In Search of the

Jay S. Kaufman, PhD
Richard S. Cooper, MD

SYNOPSIS

EPIDEMIOLGIC STUDIES OF racial differences sorely lack sound and explicit hypotheses. Race is a social convention, not a biological concept. Its careless use in epidemiology demonstrates a failure to generate appropriate hypotheses to study its role in health.

Studies of hypertension in blacks illustrate the point. Two underlying pitfalls plague hypothesis generation: *directionality* involving the null and alternative hypotheses and *circularity,* where efforts to understand social factors have the effect of emphasizing racial differences. The proper prescription is to identify explicitly the hypotheses of interest, including their origins and implication.

Racial differentials in health are an urgent public health problem in the United States.

Death rates are 60 percent higher among blacks than among whites, a differential that has not changed detectably since vital records were first recorded at the beginning of the century (*1*). Epidemiology has played a central role in forming our responses to racial health differentials, primarily through its descriptive power. In this paper we examine the underpinnings of epidemiologic knowledge concerning racial differences.

Although the surveillance value of population studies is self-evident, the contribution of etiologic research based on racial comparisons seems much less clear. At the most fundamental level, we would argue, there is reason for concern about the nature of the enquiry being conducted.

Where Do Etiologic Hypotheses Come From?

The answers to two fundamental questions shed light on the process of scientific discovery and the accumulation of knowledge: (*a*) Where do hypotheses come from? and (*b*) How are hypotheses evaluated? Philosophers of science have actively debated for more than a century the question of how scientific conclusions are reached (*2,3*), and an appreciation of the structure of scientific reasoning has been integrated into the discourse of epidemiology (*4*). Questions about the origins of hypotheses, however, generally have been avoided by both practitioners and philosophers of science. Instead these issues have been addressed more fully by historians and anthropologists, who are interested in the ways that science, as a

Hypothesis

social institution, reflects general social values and paradigms (*5,6*).

Within general biological sciences, some attention has been paid to the origins of hypotheses, although this issue often has been regarded as the concern of a fringe minority (*7*). Epidemiology, despite its purported focus on people in a social context, has been almost entirely indifferent to the fundamental problem of how researchers choose what questions to ask. Only a smattering of thoughtful articles have sought to bring this issue into the arena of epidemiologic thought (*8-10*). A review of current standard texts in the field reveals no attention to the origin of hypotheses. We look at the process that leads epidemiologists to construct racial comparisons and exactly what these comparisons can test.

The conduct of epidemiology requires that we rigorously scrutinize the validity and interpretation of our observations, but how does one decide what to observe in the first place? An infinite quantity of observable information exists around us, yet we select a small subset on which to form our scientific judgements. How do we decide what is interesting and relevant? How does this selection limit interpretation of the outcome? Little has been said on this problem within the canon of the philosophy of science used by epidemiologists. Karl Popper, for example, left hypothesis generation and the selection of observations enshrouded in the mysterious realm of "inspiration" (*11*). Although Imre Lakatos described science as a "social process," he focused on the sociology of scientific progress, a pattern of behaviors by scientists that determine the course of their investigations and the ways in which their theories and discoveries are evaluated and compared (*12*). This construct addresses only one aspect of how social relations define the activities and products of scientists.

...our hypotheses cannot be independent of what we "know" from living in a society in which race is among the most fundamental of social divisions.

Scientists, like all human beings, have a set of beliefs about the natural world that inform the development of their scientific ideas. The answer to the question, "Where do hypotheses come from?" is, that in large measure, they come from ambient intellectual material: the subset of ideas to which we have access through membership in a particular society. Thus the process that creates research hypotheses exploits ideas from the particular field of research and others that are the product of our cultural, social, and economic systems. Just as the physical scientist cannot as yet travel to a parallel universe where another set of natural laws apply, social scientists cannot avoid being exposed to ideas about the natural world that are inherent in our economic and social systems.

