Abstract
This article examines how people of color can biologically inherit the deleterious effects of white racism. Drawing primarily on the field of epigenetics, I demonstrate how transgenerational racial disparities are in fact racist disparities that can be manifest physiologically, helping constitute the chemicals, hormones, cells, and fibers of the human body. Epigenetics can be used to demonstrate how white racism can have durable effects on the biological constitution of human beings that are not limited to the specific person who is the target of white racism, but instead extend to that person’s offspring. In this way, the field of epigenetics can help philosophers and others understand the transgenerational biological impact of social forces such as white racism. It reveals that the damage done by white racism is more extensive than critical philosophers of race might have realized, and also that interventions against white racism must address not just the economic, geographical, social, and psychological, but also the biological aspects of human existence. In particular, the article examines racist
disparities in preterm birth rates and argues that the scope and significance of prenatal care for African American women must be expanded intergenerationally and include wide-scale forms of racial justice.

When you’re confronted with racism, that covert racism, your stomach just gets like so tight. You can feel it almost moving through your body; almost you can feel it going into your bloodstream.

—Kim Anderson, *African American Lawyer and Mother*

This article examines how people of color can biologically inherit the deleterious effects of white racism: how the harmful physiological impact of white supremacy and white privilege can reach across multiple generations, from grandparents to their children and their grandchildren. In at least one respect, of course, the general concept of transgenerational inheritance of white racism is nothing new, at least not to critical philosophers of race and others who study—and often live—radical disparities in wealth across racial lines. On average, in the late 1980s white people in the United States had twelve times the net worth of African Americans, in the form of property, savings, et cetera (Mills, C., 1997, 37–39). The gap only widened in the wake of the 2008 recession. In 2010, the median net household worth of white American families was twenty-two times that of black American families: $110,729 compared to $4,955 (Luhby 2012). The roots of this disparity reach far back in American history to the days of slavery, when black slaves did not own their labor and thus could not profit from it, to the days of Reconstruction, when newly freed slaves did not receive their promised forty acres and a mule. These patterns have not disappeared, but the issue of economic transgenerational inheritance tends to be discussed only indirectly—if at all—with mention of neither race nor centuries of white domination. While communities of color occasionally revive related questions concerning reparations to African Americans, Native Americans, and Japanese Americans, generally the subject of transgenerational inheritance is relegated to the seemingly color-blind realm of tax codes governing estate and inheritance taxes.²

Economic racial disparities constitute only one form of transgenerational inheritance, however. Another equally significant form is transgenerational
inheritance along biological or physiological lines. We could call this phenomenon “racial disparities in health,” as opposed to (or actually, often in conjunction with) “racial disparities in wealth.” As the biological and medical sciences increasingly are demonstrating, there exist significant differences in the general health of white and nonwhite Americans. For example, African Americans, Native Americans, and Pacific Islanders generally have higher rates of coronary artery disease, diabetes, stroke, HIV/AIDS, and infant mortality than do white Americans (Smedley et al. 2012, 1). Health differences between black and white Americans in particular have been well studied in the United States, and thus we know that African Americans under fifty years of age are twenty times more likely to experience heart failure than white Americans in the same age group, and they have higher rates of the accompanying conditions of high blood pressure, obesity, kidney disease, and low levels of LDL, or “good” cholesterol (Gravlee 2009, 48; Kam 2010). Racial health differences between black and white Americans often persist, moreover, even after adjusting for differences in socioeconomic status (Williams et al. 1997, 337; Lu and Halfon 2003, 14).

These are health differences related to race, but just as accurate as labeling them *racial* disparities would be to call them *racist* disparities. Nowhere in the scientific world has this been recognized more forcefully but also debated more vigorously than in the health sciences. As early as 1991, for example, a pair of American neonatologists called for “a shift of focus from ‘race’ to ‘racism’” in studies of racial health differences (David and Collins 1991, 240). In the past decade, their request has been honored somewhat as various social and life scientists have documented the effects of white racism on nonwhite people’s physical and mental health, challenging the assumption that racial health differences can be explained by race apart from the social phenomena of racial discrimination and racial prejudice. This shift in the health sciences is intensely disputed, however. Although some studies on race and health disparities increasingly are “unmask[ing] racism as a bona fide public health problem” (Drexler 2007; see also Krieger 2008), large amounts of money and time are being devoted to the search for genetic causes of racial differences (Duster 2003 and 2006; Kahn 2012; Krieger 2005b; Lee, Koenig, and Richardson 2008, 89–200; Roberts 2012, 104–22). While the connection between racism and the risk of poor health isn’t news to most people of color, it is profoundly challenging the way that the life sciences thinks about the etiology of many diseases and grapples with questions of how to measure racial discrimination.
Understanding the biological inheritance of the effects of white racism means first and foremost appreciating how something social can become physiological. (I use “social” here very broadly to encompass political, cultural, economic, aesthetic, and other facets of human existence that often are assumed to be nonbiological or nonphysiological.) As a briefing paper for the American Nurses Association recently has asked, “How does race get under the skin and influence our physiology if it isn’t biological?” (Smedley et. al. 2012, 6). The paper’s authors rightly answer that it is not biology alone that constitutes our body’s physiology. Existing health disparities between races, for example, are not the result of any innate biological or genetic differences—indeed, in that sense, race does not exist—but rather the result of being harassed, oppressed, and discriminated against because one is not white. As Kim Anderson attests in the epigraph above, when a person is the target of racial oppression, racism can move through his or her body, seeping into the bloodstream and wrenching the gut.

We should understand Anderson literally: the experience she depicts is not merely metaphorical or figurative. The effects of white racism include physiological changes for the people who are confronted by it, changes that typically are very damaging to their physical (as well as psychological) health. The sociopolitical phenomenon of white racism can be and often is a physiological, biological phenomenon, in other words. Nonmaterial things that are “outside” the body can get “inside” and help compose it. Or better yet, we need to develop ways to think about biology, including genetics, that neither assume they are radically divorced from the social nor completely collapse them into it. But how exactly should we understand their relation? More specifically, how can the effects of white racism be simultaneously social and biological such that they can get “under the skin” and into the stomachs and bloodstream of people of color? And how are these physiological effects sometimes inherited by subsequent generations, getting “under their skin” too?

