population life expectancy in the past 50 years (Gortmaker and Wise 1997), the gap in health outcomes between Whites and certain minority groups remains substantial. According to the Center for the Disease Control's most recent report, rates of morbidity and mortality over the life course remain higher for African-Americans than for most other race/ethnic groups (CDC/NCHS 2011). Moreover, African-Americans have the highest rates of low birth weight births and substantially higher rates of infant mortality than other racial groups (Williams 2002). African-American children also experience higher rates of chronic health conditions such as asthma and obesity across socioeconomic status (CDC/NCHS 2011), and during adulthood, African-Americans exhibit the highest rates of hypertension and cancer mortality (Williams 2002). Though life expectancy has risen for all populations in the USA, both African-American men and women still exhibit the lowest life expectancy across race ethnic groups with men's average life expectancy at 70 years old and women's at 77 compared with 76 for White men and 81 for White women (CDC/NCHS 2011).

Sociologists systematically demonstrate the pervasive, harmful nature of social inequality for minority disparities across a range of outcomes including health, education, income, and family processes (Reskin 2012). There is an increasing awareness that racial discrimination is a salient mechanism perpetuating racial gaps in health with African-Americans showing some of the most substantial differences in prevalence and severity of certain health conditions across the life course (Williams 2012). The Biopsychosocial Model of Racism as a Stressor (BMRS) elucidates the psychosocial and biological pathways through which racism functions as a mechanism for perpetuating declines in health, particularly among African-Americans (Clark *et al.* 1999). Though there have been important strides in uncovering the life course pathways through which racial inequality perpetuates health outcomes across

the life course of racial minorities, the sociological literature has yet to effectively examine the degree to which the health consequences of racial discrimination are transmitted *transgenerationally*.

The health consequences of racism and discrimination can be persistent and passed from one generation to the next through the body's "biological memory" of harmful experiences (Thayer and Kuzawa 2011). Specifically, the psychosocial and biological pathways through which racism affects racial minority health can also have consequences for their offspring, potentially perpetuating the existing disparities in the next generation, in part, by the *embodiment* of inequality transmitted through epigenetic influences (Kuzawa and Sweet 2009). In other words, stressful conditions and poor health experienced by mothers can lead to alterations in her offspring's gene expression without changing his or her genotype. These changes in gene expression can have important implications for the healthy functioning of bodily systems in mothers and their offspring.

The goal of this paper is to elaborate on the contribution of the complex, interactive dynamics of racism and racial discrimination for driving persistent racial disparities in health across generations. In this review, we draw upon an interdisciplinary literature to inform our sociological understanding of the persistence of health disparities across generations. We use African-American women's health as a descriptive example illustrating the importance of eradicating racial inequality as a key solution to improving health outcomes of marginalized groups. Specifically, we argue that discrimination is a socially generated but physiologically disruptive force that influences health across generations.

The biopsychosocial consequences of racial discrimination as a stressor

Racism refers to the beliefs, attitudes, institutional arrangements, and interpersonal acts that malign a person or group people based on their racial or ethnic affiliation (Clark *et al.* 1999). Racist ideology and beliefs have been historically used as justification for discrimination both at the individual and institutional levels in the Unites States and remain ubiquitous (Reskin 2012; Williams 2012). The consequences of this racially discriminatory structure have given rise to system-wide inequities and disparate racial differences in health outcomes. Structurally, discrimination was instrumental in creating the most harmful conditions for minority health-economic marginalization and segregation, which have been examined at length in the sociological literature (Williams and Sternthal 2010). While structural inequality is instrumental in creating unequal access to goods and resources including quality education and health care, safe communities, and economic mobility (Geronimus and Thompson 2004), racial discrimination also takes a toll on the physiological and psychological health of minority group members who may internalize racial discrimination (Clark *et al.* 1999).

The BMRS posits that the stress of racial discrimination is instrumental in African-American health disparities due to the physiological responses to chronic and acute stress brought on by racist experiences (Clark *et al.* 1999). Specifically, the exposure to environments perceived as racist can result in an overactive physiological stress response. In other words, normal bodily systems responsible for adjusting to stressful conditions remain perpetually activated in the presence of chronic stress, leaving individuals vulnerable to illness due to elevated wear and tear on the body (McEwen 1998; McEwen and Seeman 1999); conditions which we will elaborate in more detail in the following section. We expand the original BMRS model to suggest that not only is the perception of racial discrimination important for African-American health but also just as important is the disproportionate exposure to social stressors brought on by being part of a racial group that is systematically discriminated against and marginalized (Geronimus 2001).

Recent findings indicate that African-Americans are disproportionately exposed to social stressors of higher frequency and severity (Sternthal *et al.* 2011). The high prevalence of African-Americans living in racially segregated conditions is a key structural mechanism through which they are exposed to elevated numbers of stressors (Williams and Sternthal 2010). African-Americans, even in the middle class, are more likely to live in conditions where they are exposed to, or in close proximity, to concen-

trated disadvantage, high unemployment rates, pollution, violent crime, and poor housing conditions (Reskin 2012; Geronimus and Thompson 2004; Sternthal *et al.* 2011). Subjection to such stressful, noxious conditions can play a harmful role in overall well-being and illness onset and progression.

Although disproportionately represented in poverty, African-Americans across socioeconomic strata exhibit poorer health on average relative to Caucasians. Middle class African-Americans are more likely to be exposed to discrimination at the individual or interpersonal levels, with more frequency because they are more likely to work in predominantly White environments, elevating the risk of discriminatory experiences and racist microaggressions (Colen 2011). Furthermore, socioeconomic status does not share equivalent meanings across race/ethnic groups with middle class African-Americans having less overall wealth than their Caucasian counterparts thereby living in more economically tenuous conditions (Williams and Sternthal 2010). Consequently, middle class African-Americans are more vulnerable to descending into poverty during economic downturns and are at an elevated risk for experiencing perpetual stress (Sternthal *et al.* 2011). In the following section we elaborate on the physiological consequences of stress for African American health and Table 1 defines key terms salient in the discussion of such processes.

