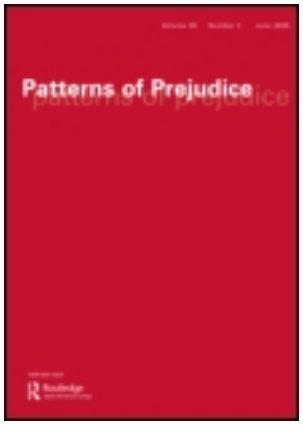

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Against racial medicine

JOSEPH L. GRAVES, Jr AND MICHAEL R. ROSE

ABSTRACT Some scholars claim that recent studies of human genetic variation validate the existence of human biological races and falsify the idea that human races are socially constructed misconceptions. They assert that analyses of DNA polymorphisms unambiguously partition individuals into groups that are very similar to lay conceptions of race. Furthermore, they propose that this partitioning allows us to identify specific loci that can explain contemporary health disparities between the supposed human races. From this, it appears that racial medicine has risen again. In this essay Graves and Rose construct a case against racial medicine. Biological races in other species are strongly differentiated genetically. Because human populations do not have such strong genetic differentiation, they are not biological races. Nonetheless, the lack of population genetic knowledge among biomedical researchers has led to spuriously racialized human studies. But human populations are not genetically disjoint. Social dominance may lead to medical differences between socially constructed races. In order to resolve these issues, medicine should take both social environment and population genetics into account, instead of dubious 'races' that inappropriately conflate the two.

KEYWORDS biomedicine, human classification, genetics, race, racial medicine, racial profiling

Racial medicine has risen again

Charles B. Davenport, one of the most respected scientists of the first decades of the twentieth century, argued that laziness was a hereditary trait. Davenport claimed in particular that laziness was a heredity character of Southern Whites. Later epidemiological studies determined that 'white trash' laziness was actually the result of heavy infections caused by the nematode necator americanus.[1] But, for Davenport and many other biologists of his time, the phenotypic differences displayed by particular populations were proof positive of the existence of human races. They believed that these races differed in readily observable features, such as skin colour and body proportions, and also in those they could not directly observe, such as intellect, morality, character, disease predisposition and

1 Charles B. Davenport, 'The hereditary factor in pellagra' (1917), quoted in Allan Chase, *The Legacy of Malthus: The Social Costs of the New Scientific Racism* (New York: Alfred A. Knopf 1977), 203.

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resistance. In 1921, for example, Ernest Zimmerman published a report on differences in the manifestation of syphilis in Blacks and Whites. Such thinking helped to sanction the now infamous Tuskegee syphilis experiment.[2] Modern biologists recoil with horror when asked to revisit this sad episode in the history of science.

The rationale for the Tuskegee experiment was the underlying assumption that the Negro was genetically inferior to Whites. Thus, the perceived differences in incidence rates and progression of disease were thought to reside in characteristics intrinsic to the race, as opposed to the social conditions under which visibly darker-skinned persons of African descent lived in the United States. (It is significant that many 'white Americans' have African ancestors but 'pass as white', an anomaly to which we will return below.) Therefore, the Tuskegee experiment suffered not only from its moral shortcomings, but also from poor experimental design. The results of the experiment could not have distinguished between any genetically based difference in disease progression, since many environmental and social differences between African Americans and the Swedish cohorts with which they were to be compared were not properly controlled. With hindsight, the scientific problems of this experiment are obvious. What is not recognized is that modern discussions of race and medicine have not moved very far beyond the misconceptions that gave birth to the Tuskegee research programme.

In 2001 the *New England Journal of Medicine* featured an exchange between two physicians, Robert Schwartz and Alistair Wood. Schwartz argued that racial profiling in medical research was unsound because biological races did not exist in the human species, citing the 1999 statement of the American Anthropological Association. Wood attempted to rebut Schwartz. He pointed out that human populations have different frequencies of genes. For example, he argued that both black Americans and Africans have a high frequency of a cytochrome P allele (CYP2D6) that encodes low activity for that enzyme. Furthermore, this allele is virtually absent in European and Asian populations. Knowing whether groups differ in this enzyme is important because it affects the metabolic effects of several drugs. Wood continued with several examples of this kind, yet he never explained how biological races were defined, whether those definitions applied to our species or, finally, whether the groups he described in his essay fit conventional biological definitions of 'race'.[3]

Likewise, in 2002, Sally Satel, a fellow of the American Enterprise Institute, wrote, citing essentially the same arguments as Wood, that she used racial

2 James H. Jones, *Bad Blood: The Tuskegee Syphilis Experiment* (New York: The Free Press and London: Collier Macmillan 1981), 28.

