

Invited Commentary: "Race," Racism, and the Practice of Epidemiology

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Imagine that you are an epidemiologist investigating the health status of the city Metropolis. You document that the east side of the city has twice the prevalence of disease X as the west side of the city. You become interested in the determinants of disease X. How will you proceed?

1. Vigorously explore the basis of the east-west difference in the prevalence of disease X, using all currently available data and collecting new data as necessary.
2. Focus on understanding other determinants of disease X in Metropolis, adjusting for the side of the city from which subjects come or restricting analyses to just one side of the city.

Most readers would agree that option 1 is the preferable response to the findings in Metropolis. That is because option 1 treats the large east-west difference as an important clue to understanding the etiology of disease X, and it attempts to vigorously explore the basis of that difference. Option 2, on the other hand, merely treats the east-west difference as a confounder in understanding other determinants of disease X. Under option 2, the basis of the large east-west difference goes unexamined and therefore is likely to remain unknown.

So consider now how we as epidemiologists typically deal with differences in health status observed by "race." Race-associated differences in health outcomes are routinely documented in this country (1–4), yet for the most part their basis remains poorly explained. Instead of vigorously investigating the basis of these differences, we tend to simply adjust for race in our analyses or restrict our studies to a single "racial" group (5).

In this issue of the *Journal*, Kaufman and Cooper (6) provide guidelines for the appropriate use of race and ethnicity in etiologic research. However, their discussion is limited in two important ways. First, even as Kaufman and Cooper admit that race is a social construct and not a biologic reality (7, 8), they still conceptualize race as an individual characteristic and a matter of self-identity akin to ethnicity. Second, even as they thoughtfully assess the potential valid-

ity of five different uses of race by epidemiologists, they do not discuss how two of these uses that they judge to have high validity may actually diminish our ability to understand root causes of racial disparities.

In this invited commentary, I discuss the meanings of race and make the case that race is a contextual variable, not a characteristic of the person. I also examine the practice of epidemiology with regard to the use of race and place the five uses of race discussed by Kaufman and Cooper in the larger context of the questions that we decide or neglect to ask. Finally, I urge epidemiologists to take an interest in elucidating the underlying causes of race-associated differences in health outcomes and conclude with eight recommendations for practice that will move us toward that goal.

WHAT DO WE MEASURE WHEN WE MEASURE RACE?

The variable race is generally thought to measure some combination of social class, culture, and genes (9). Yet race is only a rough proxy for each of these. Consider the following examples for those labeled "Black" in this country. With regard to social class, Black people in the United States are overrepresented in poverty, but the majority of poor people in this country are White and not all Black people are poor. Black race therefore serves as only a very rough proxy for poverty. With regard to culture, people who are labeled Black in this country may have just arrived from Ethiopia, recently immigrated from Haiti, or been raised in the rural South or the urban North and have very different cultures with regard to diet, physical activity, and other health-related practices. There is no single Black culture, just as there is no single White, Hispanic, or Asian culture. With regard to genes, it is clear that people who are labeled Black in this country represent a genetic admixture of geographic stocks from all over the world. (Indeed, people who are labeled White in this country also represent a genetic admixture from many parts of the world.) In addition, an Aboriginal person from Australia, a Zulu person from South Africa, and a Kikuyu person from Kenya are all labeled Black in this country, yet they arise from very different geographic genetic stocks. There is no denying that there is genetic variability on the planet. However, the pie slicer that we call race does not capture that genetic variability (10).

ACKNOWLEDGING AND MEASURING RACISM

If race is only a rough proxy for social class, culture, and genes, why is it such a good predictor of health outcomes in

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the United States? It is because the race that we measure in our studies is the same race that is noted by a taxi driver, a police officer, a judge in a courtroom, or a teacher in a classroom. That is, race is a social classification in our race-conscious society that conditions most aspects of our daily life experiences and results in profound differences in life chances (11).

