Race on Both Sides of the Razor

Terence D. Keel

Since at least the nineteenth century, the social constructionist critique of race in science has been built on the belief that human differences are the result of social rather than innate biological factors. Guided by this understanding, activists, scholars, and social scientists have identified the structural conditions that shape biological outcomes often mistaken as merely natural occurrences in the so-called human races. The social and historical conditions of Black enslavement and poverty, for example, are what Frederick Douglass had in mind when he wrote in 1854, "I think it will ever be found, that the well or ill condition of any part of mankind, will leave its mark on the physical as well as on the intellectual part of man."[1] When Franz Boas claimed at the start of the twentieth century that social processes produce biological outcomes, he argued that "the mental make-up of a certain type of man may be considerably influenced by his social and geographical environment."[2] In 1945, shortly after the synthesis of Darwinian evolution and modern genetics, Ashley Montagu wrote: "Differences of behavior and character as seem to exist between ethnic groups are due principally to inequalities in the opportunities for social and economic betterment which have been afforded them, not to unalterable inborn or hereditary differences."[3] To say that human variation is socially derived is to say that the so-called races are not natural groupings but result instead from the interaction of biology, social practices, and institutions that are perceived through socially constructed categories and patterns of reason inherited from the past.

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Governmental shifts toward closing the gap in racial health disparities and new forms of genetic analysis that emphasize difference over similarity have created an interesting set of challenges for the social constructionist position on race. Since the early 2000s, genetic research on health and behavioral differences has tended to naturalize human differences and erode the notion that "racial categories do not necessarily reflect biological categories."[4] Present-day scientists routinely draw correlations between continental ancestry (i.e., African, Asian, and European) and genetic traits in ways that often efface the social and political factors that also shape our differences. Dorothy Roberts has made this point, arguing: "Genomic science is reinforcing the concept of race as a biological category even as Americans ignore the devastating effects of racial inequality on our society."[5] The consequences are remarkably problematic: scientists have been unearthing biological differences without linking these variations to past legacies of social and political discrimination known to have biochemical, neurological, and epigenetic consequences.[6]

The proliferation of studies declaring that there is a genetic basis to health disparities and behavioral differences across the so-called races has encouraged the opponents of social constructionism to assert a victory for scientific progress over political correctness.[7] I am not concerned in this essay with providing a response to critics who believe races are expressions of innate genetic or biological differences. Instead, I am interested in how genetic research on human differences has divided social constructionists over whether the race concept in science can be used for social justice and redressing embodied forms of discrimination.

On one side, there is the position that race is an inherently flawed concept and that its continued use by scientists, medical professionals, and even social activists keeps alive the notion that it has a biological basis.[8] Dorothy Roberts, Michael Yudell, Sarah Tishkoff, and Rob DeSalle have recently called for the complete elimination of race in genetic research. They argue:

> Phasing out racial terminology in biological sciences would send an important message to scientists and the public alike: Historical racial categories that are treated as natural and infused with notions of superiority and inferiority have no place in biology.[9]

Similar calls have been made for eliminating race within medical practice, where researchers and practitioners often revert to overly simplistic ideas about human difference to study, diagnose, and deliver care to patients.[10] Those who oppose the use of race in science argue that discrimination remains a possibility when pragmatism reduces the complexity of our biohistories to highly flawed common-sense notions of race.

On the other side of this debate are those who maintain a social constructionist position yet argue that not all instances of race in science stem from dis-

criminatory politics or the desire to prove that humans belong to discrete biological units that can then be classified as superior or inferior. Proponents of this position note that social actors, politically progressive geneticists, and a public increasingly interested in the social history of their own genetic ancestry have demonstrated new forms of engagement around race and science that cannot be reduced to eugenics, social Darwinism, or other deleterious ideologies. Alondra Nelson argues in *The Social Life of DNA* that "concerns about racial reification and genetic determinism, while well warranted," fail to capture "the myriad purposes to which genetic ancestry is being put."[11] Nelson has observed the powerful social utility of race in science in African Americans' attempt to reconcile their diasporic identities with the trauma of slavery, establish meaningful ties with Africa, and make claims for reparations. Nelson argues that in these endeavors "genetic analysis is used to contribute to community cohesion, collective memory [and] social transformation."[12]