Table 1. Key physiology terms

Stress reactivity	Environmental or social conditions that affect mental and physical well-being.
Allostasis	Bodily systems maintaining stability by adapting to change.
Allostatic load	Wear and tear experienced by the body as a consequence of inefficient shutting down or turning on of responses stressors to maintain allostasis.
HPA axis (hypothalamic–pituitary–adrenal axis)	Feedback interactions between hypothalamus and the pituitary and adrenal glands. Controls responses to stress and regulates immune system, mood and emotion, and energy storage/expenditures.
Cortisol	Glucocorticoid hormone released in response to stress; it increases blood sugar (glucose) circulation, suppresses immune system, aids in fat, protein, and carbohydrate metabolism.
Metabolism	Chemical reactions that occur in the human body that maintain life.
Metabolic syndrome	Combination of co-occurring medical conditions that elevate the risk of cardiovascular disease and diabetes.
Atherosclerosis	Thickening and inflammation of artery walls as a consequence of accumulation of cholesterol or other fatty materials.
Insulin	Hormone produced by the pancreas that helps regulate fat and carbohydrate metabolism by causing certain cells to take up glucose (blood sugar) circulating in blood.
Insulin resistance	Condition where cells do not respond to insulin causing blood glucose to rise. If not regulated can elevate risk of type 2 diabetes and cardiovascular disease.
Phenotype	An organism's observable traits and characteristics.
Genotype	Genetic composition of a cell, organism, or individual, made up of alleles or various forms of the same gene.
Epigenetics	Heritable changes in gene expression without changing underlying DNA sequence.
Telomere	Region at the ends of a chromosome containing repetitive nucleotide sequences that protect chromosome from deterioration. Telomeres become shorter over time due to cell division.
Oxidative stress	Imbalance in a biological system's ability to repair damage or detoxify reactive conditions within a cell. Associated with conditions such as cancer, cardiovascular disease, and chronic fatigue.
Fetal environment	Gestational conditions during prenatal development.
Low birth weight (microsomia)	Birth weight of less than 2500 g (5.8 lb)

The physiologic consequences of stress

The BMRS asserts that African-Americans who perceive certain circumstances as racist experience physiological stress responses that can be exacerbated by sociodemographic (e.g., socioeconomic status) and psychological characteristics (e.g., depression) along with behavioral factors (e.g., smoking, alcohol use), and coping responses (e.g., ability to mobilize social support) to such experiences (Clark *et al.* 1999). Consequently, perpetual stress responses to acute and chronically stressful conditions elevate one's vulnerability to disease. Both human and animal model studies of health and disease progression demonstrate that individuals lower in the social hierarchy have poorer health and higher disease risk than those who occupy higher social strata (Sapolsky 2006; McEwen and Seeman 1999). Although among humans, health risk behaviors such as smoking, drinking, and unhealthy eating habits are more likely to happen among socially marginalized groups, these behaviors do not completely account for health differences across social strata (Williams 2003). The psychosocial stress and burden of coping with limited access to resources and the harmful challenges associated with social marginalization can place substantial wear and tear on bodily systems important for managing stress and coping (allostatic load), thus leaving individuals vulnerable to disease (McEwen and Seeman 1999).

The human body's physiologic response to stressful conditions can elevate vulnerability to disease through metabolic, cardiovascular, and immune function (McEwen and Gianaros 2010). Moreover, the body's stress response is also tied to accelerate aging and cellular degradation (Epel *et al.* 2004). The body is made up complimentary systems that work synergistically to maintain normal functioning or homeostasis. In order to enable stability, the body works to create *allostasis* by adjusting bodily systems according to changes in the environment. In the event of abnormal functioning in the body, for instance, when the body experiences chronic stressors, wear and tear may occur as a consequence of the of the body attempting to turn on or shut down certain systems to maintain allostasis – this condition is called *allostatic load* (McEwen and Seeman 1999).

Cardiovascular and metabolic function

The cardiovascular and metabolic systems are linked to diseases including atherosclerosis, insulin resistance, and cardiovascular disease. In the presence of negative stressors, the body's cardiovascular system can respond by increasing blood circulation through elevated heart rate, a protective measure in the presence of acute stress, but if left unchecked can lead to the thickening and rigidity of the arterial walls (Sapolsky 2006). This process happens in concert with the mobilization of metabolic hormones such as cortisol, which are controlled by the hypothalamic–pituitary–adrenocortical (HPA) axis (McEwen 2000). Cortisol is a glucocorticoid hormone that is, in large part, responsible for managing energy storage and expenditures, immune function, mood, and the processing of fat, protein, and carbohydrates in the body. Cortisol also regulates the release of glucose (i.e., blood sugar) into the blood stream in order to provide energy during times of stress (Sapolsky 2006).

Chronically high levels of glucose in the blood stream can escalate arterial thickening due to inflammation and scarring from the viscous blood containing high levels of glucose and cholesterol that damage the arterial walls. The consequences are twofold – first, the combinations of elevated blood pressure with high levels of glucose and cholesterol particles circulating in the blood exacerbates the risk of cardiovascular conditions including hypertension and atherosclerosis (Brindley and Rolland, 1989). Second, high levels of unregulated glucose circulation elevate the risk of other conditions associated with cardiovascular disease including insulin resistance, abdominal obesity,

and type 2 diabetes, which when present together are defined as *metabolic syndrome* (Seeman *et al.* 2010; Black 2003).

