CONCEPTUALIZING RACIAL DISPARITIES IN HEALTH

Advancement of a Socio-Psychobiological Approach

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Abstract

Although racial disparities in health have been documented both historically and in more contemporary contexts, the frameworks used to explain these patterns have varied, ranging from earlier theories regarding innate racial differences in biological vulnerability, to more recent theories focusing on the impact of social inequalities. However, despite increasing evidence for the lack of a genetic definition of race, biological explanations for the association between race and health continue in public health and medical discourse. Indeed, there is considerable debate between those adopting a "social determinants" perspective of race and health and those focusing on more individual-level psychological, behavioral, and biologic risk factors. While there are a number of scientifically plausible and evolving reasons for the association between race and health, ranging from broader social forces to factors at the cellular level, in this essay we argue for the need for more transdisciplinary approaches that specify determinants at multiple ecological levels of analysis. We posit that contrasting ways of examining race and health are not necessarily incompatible, and that more productive discussions should explicitly differentiate between determinants of individual health from those of population health; and between inquiries addressing racial *patterns* in health from those seeking to explain racial *disparities* in health. Specifically, we advance a socio-psychobiological framework, which is both historically grounded and evidence-based. This model asserts that psychological and biological factors, while playing a central role in determining individual risk for poor health, are relatively less consequential for understanding racial disparities in health at the population level. Such a framework emphasizes the etiologic role of social inequities in generating and perpetuating racial disparities in health and highlights their impact on psychological, behavioral, and biological disease processes.

Keywords: Race, Racism, Racial Health Disparities, Psychobiology, Stress

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. . . models of disease emergence need to be dynamic, systemic, and critical. They need to be critical of facile claims of causality, particularly those that scant the pathogenic roles of social inequalities. Critical perspectives on emerging infections must ask how large-scale social forces come to have their effects on unequally positioned individuals. . .

—Paul Farmer, *Infections and Inequalities: The Modern Plagues* (1999, p. 5)

INTRODUCTION

Diet. *Genetics*. These responses are often solicited when asking about factors that contribute to a number of the most pressing public health problems facing the United States today. "Diet" and "genetics" reflect popular sentiment regarding the importance of both behavioral and biological influences on health. They are also commonly invoked in response to questions about the causes of racial disparities in health (Bonham 2010; Taylor-Clark et al., 2007). However, although behavior and biology indeed matter as individual-level risk factors for disease, they hold considerably less weight in understanding the population distribution of disease and racial disparities, and in designing interventions to improve the *public* health (Krieger 1996; Syme 1987, 1996). Along these lines, interest in the "social determinants of health" has gained foothold in public health discourse around race and health (Satcher 2010).

Individual-level explanations including psychological and biological analyses of race and health tend to be seen as being in conflict with those focusing on the impact of broader social factors. For example, behavioral explanations have been criticized for placing the onus of disease burden on the individual; similarly, genetic reasons imply that the poorer health of some groups is inherent. Accordingly, debates around race and health are often mired in controversy about the relative contribution of biological versus social factors (Krieger 2005), conflating terms such as "determinants of health" and "determinants of population health" as well as "patterns in disease distribution" and "disparities in health" (Braveman 2006; Kaplan 2004; Krieger 2008a). These discussions also reflect the "proximal-distal divide" that permeates conversations around the determinants of health. However, as Krieger (2008b) posits, arguments around proximal versus distal, or downstream versus upstream factors, also carry with them a set of presumptions about primacy, temporality or direction, and causality and causal strength. In addition to explicitly stating ecological levels of analysis and their interrelationships, more productive frameworks for examining race and health should also specify the level of outcome (e.g., individual, community, or population).

Furthermore, clearer distinctions should be made between the terms population *patterns* in health and *disparities* in health. Consistent with the World Health Organization and other definitions (Braveman 2006), health disparities are conceptualized as unequal patterns in the distribution of disease between groups that are "systemic" (Graham 2004), "avoidable," "unjust and unfair" (Whitehead 1992), and therefore subject to remediation and intervention. Along these lines, although racial disparities are indicated by racial differences in health, these patterns in disease distribution do not necessarily connote racial disparities. For example, stark racial differences exist in the incidence of malignant melanoma, with Whites having more than tenfold incidence compared to Blacks (Cress and Holly, 1997). However, it is debatable whether this racial pattern can be considered a racial *disparity*. Arguably, social forces play less of a role in the etiology of some racial patterns in disease. Equally important to note, however, is that social inequities do not necessarily manifest in health

