Race and IQ

Molecular Genetics as Deus ex Machina

Richard S. Cooper
Loyola University Chicago Stritch School of Medicine

During the last hundred years, the debate over the meaning of race has retained a highly consistent core, despite evolution of the technical details. Non-Europeans, and in particular, Africans, are assigned the role of deviants and outcasts, whose claim on our common humanity remains in doubt. Each time the technical facade of these racialist arguments is destroyed, the latest jargon and half-truths from the margins of science are used to rebuild them around the same core belief in Black inferiority. Because race is in part a genetic concept, the advent of molecular DNA technology has opened an important new chapter in this story. Unfortunately, the article by D. Rowe (2005, this issue) begins from mistaken premises and merely restates the racialist view using the terminology of molecular genetics. No technology—even the awe-inspiring tools now available to DNA science—can overcome the handicap of fundamental conceptual errors. Race is not a concept that emerged from within modern genetics; rather, it was imposed by history, and its meaning is inseparable from that cultural origin. By ignoring its cultural meaning the reductionist narrative about race fails—both in the narrow terms of science and as a contribution to the broader social discourse.

Rowe (2005, this issue) has revisited the controversy surrounding racial differences in IQ, arguing that molecular genetics has now created an opportunity to test the most basic unanswered questions. Much has been staked on the potential of DNA science to solve complex problems in biology, and the molecular revolution has without a doubt brought a sea change to many disciplines. As one might expect with any new technology, however, there is the associated risk that the hype will outrun the reality (Cooper & Psaty, 2003). To date, however, change in biology is being driven primarily by opportunities inherent in the technology, not by fundamental insights into the nature of the world around us. Revisiting old problems with new methods can be very fruitful if the obstacle has been the inability to generate the necessary data. But not every problem is a nail, even if it looks that way to a person with a hammer, and it is unrealistic to expect that the "race problem" can be solved with data from a genotyping machine. Although a new approach to the examination of racial inequality is surely needed, Rowe asked the wrong questions of molecular genetics and in effect encouraged the continuation of a discredited research agenda by other means (Chase, 1977; Hearnshaw, 1981; Hernstein & Murray, 1994). A fresh new look at the Black–White gap in educational achievement will require a deeper understanding of its social origins and a break from the assumptions underlying the hierarchical theory of continental race. In addition to its technical and scientific flaws, the article by Rowe fails on both those counts.

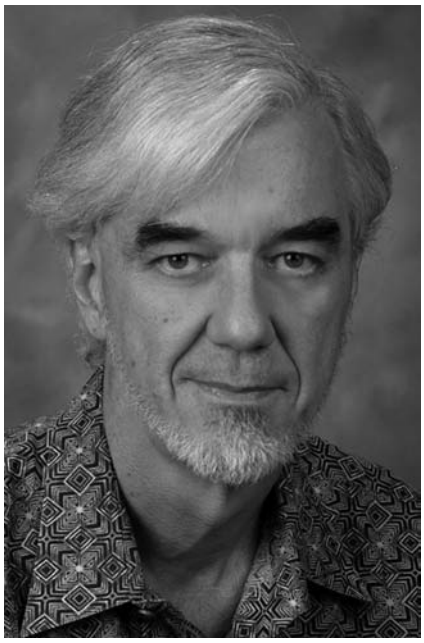
The technical errors contained in Rowe's (2005) article include both misuse of broad scientific concepts and incorrect or biased misinterpretation of specific scientific data. The author's broad argument assumes that a quantity definable as "intelligence" exists (in contradistinction to the view that multiple types of cognitive functioning can be identified that are valued and manifested differently, conditional on the setting and the observer), that intelligence can be measured with "IQ tests," that demographic groups known as "continental races" divide humans into discrete categories on the basis of important concordant variation in genetically determined traits, that molecular genetics can (or will) make it possible to define the architecture of complex traits in terms of "genes for X or Y" (i.e., "genes for intelligence"), and that significant variation in polymorphisms in those genes overlap with the traditional demographic categories, such as those promulgated by the U.S. government. I argue instead that the joint product of all of those assumptions yields something of vanishingly small scientific value. In this rejoinder I confine my comments to specific instances of disagreement related to genetics as applied to the concept of race and do not engage the issues surrounding structural equation models, psychometrics, or the social context of racial definitions. I assume that most readers of *American Psychologist* are sufficiently familiar with those latter issues to formulate their own views.