Immune function

Adrenal steroids including the hormone cortisol also help regulate immune function by controlling movement of immune cells to organs or tissues that require protection from infections or other noxious conditions (McEwen and Seeman 1999). In the same way that stress responses can lead to dysregulation among the metabolic and cardiovascular systems, unchecked immune dysregulation can elevate disease risk through immune suppression or immune overactivity by way of a complex feedback loop. Specifically, in response to stress, the body prepares for infection by increasing immune function for wound healing by mobilizing energy (i.e., releasing glucose into the blood stream) and increasing levels of hormone secretion related to immune function including cortisol (Sapolsky 2006). When there is chronic overactivity of these processes, perpetually high levels of cortisol secretion actually suppresses the immune system thus leading to elevated risk of infections (McEwen 1998). In certain cases, however, exposure to stress can lead to *overactivity* of immune function where the immune system begins to attack the body in the absence of infections causing inflammation and autoimmune disorders such as rheumatoid arthritis, multiple sclerosis, and lupus (Segerstrom and Miller 2004).

Accelerated aging or cell degradation

Recently, emerging evidence points to the consequences of both perceived stress and the presence of chronic stressors for the rate at which cells age and degrade (Epel *et al.* 2004). *Telemores* are stabilizing caps at the ends of chromosomes that shorten through cell division until a cell destabilizes or begins to deteriorate with age (senescence) (Allsopp 1992; see Geronimus *et al.* 2010). *Oxidative stress* is a key biological process through which people experience accelerated aging or cell senescence. There is evidence that the presence of social stressors and psychological distress are linked to elevated levels of oxidative stress, consequently leading to shortened telomeres (Epel *et al.* 2004). Furthermore, overactivity of the HPA axis, discussed in prior sections, has been linked to oxidative stress as a consequence of excessive secretion of glucocorticoids such as cortisol (McIntosh *et al.* 1998). Shortened telomeres are particularly important for health outcomes because as telomere lengths shorten, risk of mortality increases. Moreover, there is clinical evidence that heart attack patients have similar telomere lengths to healthy individuals who are approximately *11 years older*. Strikingly, in a study assessing telomere lengths in premenopausal women who experience chronic stress, the researchers found that women who experienced chronic stressors had telomere lengths that resembled those of people who had experienced heart attacks in early middle age (Epel *et al.* 2004).

Racial discrimination and African-American health

The health consequences of racial discrimination are produced through structural (e.g., chronic poverty, poor infrastructure in non-White communities), institutional (e.g., educational institutions and employment discrimination), and individual processes (e.g., interpersonal discriminatory acts; Harrell *et al.* 2011; Clark *et al.* 1999). Stress-related chronic illness is a leading reason for the high rates of morbidity and mortality among African-Americans, particularly in urban areas (Geronimus and Thompson 2004). African-American women are especially vulnerable to the harsh conditions

brought about by the combination of racial inequality and poor socioeconomic conditions as evidenced by markedly higher rates of chronic illness and earlier onset of functional impairment across socioeconomic strata (Gorman and Read 2006). In this section, we discuss the processes through which racial discrimination contributes to stress-related illness and accelerated aging among African-Americans in general and African-American women specifically.

Structural discrimination and health

For African-Americans living in a race-conscious society, persistently experiencing stigmatization and disadvantage leaves them susceptible to physiological deterioration, subsequent illness, and early mortality (Geronimus *et al.* 2006). African-Americans that have disproportionately higher numbers of co-occurring stressors such as job discrimination, relationship stress, and financial hardship, that when experienced, are of greater intensity in their lives relative to Caucasians even after accounting for socioeconomic status (Sternthal *et al.* 2011). A key suggested structural mechanism for such differences in stress exposure and African-American health is racial segregation and the risks associated with it. African-Americans, particularly African-American women, are more likely to live in racially segregated conditions with high rates of poverty regardless of their socioeconomic status (Williams 2012). Those living in such segregated conditions are more likely to be exposed to stressors such as concentrated poverty, exposure to violent crime, limited access to institutional resources, poorer schools, and fewer safe outdoor spaces (Williams, 2002). Consequently, these conditions are associated with elevated levels of stress and hardship.

African-American women's health profiles

African-American women's health profiles show marked differences in the prevalence, onset, and severity of certain health conditions. Though African-American men have lower life expectancy relative to African-American women, women suffer from higher rates of chronic illness and experience functional impairment with earlier onset (Gorman and Read 2006). Furthermore, comorbid chronic illnesses are more prevalent in African-American women relative to White women (Williams 2002). African-American women also show different health profiles in cancer risk; African-American women have a lower incidence of breast cancer than White women but are more likely to experience a more aggressive form that is associated with substantially higher rates of cancer mortality relative to Caucasian women (Williams 2002).

African-Americans, in general, have higher rates of cardiovascular and diabetes-related mortality relative to their Caucasian counterparts (Mensah *et al.* 2005) and are more likely to experience blindness, amputations, and end stage kidney failure from diabetes (Williams 2002). There are gender differences in morbidity and mortality, however, with women being more likely to live with heart disease and other chronic conditions such as arthritis over time, while men are more likely to experience cardiovascular disease-related mortality (Crimmins *et al.* 2002). Among African-Americans, there is variation in health conditions by socioeconomic strata as well. Low-income African-American women are more likely to exhibit symptoms of accelerated aging manifested in higher allostatic load (Geronimus *et al.* 2006) compared with their Caucasian counterparts. In higher socioeconomic strata, however, African-American women show stark health differences relative to Caucasian women, with evidence that in addition to structural racial inequality, perceptions of racism and discrimination also take a toll on their health.

Discrimination and African-American women's health

There are important social pathways that contribute to African-American female health outcomes. African-American women even at higher levels of socioeconomic status are more likely to live in highly racially segregated areas (Williams 2002). Low-income African-American women experience alarming rates of early onset chronic illness and related mortality such as cardiovascular disease (Geronimus 2001); however, there are also marked Black–White health differences among African-American women occupying the upper socioeconomic echelons. Such differences are suggested to be related not only to their high risk of living in residentially segregated environments but also due to their likelihood of exposure to both structural and interpersonal discrimination. Middle class African-American women, specifically, are more likely to be exposed to discrimination in the form of structural (i.e., encountering the "glass" ceiling effect in employment mobility) and interpersonal discrimination due to their presence in predominantly White environments (Colen 2011; Thomas *et al.* 2008). The perpetual stress of being in a predominantly White work place may contribute to feelings of social isolation due to their "token" status as an African-American female (Braboy Jackson 1995; Colen 2011).