We are all born with a heritage. We have parents, grandparents, and great-grandparents behind us who give us both a genetic heritage and a cultural heritage (ethnicity), but we are assigned a race in this country. We learn our race in early childhood and it becomes part of our self-identity. However, it is clearly a contextual variable, not something inherent to the person.

This assigned race varies among countries. For example, in the United States I am clearly labeled Black, while in Brazil I would be just as clearly labeled White and in South Africa I would be clearly labeled "colored." It is likely that, if I stayed long enough in any one of these settings, my health profile would become that of the group to which I had been assigned, even though I would have the same genetic endowment in all three settings.

In addition, this assigned race may vary over time. It is instructive to examine the changing racial categories used in the United States decennial census from 1790 to the present (12). For example, in 1790 I would have been counted as a slave, in 1850 as either Black or "mulatto," in 1890 as one of Black, mulatto, "quadroon," or "octoroon" ancestry, in 1950 as "Negro," and in 2000 as "Black, African American, or Negro," plus "White" and "American Indian or Alaska Native" if I so chose. These and other changes on the census reflect changes in political climate and patterns of immigration. The only category that has remained constant over the history of the census is White, and even this is a constructed category that has had variable criteria for membership (13).

Race is a social construct, a social classification based on phenotype, that governs the distribution of risks and opportunities in our race-conscious society. Although ethnicity reflects cultural heritage, race measures a societally imposed identity and consequent exposure to the societal constraints associated with that particular identity. That is, the race that an investigator notes or a study subject has learned to self-report is an excellent measure of exposure to racism. Perhaps it is this aspect of race that profoundly impacts health and results in race-associated differences in health outcomes that are large in magnitude, occur across the life span, and involve many different organ systems.

LEVELS OF RACISM

If, indeed, racism is a root cause of observed race-associated differences in health outcomes, it is vitally important that we develop a detailed understanding of the characteristics and manifestations of racism. I describe three levels of racism, institutionalized, personally mediated, and internalized, each of which can have an impact on health (14). Understanding these three levels is useful to epidemiologists and other public health practitioners for generating hypotheses about the basis of race-associated

differences in health outcomes, as well as for designing interventions to eliminate those differences.

Institutionalized racism is defined as differential access to the goods, services, and opportunities of society by race (14). It is structural, having been codified in our institutions of custom, practice, and law so there need not be an identifiable perpetrator. Indeed, institutionalized racism is often evident as inaction in the face of need. Institutionalized racism manifests itself both in material conditions and in access to power. With regard to material conditions, examples include differential access to quality education, sound housing, gainful employment, appropriate medical facilities, and a clean environment. With regard to access to power, examples include differential access to information, resources, and voice.

It is important to note that the association between socioeconomic status and race in the United States has its origins in discrete historical events but persists because of contemporary structural factors that perpetuate those historical injustices. In other words, it is because of institutionalized racism that there is an association between socioeconomic status and race in this country. Pathways through which institutionalized racism impacts health include socioeconomic status and access to health care.

Personally mediated racism is defined as prejudice and discrimination, where prejudice is differential assumptions about the abilities, motives, and intents of others by race, and discrimination is differential actions toward others by race (14). This is what most people think of when they hear the word, racism. Personally mediated racism can be intentional as well as unintentional, and it includes acts of commission as well as acts of omission. It manifests as lack of respect, suspicion, devaluation, scapegoating, and dehumanization. Pathways through which personally mediated racism impacts health include the stresses of everyday racism (15) and differential treatment within the health care system.

Internalized racism is defined as acceptance by members of the stigmatized races of negative messages about their own abilities and intrinsic worth (14). It involves accepting limitations to one's own full humanity, including one's spectrum of dreams, one's right to self-determination, and one's range of allowable self-expression. It manifests as an embracing of "whiteness," self-devaluation, and resignation, helplessness, and hopelessness. Pathways through which internalized racism impacts health include fratricide and adoption of risky health behaviors.

The relations of institutionalized racism, personally mediated racism, and internalized racism (which taken together produce the racial climate) to health outcomes are illustrated in figure 1.