Additionally, John Hartigan has argued that the social constructionist view of race has been overly concerned with "assailing ideological notions that racial identities are both legible and fixed by our biology."[13] He notes, "The inexorable intermingling of culture and biology is not strictly encompassed by such ideological uses or interests."[14] Drawing on his study of geneticists working in Mexico to identify a common national genetic structure—which has involved "racing" both humans and nonhumans—Hartigan sees that "racial thinking does not strictly or perhaps even primarily concern itself with crafting and contemplating natural objects with which it then strives to affirm or reproduce an existing, hierarchical social order."[15]

Michael Montoya has made similar claims about social constructionists concerned almost exclusively with the ideological and discriminatory implications of race in science. In *Making the Mexican Diabetic*, he argues:

> Critics of race in medicine make present-day predictions of future social consequences based upon past abuses of race in science and medicine. Thus, the critiques of race in science on the grounds that it rebiologizes race imputes a power to "science" it does not have. The racialization and the pernicious effects of claiming that groups are biologically different are a function of racial discursive formation.[16]

Montoya contends that pernicious racial formations do not necessarily accompany scientific projects that deploy racial categories.

Those who see the value of race in science argue that new ideas about human difference are being developed that cannot be reduced to the racism of the past. Moreover, they believe that geneticists who use social categories are not necessarily driven by dangerous ideological commitments and that some biomedical researchers are capable of understanding the social and cultural factors shaping

genetic variation within and across social groups. This capacity to account for the social dimension of our local biology while using racial categories suggests that new forms of biomedical knowledge are possible. In return, scientific research on human differences might be able to redress previous legacies of discrimination and injustice.

It seems to me that part of this debate is a dispute over whether scientists can use the race concept, while also remaining committed to measuring and quantifying the social factors that shape human biology, in socially responsible ways. Presumably these findings would be integrated into claims about the sources of health and behavioral differences that could then transform our ideas about why humans vary without falling victim to the racism of the past. Those who oppose race in science doubt that this is possible, while those who support it appear more optimistic about the knowledge that can be obtained using racial categories. As Troy Duster has noted, "When race is used as a stratifying practice (which can be apprehended empirically and systematically), there is often a reciprocal interplay of biological outcomes that makes it impossible to disentangle the biological from the social."[17] Surely the social systems and practices we create have biological consequences for our own lives and those of our descendants. But the resistance of scientists, let alone the public, to consistently recognize this when it comes to race points to deeper problems tied to the forms of belief and knowing that we have inherited from the past.

I would like to shift this debate away from the question of whether race is real and move instead toward thinking about the intellectual commitments necessary for science to expose past legacies of discrimination. I believe social constructionists might be overestimating the degree to which scientists, and those who study them, think that human development (race) is driven by social practices and institutions. Perhaps the difficulty of distinguishing social causes from genetic ones is tied to one's beliefs about what moves human history. How can science reveal embodied forms of discrimination and engender conversation about sociopolitical accountability if researchers are not committed in principle to the belief that social factors largely shape biology and understandings of what it means to be human? In this sense, the issue of whether race in science can be used for social justice is not simply a question of politics but also one of epistemology and belief. This, I believe, is an issue that has gotten lost in our dispute over if and how race should be deployed in scientific research.

Indeed, I contend that the history of race in Euro-American science has transmitted reasoning strategies that prioritize and define human genetics in ways that efface the social factors that contribute to our differences. As I have written elsewhere, this reduction of "the social" has everything to do with the fact that early modern biology grew out of a Christian intellectual heritage that divinized "Nature" and reduced the role that human practices and institutions were believed to play in shaping our health, behavior, and bodily forms.[18]

The post-Enlightenment architects of today's racial categories fashioned theories of race that favored Nature over social life when it came to explaining the origins of the races. While these early modern biologists acknowledged to a certain degree that climate or culture changed the body, they did not believe humans were the protagonists of this change. Instead, humans were understood to be passive actors undergoing Nature's scripted possibilities. For example, Johann Blumenbach, the eighteenth-century German physician who first used "Caucasian" as a scientific term, claimed that racial varieties were the result of the environment diverting the "formative drive" (*Bildungstrieb*) embedded in the human body.[19] Likewise, Immanuel Kant recognized that nutrition and social status could create short-lived changes in human biology.[20] However, the true causes of difference were "numerous seeds and natural predispositions," which "must lie ready in human beings either to be developed or held back."[21] For Kant, the environment produced "a long-lasting development on [these] seeds and predispositions."[22]