Across socioeconomic status, obesity rates among African-American women are higher than that of Caucasian women, which may in part be due to structural differences in their proximity to racially segregated communities which limit opportunities for physical activity (Geronimus 2001). However, the stress of experiencing racial discrimination is also linked to cardiovascular and metabolic conditions along with immune function in African-American women. Specifically, African-Americans who reported higher levels of racism in their lives report poorer self-rated health, higher rates of diseases including immunological, infectious, or endocrine conditions and more frequent common colds (Kwate *et al.* 2003). The stress of experiencing high levels of both lifetime and everyday discrimination is positively associated with weight gain and abdominal obesity in African-American women (Cozier *et al.* 2009). Experiencing racial discrimination is also associated with coronary artery calcification (Lewis *et al.* 2006), glucose intolerance, and diabetes (Tull and Chambers 2001; Tull *et al.* 1999), suggesting long-term impacts on health.

Although not directly linked to the stressor of discrimination specifically, but to stress more generally, African-American women are more vulnerable to experiencing accelerated aging. Specifically, Geronimus and colleagues found that middle age African-American women had markedly higher rates of aging, with telomere lengths by age 49–55 that signified being approximately *7.5 years* biologically older than White women. The authors note that these findings persisted even after accounting for perceived stress, poverty, and waist to hip ratio. They posited that increased waist to hip ratio (an indicator of abdominal fat) may be a key pathway through which race and also poverty impact telomere length and overall health (Geronimus *et al.* 2010). Such conditions also linked African-American women's birth outcomes.

Biological memory of racism and the role of epigenetics

Racial discrimination is linked to birth outcomes among African-American women even after accounting for psychological well-being, educational attainment, and risk behaviors such as alcohol use and smoking. African-American women who are exposed to and internalize racial discrimination over their lifetime and during pregnancy are more likely to have preterm and/or low birth weight births (Mustillo *et al.* 2004; Collins *et al.* 2004). Kuzawa and Sweet (2009) argue that the process through which African-Americans experience elevated risks of lower birth weight and subsequent chronic health conditions such as cardiovascular disease in adulthood are the result of a complex biosocial process where fetal exposure to maternal stress is manifested through biological

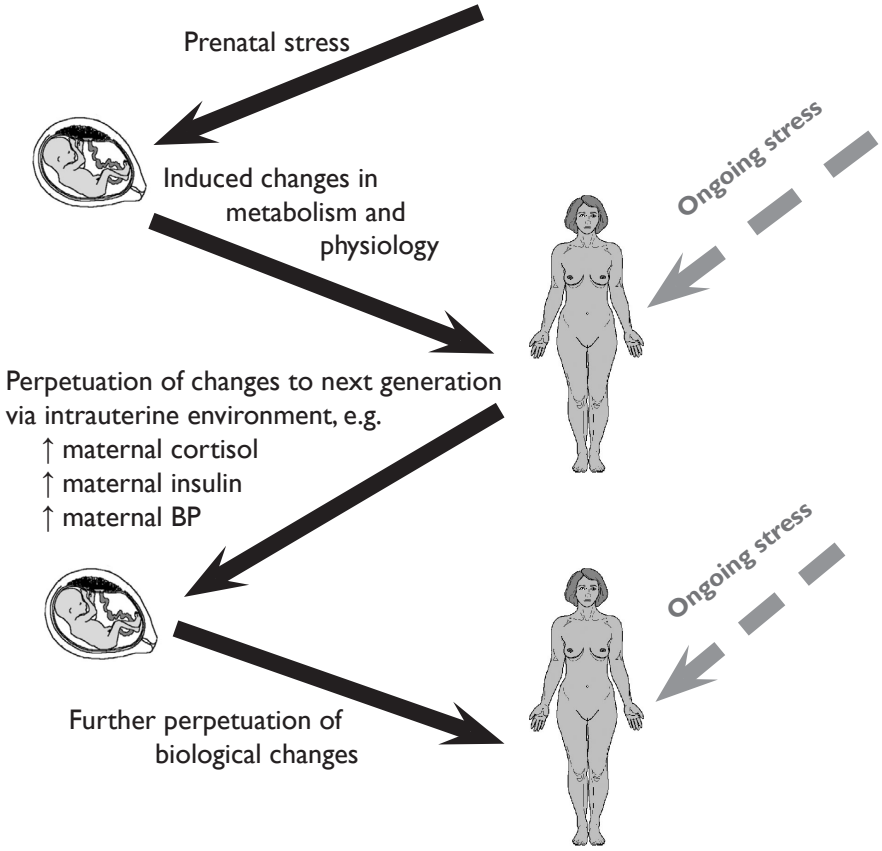

Figure 1. Transgenerational pathways of health through prenatal stress. Note: Based on Kuzawa, C. W. and E. Sweet. 2009. "Epigenetics and the embodiment of race: Developmental origins of U.S. racial disparities in cardiovascular health." *American Journal of Human Biology* 21: 2–15, figure 3.

modifications during fetal development (Kuzawa and Sweet 2009; Thayer and Kuzawa 2011).

