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How Race Becomes Biology: Embodiment of Social Inequality

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ABSTRACT The current debate over racial inequalities in health is arguably the most important venue for advancing both scientific and public understanding of race, racism, and human biological variation. In the United States and elsewhere, there are well-defined inequalities between racially defined groups for a range of biological outcomes—cardiovascular disease, diabetes, stroke, certain cancers, low birth weight, preterm delivery, and others. Among biomedical researchers, these patterns are often taken as evidence of fundamental genetic differences between alleged races. However, a growing body of evidence establishes the primacy of social inequalities in the origin and persistence of racial health disparities. Here, I summarize this evidence and argue that the debate over racial inequalities in health presents an opportunity to refine the critique of race in three ways: 1) to reiterate why the race concept is inconsistent with patterns of global human genetic diversity; 2) to refocus attention on the complex, environmental influences on human biology at multiple levels of analysis and across the lifecourse; and 3) to revise the claim that race is a cultural construct and expand research on the sociocultural reality of race and racism. Drawing on recent developments in neighboring disciplines, I present a model for explaining how racial inequality becomes embodied—literally—in the biological well-being of racialized groups and individuals. This model requires a shift in the way we articulate the critique of race as bad biology. Am J Phys Anthropol 139:47–57, 2009.

A recent cover story in *Scientific American* posed a question that has gained new life: "Does race exist?" (Bamshad and Olson, 2003). For decades, there seemed to be broad agreement among anthropologists and geneticists that the answer was "no," but some observers suggest that the consensus is unraveling (e.g., Leroi, 2005). Indeed, in both the scientific literature and the popular press, there is renewed debate over the magnitude and significance of genetic differences between racially defined groups (Jorde and Wooding, 2004; Keita et al., 2004; Ossorio and Duster, 2005; Bakalar, 2007; Drexler, 2007).

Yet much of the debate falters on the question—does race exist?—because it can be interpreted in different ways. The implicit question is usually whether race exists as a natural biological division of humankind. This question is important but incomplete. We should also ask in what ways race exists as a sociocultural phenomenon that has force in people's lives—one with biological consequences.

In this article, I take up these questions in the context of the current interdisciplinary debate over racial inequalities in health (Dressler et al., 2005a). This debate is important for three reasons. First, the magnitude of racial inequalities in health demands attention. In the United States, where debate over race is most intense, the risk of morbidity and mortality from every leading cause is patterned along racial lines (Keppel et al., 2002). The burden of poor health is especially high for African Americans: Between 1945 and 1999, more than 4.3 million African Americans died prematurely, compared to their white counterparts (Levine et al., 2001). This inequality needs to be explained and addressed.

Second, debate over race and health provides an important opportunity to advance scientific and public understanding of race, racism, and human variation. In recent years, several high-profile journals have devoted special issues to race; in each case, racial inequalities in health were a major focus of debate (*Nature Genetics*, 2004; *American Journal of Public Health*, 2005; *American Psychologist*, 2005). Moreover, when research on race and human variation makes the news, it often has to do with race, medicine, and disease (e.g., Wade, 2002, 2004; Bakalar, 2007; Drexler, 2007). Thus, if anthropologists want to reconcile race for anyone other than ourselves, we have to engage the debate over racial inequalities in health.

Third, the association between race and health exposes the inadequacy of the conventional critique of race in anthropology and other social sciences. Social scientists often dismiss race as a cultural construct, not a biological reality (e.g., Palmié, 2007; Shaw, 2007). However, this position requires more nuance. If race is not biology, some may ask, why are there such clear differences among racially defined groups in a range of biological phenomena? This question highlights the need to move beyond "race-as-bad-biology" (Goodman, 1997, p 22) to explain *how race becomes biology*.

There are two senses in which race becomes biology. First, the sociocultural reality of race and racism has biological consequences for racially defined groups. Thus, ironically, biology may provide some of the strongest evidence for the persistence of race and racism as socio-

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cultural phenomena. Second, epidemiological evidence for racial inequalities in health reinforces public understanding of race as biology; this shared understanding, in turn, shapes the questions researchers ask and the ways they interpret their data—reinforcing a racial view of biology. It is a vicious cycle: Social inequalities shape the biology of racialized groups, and embodied inequalities perpetuate a racialized view of human biology.

In this article, I address both ways that race becomes biology. To establish the significance of the problem, I begin with a brief review of the epidemiologic evidence regarding racial inequalities in health and show that these inequalities are commonly interpreted as evidence of fundamental, genetic differences between "races." Then, given the persistence of racial–genetic determinism, I argue that it is necessary to clarify and refine the critique of race in three ways: 1) to reiterate why race is insufficient for describing human genetic diversity, 2) to promote a more complex, biocultural view of human biology, and 3) to take seriously the claim that race is a cultural construct that profoundly shapes life chances. Drawing on social epidemiology and allied fields, I propose a model for anthropological research on racial inequalities in health that emphasizes the development and intergenerational transmission of racial health disparities across multiple levels of analysis. This model improves on the standard critique, which dismissed race as bad biology without offering a constructive framework for explaining biological differences among racially defined groups. It also entails a shift in how we articulate the critique of race as bad biology.

