
Critical Issues in Health and Medicine

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Growing criticism of the U.S. health care system is coming from consumers, politicians, the media, activists, and healthcare professionals. *Critical Issues in Health and Medicine* is a collection of books that explores these contemporary dilemmas from a variety of perspectives, among them political, legal, historical, sociological, and comparative, and with attention to crucial dimensions such as race, gender, ethnicity, sexuality, and culture.

For a list of titles in the series, see the last page of the book.

Mapping “Race”

Critical Approaches to Health Disparities Research

Edited by

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Rutgers University Press

New Brunswick, New Jersey, and London

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Jedi Public Health

Leveraging Contingencies of Social Identity to Grasp and Eliminate Racial Health Inequality

Eliminating racial health inequality remains seemingly impervious to efforts and intentions. Of significance in addressing this dilemma is the concept of race and how this concept is and can be linked to health. Historically, public health has conceptualized race either as static, essentialist characteristics (genetic, behavioral, cultural, or social attributes and predispositions) or as entrenched conditions (poverty and social disadvantages related to the legacies of slavery and systematized racial segregation). However, increasingly, public health researchers are approaching race dynamically. Some are looking at how ongoing and new social processes maintain race as a lived experience with health impacts, and how dominant structural and cultural processes—and the social, physical, and policy environments they create—work through a complex interplay of psychosocial, physiological, and molecular mechanisms to produce population variation in morbidity and mortality (Geronimus and Thompson 2004; Geronimus et al. 2010; Schulz et al. 2005).

In addition, race is being considered beyond the Black-White dichotomy to encompass a set of social relationships that emanate from pervasive ideologies that advantage dominant groups at the expense of others and that occur at all socioeconomic levels and in the minor ethnic, religious, or nativity divisions within racial groups (Geronimus 2000; Geronimus and Thompson 2004; James 1993; Pearson and Geronimus 2011; Viruell-Fuentes 2007). In the United States, recent decades have witnessed growing income inequality, large waves of immigration, newly emergent or intensified xenophobia, and tensions around whether our vision for a postracial society should be race-blind or multicultural. In this context, by acknowledging that marginalization of any

identified social group may have population health repercussions, by broadening the theories of how such marginalization is enacted to impact health in a growing set of groups, and by viewing marginalization and its consequences as dynamic and relational, the field can move beyond the impasse occasioned by static and binary conceptions of how race and health are entwined (see also Garcia, this volume; Geronimus 2000; Geronimus and Thompson 2004; James 1993, 1994; Pearson 2008; Pearson and Geronimus 2011; Viruell-Fuentes 2007).

Despite these promising theoretical developments, public health research and practice continue to operate from the traditional assumption that a person's race is fixed. This assumption is most widely recognized in the form that genetic predispositions are the starting point for understanding racial disparities. This viewpoint has been critiqued by social epidemiologists and population geneticists (for example, Cooper et al. 2003; Graves, this volume; Lewontin 1972). Now, most agree that the notion that everyone with the same phenotypic characteristics used to assign race—most notably skin color—would have the same health outcomes invariant to social and physical environments, access to resources, or the nature and timing of critical exposures is untenable. Now, those interested in the role genes play in population health are increasingly emphasizing the environment side of gene-environment interactions. Others are moving into areas such as epigenetics or human stress genomics, wherein the regulation of gene expression is viewed as dynamic at the molecular level (Kuzawa and Sweet 2010),¹ or are focusing on telomere length in a subset of leukocytes called peripheral blood mononuclear cells (PBMC), a measure of biological aging that appears to be sensitive to stressful life conditions (Epel et al. 2004; Geronimus et al. 2010).²

The Role of Stress Physiology

While embracing race as a social construction and assuming population differences in health along racial lines reflect social patterning, objective and subjective experiences that are socially patterned on the population level ultimately work via physiological processes and mechanisms to influence morbidity and mortality. Increasingly, public health researchers posit that prolonged psychosocial or physical challenges to metabolic homeostasis in marginalized groups can increase disease susceptibility, promote the early onset of chronic conditions (Geronimus and Thompson 2004; Geronimus et al. 2007; James 1994; Steptoe et al. 2006), and accelerate aging via a process of “weathering”—the cumulative biological impact of chronic exposure to and coping with subjective and objective stressors (Geronimus 1992, 2001; Geronimus et al. 2006; Geronimus et al. 2010; McEwen 1998; Sapolsky et al. 2000). Everyday challenges

shaped by social disadvantage may trigger chronic activation of stress processes to the health detriment of disadvantaged racial, ethnic, socioeconomic, gender, residential, or geographical ancestry groups.