I will draw primarily on the field of epigenetics to answer these questions, demonstrating how transgenerational racist disparities can be manifest physiologically and help constitute the chemicals, hormones, cells and fibers of the human body. Epigenetics is philosophically relevant because it “highlight[s] an important set of mechanisms by which social influences can become embodied, having durable and even transgenerational influences on the most pressing U.S. health disparities” (Kuzawa and Sweet 2009, 2). Epigenetics thus can be used to demonstrate how white racism...
can have durable effects on the biological constitution of human beings that are not limited to the specific person who is the target of white racism but instead extend to that person’s offspring. In this way, the field of epigenetics can help philosophers and others understand the transgenerational biological impact of social forces, such as white racism. It reveals that the damage done by white racism is more extensive that critical philosophers of race might have realized, and also that interventions against white racism must address not just the economic, geographical, social, and psychological, but also the biological aspects of human existence.

One final point before I dive into the details. The topic of the biological durability of white racism is somewhat somber and even dispiriting, but I want to underscore that “durable” is not the same thing as “permanent.” In my view, white racism is far more extensive than generally is admitted—at least by most white people, and perhaps by some people of color too—and will be far more difficult to eradicate than most (white) people think. This does not mean, however, that it can’t be challenged successfully, at least in some situations some of the time. (I do not share Derrick Bell’s [1993] view that white racism is ineradicable, although I appreciate why he makes this claim.) Precisely to improve the chances of success, it is important to confront the fact that white privilege and white supremacy are tough, stubborn, and invidiously resourceful. One reason they have been able to endure for so long is that they can repeat their effects transgenerationally in the physiological and biological processes of human life.

Weathering

A major health disparity between black and white Americans is found in preterm birth rates, and because of the extensive and often severe consequences of preterm birth, I focus on it here. Preterm birth, which occurs when a baby is born at least three weeks before full term (forty weeks), is a leading cause of infant death and mortality. It also is associated with numerous, subsequent health problems in both childhood and adulthood, such as respiratory and heart problems (including cardiovascular disease and related maladies), cerebral palsy, intellectual disabilities, vision and hearing complications, and feeding and digestive problems (Lu and Chen 2004, 692; Centers for Disease Control 2012; Mayo Clinic 2011; Kuzawa and Sweet 2009, 3). African American women are 1.6 times more likely
than white American women to give birth prematurely (thirty-seven weeks or earlier), and 2.9 times more likely to give birth very prematurely (thirty-two weeks or earlier) (Lu and Chen 2004, 692; Mayo Clinic 2011). An African American baby is more than twice as likely to die in the first year of his or her life than a white American baby (Lu and Halfon 2003, 13). This gap has not improved since the civil rights movement and the end of Jim Crow, despite efforts to increase African American women’s access to prenatal health care (Lu et al. 2010, 62). In fact, it only has widened in the past fifty years: from 1.6 to 2.3 times a greater risk of mortality for African American children than white American children in the first year of their lives (David and Collins 2007, 1191). As one neonatologist has put it, “There’s something about growing up as a black female in the United States that’s not good for your childbearing health. I don’t know how else to summarize it” (California Newsreel 2008, 1).

What is this “something” that is so damaging to African American women’s health? The official if somewhat unhelpful answer provided by the U.S. Centers for Disease Control (2012) is that “the reasons for the differences between [the preterm birth rates of] black and white women remain unknown and are an area of intense research.” A significant portion of this research is searching for a “preterm birth gene” specific to African Americans (David and Collins 2007, 1191–92). Besides tending to work with an essentialist concept of race, this line of research is troubling because it “problematically conflates observed biological variation with inferred genetic contributions, and ignores evidence that social factors can have durable life-course and transgenerational effects on health” (Kuzawa and Sweet 2009, 9).

It wrongly assumes that “predisposition” means “genetic,” that is, that the fact that people are born with predispositions for particular health conditions demonstrates that those conditions must be genetic (Francis 2011, 55). The misguided assumption made by the quest for a preterm birth gene is that biology is both synonymous with genetics and antithetical to all things social.

Two alternative, complementary approaches understand the relationship between the biological and the social in a more sophisticated fashion: epigenetics and weathering. Let me begin with the simpler concept of weathering, which signifies the gradual wearing down of the body’s systems by stressors that accumulate over time (Blitstein 2009). These stressors can come from just about anywhere in a person’s environment, from the more chemical or physical (e.g., air pollution or intense physical strain) to the more social or interpersonal (e.g., children’s temper tantrums or an
unreasonably demanding boss). As the body experiences more and more stress, it becomes more and more weathered, making it more and more prone to disease and chronic health problems.

The notion of allostatic load helps explain how environmental stressors get under the skin and have detrimental biological effects (Blitstein 2009, 6). Allostasis is a type of physiological regulation in which the internal stability of an organism is maintained while the organism deals with a challenge or crisis. (This is in contrast with homeostasis, which refers to the maintenance of the organism’s internal stability in the ordinary ebb and flow of life.) When the body undergoes a physical or social challenge, it temporarily produces extra hormones, such as adrenaline, that help the organism meet the challenge and then ceases producing them when the challenge has passed. This would count as a manageable allostatic load. In the case of ongoing stress, however, the body doesn’t stop its extra hormone production, resulting in a high allostatic load. It is as if the organism’s allostatic capacities are forced into overdrive and never shut off, weathering the body’s systems of regulation, which produces health problems such as cardiovascular disease, diabetes, and accelerated physiological (vs. chronological) aging (Blitstein 2009, 6). What forces them to do this is not, for example, some kind of genetic quirk, but a social environment that places too much stress on the physiological capacities of the organism.