disparities for all outcomes and groups, and in fact, some relatively disadvantaged groups perform similarly if not better on various indicators of health. Examples include: psychiatric morbidity among Blacks (Williams et al., 2007); low birth weight among Latinos (Osypuk et al., 2010); and smoking among Asians (Chae et al., 2006). These observations do not mean that social inequities are unimportant for these groups and/or outcomes. As authors of these studies have pointed out, a possible alternative explanation is that aggregated statistics may mask the disparity, suggesting the need for a more thorough examination of potential confounders as well as within-race group analyses.

Embracing this line of thinking, a socio-psychobiological approach represents one way of framing and understanding associations between race and health, especially for *racial disparities* at the *population level*. This model specifically highlights the importance of social processes in generating psychological, behavioral, and biological vulnerabilities involved in processes of embodiment (Krieger and Davey Smith, 2004). In contrast to proverbial "biopsychosocial" models of health (e.g., Clark et al., 1999), a socio-psychobiological model explicitly posits directionality, in which social inequalities generate unjust patterns in disease distribution. Furthermore, present-day social forces driving racial disparities in health are conceptualized as being the result of historical legacies of social oppression (Krieger 2001). Accordingly, this view emphasizes the need for historical contextualization, and specifies causal direction between ecological levels when investigating racial disparities in health.

More specifically, a socio-psychobiological framework for examining racial disparities in health emphasizes how more traditionally examined individual-level determinants of disease, including psychological, behavioral, as well as biological risk factors, are informed by racism (Fig. 1). Racism is viewed as a broader sociocultural ideology that produces distinct patterns in disease emergence along racial lines via a

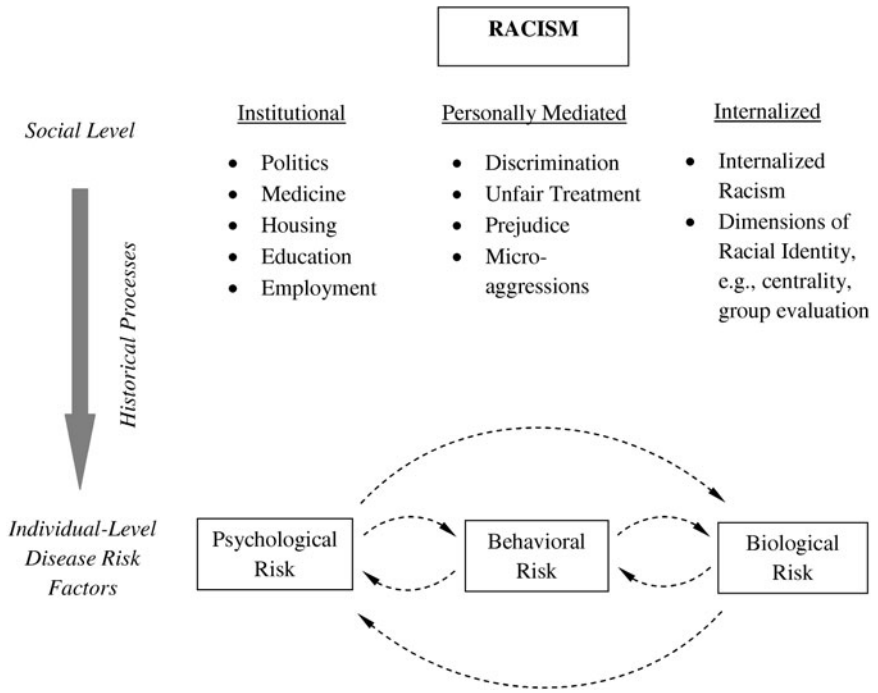

Fig. 1. Socio-psychobiological framework for examining racial disparities in health

number of mechanisms, including historical processes, institutional forces, and personally mediated as well as internalized forms of racism. Along these lines, a sociopsychobiological framework posits that unjust patterns in risk and resilience to disease are conferred on racial groups. This approach focuses not on the *problem of race*, which implies that factors inherent to race are responsible for racial patterns in disease distribution, but rather the *problem of racism*, which emphasizes how social inequalities are the primary drivers of racial disparities in health.