Early modern biologists did not believe humans created race. They believed Nature did. Scientists merely observed and classified Nature's creation according to the most useful racial categories. The legacy of this intellectual history explains why Nicholas Wade could proclaim in his 2014 book *A Troublesome Inheritance* that "it would be of the greatest interest to know how people have evolved in recent times and to reconstruct the fingerprints of natural selection as it molded and reworked the genetic clay."[23] It is no accident that "natural selection" is the personified subject of Wade's biological history. Moreover, the belief that Nature creates race is at work when we reduce the role human agency plays in shaping the body and instead discuss genetic drift, founder effects, and mutations as if these factors did not depend on our ancient ancestors making decisions to travel, flourish, and make families under specific social, historical, cultural, and environmental conditions.[24]

Indeed, the suggestion that human differences stem from social life and not Nature was radical in the nineteenth century precisely because social constructionism placed human agency as the driving force in history. This had the effect of unseating the divinized conception of biology that had so long tipped the scales of human history on the side of Nature when scientists attempted to explain so-called differences between the races. We must keep in mind that Frederick Douglass defended an early form of social constructionism against ethnologists who believed that racial differences were unalterable because they were created by God, or God's proxy Nature, rather than humans.[25] Moreover, Boas's study of human heads and the assumed fixed intelligence of Eastern European immigrants during the early twentieth century was aimed at dislodging a conception of inherited biology as a deterministic force impervious to changes from varying social settings.[26]

We continue to grapple with this intellectual history. Having socially trans-

formative political commitments, or being a descendant of a historically marginalized community, does not make one exempt from the costs of an intellectual inheritance that predisposes scientific analysis to pursue forms of inquiry that reduce and efface the social causes of human differences. God in Nature still haunts contemporary perceptions of race and human biology. This is true even for scientific research driven by transformative and progressive political commitments.

A recent and unlikely genetic study provides a case in point. Geneticists from the Slim Initiative in Genomic Medicine for the Americas (SIGMA) Type 2 Diabetes Consortium, the product of a partnership between the Carlos Slim Foundation and the Broad Institute, have been interested in understanding why "Mexican and other Latin American populations" develop type 2 diabetes at nearly twice the rate as that of "US non-Hispanic whites."[27] Inclusive and progressive political commitments appear to sit at the center of this collaboration. According to SIGMA's mission statement, the Foundation and the Institute came together in order to "make sure Latin Americans benefit from the genomic revolution."[28] To achieve this goal, they intend to promote "wider access to genomic medicine in Mexico and Latin America" through research programs that focus on health problems specific to the region. They also look to enhance the genomic research capacity in Mexico through scientific training and the promotion of "genomic diagnostics and therapeutics in Latin America."[29]

Looking for disease risk alleles that were common in Mexican and other Latin American populations—and rare in US whites—the SIGMA consortium analyzed 9.2 million single nucleotide polymorphisms (SNPs) in each of the 8,214 Latin Americans in their study. Within this group, 3,848 had type 2 diabetes, while the remaining 4,366 did not. Researchers discovered a genetic variant identified as SLC16A11 that was associated with an elevated risk for diabetes. This risk gene appeared most often in Native American populations (50 percent frequency), with the next highest frequency (10 percent) in East Asian groups. The gene was rare in Europeans and Africans.[30]

Messenger RNA from the SLC16A11 gene is expressed in the liver. These molecules carry information from DNA into the cells of the body, providing vital instructions on how to function. Scientists found that SLC16A11 alters how lipids are metabolized and increases the production of triacylglycerol, which generates triglycerides. When stored in the body at high levels, triglycerides put individuals at risk for type 2 diabetes, heart disease, stroke, and nerve damage.[31]

Geneticists found that people who carry the SLC16A11 genetic variant were 25 percent more likely to have type 2 diabetes than those who did not carry the gene. Mexicans and other Latin Americans who inherited this gene from both parents were 50 percent more likely to be diabetic. According to the study, this means that the genetic predisposition to type 2 diabetes is being passed along to Latin Americans as a result of their Native American (indigenous) ancestry.