Weathering helps demonstrate why many racial health disparities are in fact racist health disparities. While all people experience some stress from time to time, and white people also can have high allostatic loads from severe chronic stress, African Americans and many other people of color generally have to deal with a significant stressor that white Americans do not: racial oppression and discrimination. White racism contributes to the excessive weathering of non-white bodies, leading to elevated rates of disease and accelerated aging in comparison with white Americans (Geronimus et. al. 2006). For example, based on differences in telomeres, which serve as a kind of biological “mitotic clock” that measures age, black women at ages forty-nine to fifty-five years are estimated to be biologically 7.5 years older than white women in the same age group (Geronimus et. al. 2010). The ongoing struggle of African Americans against white domination and white privilege—even, or perhaps especially in their mundane, quotidian forms—means that African Americans’ allostatic systems rarely get a break. They are like an engine that is revved nonstop, burning out much more quickly than an engine that gets to rest from time to time.
Weathering thus helps explain Kim Anderson’s particular situation. A well-educated, health-conscious woman whose first baby was extremely premature, Anderson is an example of what has been called the “paradox of the well-off black mother” (David and Collins 1991, 238). Anderson and her husband enjoy(ed) a high socioeconomic status, and she ate well, exercised, didn’t smoke or drink, and otherwise did everything “right” during her pregnancy. And yet she went into labor two and a half months early, delivering a daughter who weighed only two pounds and thirteen ounces (California Newsreel 2008, 2). It seems that this shouldn’t have happened. Education and socioeconomic status are good predictors of infant mortality for women of any race, and the poorest women with the least education tend to be at greatest risk. Decrease poverty and increase education, and infant mortality will drop, or so the thinking goes. Anderson’s education, financial situation, and general good health should have protected her from having a preterm birth. But this rubric doesn’t work for African American women the way it does for white American women. The rate of infant mortality for African American with college degrees or higher is about three times higher than that of white women with the same level of education, and well-educated African American women have worse infant mortality rates than white women without a high school education (California Newsreel 2008, 3–4). The question thus is why doesn’t higher socioeconomic status reduce the rate of preterm birth for African American women the way it does for white women in the same socioeconomic class?

The answer is not likely to be found in a preterm birth gene carried by African Americans but not white Americans. This is evident when one compares the rates of preterm birth of white women and African immigrants to the United States. If people of African descent carry a preterm gene, then African women who give birth shortly after immigrating to the United States should have a similar rate of infant mortality and preterm birth as African American women. But in fact, their rates are virtually identical to that of white American women. Even more striking is the fact that within one generation of living in the United States, the rate of infant mortality and preterm birth for African immigrant women climbs to that of African American women (California Newsreel 2008, 4; David and Collins 2007, 1193; Kuzawa and Sweet 2009, 7). In other words, the problem is broadly environmental, not genetic: there is something about living as a black person in the United States that is bad for black women’s reproductive health.
Above all, that something includes the stress of enduring white racism. For women of all races, stress hormones are a normal part of pregnancy and help trigger labor when the baby has come to term. Abnormally high levels of stress, in contrast, can limit fetal growth, contribute to uterine inflammation, and trigger premature labor (California Newsreel 2008, 6; Kuzawa and Sweet 2009, 4; Lu and Chen 2004, 692; Roberts 2012, 139–40). The amount of stress experienced by a woman before her pregnancy is relevant, moreover. If a woman begins her pregnancy with a stress levels that are already unhealthily high, then the extra stress hormones produced during pregnancy can push her to the brink of labor sooner than they should. What counts is not just stress experienced during the nine months of pregnancy, but also accumulated stress across a woman’s life course (Lu and Halfon 2003). All women experience occasional stress, of course, and any particular woman can accumulate unhealthy levels of stress prior to a pregnancy. But what if a society routinely subjects a particular group of women to ongoing chronic stress, treating them as sub-persons from birth forward? This describes the general situation of black women who live in white-dominated cultures, such as the United States, regardless of their socioeconomic status. Weathered by white racism (and sexism), and thus often beginning a pregnancy with a relatively high allostatic load (Guyll et al. 2001; Lu and Chen 2004; Roberts 2012, 132–33; Sternthal et al. 2011; Woods-Giscombé 2010), even educated, well-off African American women have a high risk of pre-term birth. And because babies who survive a pre-term birth are more likely to have significant health problems, the racial stress experienced by African American women is not limited to them but “can have a life-long impact on African American families and their health” (California Newsreel 2008, 6).

With the concept of weathering, we already can see how the effects of white racism reach across a generation from mother to child. As Anderson recounts, when she enters a store and is tailed by a suspicious clerk because “they just see a black woman,” the result is a surge of adrenaline and other stress hormones that caused her stomach to tighten and her heart rate to increase (California Newsreel 2008, 7). Experiences such as these, which are common for many African Americans, are not restricted to their immediate targets. The targets’ children, such as Anderson’s daughter, indirectly will undergo the experience as well. The experience continues in the children’s lives, not just, for example, by hearing their parents talk about their confrontations with white racism or by learning from their parents.
strategies for surviving in a white-dominated world. They also undergo their parents’ experience in a more unconscious fashion, in the bodily effects that it has on their health while in the womb.

**Epigenetics**

I want now to push this argument further, demonstrating how the biological dimensions of white racism can replicate themselves across more than just one generation. This will entail a detour into the general principles of epigenetics before returning to the specific topic of white racism. *Replicate* is not a metaphor here, and for that reason my use of the term might make some readers skeptical. Weathering may have significant health effects on a mother and her fetus, but at bottom, one might object, it is merely an instance of a fetus’s environment impacting its development, not genuine biological replication across generations. The latter can happen only through genetic reproduction, isn’t that what Darwin and Mendel taught us? Wasn’t Lamarck’s theory of inheritance of acquired characteristics—for example, that giraffes’ necks lengthened over a few generations so they could reach leaves on tall trees—soundly defeated in the nineteenth century? How can one credibly claim that something broadly social and non-genetic, such as the effects of white racism and white domination, can be biologically inheritable?

Coined in the 1940s and rooted in the word *epigenesis*, the term *epigenetic* initially was used to oppose preformationism in debates about human development (Francis 2011, 136). While preformationism argued that the human self was fully preformed at the moment of conception and thus merely needed to grow, an epigenetic perspective held that development is a gradual and creative process in which a person comes to exist (Francis 2011, 120). Various religious issues swirled around these two positions, but for my purposes here the important difference between them is the role that the environment plays (or not) in the formation of a human being. Preformationism effectively dismissed the environment as unimportant to human development. As the earliest, seventeenth and eighteenth century versions of preformationism held, whatever a person would essentially be when she or he became an adult, it was fully contained in the unfertilized egg. More sophisticated versions of preformationism emerged in the eighteenth and nineteenth centuries, arguing that a human development was a
process of making “manifest” the being that was “latent” in the zygote, but the preformationist message was unchanged. Human development supposedly is a mere unfolding of what is already fully present, a process relatively independent of environmental transactions (Francis 2011, 120–21).