DEFINING RACE AND THE PROBLEM OF RACE

Explanations regarding associations between race and health are often rooted in prevailing beliefs about what constitutes "race" during a particular historical period (Krieger 2008c). Accordingly, historically evolving notions of race (e.g., as a biological or social construct) have informed understandings of race and health.

Early usage of the word "race" was heavily based on biology, to describe animal stocks or aristocratic breeds (Augstein 1996; Byrd and Clayton, 2000). Accordingly, the "slave health deficit" was largely attributed to beliefs about the innate biological inferiority of non-Whites. Biological conceptions of race endured through the establishment of several pseudosciences, such as phrenology and craniometry. Evidence of inherent and hereditary racial inferiority was supported through additional "scientific" hallmarks, such as IQ tests and family tree analyses, which were used to justify medical experimentation and sterilization (Smedley et al., 2003).

It is important to note, however, that although the biological credibility of race was buttressed under the aegis of science and medicine, subjectivity, politics, and the legal system primarily drove how race was defined. Phenotype trumped any so-called biological definition of race, even though biology was used to support racial categorizations. For example, in *United States v. Bhagat Singh Thind* (1923), the court ruled that people of Indian ancestry were non-White, and that "purity of Aryan blood" was insufficient to gain the privileges associated with White status, including citizenship. More recently, the formation of a separate "Chicano" identity was largely motivated by Mexican American activists as a reaction to systematic institutional violence (Haney-López 2003). Accordingly, legal and political processes helped to generate newer definitions of race.

Conceptions of race have gradually shifted from early biological notions to one focused on describing race as a social construct with little or no biologic basis. Accordingly, race, as viewed in the social sciences and increasingly in biological sciences, is seen as socially derived. Indeed, race as a biological concept has fallen out of favor among many anthropologists and biologists, as revealed by DNA analyses indicating greater genetic heterogeneity within rather than across racial groups (Mountain and Risch, 2004). These findings do not deny genetic diversity among humans, but do reveal that there is little evidence for a genetic or biological definition of race. From an evolutionary perspective, human genetic heterogeneity is rather the result of migratory processes and geographic adaptation (Feldman et al., 2003).

Although biological differences exist based on "continental ancestry," which might broadly correspond to racial phenotype, debates are still ongoing on whether race is a reliable proxy for genetic difference. For some diseases, biological factors associated with race do appear to play some role in driving racial patterns, as in the aforementioned case of melanoma. Another example where "biological race" appears to have some association with disease is sickle cell anemia, which, though classified as

a rare disease having an annual incidence of approximately one in 34,000 births, affects one in 500 Black births in the United States (National Heart, Lung, and Blood Institute 2009). Though also found in non-Black populations, the higher prevalence of sickle cell anemia among Blacks was used as a marker of "Negroid" race (Wailoo 1996). More current examples in the medical literature include racial differences found in the metabolism and tolerance of drugs (e.g., Agarwal et al., 1981; Wood 2001). However, these racial patterns are likely artifacts of factors associated with geographical environment, as in the case of sickle cell anemia which developed as a form of resistance against malaria (Tapper 1997).

Although recent science has debunked earlier notions of a biological basis for race, in favor of one which instead emphasizes genetic factors correlated with geographic ancestry, the study of biological factors involved in explaining associations between race and health persists. However, there are nontrivial distinctions to be made between using biological correlations to define race, versus the use of geographical ancestry as a risk factor; this also brings into question the use of biological notions of race to influence clinical decision-making and as a risk factor for particular diseases (Schwartz 2001). Nevertheless, those diseases which appear to have biological origins account for only a small part of the health issues that contribute to racial differences in health. More recent inquiries have instead examined how race as a social construct impacts health.

Observations that social group membership is associated with patterns in disease and health at the population-level are not new. But while racial patterns in disease have been historically documented, systematic inquiries addressing how social forces drive these patterns are relatively recent, marking a shift in focus to examining how race as a socially defined phenomenon influences health. Along these lines, recent approaches to studying race and diseases have moved from an examination of how race impacts health to how social/cultural processes and the hazards associated with racism contribute to racial disparities in health.