On a biological level, persistent high-effort coping with acute and chronic stressors may have profound health effects. Stressors may be objective (for example, temperature extremes) or subjective (for example, financial anxiety) and, notably, need not be perceived as stressful to exert a physiological impact. Stress-activated biological (allostatic) systems enable people to respond to changing physical states and to cope with ambient stressors such as noise and crowding, imminent danger, hunger, extreme temperature shifts, or infection. As McEwen (1998) notes, the body's response to a stress-inducing challenge is twofold: turning on an allostatic response that introduces a complex cascade of stress hormones into the body and then shutting off this response when the threat has receded. When allostatic systems are not completely deactivated, the body experiences overexposure to stress hormones. Long periods of overexposure result in “allostatic load,” which can cause wear and tear on the cardiovascular, metabolic, and immune systems. This wear and tear increases susceptibility to infectious disease, early onset of chronic diseases such as hypertension, diabetes, morbid obesity, and metabolic syndrome, which, in turn, can lead to functional limitations or early death.

The structural positioning of a racial or ethnic group influences its exposure and vulnerability to stressors. Stressors uniquely faced by Black Americans may accumulate and interact with one another to increase allostatic load (Geronimus and Thompson 2004). U.S. studies have found that, compared to Whites, Blacks have higher levels of cortisol and sympathetic nerve activation, as well as higher levels of oxidative stress, suggesting more frequent or intense episodes of physiological stress activation. Researchers also found higher allostatic load scores, a summary biological measure of stress-mediated wear and tear on the body, among U.S. Black compared to White adults—with the disparity widening from youth through middle adulthood (Geronimus et al. 2006). Among forty-five- to sixty-year-olds, Mexican immigrants have been found to be less likely than U.S.-born Mexican Americans or non-Hispanic Whites to have a high allostatic load if they recently arrived in the United States, but more likely than either group to have a high allostatic load if they had resided in the United States for twenty years or more (Kaestner et al. 2009).³ Researchers studying telomere length estimated that, at ages forty-nine to fifty-five, Black women study participants were 7.5 biological years older than their White counterparts (Geronimus et al. 2010). Indicators of perceived stress and poverty accounted for an important share of the estimated Black-White difference in

telomere length, but data limitations precluded evaluation of objective stressors. These lines of evidence suggest that racial or ethnic health disparities may result, in part, from repeated activation of biological stress processes in members of marginalized groups who cope over long periods with stressors inherent to their structural positioning.

In addition to continued study of the physiology of stress—its impacts on molecular dynamics, biological systems, and ultimate links to poor health—investigation of the nature and potential mitigation of structurally inherent stressors that activate the stress process is needed. For Blacks, researchers have focused on interpersonal racism, crime victimization, discrimination in housing and employment, material hardship, concern for physical safety, overburdened or disrupted social support networks, toxic or decaying environments, ambient stressors in residential or work environments (such as noise, pollution, crowding), and restricted access to healthy food or physical activity (see Burton and Whitfield 2003; Geronimus 2000; Schulz et al. 2005; Shapiro 2004; Williams et al. 2003). Investigators have also considered how persistently difficult conditions contribute to an increased tendency to engage in unhealthy behaviors, to feel hostility or anger, to suffer depression, or to engage in persistent high-effort coping—all risk factors for stress-related diseases (Dallman et al. 2003; James 1994; Northridge et al. 1998).