Race and the Distribution of Disease

Although it is universally applied as a stratification variable, race has absolutely no scientific meaning. Like national boundaries, the definitions of human races are strictly social conventions, evolving over time and space to accommodate the flow of human history. Physical anthropologists have long since abandoned the quest for a schema that permits the classification of *homo sapiens* into subspecies (*13*). While accepting that news travels slowly across the interface of academic disciplines, it is worth noting that among the various fields that study human populations, only medicine and epidemiology stubbornly adhere to a biological interpretation of race (*14*).

Our beliefs about race and its relationship to health and disease are influenced by the particular mythology of innate racial differences that has evolved within our society (*15-16*). As epidemiologists, our hypotheses cannot be independent of what we "know" from living in a society in which race is among the most fundamental of social divisions. Our hypotheses concerning race cannot avoid reflecting our present system of social categories; scientists cannot transcend these categories because there are no objective criteria for racial classifications other than the "official" categories such as those used in the U.S. census (*17*). It is equally pointless to abandon these categories altogether, because they capture important information on differences in disease risk and etiology. A better alternative is to recognize that the social reality of race *causes* a differential

distribution of disease by racial category and that biological explanations of population differences tend to reflect ideas that are deeply rooted in our consciousness, including the belief in inherent differences (*18*).

The Paradigm of Racialized Thinking

The conventional wisdom is that blacks are more prone than whites to hypertension because they are genetically predisposed, and essentially all research on black hypertension addresses this hypothesis (*19*). Open recent issues of the most influential biomedical journals and you find statements such as "Blacks differ in many ways from non-blacks and these differences may explain their propensity to hypertension" (*20*). The logical structure of this argument is circular. When we set out to measure phenotypic traits in blacks and whites and find them "different," we conclude that blacks are "different" because they are black. This conclusion reinforces the belief that biology is consistent with the social definition of race—namely, distinctions on the basis of skin color. The rationale for racial criteria is rooted in the history of colonialism and slavery; there is no basis for a scientific theory which posits that skin color has an all-encompassing biological relevance.

To believe that skin color has a unique association to outcomes ranging from IQ to blood pressure to prostate cancer *by sheer chance* is a questionable, if not preposterous, proposition.

In practice, however, we observe that population distributions of disease vary on the basis of skin color. If we pick a physical characteristic other than race at random, we cannot replicate the same degree of variation in disease occurrence. Blood-type, for example, is easily measured and could provide an alternate classification scheme, but there are few population differences in the distribution of common diseases along this axis. To believe that skin color has a unique association to outcomes ranging from IQ to blood pressure to prostate cancer *by sheer chance* is a questionable, if not preposterous, proposition. Yet, given the waning interest in exploring the absurd hypotheses that melanin itself influences cerebral function, vascular tone, or DNA repair, what alternative explanation to chance can we pursue (*21*)? And how do we formulate appropriate hypotheses to allow us to pursue them?

Avoiding the Useful Hypothesis

Epidemiology journals are filled to overflowing with direct black-white comparisons. But what is the *testable* hypothesis underlying such a comparison? There are two obvious possibilities. A difference in biology, that is, a genetic predisposition, could be at the root of any observed differences.

Genetic effects can be tested by looking at monogenetic traits where the role of the environment is limited, as with sickle cell disease and cystic fibrosis. This class of diseases taken together, however, make only a modest contribution—around two percent—to total mortality in the United States. Their combined occurrence is held at a similar aggregate level in all populations by evolutionary forces (*22*). It is the common chronic diseases of adulthood that need to be better understood. Given their polygenic character and the determining role of environmental factors, cross-population comparisons of the genetic contribution with chronic diseases are well beyond our grasp at the present time.