CONTEMPORARY APPROACHES AND THE PROBLEM OF RACISM

Frameworks for Racial Disparities in Health

The idea that health and disease have multiple determinants is often considered to be central to contemporary epidemiological sciences. Conceptualizing the relationships between these various factors, their relationships to one another and to disease outcomes, however, has been problematic. "Web of causation" models of disease take into account aspects of host, agent, and environment at different ecological levels. However, these frameworks run the risk of overemphasizing close or immediate predecessors of disease, such as those related to biology or individual behavior (Krieger 1994). Along these lines, web of causation models do not explicitly differentiate between individual-level causes of disease versus population determinants of disease distribution, and may inadvertently conflate the two as being synonymous. Although individual risk profiles are indeed closely associated with health outcomes, frameworks that marginalize the role of broader social factors reduce racial disparities in health to differences in individual biology or behavior.

In contrast, population-level ways of framing disease emphasize social determinants of health. "Social production of disease" theories (Krieger 1994, 2001), including the one outlined by Doyal (1979) in *The Political Economy of Health*, were primarily "materialist" in scope, positing that economic class shapes disparities in health. A social production of disease perspective argues that features of the social structure

position groups relative to one another, which are reflected in the population distribution of health. Political and economic forces determine the health of groups through the systematic differential consumption of health resources and exposure to health hazards. Importantly, this perspective also posits that contextual factors influence health beyond that which could be explained by individual-level variables.

While social production of disease frameworks have somewhat expanded beyond their materialist origins, the tradition of studying socioeconomic factors in explaining racial disparities in health continues to be a rich area of research focus (Farmer and Ferraro, 2005; LaVeist 2005). Material-reductionist approaches, however, imply that socioeconomic position (SEP) is an intermediate variable in the pathway connecting race and health, suggesting that either SEP should not be modeled because doing so would obscure differences in health between races, or that the effect of race should not be estimated because observed differences would merely be the result of residual confounding by imprecise measurements of material circumstances (Kaufman et al., 1997). The associated counterfactual in such a perspective—that the health of a Black person compared to the health of a White person would be equivalent given identical SEP—however, is debatable (Krieger and Davey Smith, 2000). Arguably, such material-reductionist approaches to studying race and health ignore the complexity of race and racism—its persistence as a socially meaningful predictor of health even after controlling for SEP—and the historical, sociocultural, and contextual forces that have direct effects on disease risk and which would continue to inform material vulnerability (Krieger 2000; LaVeist 2005).

Moving beyond SEP, a socio-psychobiological approach explicitly posits that racial disparities in health are reflections of underlying social inequalities, expressed in inequitable relationships of dominance and oppression, and privilege and deprivation—not only with regard to material resources, but also in terms of other forms of social power. In contrast to physical and material power, ideological hegemony is the dominance of ideas, beliefs, and culture, and contributes to the reification of oppressive social structures (Althusser 1971; Freire 1970). Here, racism is conceptualized as an ideology through which unjust relationships between racial groups are sustained and perpetuated (Krieger 2008c). A socio-psychobiological approach is not meant to represent all mechanisms involved in linking race, racism, and health, and is not designed to be a comprehensive model of disease emergence. Rather, this framework highlights one particular pathway for studying racial disparities in health, and helps to declutter web of causation models. Specifically, this approach emphasizes how racism is directly embodied and also shapes psychological and biological vulnerability to disease. This framework is theoretically informed, and based on empirical findings on racism and health.

Racism and Health

While definitions of racism have varied and been contested, its contemporary usage has described unjust social relationships between racial groups, often having connotations of power, and superiority and inferiority (Fredrickson 2002). Here, racism is defined as a sociocultural ideology that is premised on the belief of the inherent inferiority of non-Whites.

As an ideology, racism operates and manifests itself on multiple ecological levels. Jones (2000) outlines three specific levels of racism: institutionalized, personally mediated, and internalized. Institutionalized racism is considered "structural," and does not have an individual perpetrator per se. It is historical in origin, and reflected in current institutions, including politics, medicine, housing, education, and employ-

ment. For example, contemporary patterns in housing and education are the result of historical legacies of forced segregation and legalized discrimination. Medicine continues to reflect the vestiges of unequal treatment and care of racial and ethnic minorities, with regard to access to health care and differential quality of care. Personally mediated racism, on the other hand, refers to attitudes and beliefs about the inferiority of racial groups (prejudice) and differential treatment of people on the basis of race (discrimination) which is directly experienced at the individual level. Internalized racism, as opposed to personally mediated racism which has an identifiable perpetrator, refers to the acceptance of negative sociocultural beliefs about the intrinsic worth of one's own racial group.