THE PRACTICE OF EPIDEMIOLOGY

Figure 2 diagrams the practice of epidemiology with regard to the use of race. The first decision we make is whether or not to collect data by race. Many investigators make the decision, "yes." I speculate that epidemiologists in the United States routinely collect data by race for the following reasons:

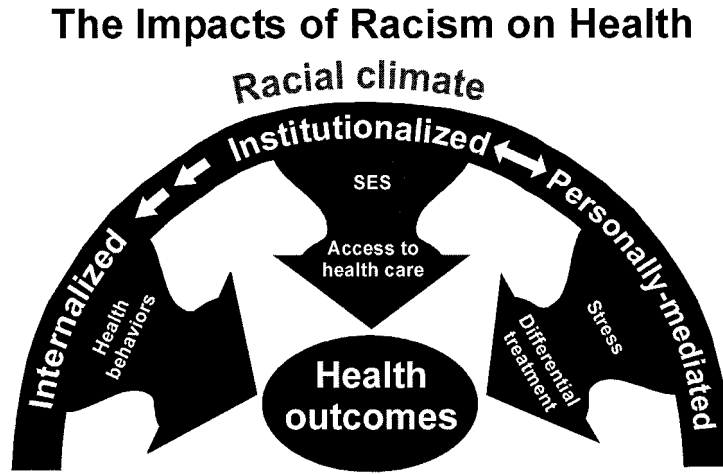


FIGURE 1. The impacts of racism on health, illustrating the relation between institutionalized racism, personally mediated racism, and internalized racism and various factors that contribute to race-associated differences in health outcomes. SES, socioeconomic status.

1. This is what we are taught to do—it is the customary practice. The routine stratification of data by race is modeled and reinforced ubiquitously in our society, including in our introductory epidemiology courses

and textbooks. This is in contrast to the customary practice in Canada and Great Britain, where data are routinely collected by social class.