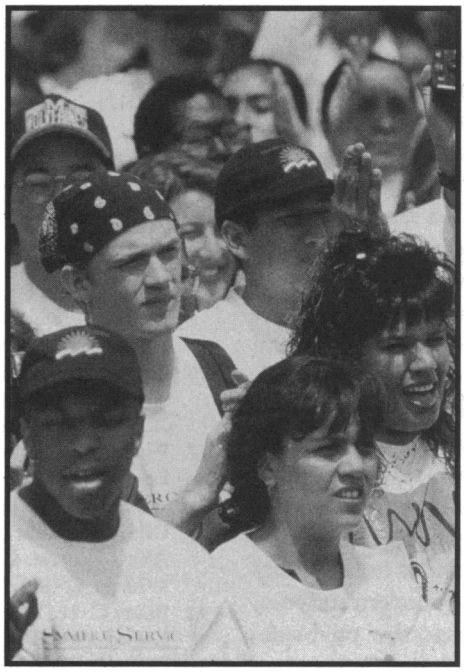
One could, of course, approach the gene-environment problem from the other side. Given a set of environmental exposures, researchers often attempt to determine what part of the between-group difference is explained by environmental exposures. Researchers have looked at topics as broad as the impacts of socio-economic status on mortality risk to as narrow as the effect of obesity on diabetes prevalence. What is the null hypothesis in such analyses? How much of the between-group differences *should* we expect to "explain?" It is not at all clear that by accounting for any given set of environmental factors we should eliminate a difference between groups. Nor do we know exactly how residual confounding and interactions will affect the interpretation of results. These designs pose many interpretive quandries. For example, how does the researcher interpret an outcome for which the risk ratio goes in the opposite direction after adjustment for social and environmental variables?

In effect, racial comparisons can be seen as an example of the ecological fallacy, where we have individual-level variables for some factors but not for others. In the end we can never know if the partcular environmental factors we were able to measure account for the differences observed. Because we cannot be sure that we have identified the important exposures, characterized them accurately, or addressed all interactive effects, this class of studies lacks testable, quantitative hypotheses. Yet how seductive in their simplicity are such black to white comparisons, which only serve to reinforce our system of social categorizations.

Directionality: the null and alternative hypotheses. There are two underlying pitfalls inherent in constructing hypotheses for black to white comparisons: *directionality and circularity*. Directionality has to do with the two mutually

exclusive propositions that form the classical Neyman-Pearson dyad: the null and alternative hypotheses. The first of this pair is the proverbial straw man, which is to be cast out by the "significant" result of the study, thus establishing the weight of evidence in favor of the specified alternative. The null hypothesis is supposed to embody the existing state of knowledge, which is then rejected in favor of some new finding. Nobody is particularly interested in failure to reject the null hypothesis, or so it would seem based on the difficulty of publishing such "null findings" (*23*). And this would appear to make sense, in the "logic of scientific discovery," since we do not need to be constantly informed of the existing state of knowledge.

The problem as it bears on the question of black to white comparisons is that the equality of groups is universally relegated to the null hypothesis. The assignment of group similarity to the status of current wisdom must be considered either a convenient fantasy or a disingenuous ploy, however, after decades of studies showing one "significant" black-white difference after another. More importantly, should one happen to be studying a biological system where the two groups were found to be indistinguishable, it would be difficult to publish this result because of this well-known bias against null findings. The end result of these processes is that scientific journals are full of the "proof" of essential racial difference.



Were we simply to invert the hypotheses and make group difference the null, it is easy to imagine that our journals would soon be full of articles discovering that, in one outcome after another, blacks and whites were in fact more similar than previously believed. The problem is only magnified by the habit of comparing a null hypothesis defined as a point ($a = b$) with a probability of zero to an alternative hypothesis that includes all other points ($a \neq b$), which therefore has a probability equal to one. With sufficient statistical power, therefore, it is *always* possible to demonstrate a black-white difference by using hypotheses constructed in this fashion (*24*).

Circularity. The second major pitfall of black to white comparisons is the problem of *circularity*. Consider, for example, the role of socioeconomic status as an explanatory variable. Although few would dispute the criticism that descriptive epidemiology lacks precision and that this lack of precision is a threat to the validity of between-population comparisons, a special niche has been created for studies that attempt to adjust for social status. After all, the argument goes, if health differentials between races are socially determined, they should disappear once social status is equalized. These studies of "social causation," however, can serve to reinforce the etiologic pathway that they seek to identify: social distinctions between races.