Recent research suggests that these three levels of racism have detrimental consequences for racial minority groups across a number of health and disease outcomes, and has identified pathways through which racism may operate. Below, we discuss three specific areas of inquiry that represent contemporary areas of research on race and health at these levels: area-level or neighborhood effects, experiences of racial discrimination, and racial identity.

Area-Level Effects

Research on neighborhoods suggests that residential segregation, particularly with regard to poverty and racial concentration among Blacks, has a negative impact on health (Williams and Collins, 2001). Specific area-level factors are related to physical neighborhood characteristics, such as the quality of housing, availability of recreational facilities, and the presence of environmental toxins; characteristics of the service environment, including the presence of supermarkets, liquor stores, fast food stores, clinics, and hospitals; and the social environment, such as the presence of crime. These neighborhood contextual factors have been hypothesized to impact health directly, as well as via their associations with access to health care services, availability of employment opportunities, quality of education, and access to food sources, which may in turn impact dietary habits, rates of physical activity, and substance use (Ellen et al., 2001; Williams and Collins, 2001).

Increasing evidence suggests that poverty concentration and racial residential segregation have a deleterious effect on health that is above and beyond compositional effects, i.e. that poor health associated with living in distressed neighborhoods is more than an artifact of the individual-level characteristics of their inhabitants (Acevedo-Garcia et al., 2003). Even after controlling for demographic factors, those living in poorer neighborhoods fare worse than those living in more affluent areas (Katz et al., 2001). Furthermore, studies have found that while poor Whites are more spatially dispersed, low-income Blacks are more likely to be concentrated and reside in poorer quality neighborhoods, being characterized along multiple dimensions of segregation (Acevedo-Garcia et al., 2003). Accordingly, hypersegregation among Blacks is considered to contribute to racial disparities in health and socioeconomic status by generating distinct pathological residential conditions (Wilson and Hammer, 2001).

Research findings on the effects of area-level factors and neighborhoods, however, are somewhat equivocal. For example, research on other racial minority groups in the United States, including Asians and Latinos, are in their infancy, and findings in these groups have been mixed (Osypuk et al., 2009, 2010; Walton 2010). In fact, some of the literature suggests that ethnic enclaves in these groups may have protective properties by serving as a buffer for acculturative stressors, providing a source of social support, or mitigating race-related stressors (Walton 2010). Additional

research examining the effects of area-level characteristics in these racial groups is especially warranted.

Racial Discrimination

Interpersonal experiences of discrimination, including racial profiling by police, in domains such as employment, housing, education, and health care, remain salient in the lives of racial minorities (Gee et al., 2009; Williams et al., 2003). These experiences may impact health directly, as in the case of physical victimization (i.e., hate crimes), which continues despite the existence of protective legislation. Racial discrimination in the form of microaggressions are also experienced more chronically in everyday interactions, including routine experiences of being treated with less respect or courtesy (Chae and Walters, 2009; Lincoln and Chae, 2010; Sue et al., 2007; Williams et al., 2003). But in addition to overt forms of racially motivated discrimination, discrimination can also be experienced more subtly. "Laissez-faire" discrimination (Bobo et al., 1997) is based on assumptions about the work ethic, habits, and dispositions of racial groups, and is distinguished by the lack of an intentional motive to subvert a particular racial group. This more insidious form of discrimination rationalizes racial inequalities as being motivated not structurally but rather on the basis of the qualities ascribed to racial group members. Research suggests that this "free market" discrimination also directly impacts health and disease outcomes. For example, race plays a role in clinical decision-making based on preconceptions about medication adherence, tolerance, and effectiveness, justified using racially based notions of behavior and biology (Kressin and Petersen, 2001; Smedley et al., 2003).