A major technical focus of Rowe's (2005) article is on admixture analysis. Although the technology exists to estimate the overall contribution of continental (i.e., "racial") origin to some U.S. subpopulations, given the large geographic contrasts of the ancestral populations, the meaning

I thank Lena Hatchett and Joan Kennelly for helpful comments on a draft of this article.

Correspondence concerning this article should be addressed to Richard S. Cooper, Department of Preventive Medicine and Epidemiology, Loyola University Chicago Stritch School of Medicine, 2160 South First Avenue, Maywood, IL 60153. E-mail: rcooper@lumc.edu

Vol. 60, No. 1, 71–76 DOI: 10.1037/0003-066X.60.1.71



Richard S. Cooper

of that quantity in relation to traits like IQ remains completely obscure. The molecular basis of the inheritance of complex polygenic traits is still unknown. Is it a result of common variants with weak effects? Rare variants with moderate effects? Does it involve variation in coding or regulatory sequence? Are gene–gene or gene–environment interactions crucial? Molecular technology has changed biology precisely because it allows the reductionist paradigm (measuring the smallest causal unit) to be implemented at the level of DNA. However, without guidance as to what should be measured, that tool remains unhelpful. Admixture analysis is proposed as a shortcut—where proportion of admixture serves as a proxy for unmeasured genetic effects associated with one of the ancestral populations. Its promise lies primarily in identifying specific loci with varying effects across population subgroups. Assessment of global risk—the application proposed by Rowe—must account for environmental confounding. The intractable nature of residual confounding undermines the usefulness of admixture mapping for that purpose (Kaufman, & Cooper, 1999, 2001; Kaufman, Cooper, & McGee, 1997; Zuberi, 2001).

In a hierarchical society with rigid stratification on skin color, social status indicators and "racial genetic traits" will be correlated. Rowe (2005) acknowledged this problem in general and yet concluded that admixture analysis can, nonetheless, serve as a proxy for direct measurement. Furthermore, the methodology (mapping by admixture linkage disequilibrium, or MALD—i.e., the use of a subset of markers to generate a global "admixture" score) is based on statistical theory and is of unproven value in gene mapping (McKeigue, 1998). Under most circumstances this method is a test for association, not linkage, and suffers from an inflated Type I error rate (Zhu, Cooper, & Elston, 2004; Zhu, Zhang, Zhao, & Cooper, 2002).

The "first principle" from which the logic of racialist discourse flows is a belief in the biological concept of race. Needless to say, this concept is hotly contested in virtually all disciplines of science. (For a thoughtful and detailed technical review that takes account of molecular genetics, see Kittles & Weiss, 2003.) Rowe (2005) dismissed this problem with nothing more than a sleight of hand: "The conceptual fuzziness of racial definitions does not negate their utility" (p. 62). In contrast, Darwin (1871/1981) suggested the opposite:

> The most weighty of all the arguments against [race . . . is that they] graduate into one another [and the] naturalist, . . . if of a cautious disposition, . . . will say to himself that he has no right to give names to objects he cannot define. (p. 698)

The modern synthesis takes as its central tenet the concept that nothing in contemporary biology makes sense outside of evolution. Although itself the subject of ongoing debate, the biological species concept can be defined as a breeding pool that is protected from the intrusion of genes that do not share the same evolutionary history (e.g., "The segregation of genetic variability into discrete packages . . . [protects against] . . . incompatible gene combinations" [Mayr, 1996, p. 264]). Race, a quantitative distinction within a species, has no equivalent defining criterion—that is, genetic variability is not restricted to discrete packages (American Anthropological Association [AAA], 1998). This aversion to distinctions without meaning is what has led most geneticists and anthropologists to the conclusion that race in its common usage has no biological basis (AAA, 1998; Darwin, 1871/1981; Gould, 1996; Graves, 2001; Kittles & Weiss, 2003; Lewontin, 2000; Mayr, 1996; Montagu, 1964; Templeton, 1998). Templeton summarized this view:

> Reproductive traits have priority in defining a species. . . . Unfortunately, there is no such guidance at the subspecies level, although in practice easily observed morphological traits are used. There is no evolutionary justification for this dominance of easily observed morphological traits. (1998, p. 632)

This point bears restating: To cluster individual members of a species into groups is not the same as creating a natural biological category. One could cluster humans into an infinite number of fractal units based on size (family, clan, deme, continent, etc.) or on a physical trait (height), and the meaning of those groupings would vary in an infinite number of ways. The contemporary Euro American definition of race is based on continental geography, which simultaneously ignores enormous within-"race" heterogeneity and confounds history with population genetics (Kittles & Weiss, 2003). The history of Africa, for example, cannot be separated from the distribution of the phenotypes observed among Africans and persons of African descent in today's world—nor can the interpretation of data collected in the study of the people of Africa.