To study the hypothesis that societal discrimination leads to different disease profiles between blacks and whites, the researcher tests whether discrimination has created different environments that have imposed disproportionately greater risk of disease on blacks than on whites. The causal process begins with social distinctions by race, proceeds to differential exposures, and ends with differential disease levels. The process is circular, however, because its outcome (differential disease levels) confirms and reinforces the primary exposure (social distinctions). The results of such studies parallel the causal process being investigated: evidence is advanced for yet another difference between the groups, which forms the basis for underlying social exposure. In fact, the very existence of these studies further reifies the distinction between blacks and whites, contributing to the social underpinnings of racialism by focusing attention on racial differences.

Our prescription. What then is the solution to this problem of hypothesis specification? If it is useful to show subgroup differences in order to critique the existing social system and to identify population subgroups most in need of intervention, how can we avoid having the very conduct and results of our studies strengthening the social distinction between racial groups that was the problem in the first place? The answer to this question must at the very least begin with recognizing the need to make hypotheses *explicit* and to make *explicit* reference to the social forces at work, such as racial discrimination. Failure to identify these forces, often in the interests of maintaining a veneer of "scientific respectability," is the root cause of circularity in much of the research involving racial comparisons.

Circularity permits, if not directly contributes socially based racial distinctions. In the explicit terms of the social conflict, it is racist rather than anti-racist. We must draw the crucial distinction between studies that are racist and studies that are anti-racist by scrutiny of the specific study hypotheses. In studies that deal with race, simply cataloging differences is not helpful.

It is unfortunate, therefore, that references to underlying hypotheses all too often remain absent from published work in epidemiology. Vague statements such as "we hypothesize a difference in the outcome between blacks and whites" are not helpful either because they do not specify a process that might underlie such a difference.

Is it possible to study disease patterns in populations without falling into the directionality or circularity traps? We suggest, perhaps immodestly, an example from our own work that is based on cross-cultural comparisons. Comparing genetically similar populations across societies can free the researcher from the arbitrary nature of categorizations based on social conventions.

In the International Collaborative Study of Hypertension in Blacks, known by the acronym ICSHIB, we attempted to reduce the dependence on social categories by defining the sampling frame as specific populations of the African diaspora, thus avoiding the North American definition of blackness. This design controlled for population-level genetic differences and emphasized social processes at the societal level. Using a standardized protocol (*25*), a direct comparison of populations of common origin living in societies in Africa and the western hemisphere was undertaken. Cardiovascular disease risk ranged from among the lowest in the world to the highest as a function of measurable dietary, anthropometric, and psychosocial factors.

We attempted to identify formally the factors that predicted disease within each population. We then compared these factors across the socio-cultural transition from Africa to the United States in an attempt to account for the observed gradient in disease. Without the possibility of major genetic differences between sites, only environmental factors can logically account for the contrasts observed. Generalizing our findings to other populations, we conclude that if identifiable environmental factors can produce a range of hypertension prevalences from 7 percent in rural Africa to 32 percent in the United States within a single racial group, then there is little necessity to invoke population genetics. By comparing black populations from settings where the range of prevalence brackets all modern societies, our study considerably widens the perspective on black-white differences in hypertension in the United States.

Conclusion

This society nurtures a powerful myth that science is above the fray, an objective pursuit that can be neither racist nor anti-racist because it simply reveals the natural world. Comparative studies on racial differences in health outcomes belie this naive belief. Failure to address the context of research into racial differentials does not provide a safe solution; rather it puts one clearly on the side of accentuating group differences. The choice available to epidemiologists is to identify explicitly hypotheses as well as their origins and implications. Furthermore, as researchers, we have a complementary obligation to make the hypotheses explicit when reporting results. Otherwise we face the possibility that our work, filtered through the prevailing paradigms, will reinforce the very processes that we might have hoped to discredit.

