

The Interaction of Race, Human Variation, and Health[☆]

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'Race' and Health Inequalities: Two Causal Pathways	1
The Raciogenetic Perspective	1
The Lived Experience of Racialization and Racism	2
Why 'Race' ≠ Human Genetic Variation	2
The Idea of Race versus Evolution	3
The Structure of Human Variation	3
Human variation is continuous	3
Human traits vary independently from each other	3
Within-race-group genetic variation is much greater than variation among 'races'	3
Race: An Unrepeatable Explanatory Variable	3
Race: Conflating Lived Experience and Genetics	3
Conflating Human Genetic Variation and Race	4
Conclusions	4
References	4

'Race' and Health Inequalities: Two Causal Pathways

In the United States, where data on 'race' are routinely collected, researchers continue to find consistent, persistent, and usually marked disparity in nearly every indicator of wealth and health by race (Kochanek et al., 2013; Olshansky et al., 2012; Smedley et al., 2003). While there are some counterfactual findings and race-based health disparities that may be less glaring than in other countries, the overall pattern is clear. On nearly every indicator of health, whites in the United States do best and, conversely, African Americans do worst (Kochanek et al., 2013; Sacher et al., 2005).

Why is it that such differences in health and disease persist among races? For well over a century, occasional debates – or, more accurately, parallel arguments – have continued in scientific literatures and among various publics. Two arguments predominate: In one argument, racial differences in health are traced back to the evolutionary development of racial differences in genetics. This 'racigenetics' perspective accepts that race is a viable substitute or shorthand for genetic variation between populations and such genetic differences are assumed to be salient causes of disease as well as other racial differences.

In the more recent counterargument, race-based health disparities are causally traced to variation in the 'lived experience' of those assigned to different racial categories. Here, lived experience refers to the totality of everyday conditions that are embedded into the fabric of social, personal, and institutional relationships. Some of these experiences are rather subtle personal interactions that communicate values based on phenotypic appearances, and others still are more deeply and profoundly personal experiences of racism (Krieger et al., 2011).

I argue that the racigenetic explanation is both epistemologically flawed and counter to scientific facts because (1) genetics accounts for only a small fraction of the variation in the complex diseases of interest and, most importantly, (2) human genetic variation is extremely large within racial groups and does not easily map onto race as it is typically defined. Furthermore, in an age characterized by the celebration of genetic medicine, a compromise position in which both causal mechanisms are seen as valid is likely to have the consequence of overstating the role of genetics (Goodman, 2000; Sankar et al., 2004).

In the following, I first outline the racigenetic argument and then the lived experience/racism argument. I then present reasons why equating race with human genetic variation is epistemologically and scientifically flawed. I end with a critique of the compromise position and a statement of the need to clearly determine what we mean by race when it is used in public health research and practice.

The Raciogenetic Perspective

The perspective or worldview that racial differences in health are due to natural or inborn factors is an old one that extends back to at least the early nineteenth century. In the earliest (pre-Darwinian) versions of this worldview, by virtue of either assumed separate creation or separate evolutionary histories, whites were believed to be endowed with natural abilities to resist those diseases that were characteristic of 'civilization.' Frederick Hoffman (1896), for example, published a wealth of data on race differences in health in the United States. His influential treatise suggested that the increased morbidity and mortality of African Americans, especially in

[☆]Change History: March 2015. AH Goodman updated the text and Further Readings to this entire article.

northern US cities, resulted from their collective, inborn inabilities to survive the rigors of the contemporary world. Hoffman predicted their eventual demise.

At the time Hoffman was writing, many physicians felt that races were differentially susceptible to disease, and therefore, particular races were more likely to suffer from particular diseases. So, for example, once sickle cell disease was identified in African Americans, it was assumed to be a race-specific disease (Tapper, 1999; Wailoo, 1997). When Europeans began to present with symptoms of sickle cell anemia, it was assumed by physicians in the 1920s and 1930s that they must be part 'negro.' The possibility that sickle cell had nothing to do with race, but much to do with evolution and genetics, was not considered until the middle of the twentieth century (Livingstone, 1958).

In the twenty-first century, the idea that germs obey the color line or that diseases are specific to one race or the other has gone out of vogue. But the remnants of geno-racial determinism persist in current ideas that races are quasi-scientific units with separate disease susceptibilities. In its most common formulation, such ideas define race as a quantifiable risk factor for disease. Indeed, raciogenetics persists as a dominant explanation not only for variation in conditions such as sickle cell anemia that are caused by a single gene variant but also for variation in complex metabolic conditions such as diabetes and heart disease (Goodman, 1997 and Sankar et al., 2004). In 2005, for example, the US Food and Drug Administration approved the medication BiDil for use in African Americans, presumably because it was effective in combating congestive heart failure in this group for reasons that were thought to be intrinsic to that group (Temple and Stockbridge, 2007).