As mentioned in the previous section, chronic and acute stressors can initiate and perpetuate heightened activity in various bodily systems that impact metabolic and immune function. These processes, however, are not only harmful for mothers experiencing such conditions but for their offspring as well. Figure 1 illustrates the pathways through which maternal stress experiences can influence health across generations through the interactions of biological and social contexts. In their discussion of the epigenetic consequences of racial inequalities for African-American cardiovascular disease risk, Kuzawa and Sweet (2009) outline the pathways through which the health consequences of racial inequality can persist across generations. The authors argue that the human body has a "biological memory" of early life exposure to harmful conditions that can impact gene expression without changing the underlying nucleotide sequences, which in the case of African-Americans includes the deleterious impact of discrimination.

Much dialog regarding genetic predispositions and phenotypic characteristics imply that genes are static; however, the epigenetic research indicates that social and physical environments can influence specific gene expression (i.e., epigenesis). For example, fetal undernourishment and other environmental conditions associated with low birth weight can lead to epigenetic alterations of genes in offspring, elevating their risk of hypertension, insulin resistance, and changing the composition of

body fat cells contained in adipose tissue (i.e., body fat; Lampl *et al.* 2002; Jensen 2007). These epigenetic changes, however, do not alter the underlying DNA sequence but instead "turn on" or "off" certain sets of genes associated with specific physiologic functions.

There is evidence that chronically stressful social conditions that impact African-American women's health in adulthood can be transferred as health risk to her children due to the fetal environment in which their offspring develop (i.e., prenatal stress), thus altering their metabolic and physiological development, demonstrated through the first and second arrows in Figure 1. Specifically, women who experience severe stress during pregnancy are at elevated risk for passing the stress hormone cortisol (third arrow) through their placenta which can restrict fetal growth and increase risk for preterm and low birth weight births (Phillips *et al.* 1998). Consequently, such exposure alters fetal HPA axis development and subsequent offspring biological stress reactivity.

In addition, women experiencing hypertension, insulin resistance, and diabetes during pregnancy are also at risk for low birth weight offspring (see Kuzawa and Sweet 2009). Because African-American mothers are more likely themselves be low birth weight, their own prenatal experience may influence the *in utero* environment of her offspring through the above health conditions. In addition to low birth weight risk, insulin resistance and diabetes in pregnant mothers elevate the likelihood of similar weight gain patterns and metabolic dysregulation in their offspring into adulthood through the transmission of high levels of glucose (e.g., blood sugar) via the placenta to the fetus (Dabelea *et al.* 2000; Lampl and Jeanty 2004; Silverman *et al.* 1995). Likewise, it is also important to note that in addition to fetal exposure to maternal stress and health conditions, transgenerational transmission of health is also passed through the "the continuity of [sic] environment" (Kuzawa and Sweet 2009, p9). In other words, offspring are born into the stressful environments in which their mothers lived and are also likely to experience similar risk factors including racial discrimination, which their mothers did, thus perpetuating a cycle of health risks across generations.

Racial inequalities in birth outcomes remain a persistent deleterious condition that disproportionately impacts African-American's long-term health prospects. A key social force driving such marked differences is African-Americans' disproportionate experiences with poverty and economic hardship, demonstrated by the similarity in birth outcomes between low-income African-American and Caucasian mothers. Specifically, among this particular population, both groups of women have similar rates of low birth weight births; however, once health risk behaviors such as smoking and drinking are accounted for, the birth weight disparity between Black and White women *widens* (Reichman *et al.* 2008) suggesting that additional factors beyond poverty and risk behavior may contribute to disparities in birth outcomes. Furthermore, more marked Black–White differences in birth outcomes emerge more starkly among middle class women.

Economic mobility among African-American women who were poor in childhood does not provide the same protections from low birth weight risk that it does for White women. Specifically, for White women who spent their childhoods in poverty, their risk of having a low birth weight child was reduced by 48 percent for every unit increase in family income, while for African-American women who experience income increases, there was not a statistically significant effect (Colen *et al.* 2006). Moreover, African-American females with a college degree have higher infant mortality rates than Hispanic and White women with a high school degree (Williams 2012). These findings compliment prior literature showing that middle class, college educated African-American women are at a substantially higher risk of having low birth weight children compared with their comparable Caucasian counterparts (Foster *et al.* 2000; McGrady *et al.* 1992; Schoendorf *et al.* 1992).

Birth weight is a particularly salient outcome to consider for later African-American health over the life course because birth weight is associated with a wide array of outcomes correlated with overall life chances. Being born with low birth weight is associated with a variety of health conditions such as obesity, diabetes mellitus, hypertension, and cardiovascular disease (Phillips *et al.* 1998). Moreover, low birth weight status elevates the risk of poor cognitive development, lower academic achievement (Goosby and Cheadle, 2009), and reduced chances of high school completion (Cheadle and Goosby 2010).

The "long arm" of discrimination

Assumptions of inherent immutable biological (i.e., genetic) differences between racial ethnic groups can lead health care providers specifically and the medical establishment in general to ignore the clear structural and social processes that perpetuate racial and ethnic differences in health outcomes. Several months ago, the popular press reported on a study released that suggested African-American girls who engaged in comparable levels of exercise relative to their Caucasian counter parts did not exhibit the same levels of weight loss. The headline of the article was "Exercise Not As Beneficial For Black Girls As Whites, Study Says" (Huffington Post 6/5/12), and the authors of the article suggested that different strategies are needed such as reducing energy (i.e., caloric) intake for African-American girls (White and Jago 2012) to address this disparity.

The popular press article went on to discuss the markedly high rates of obesity among African-Americans and then cited an editorial in the New York Times where writer Alice Randall argued that African-American women "want to be fat" in response to the article and the study it was based on (New York Times 5/5/2012). This study and the perception that African-American women want to be fat is a gross oversimplification of the complex challenges that lead to persistent issues with obesity and related health conditions. An important omission left from this dialog was the uniqueness of environmental factors facing African-American children generally and girls specifically that may predispose them to risk for obesity and chronic illness.