2. Race is thought to be easy to measure. In fact, epi-

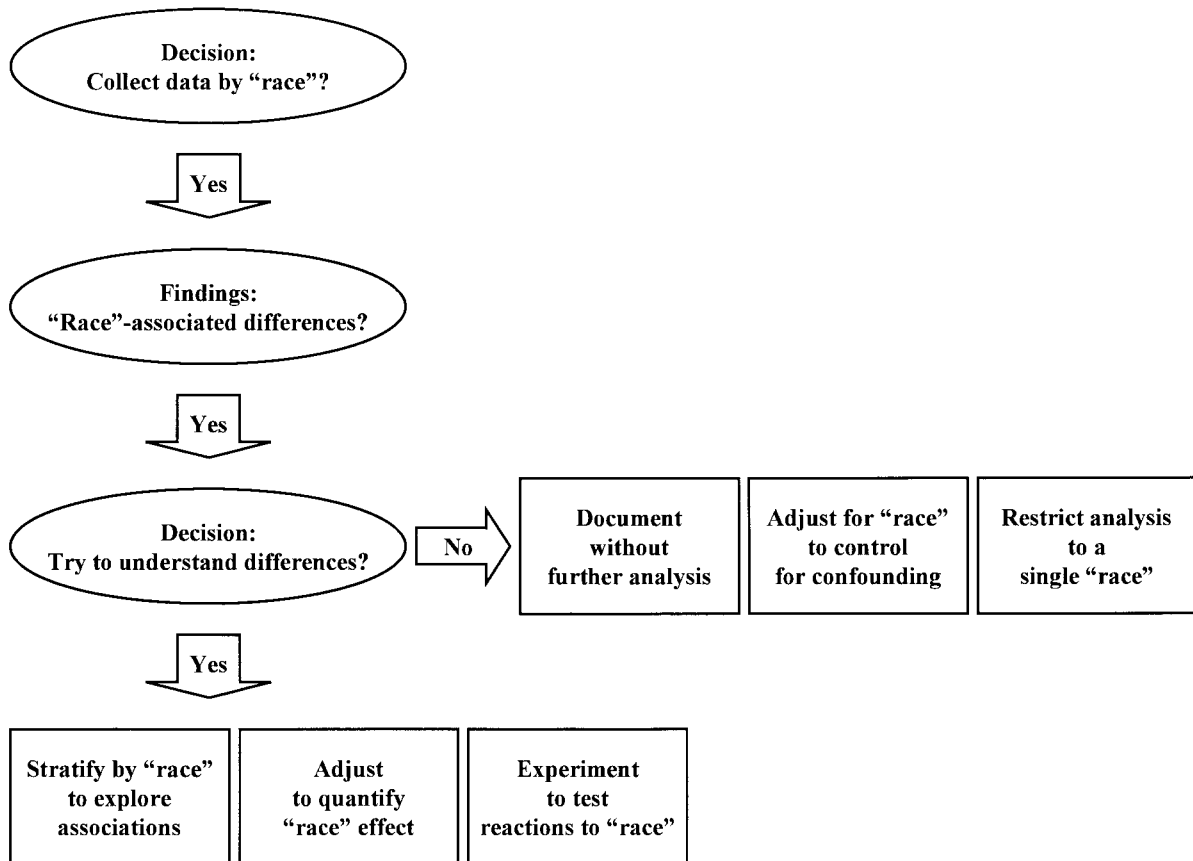


FIGURE 2. The uses of race by epidemiologists: decisions regarding the use of race by epidemiologists, including design and analytical strategies associated with these decisions.

demiologists are rarely trained in how to ascertain race, and guidelines are rarely given to subjects who are asked to self-identify race. It is assumed that anyone who grew up in the United States will know how to make the proper classifications. Interestingly, persons who did not grow up in the United States often express confusion about how to make the appropriate racial assignments.

3. Race is thought to be stable over time. However, the discordance between race on birth certificates and race on death certificates has been well documented (16). Indeed, discordance on a single medical record has been noted for some patients who have been alternately described by different observers as Black, White, and Latino (personal observation).
4. Race is indeed a good predictor of health outcomes in this country. This is true across many disease outcomes and across the life span and has been true since health data have been collected in this country.

There is increased discussion about the advisability of continuing to collect data by race, because that practice reifies race as a biologically important variable (9, 17–20). However, when we collect data by race, our findings most often reveal significant race-associated differences in health outcomes. This is important information.

The second decision we make is whether or not to try to understand the basis of the observed race-associated differences. Many investigators make the decision, “no.” I speculate that we do not vigorously investigate the basis of race-associated differences in health outcomes for the following reasons:

1. We assume race-associated differences to be unchangeable. The idea that racial differences are genetically based is widely held by the American public. Because scientists are affected by the society in which they live (21), many also infer that race-associated differences are genetic and therefore immutable. This notion of biologic determinism may inhibit efforts at vigorous investigation of race-associated differences.
2. Race-associated differences are not of primary interest to us. The majority of epidemiologists in this country are White (22). These researchers may not feel a personal stake in the racial disparities that for the most part adversely impact populations of color.
3. Race-associated differences do not surprise us. A “non-White” excess in disease burden in this country has almost grown to be expected. The differences are so ubiquitous across organ systems, over the life span, and over time that they do not surprise us or seem to require explanation. Indeed, only when there is a White excess in disease burden, as with suicide, is our professional interest piqued. It is as if these differences were endemic and do not elicit the response that would be elicited by an epidemic above the background rate.
4. The basis of race-associated differences seems intractable to us. Many health researchers may acknowledge that the basis of race-associated differ-

ences in health outcomes deserves attention, but the untangling of the reasons for the differences seems too hard, and they choose instead to devote their careers to a problem that seems more tractable to solution.

Our common practice of routinely documenting race-associated differences in health outcomes but leaving the basis of those differences poorly explained is not benign but has at least three dangerous consequences (23). It impedes the advance of scientific knowledge, limits efforts at primary prevention, and contributes to ideas of biologic determinism. Scientific understanding is robbed when clues embedded in large group differences are not mined. Efforts at primary prevention are stymied when one can only screen and treat populations defined by race rather than prevent the onset of disease by addressing root causes. The ideology of race as a biologic determinant is bolstered when scientists fail to probe the basis of race-associated differences as though this basis were already completely understood.