Fast-forward to the twenty-first century, and one can find the ghost of preformationism alive and well in modern genetics. Nowadays instead of the unfertilized egg or zygote, it is the gene—or more precisely, one’s DNA (deoxyribonucleic acid)—that often is considered the already-formed kernel of a human being. Somewhat oversimplified, each person allegedly has a genetic program for developing from conception to adulthood into the particular person that she is, and her development does not depend in any essential way on material, social, or other environments. Of course, modern genetics recognizes that the environment contributes something to human development—think, for example, of the type and quality of food that a person eats. A person couldn’t develop (she would die) if she didn’t eat. But her genes nonetheless dictate how her body will make use of the food she consumes: her metabolism, her ability to process glucose, and so on. Whether she becomes obese and/or develops diabetes, to continue the example, depends ultimately on what kinds of genes she inherited from her parents, not on her environment. On this view, the gene plays an executive role in the cell. It is something like a director of a theatrical production, unilaterally controlling the proteins (actors) and biochemicals (stagehands) of a cell that, in turn, work to fulfill the gene’s goal (Francis 2011, 18–19).

Epigenetics, in contrast, rejects the notion of an executive gene, and it does so by acknowledging the formative role that various environments play in cellular activity. Perhaps most straightforwardly, “epigenetic” refers to “somatically heritable states of gene expression . . . without alternations in the DNA sequence” (Choi and Friso 2010, 8). To explain this definition further, consider the gene in its “naked” state, which is DNA in its well-known form of the double helix. What is less well known, at least by laypeople, is that the naked gene is something of a fable. A gene is rarely if ever naked, which is to say that the double helix neither exists nor functions by itself. Chemically attached to it are various organic molecules that regulate its activity (Francis 2011, xi). One of the most common forms of DNA regulation is methylation: a gene with methyl attachments (one carbon atom and three hydrogen atoms) is said to be methylated (Francis 2011, 6). Methylation can occur in varying degrees. We can think of methyl attachments as something like power knobs, turning down (or even off) certain
genes in some cases and turning up other genes in other cases (Kuzawa and Sweet 2008, 4). The more methylated a particular gene is, the more its expression is inhibited, and vice versa (Francis 2011, 46). Thus, for example, when the genes in mice contributing to both coat color and various health problems (such as obesity, diabetes, and cancer) are unmethylated—that is, turned up—the mice have yellow coats and significant health problems. When the same genes are highly methylated—i.e., turned down/off—the mice have both darker coloration and no health problems associated with obesity, diabetes, or cancer (Francis 2011, 82–83).

How do genes become methylated or demethylated? While methyl attachments and detachments can be a product of happenstance, often they occur because of environmental factors. For example, diet tends to play a significant role in epigenetic processes. When the mice with inadequate methylation of the relevant gene were fed a diet high in a methyl donor, such as folic acid, the coat color of their offspring darkened and their offspring suffered less from obesity, diabetes, and cancer (Francis 2011, 85). (Epigenetics thus is related to why pregnant women are advised to take folic acid supplements.) Environmental factors contributing to methylation include not just elements of the physical and chemical environment, such as the food one eats and the pollution one intakes, but also social exchanges and interactions. Competitive experiences in sports or work, for example, can raise testosterone levels affecting gene expression, just as psychological trauma and chronic stress can influence genes expression via the elevation of corticotropin releasing hormones (CRH) that stimulate cortisol production (Francis 2011, 29, 33, 39–40).

The existence of methylation and other epigenetic processes helps shatter the myth of the executive gene. A person’s material and social environments can play as significant a role in cellular activities determining health, weight, appearance, and so on, as her DNA does. Considered as part of a team that includes its epigenetic attachments, genes are poorly understood in terms of unilateral causation. They are as much an effect as they are a cause of environmental, non-genetic factors (Francis 2011, 76, 124, 159). We thus could claim that “epigenetic processes occur at the interface of our environment and our genes,” which is to say that our cells—our bodies—are dynamically co-constituted by things both “inside” and “outside” of us (Francis 2011, xi).

The case of the under-methylated mice does more than just shatter the myth of the executive gene, however. It also provides an example of
epigenetic inheritance. Epigenetic modifications to genes can and do occur within the lifetime of a single being, continuing well after the developmentally crucial periods of birth and childhood (Francis 2011, 47, 74). This is medically significant, since it means it is conceivable that pathological epigenetic expressions could be changed or even reversed. But epigenetic events are not always or necessarily contained within a single being. They can be passed from one generation to another, as when the folic acid eaten by under-methylated mice changed the biochemistry of their pups. How is it that epigenetic processes can be transgenerationally inherited?

There generally are three forms of epigenetic transgenerational inheritance, ranging from more or less direct, with “direct” meaning that “the epigenetic mark is transmitted directly from parent to offspring through sperm or egg” (Francis 2011, 158). The most indirect form of epigenetic transgenerational inheritance occurs via the repetition of similar environments and social contexts in subsequent generations. In this case, the environment that triggers the (de)methylation or other alterations of a particular gene is reconstructed anew for each generation, producing epigenetic effects for offspring that are similar to those of their adult caregivers. The most powerful environment for this type of epigenetic inheritance is the so-called maternal, or uterine, environment. For this reason, indirect epigenetic inheritance is similar to weathering. In the case of epigenetic inheritance, however, the extra cortisol that a pregnant female produces when she is stressed does more than trigger early labor. It changes some of the epigenetic markers on the fetus’s DNA. When the mother’s stress hormones are transmitted to the fetus through the placenta, they program—or overtax—the fetus’s stress axis, which runs from certain neurons in the hypothalamus to the production of CRH and cortisol (2011, 39–40). The result is something like a fetal post-traumatic stress disorder (PTSD): “The elevated cortisol levels experienced by the fetus permanently adjust the settings of the stress axis of the fetus in a way that makes it more sensitive and hyperresponsive to subsequent stressful events” (2011, 42). The adult that the fetus then becomes is more likely to experience psychological difficulties, including actual PTSD, when exposed to significant stress.

Without performing specific tests on Kim Anderson’s daughter, we have no way of knowing if she inherited the effects of her mother’s racist environment via epigenetic changes. We do know, however, that something like this occurred in the case of German children whose mothers were pregnant during the Holocaust and American children whose mothers were
pregnant during the September 11 World Trade center attacks. The German children were more prone to PTSD even though they had no direct experience of the Holocaust, and the American children were born with elevated stress responses and a hypersensitive stress axis, making them more susceptible to anxiety and depression than children whose mothers did not experience PTSD while pregnant (Francis 2011, 43; Kuzawa and Sweet 2008, 6). The difference between these two examples and Anderson’s situation lies in the duration of the stressful event in question. Because they were distinct events or time periods, the Holocaust and September 11 did not contribute to weathering in the same way that ongoing quotidian racism does. Nevertheless, the Holocaust and September 11 illustrate the repetition of a stressful/traumatic environment across two generations, producing similar epigenetic effects for each of them. As in the case of the Holocaust and September 11, it is highly plausible that the racism experienced by Anderson (and many other African American women) was a cortisol-producing situation that epigenetically changed their fetus’ stress axes, producing deleterious psycho-physical results.