In an attempt to buttress claims for the biological relevance of race, Rowe (2005) placed considerable em-

phasis on the interpretation by some geneticists of the cluster analysis of Rosenberg et al. (2002; see Risch, Burchard, Ziv, & Tang, 2002). The rapidly mutating microsatellite markers examined by Rosenberg et al. (2002) are untranslated and function as a surrogate for time of separation (i.e., they have no known function and are not known to influence phenotypes). There is no necessary correlation between any of the resulting clusters and genetic variation of relevance to biomedicine. Further analysis by Rosenberg et al. (2003) demonstrated that the categories proposed as races are not that informative. Depending on the markers used, the component of variance between continents could be as low as 2.8%, whereas the component within continents was 2.5%; the remaining 95% of genetic variation was within local populations. From the hereditarian perspective, why then would IQ not be expected to vary between, say, Sicilians and Swedes as much as between Europeans and Africans? (Of course, this was once the accepted view, on the basis of intelligence tests.)

These new data do not alter the prior conclusion: Historically determined groups of various sizes can be identified, but there is no reason to assume that these categories are coterminous with any complex trait of interest to biologists. This occurs because genetic variation is overwhelmingly discordant among population groups (i.e., different variants are assorted randomly among different groups; AAA, 1998). For example, sub-Saharan Africa is home to both the tallest (Maasai) and the shortest (pygmies) people, and dark skin is found in all equatorial populations, not just in the "Black race" as defined in the United States. Contrary examples of what appear to be "racial traits" of practical relevance are actually quite rare—for example, high rates of skin cancer among white-skinned Australians and hemoglobinopathies across the distribution of malaria—and only a fixed prior belief in demographic categories could lead from that evidence to the general conclusion that races exist. Indeed, a more recent analysis (Serre & Paabo, 2004) of the data used by Rosenberg et al. (2002) confirms that sampling more evenly across the globe yields gradients in allele frequencies rather than discrete categories.

The key contention here is that the evolutionary framework within which potential racial differentiation makes sense is left unspecified by those who embrace racialist theories. Group differentiation broadly speaking can be driven by either random processes (drift) or by selection. On the assumption that the underlying architecture of mental functioning is complex and ancient, all populations of the human diaspora must share these common genetic structures. Molecular data now universally confirm that all common mutations found in the human species are represented in Africa, that current Africans have the highest degree of genetic diversity, and that even uncommon clades have deep roots in Africa (Gabriel et al., 2002; Halushka et al., 1999; Kittles & Weiss, 2003; Nei & Takezaki, 1996; Reich et al., 2001; Tishkoff & Williams, 2002; Zhu et al., 2003). Therefore the vast majority of genetic variation that is not rare (i.e., allele frequencies greater than 10%) is present in Africa, and other populations contain a subset of that African diversity.

If one hypothesizes that Africans are the "outliers" (i.e., all other groups share the "intelligence genes" except Africans), then one must argue that either (a) distinct evolutionary histories of geographic populations all led separately to "higher intelligence" for non-Africans (these "genes" developed independently and converged on the same result) or (b) Africans "evolved backward" in the period since dispersal. There is no reason to embrace either of those arguments. The hypothesis that genetic drift 50,000 to 100,000 years after dispersal caused low IQ in Africa is so improbable that it can be discarded. However, although selection can be shown to have influenced genetic differentiation of regional populations, to date the important examples are limited to immune function and red cell adaptations that protect against malaria (e.g., Sabeti et al., 2002). For complex traits, however, like blood pressure regulation, control of body fat stores, or intelligence, no such context exists, and attempted explanations of a role for selection are based on "just so stories" (Kaufman & Hall, 2003).