3 Robert S. Schwartz, 'Racial profiling in medical research', and Alistair J. J. Wood, 'Racial differences in responses to drugs—pointers to genetic differences', *New England Journal of Medicine*, vol. 344. no. 18, May 2001, 1392–3 and 1393–6, respectively.

profiling in her medical practice.[4] A year ago, the *New York Times* published an op-ed piece entitled 'A Family Tree in Every Gene' by Armand Leroi of Imperial College, London. In this article, Leroi suggested that recent genetic research supported the idea of human racial differentiation because it was relatively easy to classify humans into distinct groups if enough genetic markers were used.[5] Given these recent claims, it appears that the way has been cleared for a return to Davenport's emphasis on the genetic origins of racial disparities in susceptibility to disease: a return to racial medicine.

The identification of human races is not based on cogent biology

While humans have always recognized the existence of physical differences between groups, they haven't always described those differences in racial terms. Racial theories of human differentiation were not a consistent theme of the ancient world, and really did not begin to flourish until after the European voyages of discovery in the fifteenth century. European naturalists of the eighteenth century were divided about the characterization of human differences. Almost all agreed that there was only one human species, yet they disagreed about whether there was a legitimate way to rank the various groups of humans hierarchically. For example, Carl Linneaus's *Systema Naturae* (1735) classified human races partly on the basis of subjectively determined behavioural traits. It is not clear, however, what Linnaeus meant by the use of the term 'race'. It seems that his classification scheme was describing subspecies of humans based on morphological features. According to it, European traits were clearly superior to others, and Africans were assigned the lowest rung in the hierarchy.

Such racist ideas were transplanted to America during colonial times, along with other biological absurdities. During American chattel slavery, the socially defined race of the offspring of slavemasters and slave women was 'Negro'. Virginia law classified Eston Hemmings, who was 87.5 per cent European according to genetic ancestry, as 'Negro'. Geneticists now suspect that Thomas Jefferson was his father, based on family genealogies and a genetic marker specific to the Jefferson family found in Eston's descendants.

The one-drop rule (also called 'hypo-descent') in the United States differs from definitions of 'blackness' in Canada, Mexico, Britain and Brazil. Individuals, therefore, could move from one country to another and be classified differently according to the social custom. Indeed, in the United States, individuals have been born as a member of one race and died as a

4 Sally Satel, 'I am a racially profiling doctor', *New York Times Sunday Magazine*, 5 May 2002.

5 Armand Marie Leroi, 'A family tree in every gene', *New York Times*, 14 Mach 2005, A23; A. W. F. Edwards, 'Human genetic diversity: Lewontin's fallacy', *Bioessays*, vol. 25, no. 8, August 2003, 798–801.

member of another. European ethnic groups, such as the Irish and Italians, did not become 'white' until the twentieth century. Such 'races' are clearly based on social conventions, as opposed to biological measures of genetic ancestry. Socially produced racial ideology from the very beginning influenced the collection and interpretation of data relating to human biological variation.

Ashley Montagu was one of the first scholars to analyse the idea of race in humans explicitly as a social construction. Montagu pointed out that human physical variation was discordant.[6] For example, sub-Saharan Africans, East Indians and Australian aborigines have dark skin but differ in other anatomical traits, such as bodily proportions, skull proportions, hair type and ear wax consistency. More recently, C. Loring Brace has shown that, while physical features can be used to demonstrate the likely geographical origin of an individual skeleton, these features do not allow the unambiguous classification of races.[7] Geneticist Sewall Wright made the same point concerning discordance in his discussion of the genetic differentiation of the races of mankind.[8]

Human populations are not biological races

The measurement and apportionment of human diversity have been core issues in genetic research. This work has focused on the question of whether human genetic variation has a significant substructure.[9] Sewall Wright developed hierarchical population statistics in order to partition genetic variation within a species. Wright's statistics treat variation at the level of breeding populations (demes) within regions (DR), regions within primary subdivisions (RS), and primary subdivisions within the total species (ST). Wright argued that the last statistic was the best measure of subspecies differentiation, 'subspecies' being the term used by taxonomists as an equivalent for biological race. Such population subdivision can be calculated at individual genetic loci or at numerous genetic loci simultaneously. Wright's subspecies statistic (referred to formally as F_{st}) can range between 0 and 1.0, a value of 0 (zero) indicating complete lack of genetic differentiation between major groups. He suggested that the minimal threshold for the existence of racial differentiation would be an

6 Ashley Montagu, *Man's Most Dangerous Myth: The Fallacy of Race*, rev. 5th edn (New York: Oxford University Press 1974).

