MEDICINE

Race and Reification in Science

Troy Duster

Alfred North Whitehead warned many years ago about "the fallacy of misplaced concreteness" (*1*), by which he meant the tendency to assume that categories of thought coincide with the obdurate character of the empirical world. If we think of a shoe as "really a shoe," then we are not likely to use it as a hammer (when no hammer is around). Whitehead's insight about misplaced concreteness is also known as the fallacy of reification. Recent research in medicine and genetics makes it even more crucial to resist actively the temptation to deploy racial categories as if immutable in nature and society.

Enhanced online at www.sciencemag.org/cgi/content/full/307/5712/1050

Hypertension and Heart Disease

In the last two decades, there has been extensive publication on the differences in hypertension and heart disease between Americans of European descent and Americans of African descent (*2*–*4*). Racial designations are frequently used in efforts to assess the respective influences of environmental and genetic factors.

In November, a study was published regarding a combination of isosorbide dinitrate and hydralazine (BiDil) that was originally found to be ineffective in treating heart disease in the general population but was then shown to work in a 3-year trial of a group of 1050 individuals designated as African Americans (*5*). BiDil is likely to get FDA approval this year and has been labeled "the first ethnic drug," although in medical practice, this becomes "the first racial drug." In presenting their justification for FDA approval of an ethnic/race–specific drug, the company (NitroMed) announced, "The African American community is affected at a greater rate by heart failure than that of the corresponding Caucasian population. African Americans between the ages of 45 and 64 are 2.5 times more likely to die from heart failure than Caucasians in the same age range" (*6*).

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However, both age and survey population complicate this picture. The age group 45 to 64 only accounts for about 6% of heart failure mortality, and for those over 65, the statistical differences between "African Americans and Caucasians" nearly completely disappear (*7*). Researchers recently published a study that was explicitly designed to compare racial differences, by sampling whites from eight surveys completed in Europe, the United States, and Canada and contrasting these results with those of a sample of three surveys among blacks from Africa, the Caribbean, and the United States (*8*). Hypertension rates were measured in 85,000 subjects. The data from Brazil, Trinidad, and Cuba show a significantly smaller racial disparity in blood pressure than is found in North America (*8*).

Even within the category African American, the highly variable phenotype of skin color complicates the hypertension and race thesis. A classic epidemiological study on the topic also found differences within the African American population—with darker-skinned blacks generally having higher mean blood pressure than lighter-skinned blacks. The authors concluded that it was not the color of the skin that produced a direct causal outcome in hypertension, but that darker skin color in the United States is associated with less access to scarce and valued resources of the society. There is a complex feedback loop and interaction effect between phenotype and social practices related to that phenotype (*4*, *9*).

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Others have voiced concerns about the pitfalls of using race as anything but a temporary proxy: As the geneticist David Goldstein observed, "Race for prescription is only an interim solution to carry us through a period of ignorance until we find the underlying causes" (*10*). There is every evidence that these underlying causes interact with each other. However, race is such a dominant category in the cognitive field that the "interim solution" can leave its own indelible mark once given even the temporary imprimatur of scientific legitimacy by molecular genetics.

Studies of Human Genetic Diversity

The procedures for answering any inquiry into the empirical world determine the scientific legitimacy of claims to validity and reliable knowledge, but the prior question will always be: Why that particular question? The first principle of knowledge construction is, therefore, which question gets asked in the research enterprise.

A paper published in this week's issue of Science (*11*) is well-intentioned, well-crafted, and designed to help better understand the molecular basis of disease. The researchers were searching for and found patterns of SNPs differentially distributed in three population groups, formed from a total of 71 persons who were Americans of African, European, or Han Chinese descent.

Why was the question raised in this manner? The answer is a scientific Catch-22. This and other similar efforts (*12*) to create linkage disequilibrium and haplotype maps have a logic for choosing to study people from disparate geographic regions of the world. The purpose is to generate maps that can indicate subtle differences in the patterning or structuring of human genetic diversity across the globe. An increased understanding of these patterns of genetic diversity will help scientists doing gene-association studies by identifying new variants and reducing the likelihood of false-positive associations. The hope is that it may aid scientists to identify medically relevant genes for diseases

However, the particular groups of individuals chosen to represent each region of the world are often chosen because of their convenience and accessibility. Cell and tissue repositories are created to decrease the cost and difficulty of obtaining samples, and the archived samples will be extensively characterized and frequently utilized. Sample collections from repositories may be treated as populations in the narrow sense