In fact, the "evidence" for racial predisposition is derived exclusively from phenotypes measured in industrialized societies like the United States, where dramatic cultural influences have intervened to shape the distribution of these phenotypes. Analyses based on this indirect evidence are invalidated by the inability to control residual confounding and define an appropriate counterfactual state on which to base comparisons (Kaufman & Cooper, 1999, 2001; Kaufman et al., 1997; Zuberi, 2001). To infer genetic effects from this phenotypic evidence is simply to repeat the earlier errors of those who have used the tools and concepts of the science in vogue to buttress a belief in the inherent inferiority of persons of (recent) African origin (Chase, 1977; Hearnshaw, 1981; Hernstein & Murray, 1994).

Rowe (2005) drew further parallels between the study of IQ and "racial differences in medical traits" (p. 61). Many parallels do in fact exist with epidemiology, which shares a long tradition of unsubstantiated claims for genes as a cause of racial differentials (Cooper, 2003; Cooper, Kaufman, & Ward, 2003). In the United States, virtually all common medical conditions occur at higher rates among Blacks (see Table 1). Although investigators studying asthma, renal disease, atherosclerosis, obesity, low birth weight, dementia, diabetes, heart failure, stroke, cancer, hypertension, and so on, all make claims of genetic determinism, there are as yet no documented variants that have been shown to play an important role in the Black–White differentials. The claims made for each of the individual syndromes are plausible as hypotheses only in isolation from each other; social determinism is a much more parsimonious explanation of the root cause when seen from the public health perspective (Cooper, 2004).

To strengthen his argument regarding IQ, Rowe (2005) introduced specific examples from studies of racial health differentials, all of which are unconvincing. For

Table 1
Measures of Health Status in U.S. Racial/Ethnic Groups, 2001

Cause of death	Age-adjusted death rates[a]			
	White	Black	Hispanic	Asian
All causes	842.9	1101.2	658.7	492.1
Heart disease	245.6	316.9	192.2	137.6
Coronary heart disease	177.5	211.6	149.9	103.0
Cancer	197.4	243.1	132.3	119.5
External causes	6.2	37.6	30.7	17.4
Stroke	56.0	78.8	44.9	51.2
COPD	47.0	30.9	20.7	17.7
Pneumonia/influenza	21.7	24.1	20.5	19.0
Diabetes mellitus	22.1	49.2	36.7	16.9
Liver disease/cirrhosis	9.0	9.3	15.8	3.5
HIV infection	2.1	22.8	6.2	0.7
Infant mortality per 1,000	5.7	13.3	5.4	4.7
Life expectancy (years from birth)	77.7	72.2	>80?	>80?

Note. COPD = chronic obstructive pulmonary disease. From *Health, United States, 2003* (Tables 19, 27, & 29), by the National Center for Health Statistics, Washington, DC: U.S. Government Printing Office.
[a] Per 100,000.

brevity, I focus only on one. Low birth weight and higher infant mortality in U.S. Blacks compared with Whites have long been recognized as important features of neonatal epidemiology (Kleinman & Kessel, 1987). Blacks in the United States have a birth weight distribution shifted downward by approximately 400–500 grams relative to Whites, in parallel with a strong socioeconomic status gradient. Numerous studies, however, document very modest or absent differences between Whites and recent immigrants to the United States from Africa and the Caribbean (Collins, Wu, & David, 2002; David & Collins, 1997; Fang, Madhavan, & Alderman, 1999; Friedman et al., 1993). Among the offspring of women from Ghana and Nigeria who gave birth in Illinois, mean birth weight was 3,333 grams, compared with 3,446 grams for Whites and 3,089 grams for U.S. Blacks (David & Collins, 1997). In disadvantaged communities in New York, foreign-born Blacks actually had bigger babies than did poor Whites in the same communities (Fang et al., 1999). Immigrants from countries as poor as Haiti had 50% of the risk of low birth weight observed among U.S. Blacks (Friedman et al., 1993). Because most Black immigrants are less "admixed," these data are clearly not consistent with the deterministic genetic hypothesis but suggest an effect specific to the U.S. cultural environment.