Parenting Styles

A good chance exists that this pattern will repeat itself in the third generation, at least absent social, medical, or other interventions to change a person’s stress response. Offspring with hypersensitive stress axes can become parents who create environments for their offspring that will elevate their children’s (the grandchildren’s) cortisol levels in turn (Guerry and Hastings 2011). For one reason, a pregnant woman who has an overtaxed stress axis (courtesy of the uterine environment when she herself was a fetus) will in turn have elevated cortisol levels in her uterus that can be transmitted to her fetus via the placenta (Francis 2011, 42). In other words, Kim Anderson’s future grandchildren via her daughter are at risk of having overtaxed stress axes regardless of her daughter’s experiences with white racism and even if (miraculously) white racism was abolished before the grandchildren were conceived.

A second reason is that an overtaxed stress axis also can affect parenting styles. As the above study of methylated mice also demonstrated, mice with a hyperresponsive stress axis do not lick their babies as much as parents with a healthy stress axis do. This is significant since baby mice
that are licked amply have higher levels of a particular nerve growth factor (NGF-A) that, when bound to glucocorticoid receptors (GR) in the brain, epigenetically change the responsiveness of that gene for the better. The higher the NGF level, the less sensitive one is to cortisol, which reduces levels of CRH and results in a dampened (i.e., more resilient) stress axis (Francis 2011, 45–46, 69). In sum, mouse pups of poor lickers tend to develop the same overtaxed stress axis that their parents (and grandparents) have, and so the cycle repeats itself. Similar results have been found in studies on captive gorillas and rhesus monkeys (Francis 2011, 69). Epigenetic effects related to the genes associated with stress sensitivity can be inherited transgenerationally through the replication of post-natal as well as uterine environments. This means that Kim Anderson’s stress levels might contribute to a parenting style that makes her daughter more sensitive and less resilient to racist (and other forms of) stress.

I will return shortly to the contentious issue of parenting styles, but before I do, it is important to note that biases exist in epigenetics just as they do in any scientific field. There is no such thing as objective science if “objective” means a viewpoint that is independent of any particular perspective or interests. This is not necessarily cause for relativistic alarm, nor is it reason to dismiss what can be learned from epigenetics and other sciences. In and of itself, bias is not problematic. In fact, it is unavoidable given that human beings are in and of the world, not little gods hovering outside it. As embodied, perspectival human beings, we can still judge better and worse ways of understanding the world. The important question to ask about epigenetics thus is not whether it is biased, but which particular biases have contributed to it and whose interests they tend to serve. The biases present in epigenetics do not necessarily invalidate the field, but they need to be unearthed, acknowledged, and critically countered by other perspectives.

For example, in my discussion of the pup-rearing methods of methylated mice, I referred to parental styles of licking, when in fact the scientific studies examined and discuss maternal styles of licking. Paternal styles of parenting in mice and other mammals, especially their connections to the stress responses of their offspring, have not been studied much to this point, as if they are irrelevant to the subject of childrearing (Francis 2011, 73). This fact reflects a male-privileging bias that the job of caring for offspring is properly the work of the females of the species. This bias is in need of feminist critique, not just politically and ethically but also epistemologically. Perhaps there are different ways in which methylation
is triggered in newborns and young children given different parenting styles commonly adopted by men and women. Or perhaps parenting is just parenting and there are no meaningful differences in methylation patterns in offspring based on the gender of the parents. Rather than myopically focusing on maternal styles and the so-called maternal environment (as if pregnant women and mothers weren’t persons, but an atmospheric condition or landscape), scientists should reformulate both their implicit assumptions and their explicit hypotheses about gender and parenting so as to expand our understanding of epigenetic inheritance.

Equally pressing is the need to interrogate racist stereotypes of non-white people that could influence both epigenetic research and its subsequent interpretations. For example, in the nineteenth century craniology sought to prove scientifically the innate intellectual inferiority of people of color (Gould 1981), and in the late twentieth century Richard J. Herrnstein and Charles Murray’s inflammatory book, *The Bell Curve* (1996), appealed to genetics for much the same end. In the case of epigenetics, stereotypes of the black family as broken and dysfunctional are particularly troublesome. They probably date back to the days of chattel slavery, but they were given a kind of official credibility with Daniel Patrick Moynihan’s 1965 publication of “The Negro Family: A Case for National Action” on the part of the United States Department of Labor. In the name of advising the federal government how to improve the lives of black Americans, Moynihan’s report diagnoses black families as pathological. “At the heart of the deterioration of the fabric of Negro society is the deterioration of the Negro family,” Moynihan writes, “it is the fundamental source of the weakness of the Negro community at the present time.” Because black families supposedly are broken, they are perpetuating problems of poor education, poverty, illegitimate births, dependence on welfare, and (most problematic of all, for Moynihan) female-headed households in the black community. Moynihan blames slavery for initially breaking black families, but the fact remains for him that merely by growing up in a black family, a black child is being socialized in an unstable, pathological environment that ensures the replication of black inferiority from generation to generation. According to Moynihan, before real headway can be made against racial inequalities, black families must be repaired so that they have the same high degree of stability as white families.

Bluntly put, one of the dangers of epigenetic research in connection with race and racism is that it could be wielded as scientific “proof” of the
diseased or broken black family. This danger is not intrinsic to the field of epigenetics. Epigenetic inheritance can and does happen for people of all races and, moreover, it encompasses health benefits, not just health problems. But because epigenetics and the racist stereotype of the broken black family share the theme of socially and environmentally caused problems being passed down generationally, epigenetics might be seen as offering physiological evidence of the validity of the stereotype. Perhaps it is not black people’s fault—one could say it is the fault of a racist environment, just as Moynihan pointed a finger at chattel slavery—but they nonetheless are a sick people. For that reason, they are biologically inferior to white people, so the well-worn story goes. In fact, they are so fundamentally ill that they can pass down their disease to the next generation of black children regardless of what the future social environment is like.