Finally, it is clear that we live in an age of genetics, a time in which genetics has taken hold as the dominant explanation for most behaviors and conditions. Certainly, genetic medicine is big business. And so it follows that genetics might explain variation in health among races (Goodman et al., 2003).

The Lived Experience of Racialization and Racism

In part due to the emergence of social epidemiology in the later part of the twentieth century (Krieger, 2001), the impact of dietary habits, stress, pollutants, work conditions, and other aspects of daily life on health is clearer to both professional and various publics. Part of the work in this field has pointed toward how lifelong differences in living conditions might explain the bulk of the variation in health among races (see, e.g., Geronimus, 1992; Williams et al., 1997). Social class and racism, for example, are closely intertwined, and both interact to affect life courses, determining exposure to, among other things, healthy foods, pollutants, and experiences of discrimination and rendering people differentially vulnerable to exogenous risks (Krieger, 2003).

In some sense, the lived experience hypothesis is so strong and obvious and fits the data so well that even those who strongly back genetic explanations acknowledge the importance of lived experience (Satel, 2002). Yet, for reasons noted later, the tendency is not to drop a genetic explanation entirely, but to acknowledge a role for both lived experience and raciogenetics (Satel, 2002). The problem with this compromise position is that it says little about underlying etiology. Ultimately, if one wishes to address health inequities, the relative importance of these distinct etiologic pathways must be determined. Given the ascendancy of genetics in health sciences research, it is often assumed, even in the absence of empirical evidence, that somehow genetic factors are more prominent as determinants of health than those hard-to-measure lifestyle or social experience.

The debate among the competing causes persists, and it is both scientific and political in the sense that each hypothesis points toward a series of actions that have political and ethical implications. In the following section, I present a number of reasons why race should not be used as shorthand for human genetic variation. If race maps poorly onto genetic variation, then the raciogenetic explanation is scientifically flawed.

Why 'Race' ≠ Human Genetic Variation

Race is a powerful idea and a worldview that was invented and reified to explain variation in human biology, culture, and behavior (Smedley, 1999). The underpinnings of this idea can be traced to classic Greek philosophical notions of ideal types and Christian ideas about a great chain of being. However, in the view of most historians of race and slavery, the idea of biologically based human races was itself a more recent invention (Smedley, 1999). With the development of ocean travel and international migration, human differences were magnified. Colonial factors, particularly the desire to exploit lands and people, may have also contributed to the tendency toward value-laden, racialized thinking. Starting in the eighteenth century, natural historians such as Linnaeus began to classify humans into subspecies or races. These classifications persist today.

However, we now know that the raciogenetics

- is antithetical to the idea of evolution,
- does not fit the measurable reality of the structure of human variation,
- does not translate into a concept that is epidemiologically repeatable, and
- leads to a series of confluents that inhibit understanding of disease cause, treatment, and prevention.

The Idea of Race versus Evolution

Race, as noted earlier, is largely a socially constructed idea about how human genetic variation is structured. It is, in fact, an idea that should have been cast aside with the development of evolutionary thinking (Goodman, 1997). What is perhaps most problematic about the idea of race is that it is not a process; rather, it is cast as a thing or an end result of a process. To say that race differences exist because of race is a tautology. There are in these explanations no explicitly theorized arguments relating to the processes by which race differences came into being. Yet we know now that genetic variation arose and is a result of human evolution and history. The idea of race, of stable and unchanging types, inhibits rather than advances studies of the evolution of human differences.

The Structure of Human Variation

If the idea of race – dividing humans into some three or more racial groups – approximated in a useful way the geographic structure of human variation, then one might support the notion that race is an imperfect but acceptable stand-in for human genetic variation. So framed, the association of place and genetic variation does not explain everything, but it is a sort of ‘quick and dirty’ approximation (Satel, 2002). This position may have been defensible prior to the application of modern genetics to human evolutionary studies. However, it is not defensible now for the following rules of human variation.

Human variation is continuous

Allelic and phenotypic frequencies tend to vary gradually across human populations. Definitions of race as a discontinuous category, reflecting clear ‘breaks,’ are thus conceptually flawed: It is impossible to identify where one race begins and another ends. Skin color, for example, varies widely by latitude and degree of exposure to ultraviolet. Since Africa covers such a wide span of latitude, it is reasonable that African groups exhibit a wide range of skin colors that overlap tremendously with individuals from other continents (Jablonski, 2012).