WHAT IS RACE?

Debate about race often founders on ambiguity in the definition of race. Following Smedley (2007, p 18), I define race as a worldview: "a culturally structured, systematic way of looking at, perceiving, and interpreting" reality. In North America, a central tenet of the racial worldview is that humans are naturally divided into a few biological subdivisions. These subdivisions, or races, are thought to be discrete, exclusive, permanent, and relatively homogenous (Keita and Kittles, 1997; Banton, 1998; Smedley, 2007). The race concept also implies that the superficial traits used to distinguish races reflect more fundamental, innate biological differences (Smedley, 2007). This definition should not be taken to mean that race is merely a bad idea. Race emerged from unique material circumstances in English North America (Harris, 1964), and racism remains embedded in social, political, and economic structures in the United States (Feagin, 2006).

Some researchers (e.g., Long and Kittles, 2003) distinguish between folk and scientific definitions of race. This distinction may be misleading, because scientists have played a pivotal role in constructing and legitimating race for centuries (Brace, 2005). The key elements of the racial worldview persisted in anthropology well into the twentieth century (Caspari, 2003), and it still shapes much research on race and health.

RACE AND HEALTH: EPIDEMIOLOGICAL EVIDENCE

There is abundant evidence of health inequalities among racially defined groups in many societies (e.g., Brockerhoff and Hewett, 2000; Cutter et al., 2001; Pan American Health Organization, 2001; Nazroo et al., 2007; Harding et al., 2008). Here, I focus on the United States, where epidemiological data has reflected and reinforced scientific thinking about race for more than 200 years (Krieger, 1987).

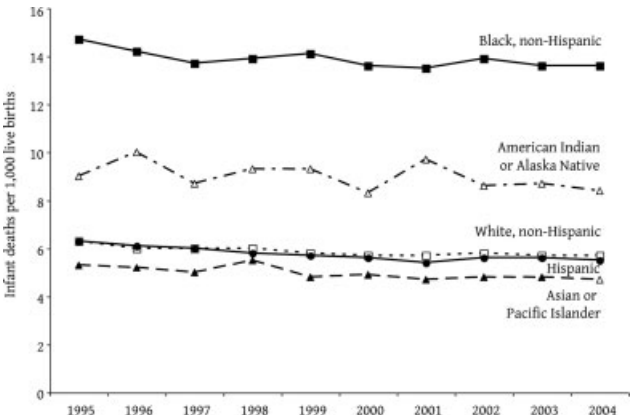

Fig. 1. Infant mortality in the United States, 1995–2004, by race and ethnicity (Data source: National Center for Health Statistics. 2007. Health, United States, 2007. Hyattsville: National Center for Health Statistics).

Epidemiological evidence in the United States shows that there are substantial racial inequalities in morbidity and mortality across multiple biological systems. The mortality profile is bleakest for African Americans: In 2004, the overall age-adjusted death rate for black Americans was more than 30% higher than it was for white Americans; for some leading causes of death, the disparity was substantially higher. Age-adjusted death rates from diabetes, septicemia, kidney disease, and hypertension and hypertensive renal disease were all more than two times higher among African Americans than among whites (Miniño et al., 2007). Cardiovascular disease accounts for the largest share of black–white difference in mortality (34.0%), but there are also substantial contributions from infections (21.1%), trauma (10.7%), diabetes (8.5%), renal disease (4.0%), and cancer (3.4%) (Wong et al., 2002).

Similar inequalities exist in infant mortality and life expectancy. From 1990 to 2004, infant mortality declined by 26% (9.2 to 6.8 per 1,000 live births) for the United States as a whole, but the gap between black and white Americans remained approximately the same (see Fig. 1). In 2004, the infant mortality rate among African Americans was 2.4 times the rate of other groups, as compared to 2.3 in 1990 (Keppel et al., 2002; Mathews and MacDorman, 2007). Black–white inequalities in life expectancy at birth narrowed dramatically in the early twentieth century—from 17.8 years in 1903 to less than seven in 1995—but changed relatively little in the second half of the century (Fig. 2). In 1995, the black–white gap in life expectancy was the same as it was 40 years earlier—6.9 years. Only recently has the gap narrowed to its historic low of just over 5 years (National Center for Health Statistics, 2007).