The echo of white supremacist eugenics here is loud, clear, and alarming. There is no way to sidestep this fact, and so it needs to be faced head-on. As the quantity and scope of epigenetic research increases, biological and health scientists need to be in critical dialogue with each other and with feminists, critical philosophers of race, and other scholars about how echoes of white supremacy and white privilege could be impacting their research. They also need to be mindful and vocal about how politicians, health specialists, academics, and others might take up and (mis) construe their work. There are no guarantees to be offered or found, no way to ensure that racist, sexist, and other pernicious stereotypes won’t fundamentally shape the “breakthrough” scientific era of epigenetics (Kennedy 2002, 2283; Baylin and Schuebel 2007, 548–49). But epigenetics does not have to be eschewed for that reason, and it indeed can be part of a critical race and feminist arsenal for combating white racism by fully comprehending the extent of white supremacy and white privilege. Simply put, whether we like it or not, the social often becomes durably and transgenerationally biological, and thus so do social injustices such as white domination. Epigenetics shows us one way that this happens.

Genomic Imprinting and Direct Epigenetic Inheritance

We can see this point again in the second general form of transgenerational epigenetic inheritance, which is genomic imprinting. Genomic imprinting is considered the more direct of the two indirect forms of
epigenetic inheritance because it is not dependent on uterine or post-natal environments. It nevertheless does not involve direct transmission of epigenetic attachments from parent to offspring. The epigenetic attachments in the offspring from its parent(s) are reestablished anew. What is distinctive about genomic imprinting is that this reiterative process happens during the development of sperm and eggs.

During the body’s process of making sperm or eggs and also shortly after fertilization of an egg by sperm, most epigenetic marks contributed by the female and male parents are erased, bestowing each zygote with a fresh set of naked DNA to clothe anew (Francis 2011, 86). This process is called epigenetic reprogramming, and it is why epigenetic processes once were thought to begin and end within a single lifetime. We now know, however, that the epigenetic slate isn’t always wiped clean during sperm and egg production. In the case of imprinted genes, the epigenetic marks (for example, degrees of methylation) are erased from the sperm or egg, but they subsequently are restored before the sperm or egg matures (Francis 2011, 111). This means that the methylation patterns inherited from the parent(s) are present at the point of fertilization. All genes then must undergo a second round of epigenetic reprogramming prior to implantation, but what is significant about imprinted genes is that their epigenetic attachments withstand this process. The result is that “by the time the embryo implants, imprinted genes are already epigenetically fixed in their expression pattern” (Francis 2011, 111).

The fact that imprinted genes survive epigenetic reprogramming can be good news since they are responsible for a great deal of crucial developmental work in the fetus prior to birth. But the situation is grimmer when the imprinted genes in question are linked to major health problems. Environmental toxins such as endocrine disruptors, for example, can have a significantly negative impact on imprinted genes (Francis 2011, 113–14; Thayer and Kuzawa 2011, 3–4). Chemicals such as polychlorinated biphenyls (PCBs) and bisphenol A, used in the production of clear durable plastics, and weed killers and fungicides, used in many lawn care products, can produce kidney disease and immune system problems and, in males, prostate cancer, abnormal testes, defective sperm, and low fertility. They do this by disrupting physiological processes that involve hormones, in this case by mimicking (and thus overdosing on) estrogen. What is especially alarming about endocrine disrupters is that their effects are transgenerational. Male rats that were exposed as fetuses to the fungicide vinclozolin
not only suffered from defective sperm and low fertility, but their male pups and grand pups did too even though they were not exposed to fungicides. The fungicide “alter[ed] the imprinting process during sperm development . . . not only alter[ing] normal imprints but establish[ing] new ones in parts of the genome that are not usually imprinted” (Francis 2011, 115). This imprinting pattern survived epigenetic reprogramming not just once, but for at least four generations of male offspring (Francis 2011, 115). Although the parent(s)’ methylation patterns had to be reconstructed in their offspring’s sperm or eggs, genomic imprinting offers a stunning example of the transgenerational reach of the physiological effects of social and environmental forces.

Perhaps even more powerful, however, is the third form of transgenerational epigenetic inheritance: the direct transmission of methylated genes from one generation to another, without any erasure (and thus no re-imprinting) of epigenetic attachments. Direct epigenetic inheritance is much more common for plants than for mammals. This is because the epigenetic reprogramming in plants is far less extensive or effective than it is in mammals (Francis 2011, 90). But there is compelling evidence of direct epigenetic inheritance in mammals, including human beings. One of the best cases comes from the methylated mice discussed above. The folic acid that the mother mice consumed not only changed their pups’ appearance and health via their experience in the womb. It also changed the pups’ epigenetic attachments independent of the uterine environment. When the fertilized eggs of yellow (severely undermethylated) mother mice were transferred to the wombs of dark (sufficiently methylated) mice, the offspring remained yellow. And when the offspring who had received folic acid in the womb and thus were darker colored became mothers themselves, their offspring (the “grand-pups”) also were darker colored even though they did not receive any methyl supplementation in utero (2011, 85). The upshot is that neither the methylation of the pups’ relevant genes nor the methylation of the grand-pups’ relevant genes was an effect of their own consumption of methyl donors while in the womb. The folic acid consumed by the mother mouse had a direct effect on the composition and activity of the genomes of both her children and her grandchildren. A particular methyl-DNA combination was inherited from her by at least two subsequent generations. This type of inheritance was not genetic because the DNA itself did not mutate. It is, in other words, an instance of physiological transgenerational inheritance on a non-genetic level.
In human beings, the study of true epigenetic inheritance is quite recent, but historical evidence from Sweden offers an interesting case study even though it is difficult to be certain whether it is an instance of genomic imprinting or true inheritance. An isolated Swedish population that keeps very accurate records of crop harvests (and thus calorie consumption per person) occasionally experienced severe famine in the nineteenth century, and scientists found that the grandsons of men (on their paternal side only) exposed to famine before they were adolescents experienced less cardiovascular disease than the grandsons whose paternal grandfathers did not experience famine as prepubescent boys (2011, 89). In other words, severe malnutrition undergone by one’s paternal grandfather meant better heart health for Swedish men.