Dr. Kaufman and Dr. Cooper are members of the Department of Preventive Medicine and Epidemiology, Loyola University Stritch School of Medicine, Maywood, IL.

The many provocative discussions with Dr. Sherman A. James and Dr. James S. Koopman helped to form the foundation for the ideas expressed in this article.

Tearsheet requests to Dr. Kaufman, Department of Preventive Medicine and Epidemiology, Loyola University Stritch School of Medicine, 2160 S. First Ave., Maywood IL 60153; tel: 708-327-9008; fax 708-327-9009.

References

1. Cooper, R. S.: Health and the social status of blacks in the United States. Ann Epidemiol 3: 137–144 (1993).
2. Popper, K.: The logic of scientific discovery. Hutchinson, London, 1959.
3. Kuhn, T. S.: The structure of scientific revolutions. Ed 2. University of Chicago Press, Chicago, 1970.
4. Rothman, K. J., editor: Causal inference. Epidemiologic Resources, Inc., Chestnut Hill, MA, 1988.
5. Foucault, M.: The archeology of knowledge. Pantheon, New York, 1972.
6. Foulcault, M.: The order of things: an archeology of the human sciences. Tavistock, London, 1970.
7. Lewontin, R. C.: Biology as ideology. Harper Perennial, New York, 1991.
8. Krieger, N.: Epidemiology and the web of causation: has anyone seen the spider? Soc Sci Med 39: 887–903 (1994).
9. Krieger, N., and Bassett, M.: The health of black folk: disease, class, and ideology in science. Monthly Rev 38: 74–85 (1986).
10. Krieger, N.: Shades of difference: theoretical underpinnings of the medical controversy on black/white differences in the United States, 1830–1870. Int J Health Services 17: 259–278 (1987).
11. Popper, K.: Conjectures and refutations. Routledge and Kegan Paul, London, 1963.
12. Lakatos, I.: The methodology of scientific research programmes. *In* Criticism and the growth of knowledge, edited by Imre Lakatos and Alan Musgrave. Cambridge University Press, Cambridge, England, 1970. pp. 91–196.
13. Brace, C. L.: The stages of human evolution. Ed. 4. Prentice–Hall, Englewood Cliffs, NJ, 1991.
14. Williams, D. R., Lavizzo–Mourey, R., and Warren, R. C.: The concept of race and health status in America. Public Health Rep 109: 26–41, January–February 1994.
15. Gould, S. J.: The mismeasure of man. Norton, New York, 1981.
16. Lewontin, R. C., Rose, S., and Kamin, L.: Not in our genes: biology, ideology and human nature. Pantheon Books, New York, 1984.
17. Lott, J. T.: Policy purposes of race and ethnicity: an assessment of federal racial and ethnic categories. Ethn Dis 3: 221–228 (1993).
18. Herrnstein, R. J., and Murray, C.: The bell curve: intelligence and class structure in American life. Free Press, New York, 1994.
19. Cooper, R. S., and Rotimi, C.: Hypertension in populations of West African origin: is there a genetic predisposition? J Hypertens 12: 215–227 (1994).
20. Kaplan, N. M.: Ethnic aspects of hypertension. Lancet 344: 450–452, Aug. 13, 1994.
21. Robins, A. H.: Biological perspectives on human pigmentation. Cambridge University Press, Cambridge, England, 1991.
22. Neel, J. V.: Physician to the gene pool: genetic lessons and other stories. John Wiley & Sons, New York, 1994.
23. Dickersin, K.: The existence of publication bias and risk factors for its occurrence. JAMA 263; 1385–1389, Mar. 9, 1990.
24. Oakes, M.: Statistical inference. Epidemiology Resources, Inc., Newton Lower Falls, MA, 1990, pp. 30–38.
25. Ataman, S. L., et al.: Standardization of blood pressure measurement in an international comparative study. J Clin Epidemiol. In press.