7 C. Loring Brace, 'Region does not mean ''race''—reality versus convention in forensic anthropology', *Journal of Forensic Sciences*, vol. 40, no. 2, March 1995, 29–33.

8 Sewall Wright, *Evolution and the Genetics of Populations. Vol. 4: Variability within and among Natural Populations* (Chicago: University Chicago Press 1978).

9 Robert H. Podolsky and Timothy P. Holtsford, 'Population structure of morphological traits in *Clarkia dudleyana*: I. Comparison of F_{st} between allozymes and morphological traits', *Genetics*, vol. 140, no. 2, June 1995, 733–44.

F_{st} of 0.25 or more, with moderate variation between populations arising with F_{st} values between 0.15 and 0.25. He examined individual loci from a variety of species, finding a range of differentiation of about 0.02 to 0.50. Using six human blood group loci, he estimated that F_{st} averaged about 0.12 between the human races. This F_{st} value did not come close to 0.25, Wright's pre-established threshold for the existence of subspecies, though human populations within geographic regions share more alleles with each other than they do with populations from other regions.

Subsequent studies of human genetic variation, including whole genome analyses, have generally also found estimates of F_{st} to be much less than Wright's critical value. Genetic substructure does exist in humans, but there are no natural divisions in our species equivalent to biological races. Recently, Jeffrey Long and Rick Kittles demonstrated that the calculation of Wright's F_{st} statistic is biased towards smaller values due to a failure of certain core assumptions, such as equal population sizes among the subpopulations. They showed that, by relaxing those assumptions, F_{st} values may become larger. Nevertheless, they concluded that all human populations derive from a recent common ancestral group, that there is great genetic diversity within all human populations and that the geographic pattern of variation is complex and has no major discontinuities.[10] These points all that undermine the application of subspecies or race concepts to human populations.

In contrast, consider the subspecies differentiation of other mammalian species. Some large-bodied mammalian species show much higher values of population subdivision: white-tailed deer have a F_{st} of 0.60, the Grant's gazelle's F_{st} is 0.65 and North American grey wolves have a F_{st} of 0.75. Our closest relatives, chimpanzees and gorillas, also have much more subdivision between their populations than humans.[11] It would be legitimate to identify geographically based races in these particular mammalian species.

Various studies estimate that, during prehistoric times, our species spent most of its evolutionary time in a small region of northeastern Africa (70,000–100,000 out of 160,000–200,000 years). It was here that most of human genetic diversity evolved. As climates changed, successive groups of migrants left the region, and each migrating group by necessity contained only a part of the total accumulated genetic diversity. This loss of alleles during migration has been shown in other species as well.[12]

10 Jeffrey C. Long and Rick A. Kittles, 'Human genetic diversity and the non-existence of biological races', *Human Biology*, vol. 75, no. 4, August 2003, 449–71.

11 Henrik Kaessmann, Victor Wiebe and Svante Pääbo, 'Extensive nuclear DNA sequence diversity among chimpanzees', *Science*, vol. 286, 5 November 1999, 1159–62.

12 Sonya M. Clegg, Sandie M. Degnan, Jiro Kikkawa, Craig Moritz, Arnaud Estoup and Ian P. F. Owens, 'Genetic consequences of sequential founder events by an island-colonizing bird', *Proceedings of the National Academy of Sciences*, vol. 99, no. 12, June 2002, 8127–32.

Medical research is often conducted on a spurious racial basis

The absence of human races should be obvious, but the lack of scientific understanding of human population genetics among some researchers has led them to structure their studies as if conventional racial categories were biologically meaningful. The problems arising from such flawed data collection become obvious from close examination of the evidence that is used to support the 'human races' assumption. For example, some have argued that differences in the frequency of rare genetic diseases in socially defined races is positive proof that these groups can be reliably distinguished.[13] Others consistently err by assuming that biological differences that may exist between socially defined groups, such as disease incidence, are proof of an underlying genetic difference. Conversely, others make the mistake of believing that finding a genetic difference between socially defined groups automatically implies that such differences are responsible for whatever specific biological phenomenon they are studying.