Much of the epidemiological literature focuses on such black–white comparisons. This focus is justified on grounds of the magnitude and historical depth of inequalities between black and white Americans, but crude black–white comparisons are limited in at least three ways. First, they conceal variation in morbidity and mortality profiles within racial categories. Second, they neglect the changing racial demography of the

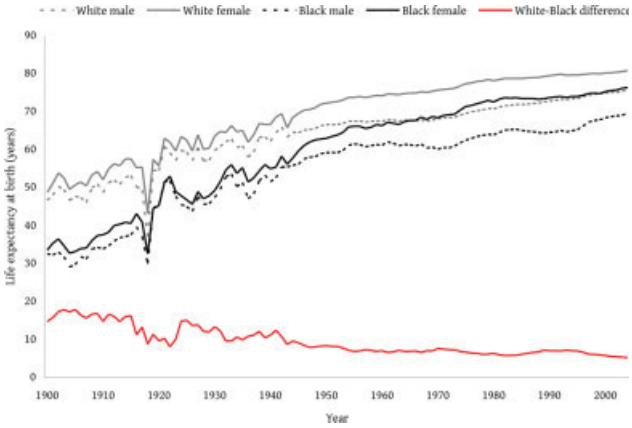

Fig. 2. Life expectancy at birth in the United States, 1900–2004, by race and ethnicity (Data source: Arias E. 2006. United States life tables, 2003. Natl Vital Stat Rep 54:1–40; National Center for Health Statistics, 2007). [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]

United States, where African Americans are no longer the largest ethnic minority group (Smelser et al., 1999). Third, they imply that race per se is an important cause of health inequalities, rather than focusing on the specific causal factors that shape racial inequalities in health (Kaufman and Cooper, 1995). Both genetic and social epidemiologists are developing new approaches to overcome these limitations (Gonzalez Burchard et al., 2005; Krieger et al., 2005; Murray et al., 2006), but much of the debate is still framed in black and white.

PERSISTENCE OF RACIAL–GENETIC DETERMINISM

In a recent review, Dressler et al. (2005a) identified five major models that researchers use to explain racial inequalities in health. Four models emphasize environmental factors, including 1) socioeconomic status, 2) health behaviors, 3) psychosocial stress, and 4) social structure and cultural context. The fifth model assumes that genetic factors contribute substantially to racial inequalities in health. This racial–genetic model continues to inform much biomedical research and clinical practice (Braun, 2006; Frank, 2007).

Racial–genetic determinism persists in part because of the uncritical use of race in biomedical sciences and public health. Systematic reviews in health-related disciplines show that race is widely used—appearing in ~80% of recent articles—but that it is seldom defined (Anderson and Moscou, 1998; Drevdahl et al., 2001; Comstock et al., 2004; Gravlee and Sweet, 2008). For example, in three independent reviews of literature in genetics (Sankar et al. 2007), infant mortality research (Anderson and Moscou, 1998), and health services research (Williams, 1994), not a single article defined race.

In lieu of explicit definitions, researchers typically use race as a proxy for some unspecified combination of environmental, behavioral, and genetic factors (Lin and Kelsey, 2000). Such usage not only obscures the causes of racial inequalities in health; it also favors the default assumption that racial differences are genetic in origin. Consider the implicit racial essentialism in a recent report from *The American Journal of Surgery*: "Is breast cancer in young Latinas a different disease?" (Biffl et al., 2001). Biffl et al. begin with the premise that "race may further influence breast cancer prognosis," and they seek to "clarify the relationship between race/ethnicity and disease severity" (p 596). Despite this aim, the paper concludes simply that "young Latinas might have more aggressive disease compared to other young women" (p 598). Biffl et al. do not suggest what biological process might account for this difference. They also do not explain what they mean by the term "race/ethnicity."

Discussants of the paper picked up on this point, however, and their published comments reveal the default assumption that race refers to genetic differences. Dr. Zannis was struck by "how primitive we are in identifying what patient sample we're talking about" (Biffl et al., 2001, p 600). He suggested that "how we racially profile our patients in these studies is important," and added: "I think in the future, we're going to have to get more sophisticated with identifying gene pools and not use the color of the patient's skin." Likewise, Dr. Allo cautioned:

> I think it's really important that you define what you mean by Latina because this could mean Mexican, it could mean Central American, it could mean Puerto Rican, and I don't think that you're dealing with a genetically identical gene pool in the best of circumstances (Biffl et al., 2001, p 600).

Both commentators are unquestionably right, but their remarks are most significant because they disclose the assumption that "race/ethnicity" means "gene pools." This assumption pervades much biomedical research, although it usually focuses on black–white comparisons (Rebbeck et al., 2006). For example, many researchers assume that African Americans' poorer survival after a cancer diagnosis, compared to whites, "reflects fundamental differences in the biology of the host or the attendant cancer or both" (Bach et al., 2002). Similarly, Pickering (2001, p 50) notes that "almost all" of the work to explain excess hypertension among African Americans "has involved the underlying assumption that there is some genetically determined physiological difference."

This assumption is most problematic when untested. Consider a recent, widely publicized study of racial inequalities in preterm birth. The study claimed to provide evidence for "important genetic contributors to the timing of birth" (Kistka et al., 2007, p 131.e1) and was featured in the *New York Times* under the headline, "Study points to genetics in disparities in preterm births" (Bakalar, 2007). However, the study actually presented no genetic data. Instead, researchers inferred a genetic cause from the residual difference between black and white mothers, after controlling for a few health behaviors and crudely measured socioeconomic variables. This finding does not warrant the conclusion that racial inequalities are genetic in origin; genetic hypotheses require genetic data. Yet, in a published roundtable discussion, several commentators agreed that "the genetic link is very strong" and that the black–white gap "may best be explained by a genetic etiology" (Stamilio et al., 2007, p e4, e5).