Similar observations have been made for other medical conditions, like hypertension and diabetes (Collins et al., 2002; Cooper, Rotimi, Ataman, et al., 1997; Cooper, Rotimi, Kaufman, et al., 1997; Cooper et al., in press; Wolf-Maier et al., 2003). Rowe (2005) further cited a German report of an association between a G protein single nucleotide polymorphism (SNP) and birth weight, advancing this as evidence that the U.S. Black–White differential is genetic (Hocher et al., 2000). SNP association studies are notoriously unreliable, however ("Freely Associating," 1999; Hirschorn, Lohmueller, Byrne, & Hirschorn, 2002), and as with many others, this finding was not replicated (Vásárhelyi, Kocsis, Schuler, Nobilis, & Tulassay, 2000). This example does not suggest that the epidemiology of medical traits supports the notion that racial differences in IQ are genetically based; rather, it lays out the transparent pattern of bias that can emerge from pulling together scraps of evidence from the biomedical literature.

There are other aspects of Rowe's (2005) article related to the style of argument with which I find myself in disagreement. For example, in the second sentence of the abstract, Rowe stated that "in science, viable, alternative hypotheses are ideally given equal Bayesian prior weights" (p. 60). If in fact the structure of knowledge is Bayesian, and hypotheses are formulated, tested, and evaluated on the basis of prior information, then that statement is patently incorrect. Only in the situation in which one has no prior information would hypotheses be given equal weight, and that is clearly not the case here. Invoking Bayesian principles is an attempt to make the author's preferred hypothesis seem more plausible than it really is. To repeat: Despite substantial effort, no genetic polymorphism has yet been found that accounts for any significant proportion of the "racial differences" in the rates of common diseases, IQ, or any other similar trait—nor is there any reason within an accepted evolutionary context to expect that to be the case. Contrariwise, there is massive and highly consistent evidence of social influences. Those who advocate "objectivity" and the use of "Bayesian prior weights" in the test of racial theories should factor those observations into their accounting.

Even more serious problems arise in Rowe's (2005) discussion of the potential for social forces to shape the biological outcomes associated with race. While acknowledging that skin color, socioeconomic status, and IQ are intercorrelated, Rowe dismissed the explanation of "greater racial discrimination against dark-skinned than against light-skinned Blacks" (p. 67) with the assertion that "no mechanism for the discrimination effect has been proposed that is viable" (p. 67). Abundant historical and social science data exist to demonstrate the impact of skin color gradients as a marker of social status. What Rowe would apparently like to do is dismiss the role of institutionalized racism in shaping the structural determinants of success in U.S. society, like the job, housing, and educational markets (Darity & Myers, 1998). Although that line of reasoning blurs the distinction between opinion and factual argument, what follows is a canard worthy of a "shock-jock" radio host: "In the United States, Jews and Asians have both endured significant discrimination but without apparent harm to the their IQs" (pp. 67–68). This blindness to the

historical patterns of cultural assimilation and anti-Black discrimination in the United States and its influence on socially determined traits voids any claim the author might make to integrity and rigor in examining this question.

Molecular genetics works by modeling genotype–phenotype relationships and then defining the DNA variants that create this connection. In the case of intelligence, it is not known what form the genotype takes or how to conceptualize or measure the phenotype. In fact, it is not known what a "race" is. It is, at the very least, premature to argue that useful answers should be expected from research on whether there is a molecular basis for racial differences in IQ scores. Until the DNA–phenotype relationship is understood, such questions should remain in the realm of nescience—the unknown and the unknowable—not science. Rather than providing useful answers, further research on race and IQ along the lines promoted by Rowe will instead add to the existing morass of Type I error and willful falsification.

For the last four centuries Western science has been obsessed with the need to justify White privilege and in so doing has provided crucial support for racist ideas in society at large. To use the rhetoric of science to sell the idea that historical inequity should be embraced as biological inevitability is an insult to those who value a common humanity.

REFERENCES

American Anthropological Association. (1998). *Statement on "race."* Retrieved December 12, 2003, from www.aanet.org/stmts/racepp.htm

Chase, A. (1977). *The legacy of Malthus: The social costs of the new scientific racism.* New York: Knopf.

Collins, J. W., Jr., Wu, S. Y., & David, R. J. (2002). Differing intergenerational birth weights among the descendants of U.S.-born and foreign-born Whites and African Americans in Illinois. *American Journal of Epidemiology, 155,* 210–216.

Cooper, R. S. (2003). Race, genes, and health—New wine in old bottles? *International Journal of Epidemiology, 32,* 23–25.

Cooper, R. S. (2004). The role of genetics in ethnic disparities in health. *Disparities in aging health* (pp. 269–309). Washington, DC: National Academy of Sciences/National Research Council Press.