In addition to the indirect health effects of racial discrimination (e.g., through its impact on housing, employment, education, and other socioeconomic indicators), as a source of psychosocial stress, racial discrimination may have direct effects on mental health and maladaptive health behaviors. Unfair treatment may lead to negative psychological reactions, including depression and anxiety (Chae and Yoshikawa, 2008; Noh and Kaspar, 2003; Williams and Williams-Morris, 2000). Indeed, findings on the negative mental health implications of racial discrimination have been the most consistent (Williams et al., 2003). Discrimination may also increase the risk of engaging in maladaptive health behaviors, including illicit substance use, smoking, and alcohol consumption (Bennett et al., 2005; Chae et al., 2008a, 2008b). Prior studies have found that drinking is often used to relieve and manage psychosocial strains particularly in response to those that are more severe and chronic, and when stressors are perceived as being unavoidable, uncontrollable, or occur in the absence of social support (Pohorecky 1991). Along these lines, racial discrimination, as a source of stress, may contribute to maladaptive substance use patterns.

Research findings also suggest that experiences of racial discrimination may directly impact the regulation of biological systems engaged in the stress response (Harrell et al., 2003). Studies in this area challenge the traditional stress-diathesis model, which posits that an existing vulnerability or predisposition, commonly believed to be of genetic or biological origin, requires challenging or stressful life experiences to precipitate pathology or disease state (Lydeard and Jones, 1989). This approach assumes that such predispositions or vulnerabilities are inherited and therefore "fixed," and that environmental stressors act upon these biologic/genetic vulnerabilities. However, recent findings indicate that racial stressors themselves may generate increased biological vulnerability to disease. For example, reported experiences of

racial discrimination have been associated with heightened blood pressure, increased cardiovascular reactivity, and a range of biological markers of stress, including neuroendocrine risk markers for poor birth outcomes, glucocorticoids, and proinflammatory cytokines (Harrell et al., 2003; Hilmert et al., 2008; Lewis et al., 2010). A longitudinal study also found that reports of both chronic and routine experiences of discrimination as well as major experiences of discrimination in specific domains (in employment, housing, and by the police) were associated with increased breast cancer risk among Black women (Taylor et al., 2007). Supporting these studies, Blacks have been found to have up to threefold higher odds of allostatic load, the physiologic damage or "wear and tear" associated with the experience of repetitive stress, compared to Whites (Geronimus et al., 2006).

The advent of more recent biotechnologies has facilitated the examination of additional novel markers of the health of biological systems that may be affected by discrimination. One emerging line of inquiry has focused on leukocyte telomere length (LTL) as a marker of immune system aging and cumulative life stress, which has been posited to be a measure of physiologic burden experienced across the life course (Epel et al., 2004; Fitzpatrick et al., 2007). Telomeres undergo accelerated shortening with heightened replication which occurs in response to physiologic challenges. In this respect, LTL may be particularly relevant for studies on discrimination and the weathering of biological systems. Studies reporting racial differences in LTL provide evidence for the hypothesis that racial stressors impact racial disparities in health. Recent findings suggest that Blacks have either shorter telomeres or a higher rate of telomere shortening compared to Whites (Diez Roux et al., 2009; Geronimus et al., 2010). Current research efforts are aimed at investigating whether racial discrimination specifically may contribute to accelerated cellular aging among Blacks.

In addition, recent developments in epigenetics suggest that racial discrimination may have a negative impact on health by altering gene expression. The epigenome—consisting of DNA marks and modifications that control gene expression—is innately plastic and can be programmed or reprogrammed by environmental experiences (Dolinoy and Jirtle, 2008; Waterland and Jirtle, 2004). These epigenetic mechanisms provide the means through which social experiences can fundamentally and profoundly alter the regulation and expression of the genome without altering genotype, and which can increase disease risk development. For example, nutritional deficiencies experienced in utero and in childhood may increase cancer and cardiovascular disease vulnerability via epigenetic mechanisms (Duthie 2010). Evidence from studies examining childhood stress and the expression of genes critical to the stress axis also allow us to readily observe how individual differences in our lived social experiences are transduced into biological signals that ultimately confer risk or resiliency (McCrory et al., 2010; McGowan et al., 2009).

Epigenetic processes demonstrate that presumed genetic differences in various disease states may be due to epigenomic differences that arise in response to stressful experiences (Fraga et al., 2005; Francis et al., 2003). Along these lines, racial discrimination may be capable of directly altering the epigenomic profiles of genes important to disease onset that may persist across the life course. These observations highlight the need to study interactions between genes and environments rather than simply treating them as independent causal agents. "Cell memory" or the finding that epigenetic changes are heritable (Jablonka et al., 1992) also points to how racial disparities in health cannot be confined as biological phenomena, but should be characterized as being historically informed and primarily in the social realm.