REFINING THE CRITIQUE OF RACE

The persistence of untested assumptions about race, genes, and health requires that the critique of race be refined in three ways. First, it is important to clarify why recent findings in population genetics do not refute

the claim that race is inadequate to describe global human genetic diversity. Second, it is critical to refocus attention on the complex, environmental influences on human biology. Third, it is necessary to revise the conventional view of race as a cultural construct to stimulate new research on the sociocultural dimensions of race and racism. I discuss each point in turn.

Race ≠ Human genetic variation

The classic critique of race has focused on three claims. First, most human genetic variation is clinal, such that there are seldom clear genetic boundaries between populations (Livingstone, 1962; Serre and Pääbo, 2004; Barbujani and Belle, 2006). Second, most human genetic variation is nonconcordant, such that the traits we use to distinguish races may have no value for predicting other aspects of biology (Goodman, 2000; Jorde and Wooding, 2004). Third, human genetic variation is widely shared across our species, with relatively little variation occurring between racially defined groups (Lewontin, 1972; Long and Kittles, 2003). Our basic understanding of these patterns has not changed in 50 years, despite enormous improvements in our technical ability to describe human genetic variation (Weiss and Fullerton, 2005).

Yet some researchers still defend race as a useful framework for describing human genetic variation—and for identifying genetic influences on racial differences in disease (Risch et al., 2002; Gonzalez Burchard et al., 2003; Bamshad et al., 2004). The defense of race relies on two related lines of evidence: 1) studies of worldwide genetic variation show that individuals from the same continent reliably cluster together (Rosenberg et al., 2002; Bamshad et al., 2003; Shriver et al., 2004; Rosenberg et al., 2005), and 2) in the United States, "self-identified race/ethnicity" is a useful proxy for genetic differentiation between groups that vary in continental ancestry (Tang et al., 2005).

These findings have important implications for genetic epidemiology (Barnholtz-Sloan et al., 2008) and population history (Tishkoff and Verrelli, 2003), but they do not refute the key arguments against the race concept. First, the claim that recent genetic studies "have recapitulated the classical definition of races" (Risch et al., 2002, p 3) misrepresents the purpose of cluster analysis, which is to detect pattern in a given dataset, not determine the essential number of subdivisions in our species. An example of this error is the common interpretation of Rosenberg et al. (2002) as evidence that humans are divided into five genetic clusters (e.g., Bamshad et al., 2004; Mountain and Risch, 2004; Leroi, 2005; Tang et al., 2005). Evidence that humans *can be* divided into five clusters does not mean they *are* naturally divided, as the classical definition of race would suggest. In fact, the number of clusters necessary to describe global genetic variation has been inconsistent; some studies report five (Rosenberg et al., 2002) and others seven (Corander et al., 2004; Li et al., 2008). Even when the number of clusters is consistent, their boundaries and composition are not [compare Corander et al., (2004) and Li et al., (2008)], and finer substructures are obscured.

Second, current defenders of race position themselves against a straw-man view that "racial and ethnic categories are purely social and devoid of genetic content" (Risch, 2006, p 408). This misleading portrayal of the critique sets the bar too low for proponents of racial classification; to resuscitate race, all they must do is show that they can reliably detect some genetic differentiation between racially defined groups, but the critique of race does not imply that racial categories correspond to no genetic differentiation. On the contrary, the argument that conventional racial classification accounts for only 5–10% of human genetic variation (Lewontin, 1972; Brown and Armelagos, 2001) implies a level of genetic differentiation that clustering algorithms ought to detect. Evidence of genetic clustering, then, does not contradict the claim that most human genetic variation occurs within rather than between traditional racial categories.

Third, recent studies confirm the claim that most human genetic variation is clinal. Several researchers have shown that genetic distance is strongly associated with geographic distance between populations (Serre and Pääbo, 2004; Manica et al., 2005; Handley et al., 2007; Li et al., 2008). The association is even stronger if one takes in account probable migration routes between continents over human history. For example, Ramachandran et al. (2005) show that geographic distances based on likely migration paths explain 78% of the variation in genetic distances between populations. Other studies show that geographic distance from East Africa explains 82–85% of the genetic diversity within populations (Prugnolle et al., 2005; Li et al., 2008). This pattern is consistent with a single origin of anatomically modern humans in East Africa, followed by serial migrations to other parts of the globe. Recent studies suggest that both clines and clusters are part of the structure of human genetic variation, but clusters explain relatively little total variation (Handley et al., 2007).

Fourth, the claim that continental ancestry may help to explain racial differences in disease (Salari et al., 2005; Risch, 2006; Tang et al., 2006) poses conceptual and methodological problems: First, estimates of genetic ancestry are generally based on noncoding DNA with unknown functional effects on disease (Cooper et al., 2003). Second, many alleles associated with common, complex diseases are likely to be ancient and shared across continental clusters (Keita et al., 2004). Third, nonconcordance implies that genetic clusters based on neutral markers may differ from clusters based on susceptibility alleles (Jorde and Wooding, 2004). Fourth, in racially stratified societies like the United States, continental ancestry is likely to be confounded with many environmental factors; consequently, reported associations between genetic ancestry and disease may be mediated through unmeasured environmental mechanisms (Kaufman and Cooper, 2008). These considerations imply that researchers should test specific hypotheses about the mechanisms linking ancestry and disease and remain cognizant that complex disease involves the interaction of many genetic and environmental influences.