Yet the idea that an individual's racial assignment or identity is a fixed trait, albeit a socially constructed one, remains restrictive. If we stipulate that the social construct of race is relational and dynamic, then it is important to look to active social and psychological processes generated by racialized ideologies and their related structures and systems to understand how racial health inequality is maintained (Geronimus 2000). If stress physiology is a key link, we need to look at the ways environments, interactions, and shared intersubjectivity of race-conscious ideologies may be stressors. Concrete examples include overt acts of interpersonal racism; legacies of race-based oppression, such as accumulated disparities in wealth; race conscious, inequitable policies; glass ceilings; and public disinvestment in the polluted, decaying structures and health deserts that often characterize segregated residential areas. In addition to these, how else can we imagine the relational quality of race as a dynamic force that imparts socially patterned health advantages and disadvantages? In leaping from structural conditions and physical environments to molecular mechanisms, we may have overlooked an important set of social-psychological processes that activate physiological mechanisms precisely because the extent to which race affects biology is not constant within or across the experiences of individuals, but rather contingent upon the *situational salience* of the aspect

of an individual's social identity that is racial in specific settings, interactions, or relationships.⁴

This position assumes, then, that individuals experience their race as health-promoting or health-harming depending on situational factors and cues that vary in their daily rounds. For example, former NBA player John Amaechi (2011) describes this college experience:

In the Big Ten, I ended up a bit of a star. And I got very used to walking around campus and having people respond to me in this really effusive way . . . I started to really enjoy it. I would walk around campus and people . . . would yell . . . , “Meach!” “Yeah . . . This is cool.” . . . We had a good game the night before, walking to class, “Meach . . . Good game!” “Mmm, hmm . . . you know it.” And then one day I was walking just off campus, and . . . I was really feeling good about my life, and everything I was achieving. . . . A car goes by, window winds down, jams the brakes on, I've got my hand right here [raises hand in a wave] . . . All of a sudden, out the window, this kid goes, “Nigger!” . . . it affected everything. Some random kid, in the back of a crappy car that I can't even describe . . . someone I've never seen since, I don't think, someone I had certainly not met before . . . yells that word out of the window, and my world kind of crushed. There I was as an athlete at the time . . . But he crushed me flat. I thought I was this rounded three-dimensional person involved in all kinds of stuff, and a word from a stranger made me doubt myself.

In the course of this example, Amaechi's skin color phenotype does not change, yet his Blackness does. Its salience to and impact on him becomes more central and harmful because of its sudden social psychological relevance. What made him Black in a health-harmful way was what another person said and what they both understood it to mean. Identity group membership awareness—and the experience or valence of this awareness—is not fixed. The degree to which an individual is self-conscious of their racial identity, and whether that self-consciousness is comforting or stressful, a source of self-doubt or ethnic pride, is situational. It varies with social context, historical moment, expectations for performance, and situation-specific cues.

Steele's Concept of Contingencies of Social Identity

With the above vignette in mind, we turn to Claude Steele's concept of “contingencies of social identity.” Steele applies this concept most directly to the actions of “stereotype threat” on performance: the increased potential for

underperformance in situations where an individual experiences cues to the negative stereotypes attached to his or her social identity (Aronson and Steele 2005). Those cues have demonstrable psychosocial impacts in the moment that can result in deteriorated performance. As in Amaechi's case, such cues are fluid and an individual's sense of identity safety can be swiftly undermined, with potential long-term consequences for well-being.

How a person experiences his or her social identity at any given moment is additionally influenced by the stakes inherent in the situation for the individual. So, for example, the tragic irony, as Steele observes in the case of stereotype threat, is that the largest negative impacts on academic performance occur among individuals performing in an area where they are highly talented, trained, and personally invested, but in which members of their identified racial, ethnic, or gender group are thought to be incompetent. These high-stakes instances increase the potential of negative stereotyping to engender self-doubt, self-consciousness, or social paralysis, despite one's talent or preparation and in the face of pressure to overcome or disprove the stereotype. Steele observes that such stereotype threat is sufficiently powerful to "single out an identity and make it the center of a person's functioning, powerful enough to make it more important, for the duration of the threat, at least, than any of the person's other identities" (2010, 72). As Amaechi describes the impact of that one unexpected racist epithet: "I had felt myself to be this person that was rounded, that had everything that was going on, that was involved and connected to people in a real way . . . And some random stranger . . . made me think, is this what they really see? Is this what people see? Is it true that you can do all this stuff, that you can work this hard and try to achieve all these things, and still all people will see when they look at you, is that?"