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of the term, even when there is little evidence that they represent a geographically localized, reproductively isolated group. These samples are often subtly portrayed as representing racially categorized populations. Finding a higher frequency of some alleles in one population versus another is a guaranteed outcome of modern technology, even for two randomly chosen populations. When the boundaries of those populations coincide with the social definition of race, a delicate tightrope needs to be better navigated between: (i) acknowledging race as a stratifying practice in societies that can lead to different frequencies of alleles in different modern populations but also to different access to health-related resources, and (ii) reifying race as having genetically sufficiently distinctive features, i.e., with "distinctive gene pathways," which are used to explain health disparities between racially categorized populations.

If we fall into the trap of accepting the categories of stored data sets, then it can be an easy slide down the slope to the misconceptions of "black" or "white" diseases. By accepting the prefabricated racial designations of stored samples and then reporting patterns of differences in SNPs between those categories, misplaced genetic concreteness is nearly inevitable.

SNP Patterns and Searches for a Biological Basis for Criminal Behavior

Several countries now have national DNA databases (*13*). Although I use the U.S. criminal justice system as an example, I have no doubt that the principles being considered are universal ones.

It is now relatively common for scholars to acknowledge the considerable and documented racial and ethnic bias in the criminal justice system, from police procedures, prosecutorial discretion, jury selection, and sentencing practices—of which racial profiling is but the tip of an iceberg (*14*–*16*). If the FBI's DNA database is primarily composed of those who have been touched by the criminal justice system and that system has engaged in practices that routinely select more from one group, there will be an obvious skew or bias toward this group in this database.

If we turn the clock back just 60 years, whites constituted about 77% of all prisoners in America, while blacks were only 22% (*17*). In just six decades, the incarceration rate of African Americans in relation to whites has gone up in a striking manner. In 1933, blacks were incarcerated at a rate about three times that of whites (*18*). In 1950, the ratio had increased to about four times; in 1970, it was six times; and in 1990, it was seven times that of whites.

Among humans, gene pools and SNP patterns cannot change much in 60 years, but economic conditions and the practices of the criminal justice system demonstrably do. The comparative explanatory power of SNP patterns surely pales before the analytic utility of examining shifting institutional practices and economic conditions. However, given the body of "ethnic-estimation" research being published on behalf of forensic applications (*19*, *20*) and the exponential growth of national DNA databases (*21*, *22*), it is not at all unreasonable to expect that a project that proposed to search for SNP profiles among sex offenders and felons convicted of violent crimes would meet with some success, both for funding and for finding "something." This could begin with the phenotype of "three populations," as in the study cited above (*11*), because that is the way these data are collected by the FBI and the contributing states. We must maintain vigilance to prevent SNP profiling from providing the thin veneer of neutral scientific investigation, while reinscribing the racial taxonomies of already collected data.

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Conclusions

As I have tried to show, a set of assumptions about race has animated the development of BiDil, genetic diversity analyses, "ethnic estimation" research, and the siren's call to do SNP research on the ever-expanding databases of DNA from the incarcerated. These elements are poised to exert a cascading effect—reinscribing taxonomies of race across a broad range of scientific practices and fields. Biomedical research must resist setting off the cascade and, while still moving forward in their efforts to identify the molecular correlates of disease, climb back on the tightrope to address racial disparities in health, in all their biosocial complexity.

The ability to use genomic knowledge to deliver effective pharmaceuticals more safely to special subpopulations that have some functional genetic markers holds promise. Thus, if the FDA approves BiDil, it should do so only under the condition that further research be conducted to find the markers that have the actual functional association with drug responsiveness—thus assuring that the drug be approved for everyone with those markers, regardless of their ancestry, or even of their ancestral informative markers.

The technology will be increasingly available to provide SNP profiles of populations. When the phenotype distinguishing these populations is race, the likelihood of committing the fallacy of misplaced concreteness, in science, is nearly overwhelming. For this reason, when geneticists report population data, they should always attach a caveat or warning label that could read something like this, "allelic frequencies vary between any selected human groups—to assume that those variations reflect 'racial categories' is unwarranted." Whereas this will not completely block the tendency to reify race, it will be an appropriately cautious intervention that tries to prevent science from unwittingly joining the current march toward a biological reinscription of the concept.

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10.1126/science.1110303