Racial Identity

Racial identity is conceptualized as a psychological construct, but is informed by broader sociocultural values. Social Identity Theory suggests that racial stressors may have adverse effects on mental health via the internalization of negative social beliefs attached to membership in a marginalized group (Tajfel and Turner, 1986). Furthermore, poorer self- and group-evaluation may lead to negative affective and cognitive reactions.

Although several models of racial identity development and formation have been developed in the psychological literature, only recently have they been explicitly examined in relation to health outcomes. Racial identity has been viewed as being multidimensional, including people's personal evaluations about their racial group, racial group attachment or affiliation, and importance of race to self-definition (Sellers et al., 1998). In her model of racial identity, Helms (1995) distinguished those with less complex racial identity schemas—characterized by a minimization of race and racism, and ambivalence towards identifying as a racial minority—from those with more complex racial identity schemas, who display a greater awareness around issues of social oppression and a strong valuation or idealization of their racial group. Studies have indicated that issues of internalizing negative beliefs and attitudes represent a salient concern in racial minority populations. For example, one study found that 35% of African American men endorsed negative attitudes towards Blacks as being true (Chae et al., 2010). Findings from the Implicit Association Test, a measure of unconscious racial bias, also suggest high levels of internalized anti-Black bias (Nosek et al., 2007).

Importantly, dimensions of racial identity may have a direct impact on health outcomes. Studies have found that racial minorities who positively evaluate their racial group have better mental health compared to those holding negative beliefs (Chae and Yoshikawa, 2008; Sellers and Shelton, 2003). There is also evidence suggesting that having a negative group identity may be associated with substance use behaviors (Katz et al., 2002; Martin et al., 2003). While studies on racial identity and physical health outcomes are in their infancy, recent findings indicate that Blacks who endorse negative attitudes about their racial group may be at greater risk of cardiovascular disease (Chae et al., 2010). Other studies have reported that high levels of internalized racism may increase risk of glucose intolerance and abdominal obesity, as well as higher levels of perceived stress, poor coping, and cortisol dysregulation (Chambers et al., 2004; Tull et al., 1999; Tull et al., 2005). Pathways implicated in connecting racial identity and physical health may involve poor mental health as a potential mediator, which has been associated with greater susceptibility to and progression of disease.

Prior studies have also indicated that racial identity may be protective against race-related stressors (Romero and Roberts, 2003). For example, greater levels of racial group identification were associated with lower risk of both smoking and alcohol disorders among Asian Americans, and also buffered against the negative effects of racial discrimination on these outcomes (Chae et al., 2008a, 2008b). However, other studies have reported that greater centrality of race may be associated with poorer health outcomes or may exacerbate the negative effects of racial discrimination (Chae and Yoshikawa, 2008; Zea et al., 1999). One possible explanation for these findings is that among those who identify more closely with their racial group, negative social evaluations may be more salient and exert detrimental impact on health. In light of these contradictory results, future research may examine additional pathways through which dimensions of racial identity are associated with health.

CONCLUSION

Race matters for health, as repeatedly shown in surveillance studies which point to persistent patterns in the distribution of health along racial lines. However, there remains considerable debate on the reasons *why* race matters for health, for which health outcomes and patterns, and at what levels. In this essay, we posit that it is not race, per se, but rather the problem of racism that matters for racial disparities in health (LaVeist 2000). Herein, the impact of race is in racism—historically informed, perpetuated by institutions, and manifested in the set of assumptions, stereotypes, and biases that are attached to race, both externally and internally—positioning groups of people into relative positions of power and deprivation.

Too common are individual-level behavioral or biological reasons provided for explaining racial disparities in health. Instead, a socio-psychobiological approach emphasizes how social inequalities generated by racism impact health, directly as well as by shaping psychological, behavioral, and biological vulnerability to disease. This population-level approach emphasizes the role of racism in generating inequitable racial patterns in disease emergence by flipping the popular biopsychosocial model over on its head. A socio-psychobiological approach does not discount the importance of more individual-level pathways. Rather, this framework encourages transdisciplinary methods for studying racial disparities in health, emphasizing interactions between determinants of health at multiple ecological levels and the need to understand how psychological, behavioral, and biological risk factors are shaped by racism.

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