Amaechi's example involves his response to a charged, direct interpersonal verbal attack. Yet individuals whose social identities place them in marginalized groups relative to others in the same setting can also be negatively impacted by shared understandings of marginalizing social ideologies and environmental cues that aren't targeted specifically at them. Amaechi provides an example, referring to an e-mail he had received from a college sophomore who was a starter on the varsity basketball team:

He said, "Kobe's my favorite player" . . . And now that's all changed, because one day . . . on TV . . . he could see Kobe's lip curl with contempt, as [Kobe] said what he thought . . . is the worst thing you can say about another man. He said that F-word, right there, big as life. That word, this kid said in his email, that he heard every single day. Never

directed at him because he's a Black kid, basketball superstar . . . and therefore it's impossible for him to be gay. But . . . the word was always around him. He made it sound . . . like it swarmed him like a mosquito, sucking the life out of him one drop at a time, infecting him with despair.

This, too, is a stark example. Cues to stereotyped identity are usually more subtle than hate speech: they can be unspoken and still have measurable effects. Thus, for example, researchers found that Asian American girls perform better on tests of mathematical achievement when they are cued to remember they are Asian, and do worse on the same math tests when they are cued to remember they are girls (Amhady et al. 2001). This impact has been observed in Asian girls in the United States as young as five years old.

We may posit a similar link between contingencies of social identity and health that hinges on recognized and largely unquestioned social ideologies, hierarchies, attitudes, and expectations that are structured by identity group. Through a broad range of social classifications related to a person's or a group's phenotypic characteristics, national origin, or religious affiliation, each of us becomes a gendered, racialized actor in a pervasive racial paradigm, shaping our lives and health in countless and consequential ways. In considering how Steele's documented instances of underperformance are linked to neurological and physiological processes in real time, it is logical to consider the possibility that threatening contingencies of social identity are important triggers of chronic activation of physiological stress processes. Steele draws on the model Schmader and Johns (2003) developed of the racing mind to interpret how stereotype threat results in deteriorated performance: "First, the threat of confirming the stereotype makes us vigilant to all things relevant to the threat, and to what our chances of avoiding it are. Second, it raises self-doubt and then rumination over how warranted the doubts are. Third, these concerns lead us to constantly monitor how we are doing (something that can cause choking in athletes, for example) . . . that's a lot of mental activity and while it's going on there isn't much mind left over for other things" (Steele 2010, 124).

Poorer performance itself contributes to the cumulative disadvantage already faced by marginalized group members—manifesting in restricted or foreclosed options for advancing education or employment, with related health implications. But it also appears that the repeated experience of stereotype threat expends cognitive resources and activates physiological stress processes that can cause wear and tear on important body systems over time. Even the finite lab-induced encounters with stereotype threat that Steele and

others used in their experiments were enough to affect pulse rate and stability, raise blood pressure, increase rumination, selectively recruit neural networks, and reduce working memory, leading to the observed deteriorated performance in their experimental groups (Blascovich et al. 2001; Krendl et al. 2008).

Continuing Inquiry

Considering race in its situational dimensions opens up new opportunities for inquiry and, ultimately, for intervention. It offers researchers a possible perch from which to better understand persistent puzzles in social epidemiology. For example, racial health disparities are apparent at all income levels and regardless of access to health care (Smedley et al. 2003). Historically, the “common sense” interpretation has been to assume that some essential feature of being Black (whether genetic, behavioral, or cultural) must explain the residual “race” effect after controlling for income or health care use. This interpretation has been well-critiqued with reference to the extent to which race and socioeconomic variables are subject to considerable measurement error and, thus, to residual confounding (Kaufman et al. 1997), as well as on the grounds that interpreting a residual as due to factors that were not measured in the study (such as genes, behavior, or culture) is scientifically inappropriate (Kaufman et al. 2007; Graves, this volume). Examining how the contingencies of social identity may act as a mechanism through which lived experience in a racialized society is translated into physiological stress process activation allows researchers to identify and gauge psychosocial processes that differ between Blacks and Whites of similar socioeconomic status as alternative explanations to essentialist interpretations of residual race effects. For example, Amaechi was the son of British physicians, graduated from a Big Ten college, and became an NBA player, presumably with a top one percent salary. His high socioeconomic position certainly must have protected his health in many respects—through avoidance of material hardship and access to knowledge, health care, food security, and health-promoting physical environments, for example. Yet it did not inoculate him from feeling crushed when a stranger reduced him through racist hate speech. In that drive-by interaction, the racist ideology of Black inferiority—with all its power to undo him—was embedded in a single word.