There are two significant aspects of this case to underscore. First, the grandfathers did not experience famine while they were fetuses, and second, the cardiovascular health effects in question were not transmitted to Swedish men from their maternal grandfathers. The grandsons’ health effects thus cannot be attributed to the nutritional stress experienced by their great-grandmothers while they were pregnant, nor can it be attributed to the repetition of a similar environment (nutritional or otherwise) in the womb from one generation to another. Something more directly affecting the grandsons’ genome appears to have been passed down to them from their grandfathers via their fathers, presenting a strong case of possible true epigenetic inheritance in human beings. One admittedly cannot be 100 percent sure how direct the epigenetic inheritance was in this case. For example, it is possible that the relevant epigenetic markers were erased and re-imprinted during the maturation of the father’s sperm. But because (unlike women’s ovaries) men’s sperm matures during adolescence, we at least know that uterine epigenetic reprogramming was not involved. As other studies on the epigenetic effects of pre-pubescent boys’ smoking confirm, “there is a general [epigenetic] mechanism for transmitting information about the ancestral environment down the male line” (Cloud 2010). In the Swedish case, the DNA in the grandfathers’ sperm appears to have been epigenetically altered by the famine as the grandfathers went through puberty and—even more significantly—this alteration was maintained in their sons’ sperm which later contributed to the biochemical makeup of the grandsons.

Whether or not a case of true epigenetic inheritance, the Swedish famine offers a powerful example of a socio-historical event producing health
effects physiologically inherited at least two generations after the event. While the grandsons of the famine survivors did not directly experience the famine, it is as if their bodies nonetheless remember its hardships. And the same general point could be made for other instances of epigenetic inheritance, indirect and direct alike. As one pair of scientists has put it, epigenetic attachments can be thought of as “biological memories of past environments” (Thayer and Kuzawa 2011, 1). Knowledge of the Swedish famine persisted across generations even if this knowledge was buried in the chemical makeup of the genome, not consciously known but nevertheless expressed and experienced via a person’s cardiovascular health.

The nature of epigenetic memory can be surprising. Why would famine produce better health, not worse, in subsequent generations, for example? After all, in a different famine inflicted upon the Dutch by the Germans during WWII, people exposed to severe malnutrition in the womb during the first trimester experienced greater levels of heart disease and obesity while those exposed during the second trimester had more lung and kidney disease and those in the final trimester tended to suffer from glucose intolerance (Francis 2011, 4; ScienceDaily 2008). What accounts for these epigenetic differences? The answer to this and many other questions about epigenetics isn’t always clear, but what is evident is that, courtesy of our bodies, past social environments and situations can be vibrantly alive in the present. Epigenetics thus gives striking new meaning to William Faulkner’s (1951, 81) famous comment that “the past isn’t dead. It isn’t even past.” The past bodily lives on—is re-membered—in our epigenetic markers.

This can be a chilling thought given the years of white racism that mar the past of the United States (and many other countries). It means, for example, that the stress and trauma of Jim Crow continue to live even though Jim Crow formally ended in the 1960s. Its health effects likely persist physiologically, not only in the biochemistry of African American people who were adolescents in the 1950s, but also in the biochemistry of their children and grandchildren. Of course, ongoing racism after Jim Crow also can be blamed for contributing to the high rates of infant mortality and cardiovascular disease experienced by African Americans. But it is not just the racist present that is harming contemporary African Americans. It also is the racist past experienced by their ancestors. “Through epigenetics,” as sociologist Dorothy Roberts (2012, 143) argues, “the effects of racism on parents might be transmitted to their children, perpetuating inequalities across generations.”
If the past lives on epigenetically, then our present will become the past that endures in our children’s, and perhaps even their children’s biochemistry. The white racism that mars our society today will not necessarily stop there, in other words. It likely will impact the lives of several generations to come, physiologically and medically as well as economically, possibly through direct paternal as well as direct maternal lines. This implication of epigenetics is distressing. But there is an additional facet to this observation about the past, one that offers a measure of hope to those who wish to eradicate white racism. The effect of changes made today might not always be immediately apparent, but they can make a significant difference in the future. While a reduction in institutional and individual prejudice against adult people of color could improve their health by reducing stress levels, it is not likely to completely eliminate a person’s existing major health problems, such as cardiovascular disease, associated with years of enduring a heavy allostatic load. A severely weathered body does not rebound in this way; this is the bad news. (It also marks another moment to guard against racist stereotypes of the broken black person. The point here is the opportunity offered by the future, not that the present is unimportant or somehow “unsalvageable.”) Equitable health policies do help reduce existing racial health disparities (Lu et. al. 2010; Roberts 2012, 123–27, 144–45). Even if adult African American health were only marginally improved by reducing white racism, however, the good news is that such a reduction made today is likely to significantly benefit the health of their future children and grandchildren.

In particular, the persistence of the past and present in the future demands rethinking the scope and significance of prenatal care. Activities such as avoiding smoking and drinking alcohol and eating healthily and taking vitamins remain important, but we need to appreciate that “the benefits of prenatal care for improving birth outcomes may be more inter-generational than immediate” (Lu and Halkon 2003, 25). This means that what counts as prenatal care for women of color should be expanded trans-generationally. It’s not just a matter of nine months of healthy living and regularly visits to the obstetrician. The relevant issue also is that wide scale forms of racial justice are needed to close the racist gap in birth outcomes. Reflecting on the fact that African American women who had prenatal care still have higher rates of infant mortality than white women who received none, some health specialists thus have concluded that “to expect prenatal care, in less than 9 months, to reverse the impact of . . . cumulative
allostatic load on [African American] women’s reproductive health, may be expecting too much. . . . Prenatal care as currently prevails may do too little too late to have a major impact on [racial] disparities in birth outcomes” (Lu and Halfon 2003, 21). Seemingly nonmedical aspects of contemporary black people’s lives, such as community building and urban renewal, improved schools and educational opportunities, wage equality, and support for working mothers and families, are closely tied to the health and well-being of future generations of African Americans and thus should be considered a fundamental part of prenatal care, rather than incidental or unrelated to it (Lu et. al. 2010).

Conclusion: Beyond “Race as Bad Biology”

Human beings are composed through our transactions with our environments; the fresh insight provided by epigenetics is that those environments are not just contemporary but also historical ones (Kuzawa and Thayer 2011, 222). Social environments inhabited by one’s ancestors can constitute the person that one is today, and this can occur physiologically via inherited chemical and hormonal markers that affect the expression of one’s genes. The fight against white racism thus needs to be waged on biological and medical levels, as well as economic, political, aesthetic, and other social levels. Or rather, since no rigid lines separate the biological and the social, we could say that due to the existence of racist health disparities, the medical already is implicated in political struggles against white domination. By illuminating the transgenerational scope of white racism, epigenetics can be a useful ally in that fight.