Human traits vary independently from each other

Traits tend to vary independently of other traits. Race categories will therefore vary by the traits used to classify. A classification based on sickle cell trait might include equatorial Africans, Greeks, and Turks, while another classification based on lactase enzyme deficiency might include eastern and southern Africans with southern Europeans, Japanese, and Native Americans. There is no possibility for consistency. As skin color is only correlated with a few other phenotypic traits such as hair and eye color, it is true to say that ‘race is only skin deep’ (Diamond, 1994).

Within-race-group genetic variation is much greater than variation among ‘races’

Starting with Lewontin (1972), studies have statistically apportioned variation in different genetic systems to different levels: among ‘races’ and within ‘races’ and smaller populations such as the Hopi, the Ainu, and the Irish. Lewontin collected data on blood group polymorphisms in different groups and races. He found that blood group variation between races statistically explains only about 6% of the total variation. These results show that if one is to adopt a racial paradigm, one must acknowledge that race will statistically explain only a small proportion of genetic variation. Moreover, this small variation is better explained by geographic distance (Templeton, 1998).

Yu et al. (2002) more recently compared a large sequence of DNA, 25 000 letters or base pairs long, of ten individuals from each of the three main ‘races’ typically used in medical studies: Asian, European, and African. They counted out the number of differences between any two individuals and found that the average number of differences between any two individuals from Africa was greater than the average number of differences between an African and a European or Asian. These results support the understanding that there is greater genetic variation in Africa because of the increased evolutionary time humans have spent in Africa. Most startling perhaps is that Europeans and Asians, rather than being genetically separable, appear more accurately to be subsets of Africans. We truly are, it seems, all Africans.

Race: An Unrepeatable Explanatory Variable

Race is impossible to define in a stable and universal way because ‘race as biology’ varies with place and time, and the socially determined color line is even more dynamic.

Other continuous variables such as head and foot size are classified into hat sizes and shoe sizes, and these systems work. A problem with race in practice is that there is no agreed-upon ‘race scale’ as there are hat and shoe size scales. Ideas about race are fluid and based on different phenotypic cues. The salient cues change over time, place, and circumstance and are subject always to social and cultural processes.

Race: Conflating Lived Experience and Genetics

Other key methods of classification such as social class may also differ widely. Although always imperfect, measures of social class begin to provide a glimpse at the underlying processes through which social and economic positions affect lived experience and

health. Where race critically differs is in the breadth of potential interpretations of the underlying processes. As previously noted, some individuals view racial differences in disease as due to genes, while others view race differences as the consequence of the lived experience of race and racism. Obviously, this confusion has serious implications for theory and practice. One cannot do predictive science based on a changing and indefinable cause.

Conflating Human Genetic Variation and Race

Human genetic variation does exist: It is real and measurable. But it is also more dynamic than one might assume. For example, the genetics of Amherst, Massachusetts, in 1615 was very different from 1815 to 2015. And the road from genetic factors to complex diseases and behaviors is exceedingly interactive and less than fully determined. Where we end up – whether a behavior or disease becomes manifest – is undoubtedly related, though partly and incompletely, to genetics. But none of this has anything to do with race.

A reasonable compromise position would be to accept the fact that racial inequalities in health are likely a result of both causal pathways: genetic and lived experience. In fact, this is precisely the position advocated by Francis Collins (2004), the powerfully placed head of the US National Human Genome Research Institute. But Sankar and colleagues (2004) argued that such a position has the real consequence of overstating the importance of genetics, as well as continuing to conflate race with human genetic variation. In their analysis, such a position is likely to divert research funds away from studies of socioeconomic causes of health disparities.

Conclusions

No single reason noted earlier may be sufficient to throw race as genetics, or radiogenetics, onto the scrap heap of surpassed scientific ideas. But considered in combination, the critical discussion in the preceding text clearly suggests that it is time to move beyond radiogenetic thinking in the health sciences. Such a move not only finally jettisons an outdated paradigm but also provides the space to explore more fully the complex and critical connections between the experience of racism and health and, ultimately, the full range of causes of health inequities.

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Relevant Websites

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- <http://www.pbs.org/race> — PBS, Race: The Power of an Illusion.
- <http://raceandgenomics.ssrc.org/> — Social Science Research Council, Is Race "Real"?
- <http://www.understandingrace.org> — American Anthropological Association, Race: Are We So Different?