As discussed in previous section, African-American health trajectories are shaped in part by maternal life conditions including the experiences of racism and discrimination that can shape later outcomes of their offspring through potential changes in the child's metabolic functioning, subsequently elevating the risk of experiencing obesity and other chronic illnesses. So, in fact, the metabolic differences exhibited in the medical literature suggesting that African-Americans have phenotypes predisposed to obesity and other risk factors for cardiovascular disease (White and Jago 2012) makes a remarkable omission by not accounting for *or even mentioning* the harmful nature of structural inequality and discrimination for the differences among youth obesity rates. Indeed, there is evidence that among adolescents of African descent experiencing racism associated with body fat distribution and insulin resistance (Chambers *et al.* 2004), two conditions strongly associated with the body's physiological response to *chronic stress* (Sapolsky 2006). Taken together with early risk consequences minority youth may experience as early as *in utero*, the "long arm" of discrimination becomes increasingly more evident.

While there is growing acceptance that racial inequality does play a role in differential minority health outcomes, the example above demonstrates that the assumption of inherent genetic differences between racial groups still exists. These arguments do not account for deep systemic inequalities established based on racist ideology and the fact that much of the socioeconomic disadvantages and stressful conditions brought about by social marginalization can have lasting impacts on the overall health of people in marginalized groups. It has been established and accepted in the field of

sociology that racial categories in the USA are socially constructed and have been used as a tool to maintain power and oppress disenfranchised groups. Interestingly, however, there is *more* genetic variation within racial groups than across groups (Krieger 2005) indicating flawed assumptions in how racial differences in health occur.

Racial discrimination is persistent, systemic, and pervasive – emerging in multiple social contexts over life course for African-American youth. For those already susceptible to stress-related conditions, additional noxious experiences related to racial inequality can exacerbate health risks. There is an expanding literature demonstrating that interpersonal interactions as well as larger structural environments such as neighborhoods and schools can also play a salient role for shaping African-American life course health. There is evidence regarding the youth physiological stress responses to racism and discrimination, where particularly African-American girls who experience racism have higher waist circumference (Chambers *et al.* 2004). Moreover, economic and neighborhood disadvantage are linked to gender differences in physiological stress responses among African-American youth (Hackman *et al.* 2012) both conditions in which African-American youth are disproportionately represented.

Interestingly, there is also evidence that school context is a key factor during childhood and adolescence that can exacerbate existing health risks among African-American youth by perpetuating exposure to racism and discrimination as well as social alienation (Goosby and Walsemann 2012; Juvonen *et al.* 2006). Specifically, African-American adolescents attending predominantly White schools report more depressive symptoms and somatic complaints (stomach aches, nausea, headaches, etc.) and poorer self-rated health in early adulthood relative to youth in more integrated schools (Walsemann *et al.* 2011a, 2011b; Goosby and Walsemann, 2012). Such studies illustrate the emerging cycle of racial discrimination across generations and can exacerbate already existing health risk brought about by their parents' experiences with discrimination.

Conclusion

While protective social conditions such as family support and higher income may offset some of these risks for African-Americans, the added stress of being part of a marginalized group and experiencing discrimination may curtail potential gains made by economic mobility, thus leading to continued health risks associated poor birth outcomes in the next generation. Increasingly, policy makers are escalating strategies to address health disparities through eliminating differential treatment among health care providers, increasing access to care, and altering health risk behaviors. While these are important systemic changes, more attention is required to target the underlying social mechanism of racial discrimination at the structural and interpersonal levels that contribute to these disparities. The physiological consequences created by racial and economic inequality are not inalterable, however. But without addressing the harmful consequences of racial discrimination, improving the health of African-Americans as well as other marginalized groups will remain inadequately addressed.

Acknowledgements — This research is part of a larger study funded by the National Institute of Child Health and Human Development (K01 HD 064537; Bridget Goosby, PI).

References

Allsopp, R. C., H. Vaziri, C. Patterson, *et al.* 1992. "Telomere length predicts replicative capacity of human fibroblasts." *Proceedings of the National Academy of Sciences* 89: 10114–10118.

Black, Paul H. 2003. "The inflammatory response is an integral part of the stress response: Implications for atherosclerosis, insulin resistance, type II diabetes and metabolic syndrome X." *Brain, Behavior, and Immunity* 17: 350–364.

Braboy, Jackson, Pamela P. Peggy Thoits, and Howard F. Taylor. 1995. "Composition of the workplace and psychological well-being: the effects of tokenism on America's Black elite." *Social Forces* 74: 543–557.

Brindley, D. N., and Y. Rolland. 1989. "Possible connections between stress, diabetes, obesity, hypertension, and altered lipoprotein metabolism that may result in atherosclerosis." *Clinical Science* 77: 453–461.

Chambers, Earle C., Eugene S. Tull, Henry S. Fraser, *et al.* 2004. "The relationship of internalized racism to body fat distribution and insulin resistance among African adolescent youth." *Journal of the National Medical Association* 96: 1594–1598.

Cheadle, Jacob E., and Bridget J. Goosby. 2010. "Birth weight, cognitive development, and life chances: A comparison of siblings from childhood into early adulthood." *Social Science Research* 39: 570–584.

Clark, Rodney, N. B. Anderson, V. R. Clark, and David R. Williams. 1999. "Racism as a Stressor for African Americans: A Biopsychosocial Model." *American Psychologist* 54: 805–816.

Colen, Cynthia G. 2011. "Addressing Racial Disparities In Health Using Life Course Perspectives." *Du Bois Review* 8: 79–94.

Colen, Cynthia G., A. T. Geronimus, J. Bound, and S. A. James. 2006. "Maternal upward socioeconomic mobility and black-white disparities in infant birth weight." *American Journal of Public Health* 96: 2032–9.

Collins, James W., Richard J. David, Arden Handler, *et al.* 2004. "Very Low Birthweight in African American Infants: The Role of Maternal Exposure to Interpersonal Discrimination." *American Journal of Public Health* 94: 132–2138.