Turning to physiological impacts of contingencies of social identity for other socially marginalized groups, studies document that poor Mexican immigrants often have better health than other poor people in the United States (Markides and Eschbach 2005), and also that this health advantage disappears

in the next generation or even for the immigrants themselves the longer they reside in the United States (Collins and Shay 1994; Kaestner et al. 2009). Perhaps, as new immigrants, Mexicans in America are impervious to racialized contingencies of social identity (Pearson 2008), either because they have not internalized the U.S. racial hierarchy or because their social interactions are primarily with other Mexicans (Viruell-Fuentes 2007). As they reside in the United States longer, or, as the children of immigrants, they are raised here, they become more integrated with Whites, more aware of racial hierarchies and ideologies, and more vulnerable to contingencies of social identity pursuant to being of Mexican descent in the United States. Some evidence across the minor ethnic divisions within racial groups may also be interpreted from this perspective. One study found the self-reported health of Jewish Americans, who are highly educated and economically well-off as a group, is comparable to that of other Whites until socioeconomic resources are controlled, where upon their reported health more closely approximates that of Black Americans (Pearson and Geronimus 2011). This disparity might be explained by the impacts of contingencies of social identity for the stigmatized White group—in this case, Jewish Americans.

In another intriguing example, Arabic-named women in California suffered higher rates of poor birth outcome in the six months after September 11, 2001, than they had in the same six months the previous year, while rates for other ethnic groups did not change (Lauderdale 2006). One might hypothesize that the contingencies of having an Arabic social identity in the United States underwent a major transformation in the wake of 9/11, with concomitant stress process activation showing rapid health affects through its negative impact on pregnancy.

The above examples provide suggestive, indirect evidence, and there is much more research to be done. Whether temporary activation of the physiological stress process in response to specific, sometimes laboratory-manipulated cues really progresses into the early onset of chronic disease remains to be tested (although animal studies also suggest the biological plausibility of this hypothesis, see Blanchard et al. 2001; Jayo et al. 1993; Sapolsky 1998; Sekl and Meany 2004). To test hypotheses stemming from a focus on contingencies of social identity, interdisciplinary, collaborative efforts are needed, including between population health researchers and social psychologists. Together they could devise ways of monitoring the extent to which members of marginalized groups experience stress process activation secondary to changing contingencies of social identity and then identify the situations or cues that trigger these processes.

Translational Research

To determine best practices, translational research is also needed. Translational research refers to the transformation of interdisciplinary research findings into practical applications to improve health. Increasingly, translational research related to racial health disparities investigates biochemical pathways, molecular dynamics, and potential pharmaceutical treatments. However, findings from the social and behavioral sciences suggest alternate translational routes. In the current case, several lessons may be drawn from the research on the effect of stereotype threat on performance. First, the stress activation process that accompanies deteriorated performance appears to be triggered by contingency signaling cues in a specific setting. Members of groups at risk of devaluation based on their racialized social identity are attuned to such cues. For example, Purdie-Vaughns et al. (2008) found low minority representation in workplace settings coupled with an explicit organizational creed of “color blindness” (as opposed to “valuing diversity”) apparently led African American professionals to become distrustful of the setting and perceive threatening identity contingencies within it. Such findings point to the ubiquity of encounters with threatening contingencies of social identity for members of underrepresented groups but also to potential foci for ameliorative interventions to preserve identity safety in potentially threatening institutional environments, including schools, workplaces, doctors’ offices, health care facilities, and neighborhoods. As Steele notes,