Several studies have examined genetic variants that show large differences in frequency of hypertension between European and African-American populations, yet found no association between the alleles at candidate loci and the occurrence of hypertension.[14] While one can use non-coding portions of the genome to cluster human populations, this does not mean that these clusters coincide with socially defined racial groups, nor that these groups necessarily differ in genes that account for disease disparity between such socially defined groups.

For example, Hua Tang *et al.* utilized 326 microsatellite markers and found that they could cluster individuals of self-identified ancestry into groups that roughly corresponded with socially defined races. The details of such clustering are important, and indeed revelatory. Tang *et al.*'s study utilized the computer algorithm STRUCTURE. This piece of software allows users to define *in advance* how many clusters should exist at the end of the analysis. With the two-cluster option chosen at the start of the analysis, the software lumped self-identified individuals of African-American, Hispanic (Texas) and European ancestry together, the second cluster being made up of individuals who designated themselves as Chinese-American (Hawaii) and

13 Vincent Sarich and Frank Miele, *Race: The Reality of Human Differences* (Boulder, CO and Oxford: Westview 2004).

14 J. Barley, A. Blackwood, M. Miller, N. D. Markandu, N. D. Carter, S. Jeffery, F. P. Cappuccio, G. A. MacGregor and G. A. Sagnella, 'Angiotensin converting enzyme gene I/D polymorphism, blood pressure and the renin-angiotensin system in Caucasian and Afro-Caribbean peoples', *Journal of Human Hypertension*, vol. 10, no. 1, January 1996, 31–5; Hong-Guang Xie, C. Michael Stein, Richard B. Kim, James V. Gainer, Gbenga Sofowora, Victor Dishy, Nancy J. Brown, Robert E. Goree, Jonathan L. Haines and Alastair J. J. Wood, 'Human β2-adrenergic receptor polymorphisms: no association with essential hypertension in black and white Americans', *Clinical Pharmacology and Therapeutics*, vol. 67, no. 6, June 2000, 670–5.

Japanese-American (Stanford, CA). With the alternative three-cluster setting, the programme clustered African Americans separately from Hispanics and European Americans. With four clusters selected, a Hispanic cluster appeared in the output. It should be further noted that each of these three rather different clusterings included a few individuals who did not identify themselves as the majority of individuals in the inferred cluster. For example, cluster A, which was made up of 1,448 self-identified European Americans, also included three self-identified African Americans, one Hispanic and one person described as 'other'.[15]

A naive interpretation of these results supports the idea that medically significant genetic clustering exists within modern humans, and that these clusters reasonably coincide with self-identified race. But this interpretation is deeply flawed. The study was structured in a way that guaranteed that clustering would be found: the software was designed to cluster. Furthermore, the data were chosen so as to represent human populations that were either contemporaneously well separated or had geographically distinct recent origins. These groups, such as East Asians, Europeans and sub-Saharan Africans, are very far apart in the continuum of human genetic diversity. Furthermore, the roles of ancestry, genetic drift and geographically specific selection are conflated in such data. Non-coding portions of the genome may accumulate neutral mutations that are unrelated to how selection operates in coding portions, if these regions are far enough apart. For example, non-coding variation at eighty independent loci clustered Ashkenazi Jews very closely to Russians.[16] However, the genetically based disease distribution of the former is very different from the latter group.[17]

At present we do not have data that have been collected in a systematic way from the entire spectrum of human populations. For example, David Hinds *et al.* surveyed over two million single nucleotide polymorphisms in the human genome, but did *not* continuously sample human diversity. Instead, they examined DNA from individuals identified as African American, European American and Han Chinese.[18] Yet Africa, Europe and

15 Hua Tang, Tom Quertermous, Beatriz Rodriguez, Sharon L. R. Kardia, Xiaofeng Zhu, Andrew Brown, James S. Pankow, Michael A. Province, Steven C. Hunt, Eric Boerwinkle, Nicholas J. Schork and Neil J. Risch, 'Genetic structure, self-identified race/ethnicity, and confounding in case-control association studies', *American Journal of Human Genetics*, vol. 76, no. 2, February 2005, 268–75.

16 Sarah A. Tishkoff and Kenneth K. Kidd, 'Implications of biogeography of human populations for ''race'' and medicine', *Nature Genetics*, vol. 36, no. 11 (Supplement), November 2004, S21–S27.