Cozier, Yvette C., Lauren A. Wise, Julie R. Palmer, and Lynn Rosenberg. 2009. "Perceived Racism in Relation to Weight Change in the Black Women's Health Study." *Annals of Epidemiology* 19: 379–387.

Crimmins, Eileen M., Jung Ki Kim, and Aaron Hagedorn. 2002. "Life with and without disease: Women experience more of both." *Journal of Women & Aging* 14: 47–59.

Dabelea, Dana, William C. Knowler, and David Pettit. 2000. "Effect of diabetes in pregnancy on offspring: Follow-up research in the pima Indians." *The Journal of Maternal-Fetal & Neonatal Medicine* 9: 83–88.

Epel, Elisa S., Elizabeth H. Blackburn, Jue Lin, *et al.* 2004. "Accelerated telomer shortening in response to life stress." *Proceedings of the National Academy of Science* 101: 17312–17315.

Foster, H. W., Lawrence Wu, M. B. Bracken, K. Semenya, *et al.* 2000. "Intergenerational effects of high socioeconomic status on low birth weight and preterm birth in African Americans." *Journal of the American Medical Women's Association* 92: 213–221.

Geronimus, Arline. T. 2001. "Understanding and Eliminating Racial Inequalities in Women's Health in the United States: The Role of Weathering Conceptual Framework." *Journal of the American Medical Women's Association* 56: 133–136.

Geronimus, Arline T., and J. Phillip Thompson. 2004. "To Denigrate, Ignore, or Disrupt: Racial Inequality in Health and the Impact of a Policy-induced Breakdown of African American Communities." *Du Bois Review* 1: 247–279.

Geronimus, Arline. T., Margaret Hicken, Danya Keene, and John Bound. 2006. "'Weathering' and age patterns of allostatic load scores among blacks and whites in the United States." *American Journal of Public Health* 96: 826–33.

Geronimus, Arline. T., Margaret Hicken, Jay A. Pearson, Sarah J. Seashols, Kelly L. Brown, and Tracey Davidson Cruz. 2010. "Do US Black Women Experience Stress-Related Accelerated Biological Aging?: A Novel Theory and First Population-Based Test of Black-White Differences in Telomere Length." *Human Nature* 21: 19–38.

Goosby, Bridget J., and Jacob E. Cheadle. 2009. "Birth Weight, Math and Reading Achievement Growth: A Multilevel Between-Sibling, Between-Families Approach." *Social Forces* 87: 1291–1320.

Goosby, Bridget J., and Katrina M. Walsemann. 2012. "School racial composition and race/ethnic differences in early adulthood health." *Health & Place* 18: 296–304.

Gorman, Bridget, and Jen'Nan Ghazal Read. 2006. "Gender Disparities in Adult Health: An Examination of Three Measures of Morbidity." *Journal of Health and Social Behavior* 47: 95–110.

Gortmaker, Steven L. and Paul H. Wise. 1997. "The First Injustice: Socioeconomic Disparities, Health Technology, and Infant Mortality." *Annual Review of Sociology* 23: 147–170.

Hackman, Daniel A., Laura M. Betancourt, Nancy Brodsky, *et al.* 2012. "Neighborhood disadvantage and adolescent stress reactivity." *Frontiers in Human Neuroscience* 6: 1–11.

Harrell, Camara Jules P., Tanisha I. Burford, Brandi N. Cage, Travette McNair Nelson, Sheronda Shearon, Adrian Thompson, and Steven Green. 2011. "Multiple Pathways Linking Racism to Health Outcomes." *Du Bois Review* 8: 143–157.

Jensen, Christine B., Heidi Storgaard, Sten Madsbad, *et al.* 2007. "Altered Skeletal Muscle Fiber Composition and Size Precede Whole-Body Insulin Resistance in Young Men with Low Birth Weight." *Journal of Clinical Endocrinology and Metabolism* 92: 1530–1534.

Juvonen, J., A. Nishina, and S. Graham. 2006. "Ethnic diversity and perceptions of safety in urban middle schools." *Psychological Science* 17: 393–400.

Krieger, Nancy. 2005. "Stormy Weather: Race, Gene Expression, and the Science of Health Disparities." *American Journal of Public Health* 95: 2155–2160.

Kuzawa, Christopher W., and Elizabeth Sweet. 2009. "Epigenetics and the embodiment of race: developmental origins of US racial disparities in cardiovascular health." *American Journal of Human Biology* 21: 2–15.

Kwate, Naa Oyo A., Heiddis B. Valdimarsdottir, Josephine S. Guevarra, Dana H. Bovjerg. 2003. "Experiences of Racist Events Are Associated with Negative Health Consequences for African American Women." *National Medical Association* 95: 450–460.

Lampl, Michelle and Phillipe Jeanty. 2004. "Exposure to maternal diabetes is associated with altered fetal growth patterns: A hypothesis regarding metabolic allocation to growth under hyperglycemic-hypoxemic conditions." *American Journal of Human Biology* 16: 237–263.

Lampl, Michelle, Christopher W. Kuzawa, and Phillipe Jeanty. 2002. "Infants thinner at birth exhibit smaller kidneys for their size in late gestation in a sample of fetuses with appropriate growth." *American Journal of Human Biology* 14: 398–406.

Lewis, Tene T., Susan A. Everson-Rose, Lynda H. Powell, Karen A. Matthews, Charlotte Brown, Kelly Karavolos, Kim Sutton-Tyrrell, Elizabeth Jacobs, and Deidre Wesley. 2006. "Chronic exposure to everyday discrimination and coronary artery calcification in African-American women: the SWAN Heart Study." *Psychosomatic Medicine* 68: 362–8.

McEwen, Bruce S. 1998. "Protective and Damaging Effects of Stress Mediators." *The New England Journal of Medicine* 338: 9.