To be clear, the critique of race is neither a denial of human biodiversity, nor a claim that genes are irrelevant to racial inequalities in health. Rather, the central argument is that the race concept is inadequate for describing the complex structure of human genetic variation. Clearly, there is geographic structure to human genetic variation. This structure is most consistent with a model of serial founder effects beginning with a single African origin of our species. Relatively low levels of genetic differentiation across major barriers to gene flow (e.g.,

Himalayas, the Sahara desert) appear to produce minor discontinuities that can be detected by clustering algorithms (Rosenberg et al., 2005), but to emphasize clustering at the expense of clinal variation and within-region diversity—the dominant signals—is to privilege a typological view of human genetic variation with pre-Darwinian roots (Caspari, 2003).

Biology ≠ Genetics

The argument that race does not correspond to global patterns of human genetic variation has come to dominate the critique of race. Yet, as important as the genetic evidence is, it understates the case against race. Indeed, the emphasis on genetic evidence may undermine the critique, because it tacitly accepts the primacy of genes in describing and explaining human biological variation. Thus, it is important to expand the critique of race by rejecting naïve reductionism and replacing it with a more complex view of human biology that acknowledges the interplay of organisms and environments over the life course.

This goal may require a shift in the way we articulate the critique of race. Often the critique is condensed to the idea that "race is not biology." Sometimes, this idea appears in the context of more subtle arguments about the complexity of human biology (e.g., Goodman, 2000), but more often it stands alone as a ritual repudiation of the race myth. Despite its popularity in scholarly circles, this ritual has failed to sway public understanding of race. As one observer put it, "Clearly for mainstream popular culture, the idea that race is not biology is still 'surprising' news" (Caminero-Santangelo, 2004, p 207).

The debate over racial inequalities in health brings this problem into sharp relief. Epidemiologic evidence shows that, in a very certain sense, race *is* biology. There are, in fact, well-defined differences between racially defined groups for a range of biological outcomes—cardiovascular disease, diabetes, renal failure, cancer, stroke, and birth outcomes, to name a few. In the face of this evidence, the refrain that race is not biology is impotent at best, counterproductive at worst. The challenge is to move beyond the pat assertion that race is not biology to explain how race *becomes* biology.

This shift in emphasis suggests that we may need to devote as much attention to revising our conception of biology as we do to our conception of race. Some observers may be uneasy with talk of biological differences among racially defined groups. They may worry—with good cause—that such talk reinforces the perception of intrinsic, genetic differences between alleged races. This well-founded concern is important, because it reveals how deeply entrenched the twin assumptions of reductionism and genetic determinism are in our understanding of race (Caspari, 2003) and biology in general (Lewontin, 2000). The idea that it is politically dangerous to discuss biological differences among racially defined groups makes sense only if we (or our audience) implicitly reduce biology to genetics and minimize or ignore the causal influence of external, environmental factors on human biology. The tacit conflation of genes and biology in the conventional critique of race unwittingly perpetuates this form of reductionism.

Recent research on racial inequalities in health provides a counterweight to reductionism and lends support for renewed attention to phenotypic plasticity and a complex view of human biology as biocultural. One influential model is Krieger's ecosocial theory for social epidemiology (Krieger, 1994, 2001). To comprehend humans' dual status as biological organisms and social beings, Krieger proposes the construct of *embodiment*:

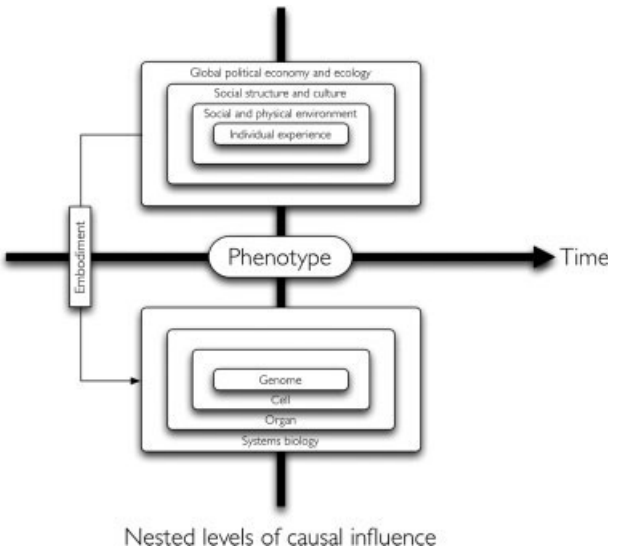

Fig. 3. Conceptual model for the study of multilevel and developmental influences on phenotype.

> a concept referring to how we literally incorporate, biologically, the material and social world in which we live, from conception to death; a corollary is that no aspect of our biology can be understood absent knowledge of history and individual and societal ways of living (Krieger, 2005, p 352).