In attempting to locate the origins of this risk allele, SIGMA geneticists made a discovery that led them to a remarkable conclusion. The SLC16A11 variant is believed to have originated in a common ancestor roughly 800,000 years ago. This would have been well before the major migration of humans out of Africa, which is estimated to have happened between 90,000 and 100,000 years ago. With the SLC16A11 variant being absent in Africa and rare among Europeans, SIGMA geneticists concluded that this variant could have come from human mixing with Neanderthals.[32] Confidence for this conclusion grew after geneticists analyzed the unpublished genome of a Neanderthal and found this archaic human also in possession of the SLC16A11 variant.[33]

Geneticists noted that they did not discover a direct causal relationship between this Neanderthal gene and diabetes. They also claimed that this variant should be understood as one of many factors contributing to disease outcomes in Mexicans and other Latin Americans. Researchers went on to conclude that this gene would have been missed had it not been for their conscious effort to look outside the European genome, where SLC16A11 was rare. By decentering Europe, researchers produced one of the first studies "providing an example of Neanderthal admixture affecting physiology and disease susceptibility today."[34]

The SIGMA study is merely one among many recent efforts undertaken in the past two decades to locate the genetic basis of diseases thought to disproportionally afflict people of color. In the United States, this focus is the logical consequence of a shift in government priorities ushered in by the Clinton administration. In 1993, under President Bill Clinton, the US government set into law the National Institutes of Health Revitalization Act, which mandated that all NIH-funded research include women and minorities among the subjects of study.[35] The spirit of the act was to increase scientific and medical knowledge about the causes of high disease rates facing women and minorities. The Revitalization Act was a long-awaited response to the landmark 1985 "Malone-Heckler Report," which captured the gross disparity in the excess death rates facing Black and other minority groups when compared to whites.[36] In the report, researchers found that nearly half the deaths of Black people under the age of seventy could have been avoided if more research, preventative care, and treatment had been available. The Revitalization Act of 1993 provided incentives for a range of scientific and medical researchers to use race as an analytic tool to better understand health outcomes and incidences of disease. By the late 1990s, the federal government had helped make the scientific study of race a matter of national interest and a market opportunity for private investors and the pharmaceutical industry.[37]

This increased attention to race was intensified shortly after the sequencing of the human genome in 2000. Although it was declared that humans share 99.9 percent of the same genetic information, geneticists developed tools that allowed them to "molecularize race" by identifying SNPs that place people into

one of the four major continental populations (African, Asian, European, and Native American).[38] As the anthropologist Duana Fullwiley has noted, by 2003 a vocal group of scientists, many of whom were racial minorities, were convinced that there was a genetic basis for racial disparities in health.[39] The commitment of these scientists reflected the spirit of the Revitalization Act, as they actively looked to design research projects that studied underrepresented groups with the intention of redressing past legacies of exclusion from scientific research. Their ultimate goal was to uncover the genetic basis of present-day disease outcomes in minorities.[40]

I will leave it to others to contest the technical findings of the SIGMA consortium. I would like, however, to make a few observations and raise some questions about the disquieting suggestion that Neanderthal genes put Mexicans and other Latin Americans at risk for type 2 diabetes as this relates to implied beliefs about the social or biological drivers of human development.

If we understand disease risk to be a product of social life—with genes as the canvas that records, embodies, and transmits these social processes over time—then Neanderthal variants are useless and dangerous for illuminating disease risk in Mexicans and other Latin Americans. This is why: Understanding the connections among disease expression, genetics, and race ought to involve uncovering the social and environmental processes that led to the manifestation of this allele, its rise in frequency, its transmission over time, and its causal connection to disease outcomes. In the case of the Neanderthal we are talking about orders of time in the tens of thousands of years. These scales of time make it impossible to recover with any nuance or certainty the lived social experience of those Neanderthal individuals who struggled with illnesses or changes to their diet and passed along these experiences in the form of presumed risk alleles to humans during the past forty thousand years. We cannot recreate the vital social history that accompanied the perceived changes in allele frequencies that are now believed to increase disease risk in Latin Americans.

Social constructionism commits us to saying more than that racial science is political ideology or eugenics by other means. Social constructionism commits us to a way of knowing that affirms that human ideas and practices fundamentally shape and make intelligible our biological outcomes. Recognizing this requires that we dedicate ourselves, much like Douglass, Boas, and Montagu, to renouncing ideas, patterns of reasoning, and beliefs that diminish or negate the role of the human hand in producing biological outcomes and making them intelligible.

If we are to be consistent in our social constructionism, we might ask: Are there then temporal limits to how far back we can reasonably trace the genetic basis of disease risk while using race as an analytic tool? This is to say, how useful or meaningful is information about inherited risk alleles without the corresponding knowledge of the social, cultural, environmental and historical condi-

tions under which our ancestors lived? It seems to me that in the absence of this knowledge we are left to say that a gene with no clear social history is responsible for why groups now living are predisposed to disease—a line of reasoning of the same species as Blumenbach's formative drive and Kant's "numerous seeds and natural predispositions." The scientific pursuit of said genes may stem from progressive commitments, but devaluing the lived experiences of those who carried these genes surely does not.