Cooper, R. S., Kaufman, J., & Ward, R. (2003). Race and genomics. *New England Journal of Medicine, 348,* 1166–1170.

Cooper, R., & Psaty, B. (2003). Genomics and medicine: Distraction, incremental progress, or the dawn of a new age? *Annals of Internal Medicine, 138,* 576–580.

Cooper, R., Rotimi, C., Ataman, S., McGee, D., Osotimehin, B., Kadiri, S., et al. (1997). Hypertension prevalence in seven populations of African origin. *American Journal of Public Health, 87,* 160–168.

Cooper, R., Rotimi, C., Kaufman, J. S., Owoaje, E. E., Fraser, H., Forrester, T., et al. (1997). Prevalence of NIDDM among populations of the African diaspora. *Diabetes Care, 20,* 343–348.

Cooper, R. S., Wolf-Maier, K., Adeyemo, A., Banegas, J. R., Forrester, T., Giampaoli, S., et al. (in press). An international comparative study of blood pressure in populations of European vs. African descent. *BMC Medicine.*

Darity, W. A., Jr., & Myers, S. L., Jr. (1998). *Persistent disparity: Race and economic inequality in the United States since 1945.* Cheltenham, England: Edward Elgar.

Darwin, C. (1981). *The descent of man, and selection in relation to sex.* Princeton, NJ: Princeton University Press. (Original work published 1871)

David, R. J., & Collins, J. W., Jr. (1997). Differing birth weight among infants of U.S.-born Blacks, African-born Blacks, and U.S.-born Whites. *New England Journal of Medicine, 337,* 1209–1214.

Fang, J., Madhavan, S., & Alderman, M. H. Y. (1999). Low birth weight: Race and maternal nativity—Impact of community income. *Pediatrics, 103,* e5.

Freely associating. (1999) [Editorial]. *Nature Genetics, 22,* 1–2.

Friedman, D. J., Cohen, B. B., Mahan, C. M., Lederman, R. I., Vezina, R. J., & Dunn, V. H. (1993). Maternal ethnicity and birthweight among blacks. *Ethnicity & Disease, 3,* 255–269.

Gabriel, S. B., Schaffner, S. F., Nguyen, H., Moore, J. M., Roy, J., Blumensteil, B., et al. (2002, May 23). The structure of haplotype blocks in the human genome. *Science, 296,* 2225–2229.

Gould, S. J. (1996). *The mismeasure of man.* New York: Norton.

Graves, J. L., Jr. (2001). *The emperor's new clothes: Biological theories of race at the millennium.* New Brunswick, NJ: Rutgers University Press.

Halushka, M., Fan, J. B., Bentley, K., Hsie, L., Shen, N., Weder, A., et al. (1999). Patterns of single-nucleotide polymorphisms in candidate genes for blood pressure homeostasis. *Nature Genetics, 22,* 239–247.

Hearnshaw, L. S. (1981). *Cyril Burt, psychologist.* New York: Vintage Books.

Hernstein, R., & Murray, C. (1994). *The bell curve: Intelligence and class structure in American life.* New York: Free Press.

Hirschhorn, J. N., Lohmueller, K., Byrne, E., & Hirschhorn, K. A. (2002). Comprehensive review of genetic association studies. *Genetics in Medicine, 4,* 45–61.

Hocher, B., Slowinski, T., Stolze, T., Pleschka, A., Neumayer, H. H., & Halle, H. (2000). Association of maternal G protein β3 subunit 825T allele with low birthweight. *The Lancet, 355,* 1241–1242.

Kaufman, J. S., & Cooper, R. S. (1999). Seeking causal explanations in social epidemiology. *American Journal of Epidemiology, 154,* 291–298.

Kaufman, J. S., & Cooper, R. S. (2001). Considerations for use of racial/ethnic classification in etiologic research. *American Journal of Epidemiology, 154,* 291–298.

Kaufman, J. S., Cooper, R. S., & McGee, D. (1997). Socioeconomic status and health in Blacks and Whites: The problem of residual confounding and the resiliency of race. *Epidemiology, 6,* 621–628.

Kaufman, J. S., & Hall, S. A. (2003). The slavery hypertension hypothesis: Dissemination and appeal of a modern race theory. *Epidemiology, 14,* 111–118.

Kittles, R. A., & Weiss, K. M. (2003). Race, ancestry and genes: Implications for defining disease risk. *Annual Reviews of Genomics and Human Genetics, 4,* 33–67.