There is an obvious affinity between *embodiment* and a century of anthropological research on human biology in the context of culture. Indeed, Franz Boas might be seen as a pioneer in the study of embodiment. His demonstration that descendants of immigrants embodied the new American environment (Boas, 1912) established plasticity as a central construct in human biology and turned the tide against biological determinism in anthropology (Gravlee et al., 2003). Yet the construct of *embodiment* does work that *plasticity* alone does not. In particular, Krieger's model reflects an emerging consensus that the next wave of research needs to integrate 1) multiple levels of analysis with 2) developmental and life-course perspectives. The conceptual model in Figure 3 illustrates the approach, drawing on previous recommendations for research on the social patterning of health (e.g., Kaplan, 2004; Glass and McAtee, 2006; Diez Roux, 2007; Krieger, 2008).

A key feature of this model is that it situates phenotype at the intersection of two axes. The first (horizontal) axis represents time. This axis may reflect life-course, developmental processes at an individual level or historical change at a population level (Glass and McAtee, 2006). The second (vertical) axis represents the nested hierarchy of causal influences on phenotypes, ranging from the genome to global political economy and ecology. The line depicting embodiment represents the direct and indirect influences of sociocultural context at multiple scales and levels (Krieger, 2008) on gene expression and biological functioning. Although the model draws on cur-

rent developments in health-related social sciences, the main elements and connections are also recognized in anthropology (e.g., Baker, 1997; Goodman and Leatherman, 1998; Kuzawa and Pike, 2005).

The model applies to population health in general, but a growing body of evidence establishes its importance for explaining racial inequalities in health in particular. First, recent research on the health effects of racism points to direct and indirect effects of racism across multiple levels of analysis. At an individual level, the experience of unfair treatment or interpersonal discrimination has a wide range of embodied consequences (Krieger, 1999). Researchers in several societies have linked self-reported experiences of discrimination to elevated blood pressure (Steffen et al., 2003; Brondolo et al., 2008), breast cancer (Taylor et al., 2007), coronary artery calcification (Lewis et al., 2006), body mass index (Gee et al., 2008), abdominal adiposity (Vines et al., 2007), preterm birth (Dole et al., 2004), low birth weight (Mustillo et al., 2004), depression (Williams et al., 2003; Borrell et al., 2006; Kelaher et al., 2008), and other aspects of mental and physical health and health-related behaviors (Harris et al., 2006; Borrell et al., 2007; Chae et al., 2008; Ryan et al., 2008).

At a higher level of analysis, studies show that institutionalized racism contributes to racial disparities in health, above and beyond individual factors. In particular, Williams and Collins (2001) argue that racial residential segregation is a fundamental cause of racial inequalities in health, because it a) constrains opportunities for success on traditional markers of individual SES such as education, occupational status, or income, and b) creates pathogenic social contexts that influence the distribution of disease. Recent studies bear out this argument. Residential segregation has been associated with overweight and obesity (Chang, 2006), low birth weight (Grady, 2006), fetal growth restriction (Bell et al., 2006), cardiovascular disease (Cooper et al., 2001), tuberculosis (Acevedo-Garcia, 2000), and all-cause mortality (Inagami et al., 2006). A related body of research links a variety of neighborhood conditions to health, independent of individual-level risk factors (Ellen et al., 2001; Sampson et al., 2002; Diez Roux, 2003; Kawachi and Berkman, 2003; Zenk et al., 2005; Cozier et al., 2007; Primack et al., 2007; O'Campo et al., 2008). One recent study in Chicago, for example, found that the unadjusted odds of hypertension were 80% higher for African Americans than for whites; controlling for individual-level factors reduced the disparity only slightly, but adding neighborhood-level variables completely eliminated the black–white gap in prevalence of hypertension (Morenoff et al., 2007).

There is also evidence that structures and events at even higher levels of analysis reverberate to the individual level. A recent study of birth outcomes before and after September 11, 2001, provides a dramatic example. Lauderdale (2006) examined birth certificate data for all California births during the 6 months after September 2001, compared to the same period 1 year earlier. They found that women with Arabic names—and *only* women with Arabic names—experienced a 34% increased in the likelihood of having a low birth weight infant after 9/11. Moreover, the effect appeared to be moderated by parents' strength of ethnic identification: Infants who were given ethnically distinctive Arabic names had twice the risk of low birth weight after the attacks of September 2001, compared to 1 year earlier. This finding hints

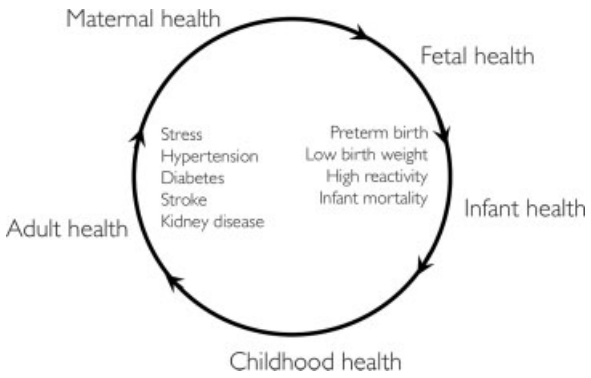

Fig. 4. Conceptual model for the emergence and persistence of health inequalities over the life course and across generations [adapted from Kuzawa (2008)].

at how events structured by global political–economic forces may have embodied consequences that are often hidden from view (Krieger, 2008).