NOTES

1. Frederick Douglass, "The Claims of the Negro Ethnologically Considered: Address Delivered at Western Reserve College, July 12, 1854," in *Frederick Douglass: Selected Speeches and Writings*, ed. Philip S. Foner (Chicago: Lawrence Hill Books, 1999), 294.

2. Franz Boas, "Instability of Human Types," in *The Idea of Race*, ed. Robert Bernasconi and Tommy L. Lott (1911; repr. Indianapolis: Hackett Publishing, 2000), 88.

3. Ashley Montagu, *Man's Most Dangerous Myth: The Fallacy of Race* (New York: Columbia University Press, 1945), 240.

4. American Sociological Association, "Statement of the American Sociological Association on the Importance of Collecting Data and Doing Social Scientific Research on Race," 2003, http://www.asanet.org/sites/default/files/savvy/images/press/docs/pdf/asa_race_statement.pdf.

5. Dorothy Roberts, *Fatal Invention: How Science, Politics, and Big Business Re-create Race in the Twenty-First Century* (New York: New Press, 2011), xii.

6. For epidemiological work explaining how "the social" is embodied in health outcomes, see Nancy Krieger, "The Science and Epidemiology of Racism and Health: Racial/Ethnic Categories, Biological Expressions of Racism, and the Embodiment of Inequality—An Ecosocial Perspective," in *What's the Use of Race? Modern Governance and the Biology of Difference*, ed. Ian Whitmarsh and David S. Jones (Cambridge, MA: MIT Press, 2010), 225–258.

7. See Michael Levin, *Why Race Matters* (Oakton, VA: New Century Foundation, 2016); Richard Lynn, *Race Differences in Intelligence: An Evolutionary Analysis* (Augusta, GA: Washington Summit Publishers, 2015); Nicholas Wade, *A Troublesome Inheritance: Genes, Race and Human History* (New York: Penguin Press, 2014); David Reich, "How to Talk about Race and Genetics," *New York Times*, March 30, 2018.

8. Joseph Graves, "Great Is Their Sin: Biological Determinism in the Age of Genomics," *Annals of the American Academy of Political and Social Science* 661, no. 1 (2015): 24–50; Matthew Hughey and W. Carson Byrd, "Beautiful Melodies Telling Me Terrible Things: The Future of Race and Genetics for Scholars and Policy Makers," *Annals of the American Academy of Political and Social Science* 661, no. 1 (2015): 238–258; Catherine Bliss, "Science and Struggle: Emerging Forms of Race and Activism in the Genomic Era," *Annals of the American Academy of Political and Social Science* 661, no. 1 (2015): 86–108; Pilar Ossorio and Troy Duster, "Race and Genetics: Controversies in Biomedical, Behavioral, and Forensic Sciences," *American Psychologist* 60, no. 1 (2005): 115–128; Nancy Krieger, "Refiguring 'Race': Epidemiology, Racialized Biology, and Biological Expressions of Race Relations," *International Journal of Health Services* 30, no. 1 (2000): 211–216. See also an earlier debate among social scientists: American Social Science Research Council, "Is Race Real?," http://raceandgenomics.ssrc.org/, accessed August 8, 2016.

9. Michael Yudell, Dorothy Roberts, Rob DeSalle, and Sarah Tishkoff, "Taking Race Out of Human Genetics," *Science* 351, no. 6273 (2016): 564–565.

10. Lundy Braun et al., "Racial Categories in Medical Practice: How Useful Are They?," *PLoS Medicine* 4, no. 9 (2007): e271, doi:10.1371/journal.pmed.0040271; Robert Schwartz, "Racial Profiling in Medical Research," *New England Journal of Medicine* 344, no. 18 (2001): 1392–1393, doi: 10.1056/nejm200105033441810.

11. Alondra Nelson, *The Social Life of DNA: Race, Reparations, and Reconciliation after the Genome* (Boston: Beacon Press, 2016), 17.

12. Ibid., 8.

13. John Hartigan Jr., "Mexican Genomics and the Roots of Racial Thinking," *Cultural Anthropology* 28, no. 3 (2013): 372.