17 Inbal Kedar-Barnes and Rozen Paul, 'The Jewish people: their ethnic history, genetic disorders and specific cancer susceptibility', *Familial Cancer*, vol. 3, nos 3–4, September 2004, 193–9.

18 David Hinds, Laura Stuve, Geoffrey Nilsen, Eran Halperin, Eleazar Eskin, Dennis Ballinger, Kelly Frazer and David Cox, 'Whole-genome patterns of common DNA variation in three human populations', *Science*, vol. 307, 18 February 2005, 1072–9.

Asia are large continents, while the ancestry of African Americans is primarily from a portion of the West Coast of Africa, Han Chinese represent only one ethnic Chinese group, and the ancestry of the European Americans in the study was not described. If one believes that biological races exist, and that Africa, Europe and Asia represent different biological races, then one might falsely believe that studies such as this one adequately characterize their genetic diversity. The authors of this paper did not claim that their findings revealed the detailed genetic structure of human populations. But many biomedical researchers act as if such systematically misleading samples adequately represent the spectrum of human diversity.

Many studies suggest that human genetic variation is continuous. Luigi Luca Cavalli-Sforza, Paolo Menozzi and Alberto Piazza illustrated this well in their use of maps of allele frequencies at multiple loci.[19] Their maps show continuous gradients in allele frequency from Africa to the Americas. Alan Templeton plots genetic distance versus geographic distance for 135 medically important loci. He found continuous change in genetic distance as a function of geographic distance between populations.[20] Clearly, the racial sampling of human genetic variation at widely separated points along its continuum greatly distorts the interpretation of geographic variation.

In another analysis of human population structure, Lynn Jorde and Stephen Wooding presented genetic data from individuals sampled from three geographically discontinuous regions.[21] They used 368 individuals of African, European and East Asian origin to study genetic variation within a 14,400 nucleotide sequence (14.4 kb) of the gene angiotensinogen (AGT). The alleles at this locus did not show statistically well-defined clustering. Another study that examined HLA alleles determined by DNA typing and sequencing in individuals from the Republic of Macedonia and Greece found that the Macedonians were more similar to an 'older' set of Mediterranean populations (Basques, North Africans, Italians, French, Cretans, Jews, Lebanese, Turks, Anatolians, Armenians and Iranians) and that the Greeks were closer to Ethiopians and sub-Saharan Africans (Oromo, Amhara, Fulani, Rimaibe and Mossi).[22] Thus, while individuals with shared

19 Luigi Luca Cavalli-Sforza, Paolo Menozzi and Alberto Piazza, *The History and Geography of Human Genes* (Princeton, NJ: Princeton University Press 1994). See also Alan R. Templeton, 'The genetic and evolutionary significance of human races', in Jefferson M. Fish (ed.), *Race and Intelligence: Separating Science from Myth* (Mahwah, NJ: Lawrence Erlbaum 2002); Frank B. Livingstone, 'On the non-existence of human races', *Current Anthropology*, vol. 3, no. 3, June 1962, 279–81.

20 Templeton, 'The genetic and evolutionary significance of human races'.

21 Lynn B. Jorde and Stephen P. Wooding, 'Genetic variation, classification, and race', *Nature Genetics*, vol. 36, no. 11 (Supplement), November 2004, S28–S33.

22 A. Arnaiz-Villena, K. Dimitroski, A. Pacho, J. Moscoso, E. Gómez-Casado, C. Silvera-Redondo, P. Varela, M. Blagoevska, V. Zdravkovska and J. Martínez-Laso, 'HLA genes in Macedonians and the sub-Saharan origin of the Greeks', *Tissue Antigens*, vol. 57, no. 2, February 2001, 118–27.

ancestry may be alike when it comes to correlations of large numbers of their genes, at any specific gene they may be different. In addition, the geographic proximity of populations doesn't always guarantee genetic relatedness at specific loci. These results argue against 'racial profiling' in medicine.

Patient populations are not genetically disjoint

The biological dubiousness of socially constructed races becomes most apparent when we discuss biomedical research. As mentioned above, human physical characteristics are not correlated with each other in ways that always reflect genetic relatedness. For this reason, we cannot necessarily infer that the genetic relatedness of individuals within populations means that they are certain to share the same disease phenotypes. Nor can we infer that the similarity of disease phenotypes among populations is proof that specific populations are more closely related to each other genetically than they are to other populations.