If there is nothing in these settings that you have to deal with because you are a woman, or Black, or older, or have a Spanish accent then these characteristics will not become important social identities for you in that setting. They’ll be characteristics you have. You might cherish them for a variety of reasons. But in that setting they won’t much affect how you see things, whom you identify with, how you react emotionally to events in the setting, whom you relate to easily, and so on. They won’t become central to whom you are there. (2010, 73)

Unlike studies that connect structural background factors to molecular dynamics and health status, a focus on contingencies of social identity allows consideration of guiding principles for straightforward, low-tech, but potentially fundamental primary prevention strategies. The finding that situational cues, even cues that are incidental or spontaneous representations of entrenched racialized ideology, can throw a person into self-doubt or despair with the attendant physiological burden of becoming highly vigilant, can be

turned around to suggest the ameliorative power of becoming attuned to how aspects of settings and situations affect people inequitably and, then, changing the settings so socially patterned disadvantage is not reproduced.

Jedi Public Health

Those studying the impact of stereotype threat both in labs and classrooms (K–12 and college) have concluded that large and enduring improvements in performance can be realized in youth by changing situational identity contingencies, the cues that signal them, and the narratives that students use to interpret them (for a review, see Steele 2010). In the school context, they have shown positive results in improving performance through enhancing feelings of identity safety through simple methods, including

- Improving a minority group’s critical mass (number) in an integrated setting;
- Changing ways of giving critical feedback to show confidence in the student’s ability to succeed,
- Framing the ability to meet a challenge as learnable and expandable rather than as a fixed capacity;
- Fostering intergroup conversations that substitute familiarity and first-hand knowledge for stereotype-driven assumptions;
- Having students affirm their most valued sense of self, helping inoculate them from threats;
- Helping students develop a narrative about the setting that explains their frustrations.

Many of these guiding principles for action can be applied to other institutional settings such as workplaces, health care facilities, or health promotion interventions. More broadly, they may suggest policy applications related to neighborhood environments and representations of social settings and interactions in the media. The more settings in which a person experiences identity safety instead of stereotype threat, and the fewer insults that are accumulated on a daily basis to trigger the physiological stress process, the greater the chance that positive recursive processes are set in motion instead. For example, a girl who is not exposed to threatening situational cues in the school setting about girls and math will not only perform better on a specific math test but may also enlarge her future options if she is subsequently more likely to continue math study than a girl exposed to stereotype threat in math. A talented and

committed Black college student who is not challenged by stereotype threat in his organic chemistry course may be more likely to realize his potential and ambition to become a physician when it is not unilaterally dashed by a low grade in this gateway course (Steele 2010). Moreover, practices that reduce stereotype threats to underrepresented group members in integrated settings such as schools not only improve the health and life prospects of individuals, but they help diversify the academy and the labor force, naturally improving the prospects for continued identity safety.

The power of speech that Amaechi details can also be used to provide new self-affirming narratives, instill confidence in vulnerable groups, and quell physiological stress activation. As a seven-year-old, accompanying his mother on house calls in England in 1977—the year the first *Star Wars* movie was released—Amaechi observed,

I would sit in the living room of families who are really, really stressed because somebody in their house is really, really sick. And my mother would . . . do doctor stuff, and then she would come downstairs and she would always make time to talk to these families . . . She would look at these families . . . the tension in the room was clawing at them. She would look at them and she would say, “You can cope. I’ll be back in two weeks and you will be fine . . . You can manage . . . This is what you are going to do . . . and then I will be back.” You could just feel the tension . . . drop in the room. . . .

. . . I thought, I’ve seen this before . . . in *Star Wars*. Obi-Wan Kenobi, he’s got the droid in the back and he is being stopped by the storm trooper and Obi-Wan says to the storm trooper, “These aren’t the droids you are looking for.” And the storm trooper said, “These aren’t the droids we are looking for. Move along, move along.” This is what my mother is doing. I remember going back to my room, shutting the door and sitting on my bed and thinking, my mother is a Jedi.

We can all be Jedi in this way. While doing our best to design settings and interactions to maximize all participants’ feelings of identity safety and to eliminate many episodes of health-threatening stress process activation, we can also use words once a stress-inducing challenge has occurred, to shut off the stress response and allow the feeling of threat to recede, thereby reducing overexposure to stress hormones.