14. Ibid., 373.

15. Ibid., 386.

16. Michael J. Montoya, *Making the Mexican Diabetic: Race, Science, and the Genetics of Inequality* (Berkeley: University of California Press, 2011), 67.

17. Troy Duster, "Buried Alive: The Concept of Race in Science," in *Genetic Nature/Culture: Anthropology and Science beyond the Two-Culture Divide*, ed. Alan H. Goodman, Deborah Heath, and M. Susan Lindee (Berkeley: University of California Press, 2003), 258–277.

18. Terence D. Keel, "Religion, Polygenism, and the Early Science of Human Origins," *History of the Human Sciences* 26, no. 2 (2013): 3–32.

19. Johann Friedrich Blumenbach, *On the Natural Varieties of Mankind*, trans. Thomas Bendyshe (1795; repr. London: Longman, Green, Longman, Roberts, and Green, 1865), 196.

20. Immanuel Kant, "Of the Different Human Races," in *The Idea of Race*, ed. Robert Bernasconi and Tommy L. Lott (Indianapolis: Hackett Publishing, 2000), 14.

21. Ibid.

22. Ibid., 15.

23. Nicholas Wade, *A Troublesome Inheritance*, 1.

24. *Genetic drift* and *founder effects* are concepts used by population geneticists to study human developmental history. Genetic drift describes when the frequency of alleles in a population randomly changes as a result of a sampling error reproduced across multiple generations. An allele is one possible form that a gene might take. Most genes have a dominant and a recessive allele (or form). Population geneticists are interested in how often a specific allele appears within human groups, since this allows them to track the prevalence of a physical or health trait within large groups of people. Genetic drift is one of many concepts used to account for the distribution of alleles in human groups. It is worth noting that many human traits are not controlled by a single allele but instead by multiple genes interacting with the environment. The founder effect describes the reduction of genetic variation within a population. This loss of diversity occurs when a new group of people is born from a small number of ancestors. Population geneticists believe that new groups have greater biological success when their children have a wide diversity of alleles to choose from during the genetic recombination process that happens when two parents give alleles to their descendants.

25. Douglass, "The Claims of the Negro Ethnologically Considered."

26. Boas, "Instability of Human Types."

27. SIGMA Type 2 Diabetes Consortium, "Sequence Variants in SLC16A11 Are a Common Risk Factor for Type 2 Diabetes in Mexico," *Nature* 506, no. 7486 (2014): 97.

28. Broad Institute, "Carlos Slim Center for Health Research at the Broad Institute," https://www.broadinstitute.org/scientific-community/science/programs/carlos-slim-center-health-research/carlos-slim-center-health, accessed March 15, 2014.

29. Broad Institute, "Carlos Slim Center for Health Research at the Broad Institute," https://www.broadinstitute.org/sigma, accessed March 26, 2018.

30. SIGMA Type 2 Diabetes Consortium, "Sequence Variants in SLC16A11," 99.

31. Ibid., 100.

32. Ibid., 99.

33. Ibid.

34. Ibid., 100. It is worth noting that David Reich and Nick Patterson, who were members of the SIGMA Type 2 Diabetes Consortium, were also lead geneticists involved in the groundbreaking sequencing of the Neanderthal genome in 2010. See Richard E. Green et al., "A Draft Sequence of the Neanderthal Genome," *Science* 328, no. 5979 (2010): 710–722.

35. Steven Epstein, *Inclusion: The Politics of Difference in Medical Research* (Chicago: University of Chicago Press, 2007), 80–83.

36. US Department of Health and Human Services, *Report of the Secretary's Task Force on*

Black and Minority Health, Volume 1: Executive Summary, Washington, DC: US Government Printing Office, August 1985.

37. Epstein, *Inclusion*, 82; Jonathan Khan, *Race in a Bottle: The Story of BiDil and Racialized Medicine in a Post-genomic Age* (New York: Columbia University Press, 2012).

38. Duana Fullwiley, "The Biologistical Construction of Race: 'Admixture' Technology and the New Genetic Medicine," *Social Studies of Science* 38, no. 5 (2008): 695–735; Duana Fullwiley, "The Molecularization of Race: Institutionalizing Human Difference in Pharmacogenetics Practice," *Science as Culture* 16, no. 1 (2007): 1–30.

39. Duana Fullwiley, "'Contemporary Synthesis': When Politically Inclusive Genomic Science Relies on Biological Notions of Race," *Isis* 105, no. 4 (2014): 803–814.

40. Ibid., 804.