Second, a growing body of research addresses the time axis (see Fig. 3) and suggests that inequalities across multiple levels of analysis have lingering effects across the life course and even from one generation to the next. This body of work draws on life course epidemiology (Davey Smith, 2003; Kuh and Shlomo, 2004) and on recent developments in evolutionary and developmental biology (West-Eberhard, 2003; Gluckman and Hanson, 2005; Jablonka and Lamb, 2005). The synthesis of these fields has the potential to produce a minor revolution in how we think about racial differences in biology, because it identifies the biological—but not genetic—pathways through which social disadvantage may be transmitted from one generation to the next (Schell, 1997; Drake and Walker, 2004; Gluckman et al., 2007).

Figure 4, adapted from Kuzawa (2008), illustrates the general model. The toxic effects of exposure to racism in one's own lifetime include a higher risk of hypertension, diabetes, stroke, and other conditions (Williams, 1999; Geronimus, 2001). These conditions, in turn, affect the health of the next generation, because they alter the quality of the fetal and early postnatal environment. The immediate consequence of this intergenerational effect is a higher risk of adverse birth outcomes (Rosenberg et al., 2002; Collins et al., 2004; Mustillo et al., 2004; Giscombé and Lobel, 2005; Bell et al., 2006; Dominguez et al., 2008), but there is also a lingering effect into adulthood, as adult chronic diseases like heart disease and diabetes can be traced in part to prenatal and early life conditions (Barker, 2004; Adair and Dahly, 2005; Cruickshank et al., 2005; Pollitt et al., 2005; Junien and Nathanielsz, 2007). Thus, the cycle begins again.

David and Collins (2007) provide an elegant example of how these life course and intergenerational processes unfold. They first compared birth weights across three groups of women who gave birth in Illinois during 1980–1995: U.S.-born black women, African-born black women, and U.S.-born white women. Contrary to the racial–genetic model, the distribution of birth weight for infants of African-born black women was almost identical to that for U.S.-born white women. By contrast, the entire distribution was shifted downward for U.S.-born black women (David and Collins, 1997). Within a single generation, however, the relative advantage of African- and Caribbean-born women began to disappear. The first

generation of girls born in the United States to mothers of African descent grew up to have girls of their own with lower mean birth weights—a trend that shifted the distribution toward that of U.S.-born black women (Collins et al., 2002).

This example brings us full circle to the roots of the critique of race in anthropology (Boas, 1912). The major elements of that critique still apply, but it is increasingly clear that we need new ways to articulate the failures of race. The common assertion that "race is not biology" may be correct in spirit, but it is too crude and imprecise to be effective. It does not adequately challenge the reductionism and genetic determinism of contemporary biomedical science or popular culture, and it blinds us to the biological consequences of race and racism as sociocultural phenomena.

Race ≠ Myth

The counterpart to the assertion that "race is not biology" is the mantra that "race is a cultural construct." As a growing number of cultural anthropologists recognize, this element of the critique also needs to be reexamined. The central problem is that, when biological anthropologists declared race a "myth" (Montagu, 1997), the concept lost its place in anthropology. The rise of "no-race" anthropology (Harrison 1995) came to mean not only that there were no biological races of humankind but also that there was no *discussion* of race in anthropology. Only in the last decade have race and racism reemerged as a major areas of research in cultural anthropology (Mukhopadhyay and Moses 1997; Mullings, 2005).

In advancing this line of research, I suggest that the conceptualization of race as a cultural construct needs to be refined in two ways. First, it cannot be—or appear to be—a wholesale dismissal of human biological diversity. In a recent invited commentary in *American Ethnologist*, Shaw (2007, p 236) laments that anthropology's view of race as "locally variable and socially constructed never captured the popular imagination in the United States":

> For decades, anthropologists have tried to teach the world that commonly used racial categories have little or no biological validity and that race is a social idea used in practices and institutions to give people differential access to opportunities and resources. More recently, amid reports of the Human Genome Project, anthropologists have joined others in trumpeting the homogeneity of the genetic makeup of people around the globe (Shaw, 2007, p 236).

Shaw rightly attributes the staying power of race to deeply embedded political and economic structures that sustain racial thinking and oppose "trumpeting the homogeneity" of humankind, but she does not appear to consider that there may be something wrong with the trumpet: Part of the reason people are not convinced by the claim of homogeneity is that it is false. We are indeed a less variable species than are our closest relatives, but genetic variation exists. Moreover, as current defenders of race emphasize, variation is structured in such a way that there are detectable genetic differences between people who self-identify with conventional racial categories (Risch et al., 2002; Tang et al., 2005). The denial of human genetic variation is, therefore, both false and strategically shortsighted, because it opens the door for a straightforward empirical defense of race.