If we consider skin cancer incidence around the world, we observe a gradual increase as we move from the tropics towards the more northerly latitudes. We would be correct in surmising that this has something to do with skin colour. Northern European populations, which share specific alleles at melanin-producing loci but which also have cultural habits that encourage them to seek sunlight, should have similar rates among populations. Yet, when examined at multiple genetic loci, these European populations are closer genetically to indigenous Central American Indians than to sub-Saharan Africans. However, the sub-Saharan populations and the Central American Indians, while more genetically distant from each other, share lower skin cancer rates than Northern Europeans, both as a result of having more melanin in their skin and cultural habits that discourage exposure to sunlight. Finally, variation in skin colour is not necessarily correlated with variation for other physical traits. World skin colour shows approximately 88 per cent of its variation among regions, and only 12 per cent within regions, while fifty-seven craniometric measurements show 15 per cent of their variation among regions and 85 per cent of its variation within regions.[23]

But there is much more at stake than mere 'race identification' by practising physicians. A patient's social position and genetic ancestry may combine to produce predisposition to disease.[24] The United States was not founded by people who represented the complete spectrum of the world's genetic

23 John H. Relethford, 'Apportionment of global human genetic diversity based on craniometrics and skin color', *American Journal of Physical Anthropology*, vol. 118, no. 4, August 2002, 393–8.

24 Vicente Navarro, 'Race or class versus race and class: mortality differentials in the United States', *The Lancet*, vol. 336, no. 8725, 1990, 1238–40; Ichiro Kawachi, Norman Daniels and Dean E. Robinson, 'Health disparities by race and class: why both matter', *Health Affairs*, vol. 24, no. 2, March–April 2005, 343–52.

diversity. Initially, American society was composed of Northern Europeans, indigenous North Americans and Africans. Its first socially constructed races reflected who was there and their social position.

More recently, immigrants have arrived from regions such as the Middle East and Southeast Asia. In the face of such continuing immigration, the failure to study the full range of human genetic diversity will become more significant for the practice of medicine. Sampling large numbers of Europeans, followed by individuals from one region of China and even fewer individuals with ancestry from one portion of Africa, will produce findings that are of diminishing relevance to genetic predispositions to disease in the changing mixture of ancestries present in the United States. Furthermore, because of 'racial intermarriage', the rape of slaves and even the 'multiracial' ancestry of immigrants, one patient's self-proclaimed genetic ancestry may reflect well-defined origins in a specific portion of the world while this may not be true for other patients making the same claim of ancestry. For example, a study of self-reported race and genetic admixture showed that, while 93 per cent of those who self-identified as 'white' had a predominantly European genetic background, but only 4 per cent of those who self-identified as 'black' had a predominantly African genetic background.[25]

The study of 'nature *v.* nurture' in patients is complicated by socially constructed race

To what degree is health disparity between 'human races' the result of 'nature' and to what degree the result of 'nurture'? Almost every complex phenotype is produced by the interaction of genetic, environmental and chance events. Quantitative genetics is the tool that allows biologists to estimate the importance of each of these factors.

The case of the hypertension differential between American 'Blacks' and 'Whites' is one of the most reproducible findings for prospective racial medicine. The observation of elevated blood pressures in African Americans goes back to the early 1930s. By the 1960s similar observations had been made of Afro-Caribbeans. Many concluded that hypertension was a racial feature of 'the Negro', even as data were accumulating showing that no such elevated blood pressures could be found in Africans from West Africa and that there were European populations with similar rates of hypertension as African Americans. Various theories were proposed to explain black hypertension, including the salt retention hypothesis. Supposedly, hypertension was related to a historical absence of salt in West Africa, combined with the need for salt re-uptake in the survivors of the Middle Passage. By the early 1990s this theory had been widely adopted, with no published scientific data that

25 Moumita Sinha, Emma K. Larkin, Robert C. Elston and Susan Redline, 'Self-reported race and genetic admixture', *New England Journal of Medicine*, vol. 354, no. 4, January 2006, 421–2.

specifically supported its assertions. Subsequent analyses have dismantled the salt-hypertension hypothesis.[26]