Actions that reduce stereotype threat and threats to identity safety more broadly—that expand the ways that diverse people feel valued and safe from the contingencies of their racial identities in integrated settings and that

reassure all individuals of their competence and belonging in a variety of situations—would be steps toward Jedi public health. Considerable evidence now suggests that taking such actions would improve the academic and work performance of members of underrepresented groups. Even if that is all such actions accomplished, they would contribute to reducing health disparities by mitigating structural barriers to socioeconomic resources for members of oppressed groups. But if, as one might suspect, these actions also importantly diminish the frequency or duration of acute episodes and chronic periods of physiological stress process activation and set positive recursive processes in motion instead, great and rapid gains toward eliminating racial health inequality might be achieved. While we await returns on high-stakes, speculative, molecular, and pharmaceutical translational research investments—knowing also that their products are likely to be distributed inequitably and may even reify racial health inequality (Kahn, this volume; Link and Phelan 1996; Rubin et al. 2010)—it would be relatively straightforward and inexpensive to explore the possibilities of Jedi public health.

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Notes

1. Epigenetic mechanisms influence what proteins cells make under specific conditions. To the extent that the environmental conditions that impact them are experienced at critical periods, especially in utero, they may have effects on later health, although understanding of epigenetic influences and mechanisms, as well as the degree to which they may be permanent or transgenerational, is in its infancy. Similarly, human stress genomics research suggests that stressful life experiences can influence gene expression. Both of these emerging approaches require continued elaboration and critique beyond the scope of this chapter.
2. Telomeres are the stabilizing caps of chromosomes that shorten with each mitotic division until the cell either dies or enters reproductive senescence. Research

indicates that, because breaks in the DNA structure due to oxidative stress are not easily repaired in PBMC telomeres, oxidative stress is an important mechanism by which telomeres are shortened. As oxidative stress is an important mechanism linking aging, psychosocial stress, biological stress activation, inflammation, and disease development, PBMC telomere length may serve as a powerful marker of the toll that cumulative stress takes on the body—a biological rather than chronological age marker (see Geronimus et al. 2010 for a review). The validity and utility of using telomere length as a biomeasure of aging is still being investigated.

3. These estimates were robust to controlling for measured health behaviors and health care use, and they occurred despite higher economic profiles of immigrants with increased length of residence in the United States.
4. This is different from the idea that one's reported racial label may change according to situation—although it is also true that people's self-reported racial identity in surveys such as the census can be fluid; some members of marginalized racial or ethnic groups "pass" part or full time; or their racial identity might differ from their racial assignment in the eyes of others.

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Contextualizing Lived Race-Gender and the Racialized-Gendered Social Determinants of Health

When I take my two daughters and other family members to the local hospitals in Albuquerque, New Mexico, I am sometimes asked to fill out forms regarding "race," ethnicity, and language at the registration desk.¹ As I fill out these forms, I make note of the large bright posters lining some of the registration cubicles, which feature smiling patients from a variety of backgrounds. Several captions attempt to reassure patients by explaining why it is important to collect race, ethnicity, and language data in the hospital setting: "We ask because we care. By asking your race, ethnicity, and language, we are able to deliver health care equally to all patients. What is your race? What is your ethnicity? What is your primary language?" Toward the end of the placard another heading affirms: "Respecting every difference, treating each equally. Get REAL: Race, Ethnicity, and Language."²

As an Afro-Latina and a sociologist of racial and gender stratification, I am viscerally aware of the importance of collecting data and analysis of data on "race" and ethnicity. As several of my colleagues have pointed out in this volume, one way of pursuing high-quality research on race and inequality in a variety of domains including health, education, and beyond is to take the social construction of race seriously (Gómez, this volume). While it is tempting to equate ethnicity with racial status, the conceptual and analytical distinction between race and ethnicity is of particular importance, as studies have found qualitatively different treatment and health outcomes for Latinos who self-identify or are socially defined as Black as opposed to White, or "some other race" (LaVeist-Ramos et al. 2011; Jones et al. 2008; Gravlee and Dressler, 2005). For example, I was born and raised in a New York City public housing project