Second, the view of race as a cultural construct needs to become a starting point for empirical research, rather than an end point in the dismissal of race. To say that race is a cultural construct is not to say it does not exist; cultural constructs have an objective reality despite their reliance on human thought (Searle, 2006). Two avenues for research on racial inequalities in health follow from this observation. The first—an anthropology *of* medicine (Foster, 1974)—examines the cultural construction of race in biomedical research and clinical practice. There is already important work in this area, which shows how hidden assumptions about race shape the formulation of research questions and interpretation of data (e.g., Fullwiley, 2007; Lee, 2007; Montoya, 2007; Hunt and Megyesi, 2008). It would be valuable to have more ethnography of race and racism in clinical settings, especially given evidence for systematic racial bias in the delivery of health care (Braveman and Tarimo, 2002; Smedley et al., 2002; Bhopal, 2007).

Another avenue for research—an anthropology *in* medicine—is to contribute to explaining the origin and persistence of racial inequalities in health. Chapman and Berggren (2005) argue that anthropologists have an important role to play through the "radical contextualization" of racial inequalities in health. In particular, a major thrust of current research in cultural anthropology is to understand how global political–economic structures shape the local context of people's lives and become embodied in individual sickness and suffering (Nguyen and Peschard, 2003; Farmer, 2004). Integrating this approach with the model in Figure 3 has potential to elucidate the pathways of embodiment through which race becomes biology.

In addition, cultural anthropologists can contribute to interdisciplinary research by developing measurement strategies that take seriously the view of race as a cultural construct. My work on the relationship between skin color and blood pressure illustrates this point (Gravlee and Dressler, 2005; Gravlee et al., 2005). Previous researchers had showed that, within the African Diaspora, people with darker skin had higher average blood pressures than did their lighter skinned counterparts. Some researchers interpreted this pattern as evidence of a racial–genetic predisposition for high blood pressure; others suggested that it may reflect sociocultural factors. Yet previous studies had not tested these alternatives directly, because they conflated two dimensions of skin color: the *phenotype of skin pigmentation* and the *cultural significance of skin color* as a criterion of social classification.

The distinction between cultural and biological dimensions of skin color requires a measurement strategy that incorporates the cultural meaning of skin color. In Puerto Rico, I adopted a two-phase approach (cf. Dressler et al., 2005b). I first conducted a systematic ethnographic study of the cultural model of *color* (Gravlee, 2005). The ethnography shed light on local ways of talking about skin color and on how *color* shapes Puerto Ricans' exposure to racism and other social stressors. Systematic ethnographic methods (Romney et al., 1986) made it possible to test the assumption that people shared a coherent cultural model of *color*. Colleagues and I then developed a survey measure that explicitly linked respondents to ethnographic data on the cultural model of *color* to estimate how they would be perceived

by other Puerto Ricans in everyday social interaction. In a small epidemiologic survey, we compared blood pressure to *color*, as defined by the local cultural model, and to skin pigmentation, as measured by reflectometry. The key finding was that both self-rated and culturally ascribed *color*—but not skin pigmentation—were associated with blood pressure through an interaction with income and education (Gravlee and Dressler, 2005; Gravlee et al., 2005). This finding suggests that empirical research on *how* race is culturally constructed better positions us to identify the biological consequences of cultural constructs like *race* in the United States or *color* in Puerto Rico.

CONCLUSION

Race has played a pivotal yet tortured role in the history of anthropology. In the nineteenth and early twentieth century, anthropologists were central in legitimating race as a framework for understanding human biological variation. By the mid-twentieth century, most anthropologists rejected race as biology, and the view of race as a cultural construct came to dominate the social sciences. However, the anthropological critique of race has had only partial success. In particular, current debate over racial inequalities in health exposes important weaknesses in the usual framing of the critique and points the way toward a more constructive approach to the links between race, biology, and culture.

The specific challenge is to explain *how race becomes biology*. Our response to this challenge must deal with two senses in which race becomes biology: Systemic racism becomes embodied in the biology of racialized groups and individuals, and embodied inequalities reinforce a racialized understanding of human biology. To break this cycle, I propose that the conventional critique of race needs to be refined in three ways: 1) to clarify why recent genetic findings do not warrant a return to racial thinking, 2) to promote a more complex, biocultural view of human biology, and 3) to revise the conceptualization of race so that it becomes more than a mantra.

These three claims inform a conceptual model for research on the multilevel and developmental influences on racial inequalities in health. This model crosses old fault lines and lays the groundwork for more productive collaboration between the social and biological sciences. The model does not promote a focus on social and cultural factors to the exclusion of genetic ones; rather, it implies that the embodiment of social inequality passes through biological systems regulated by genes. It does not deny human biological variation; rather, it claims that the pattern and causes of human biological variation are more complex than the race concept allows. It does not claim that race is a myth; rather, it treats race as deeply embedded in sociocultural systems. Research on the biological consequences of race and racism can help to reinvigorate the critique of race by offering a constructive framework for explaining biological differences between racially defined groups.

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