If strong selection had been responsible for increased hypertension risk in Africans of the western hemisphere, we should see clear evidence of it in allele frequencies. At least 33 genetic systems and more than 63 genetic loci have been investigated for associations with increased risk of hypertension over the last six years. A recent search on MEDLINE showed 57 studies published between 1997 and 2003 that examined loci that might be associated with hypertension and racial variation. The results of these disparate studies can only be described as inconsistent, while their methodologies were often deficient. In only 7 out of the 57 studies were genetic variants consistently correlated with hypertension.[27]

A study of racial differences in drug action produced similarly inconsistent results. The study examined twenty-two drugs that had been claimed to show racial differentiation in medical outcome, including ACE inhibitors, vasodilators and beta-blockers. Despite the deliberate attempt to find racial genetic differences in drug effects, however, only one was found.[28]

Social dominance may create health disparities for socially constructed races

There is evidence that emotional stress can cause cellular damage.[29] A recent study demonstrated that greater susceptibility to cell deterioration arises among women caring for chronically ill children, relative to women caring for healthy children. Another recent study concludes that hypertension levels rise in African immigrants as they spend more time in white-dominated American or European societies.[30] Socially mediated stress can

26 Jay S. Kaufman and Susan A. Hall, 'The slavery hypertension hypothesis: dissemination and appeal of modern race theory', *Epidemiology*, vol. 14, no. 1, January 2003, 111–26.

27 Joseph L. Graves, Jr, *The Race Myth: Why We Pretend Race Exists in America* (New York: Dutton 2005), 126.

28 James F. Wilson, Michael E. Weale, Alice C. Smith, Fiona Gratrix, Benjamin Fletcher, Mark G. Thomas, Neil Bradman and David B. Goldstein, 'Population genetic structure of variable drug response', *Nature Genetics*, vol. 29, no. 3, November 2001, 265–9; Sarah K. Tate and David B. Goldstein, 'Will tomorrow's medicines work for everyone?', *Nature Genetics*, vol. 36, no. 11 (Supplement), November 2004, S34–S42.

29 Elissa S. Epel, Elizabeth H. Blackburn, Jue Lin, Firdaus S. Dhabhar, Nancy E. Adler, Jason D. Morrow and Richard M. Cawthon, 'Accelerated telomere shortening in response to life stress', *Proceedings of the National Academy of Sciences*, vol. 101, no. 49, December 2004, 17312–15.

30 Jen'nan Ghazal Read, Michael O. Emerson and Alvin Tarlov, 'Implications of black immigrant health for U.S. racial disparities in health', *Journal of Immigrant Health*, vol. 7, no. 3, July 2005, 205-12; Jen'nan Ghazal Read and Michael O. Emerson, 'Racial context of origin, black immigration, and the U.S. black/white health disparity', *Social Forces*, vol. 84, no. 1, September 2005, 183–201.

cause illness, and such stress may be partly dependent on socially ascribed racial status. But these are only initial findings. We need to know more; medical research should address the impact of socially assigned race on health.

Another study has estimated how much the United States has to gain by eliminating health disparities among socially designated races. It compared the number of lives saved by advances in medical technology from 1991 to 2000 with the number of lives that could have been 'saved' by equalizing 'black' and 'white' mortality rates over the same period. The difference was 886,202 more lives spared without health disparity.[31] At a minimum, this finding suggests that there is a need for considerable redirection of biomedical research and public health resources towards the role of perceived race in health outcomes. This should not be interpreted as opposition to basic research on human genetics. However, if we are going to address health disparity with limited funds to achieve our ends, we need to take account of what is going to improve health the most.

Medicine should take both social environment and population genetics into account, not spurious 'human races' that inappropriately conflate the two

Human genetic variation is real. Individuals with ancestry in particular geographic regions are more likely to share genes with other individuals from the same region. But the overall amount of measured genetic differentiation between human populations is meagre. In particular, living as a 'black person' or a 'white person' in the United States may lead to substantially different health outcomes, regardless of underlying genetic differences. Modern human population genetic research demonstrates that apportioning individuals into racial groups, particularly those groupings defined by our historical conceptions of 'race', is a dubious enterprise. Dangerous consequences may follow from the implementation of racial medicine in clinical practice. In addition to fostering social inequality by underscoring racial classification, racial medicine might kill people by neglecting the substantial genetic variation within, and genetic overlap between, human populations.

31 Steven H. Woolf, Robert E. Johnson, George E. Fryer, Jr, George Rust and David Satcher, 'The health impact of resolving racial disparities: an analysis of US mortality data', *American Journal of Public Health*, vol. 94, no. 12, December 2004, 2078–81.

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