McEwen, Bruce S. 2000. "The Neurobiology of Stress: From Serendipity to Clinical Relevance." *Brain Research* 886: 18.

McEwen, Bruce S., and Peter J. Gianaros. 2010. "Central role of the brain in stress and adaptation: links to socioeconomic status, health, and disease." *Annals of the New York Academy of Sciences* 1186: 190–222.

McEwen, Bruce S., and Teresa Seeman. 1999. "Protective and Damaging Effects of Mediators of Stress: Elaborating and Testing the Concepts of Allostasis and Allostatic Load." *Annals of the New York Academy of Sciences* 896: 30–47.

McGrady, Gene A., John F. Sung, Diane L. Rowley, and Carol J. Hogue. 1992. "Preterm delivery and low birth weight among first-born infants of Black and White college graduates." *American Journal of Epidemiology* 136: 266–276.

McIntosh, Laura J., Kay E. Hong, and Robert M. Sapolsky. 1998. "Glucocorticoids may alter antioxidant enzyme capacity in the brain: baseline studies." *Brain Research* 791: 209–214.

Mensah, G. A., A. H. Mokdad, E. S. Ford, *et al.* 2005. "State of disparities in cardiovascular health in the United States." *Circulation* 111: 1233–1241.

Mustillo, Sarah, Nancy Krieger, and Erica P. Gunderson. 2004. "Self-Reported Experiences of Racial Discrimination and Black–White Differences in Preterm and Low-Birth weight Deliveries: The CARDIA Study." *American Journal of Public Health* 94: 2125–2131.

Phillips, D. I., D.J.P. Barker, C.H.D. Fall, *et al.* 1998. "Elevated Plasma Cortisol Concentrations: A Link between Low Birth Weight and the Insulin Resistance Syndrome?." *Journal of Clinical Endocrinology and Metabolism* 83: 757–760.

Reichman, Nancy E., Erin R. Hamilton, Robert A. Hummer, and Yolanda C. Padilla. 2008. "Racial and Ethnic Disparities in Low Birth weight Among Urban Unmarried Mothers." *Maternal and Child Health Journal* 12: 204–215.

Reskin, Barbara. 2012. "The Race Discrimination System." *Annual Review of Sociology* 38: 17–35.

Sapolsky, Robert M. 2006. "*Why Zebras Don't Get Ulcers: An Updated Guide to Stress and Stress-Related Diseases, and Coping*." New York: W. H. Freeman & Co.

Schoendorf, K. C., C. J. Hogue, J. C. Kleinman, and D. L. Rowley. 1992. "Mortality among infants of Black compared with White college-educated parents." *The New England Journal of Medicine* 326: 1522–1526.

Seeman, Teresa, Elisa S. Epel, Tara Gruenewald, *et al.* 2010. "Socio-economic differentials in peripheral biology: Cumulative allostatic load." *Annals of the New York Academy of Sciences* 1186: 223–239.

Segerstrom, Suzanne C. and Gregory E. Miller. 2004. "Psychological stress and the human immune system: a meta-analytic study of 30 years of inquiry." *Psychological Bulletin* 130: 601–30.

Silverman, Bernard L., Boyd E. Metzger, Nam H. Cho, *et al.* 1995. "Impaired Glucose Tolerance in Adolescent Offspring of Diabetic Mothers: Relationship to fetal hyperinsulinism." *Diabetes Care* 18: 611–617. Statistics, National Center for Health. 2011. "*Health, United States: With a Special Feature on Socioeconomic Status and Health*." Hyattsville, MD.

Sternthal, Michelle, Natalie Slopen, and David R. Williams. 2011. "Racial Disparities in Health: How Much Does Stress Really Matter?." *Du Bois Review* 8: 19.

Thayer, Zaneta M., and Christopher W. Kuzawa. 2011. "Biological Memories of Past Environments: Epigenetic Pathways to Health Disparities." *Epigenetics* 6: 5.

Thomas, Anita Jones, Karen M. Witherspoon, and Suzette L. Speight. 2008. "Gendered racism, psychological distress, and coping styles of African American women." *Cultural Diversity & Ethnic Minority Psychology* 14: 307–14.

Tull, Eugene S. and Earle C. Chambers. 2001. "Internalized Racism Is Associated With Glucose Intolerance Among Black Americans in the U.S. Virgin Islands." *Diabetes Care* 24: 1498.

Tull, Eugene S., Tissa Wickramasuriya, Jerome Taylor, *et al.* 1999. "Relationship of internalized racism to abdominal obesity and blood pressure in Afro-Caribbean women." *Journal of the National Medical Association* 91: 447–452.

Walsemann, Katrina M., Bethany A. Bell, and Bridget J. Goosby. 2011a. "The effect of school racial composition on trajectories of depressive symptoms from adolescence through early adulthood." *Race and Social Problems* 3: 131–145.

Walsemann, Katrina M., Bethany A. Bell, and D. Maitra. 2011b. "The intersection of school racial composition and student race/ethnicity on adolescent depressive and somatic symptoms." *Social Science & Medicine* 72: 1873–1883.

White, James, and Russell Jago. 2012. "Prospective Associations Between Physical Activity and Obesity Among Adolescent Girls: Racial Differences and Implications for Prevention." *Archives of Pediatrics & Adolescent Medicine* 166: 522–527.

Williams, David R. 2002. "Racial/Ethnic Variations in Women's Health: The Social Embeddedness of Health." *American Journal of Public Health* 92: 10.

Williams, David R. 2003. "The Health of Men: Structured Inequalities and Opportunities." *American Journal of Public Health* 93: 8.

Williams, David R. 2012. "Miles to Go before We Sleep: Racial Inequalities in Health." *Journal of Health and Social Behavior* 53: 279–295.

Williams, David R. and Michelle Sternthal. 2010. "Understanding racial-ethnic disparities in health: sociological contributions." *Journal of Health and Social Behavior* 51 Suppl S: 15–27.