Kleinman, J. C., & Kessel, S. S. (1987). Racial differences in low birth weight. Trends and risk factors. *New England Journal of Medicine, 317,* 749–753.

Lewontin, R. C. (2000). *It ain't necessarily so: The dream of the human genome and other illusions.* New York: New York Review Books.

Mayr, E. (1996). What is a species and what is not? *Philosophy of Science, 63,* 262–277.

McKeigue, P. M. (1998). Mapping genes that underlie ethnic differences in disease risk: Methods for detecting linkage in admixed populations, by conditioning on parental admixture. *American Journal of Human Genetics, 63,* 241–251.

Montagu, A. (Ed.). (1964). *The concept of race.* London: Collier-Macmillan.

National Center for Health Statistics. (2003). *Health, United States, 2003.* Washington, DC: U.S. Government Printing Office. Available from www.cdc.gov/nchs/data/hus/hus03.pdf

Nei, M., Takezaki, N. (1996, January). The root of the phylogenetic tree of human populations. *Molecular Biology and Evolution, 13*(1), 170–177.

Reich, D. E., Cargill, M., Bolk, S., Ireland, J., Sabeti, P. C., Richter, D. J., et al. (2001, May 10). Linkage disequilibrium in the human genome. *Nature, 411,* 199–204.

Risch, N., Burchard, E., Ziv, E., & Tang, H. (2002, July). Categorization of humans in biomedical research: Genes, race and disease. *Genome Biology, 3*(7), 1–12. Retrieved from www.genomebiology.com

Rosenberg, N. A., Pritchard, J. K., Weber, J. L., Cann, H. M., Kidd, K. K.,

Zhivotovsky, L. A., & Feldman, M. W. (2002, December 20). Genetic structure of human populations. *Science, 298,* 2381–2385.

Rosenberg, N. A., Pritchard, J. K., Weber, J. L., Cann, H. M., Kidd, K. K., Zhivotovsky, L. A., & Feldman, M. W. (2003, June 20). Response to comment on "Genetic structure of human populations." *Science, 300,* 1877.

Rowe, D. C. (2005). Under the skin: On the impartial treatment of genetic and environmental hypotheses of racial differences. *American Psychologist, 60,* 163–173.

Sabeti, P., Reich, D. E., Higgins, J. M., Platko, J. V., Richter, D. J., Schaffner, S. F., et al. (2002, October 24). Detecting recent positive selection in the human genome from haplotype structure: Two genes associated with malaria resistance. *Nature, 419,* 832–837.

Serre, D., & Paabo, S. (2004). Evidence for gradients of human genetic diversity within and among continents. *Genome Research, 14,* 1679–1685.

Templeton, A. R. (1998). Human races: A genetic and evolutionary perspective. *American Anthropologist, 100,* 632–650.

Tishkoff, S. A., & Williams, S. M. (2002). Genetic analysis of African populations: Human evolution and complex disease. *Nature Reviews Genetics, 3,* 611–621.

Vásárhelyi, B., Kocsis, I., Schuler, A., Nobilis, A., & Tulassay, T. (2000). G protein in very low birth-weight infants. *The Lancet, 356,* 254.

Wolf-Maier, K., Cooper, R. S., Banegas, J. R., Biampaoli, S., Hense, H., Joffres, M., et al. (2003). Hypertension prevalence and blood pressure levels in 6 European countries, Canada, and the United States. *Journal of the American Medical Association, 289,* 2363–2369.

Zhu, X., Cooper, R. S., & Elston, R. (2004). Linkage analysis of a complex disease using admixed populations. *American Journal of Human Genetics, 74,* 1136–1153.

Zhu, X., Yan, D., Cooper, R. S., Luke, A., Ikeda, M. A., Chang, Y.-P. C., et al. (2003). Linkage disequilibrium and haplotype diversity in the genes of the renin-angiotensin system: Findings from the Family Blood Pressure Program. *Genome Research, 13,* 173–181. Available from www.genome.org

Zhu, X., Zhang, S.-L., Zhao, H., & Cooper, R. S. (2002). Association mapping, using a mixture model for complex traits. *Genetic Epidemiology, 23,* 181–196.

Zuberi, T. (2001). *Thicker than blood: An essay on how racial statistics lie.* Minneapolis: University of Minnesota Press.