Critical philosophers of race fall into a vicious trap when we assume or endorse a dichotomy between “‘race as biological’ (now out of favor) and ‘race as merely a social construction’” (Duster 2003, 272, emphasis in original). This false dilemma profoundly misunderstands the meaning and effects of social constructionism (2003, 263). As various sociologists, anthropologists, and social epidemiologists have documented in vivid empirical detail (Duster 2003; Gravlee 2009; Krieger 2005a; Krieger and Smith 2004; Ossorio and Duster 2005; Roberts 2012), the social and political world is literally incorporated into the muscles, hormones, fluids, tissues, and chemicals of human bodies. Not only does so-called social construction not mean that what is constructed is unreal, but it also does not mean that what is constructed is
nonbiological. My hunch is that the language of “social construction” too
easily lends itself to this trap, and thus it should be avoided. Far superior
is social epidemiologist Nancy Krieger’s (2005a) development of “embodi-
ment” as an ecosocial concept designating active organisms that physiologi-
cally incorporate their world through activity it.14 If the language of social
construction is retained, however—as it generally seems to be in the empir-
ical sciences—then we must be willing to speak of biology as social con-
structed. Not merely the discipline, but biological and physiological matter
itself: cortisol levels, allostatic loads, nerve growth factors, epigenetic mark-
ers, and so on. The inequalities of the social world are no different than,
for example, the food we eat and the air we breathe: they “become literally
embodied into physio-anatomic characteristics that influence health” and
other salient aspects of human existence (Krieger and Smith 2004, 92).

The social-versus-biological trap is not neutral or innocent. It mani-
fests an ideological position, popular with conservative foundations in the
United States, that tends to be unsupportive of social justice movements
(Krieger 2005b, 2155). By casting social justice concerns (such as the health
effects of racial discrimination) as politically correct but unscientific and
the pursuit of biological facts (especially regarding genes) as rigorous even
if politically incorrect science (2005b, 2155), the false dilemma between the
social and the biological serves the interests of white supremacy and white
privilege. In the name of defending a neutral, apolitical version of science,
the dilemma deceptively attempts to paint social justice concerns as ideo-
logically biased and thus as something for “real” science to stay well clear
of. This not only furthers a racialized epistemology of ignorance that makes
it difficult to understand the white dominated world in which we live (Mills,
C., 1997), but it also leaves in place staggering health disparities across
racial groups and does nothing to reduce the shockingly high levels of mor-
bidity and mortality for African American people in particular.

Which seems to be the point of the trap, one can say without too much
cynicism. Critical philosophers of race thus need to avoid it, and that means
shelving the well-worn mantra in philosophy (and elsewhere) that race is
not biological. We no longer can simply criticize the concept of “race as bad
biology” (Gravlee 2009, 48). We should acknowledge that race is biologi-
cal—not in the pre-critical sense of a static, essential category but in the
critical, dynamic way in which “race becomes biology” through the embodi-
ment of racial inequalities (2009, 54, emphasis in original; see also Roberts
2012, 129–30).15 Racial differences are physiologically, biologically real in
that white racism has fundamentally impacted the physical health and functioning of African American and other non-white people. (It also has fundamentally impacted—that is, unfairly benefitted—the physical health and functioning of white people, but that is a story for another time [see Sullivan 2014].) This acknowledgement is not a capitulation to white racism, but instead an important part of the struggle against it. Critical philosophy of race should be also a critical physiology of race.

NOTES

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1. Hispanic Americans and Asian Americans also saw their wealth inequality gap widen in comparison with white Americans; white families’ net worth now is fifteen times that of Hispanic families and twice that of Asian families (Luhby 2012).

2. Transgenerational inheritance also affects a person’s “invisible capital,” which includes the family connections and implicit knowledge that makes it easier to succeed on the job market or as an entrepreneur (Rabb 2010).

3. As a whole, Asian Americans also have better health outcomes than African Americans, Native Americans and Pacific Islanders, but differences within this group should be noted. For example, Vietnamese and Korean American women have some of the highest rates of cervical cancer and Vietnamese American men die from liver cancer at a rate seven times that of white men (Smedley et al. n.d., 2–3).

4. Most studies have been done in the United States, but increasingly research is being performed in Finland, Ireland, South Africa, and New Zealand that demonstrates similar links between racism and negative health effects (Drexler 2007).

5. As do Kuzawa and Sweet 2012, 10.

6. Weathering also is referred to as a cumulative pathway mechanism (Lu and Halfon 2003, 16–17).

7. Although Lu and Chen “found no significant interaction effects of race-ethnicity and stress on preterm birth” (2004, 698), the conclusion of their study was that the Pregnancy Risk Assessment Monitoring System (PRAMS) measures stress too narrowly. PRAMS includes only the stressful life events that occurred during the twelve months before delivery, and thus it “may not measure stress adequately, particularly those daily hassles, chronic stressors, or contextual factors that may be more pervasive in the lives of women of color” (698). It also should be noted that even when focusing only on the twelve months prior to delivery, “a modest effect between black race and traumatic stressors” was documented (691).

8. Feminist standpoint theory is well known for its examination of scientific objectivity; see, e.g., Harding 2001. See also chapter two of Mills, C., 1998.
9. Epigenetic attachments can survive epigenetic reprogramming in plants for hundreds of generations, making plants’ epigenetic inheritance almost as stable as their genetic inheritance (Francis 2011, 90).

10. Scientists also are investigating whether true epigenetic inheritance is involved in a certain form of colon cancer (Francis 2011, 97). For a review of epigenetic contributions to hypertension, see Mills, R., 2011.


12. For more on the challenges to investigating gene-environment interactions especially from the environment “side,” see Khoury and Wacholder 2008.

13. In a different but potentially related way, psychoanalysis provides a similar insight. Unfortunately it’s beyond the scope of this paper to explore connections between the two fields.

14. Krieger’s notion of embodiment strikes me as very similar to that of John Dewey’s pragmatist concept of transaction; on the latter, see Sullivan 2001.

15. Gravlee’s neglect of epigenetic processes tends to overemphasize genetic ones, however, as when he claims, “the embodiment of social inequality passes through biological systems regulated by genes” (2009, 54).

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