DESIGN AND ANALYTICAL STRATEGIES

Three strategies derive from a decision not to vigorously investigate the basis of observed race-associated differences in health outcomes (see figure 2):

1. Document race-associated differences without further action. This is often evident in our surveillance efforts, where we collect whole libraries of tables and graphs documenting racial disparities over time. This practice identifies areas in need of further inquiry, but it does not advance our understanding of the basis of the differences. Kaufman and Cooper find that “describing disparities between racial/ethnic groups” has high potential validity (6, p. 296).
2. Adjust for the confounding effects of race on the relation between some other factor of interest and disease. This practice is commonly used (5) and treats race-associated differences as nuisance confounders rather than as important clues to be mined. Kaufman and Cooper find that “statistical adjustment for race/ethnicity in estimating the causal effect of another variable of interest” has high potential validity (6, p. 296).
3. Restrict our analyses and subsequent studies to a single racial group. This practice is also commonly used (5) and belies a total lack of interest in both the causes of the observed differences and the epidemiology of disease in other racial groups. Kaufman and Cooper do not address this research approach.

Three other strategies derive from a decision to actively investigate the basis of observed race-associated differences (see figure 2):

1. Stratify our data by race to explore the contributing factors to the differences. We can compare distributions of candidate risk factors by race (24). We can also do separate regressions on candidate risk factors and the disease of interest, compare the risk factor profiles by race, and try to explain differences in risk

factor profiles. This strategy treats race, like the side of the city on which people live, as a marker for differential experiences and exposures rather than as a factor inherent to the person. Kaufman and Cooper do not address this research approach

2. Adjust to quantify the magnitude of the race effect. This strategy does not really improve our understanding of the basis of race-associated differences, but it is often used by those who are trying to explain away race as a risk factor. Notably, Kaufman and Cooper find that the approaches where the "effect of race/ethnicity is internal to the individual study participant" and where "effect decomposition [is done] to separate direct (biologic) effects of race/ethnicity from indirect effects (relayed through social variables)" both have low potential validity (6, p. 296).
3. Conduct experiments to test reactions to race. This design strategy acknowledges the role of reactions to race in causing race-associated differences in health outcomes. Examples include trials that vary the race in case presentations (25, 26) or use testers in applying for jobs or mortgages. Kaufman and Cooper find that approaches where the "effect of race/ethnicity is external to the individual study participant" have high potential validity (6, p. 296).

Of the five research approaches discussed by Kaufman and Cooper, three were found to have high potential validity, and two of these are included among the strategies of researchers who document race-associated differences but are not interested in investigating the basis of those differences. I do not believe that these authors are calling for epidemiologists to document differences by race without further analysis, nor do I believe that they are they condoning the practice of adjusting for race without taking an interest in the existence and underlying causes of race-associated differences. However, the commentary by Kaufman and Cooper highlights the need for epidemiologists to understand our work in a larger social context. It is just as important to be sure of the validity of our research approaches in a narrow sense as it is to consider the kinds of questions that we are trying to answer and the ones we are neglecting.

RECOMMENDATIONS FOR PRACTICE

I make the following recommendations based on the preceding discussion of race, racism, and the practice of epidemiology:

1. Vigorously investigate the basis of observed race-associated differences in health outcomes.
 - a. Interpret all race-related findings instead of simply reporting them without comment or simply adjusting for race.
 - b. If there are not enough data to explicate a difference that is documented in a given study, propose follow-up studies.
 - c. View race-associated differences as important clues to be mined.
2. Acknowledge that race is a social construct, not a biologic determinant.

- a. Explicitly measure genes if there is a genetic hypothesis.
- b. Model race as a contextual variable in multilevel analyses.
3. Acknowledge the diversity within racial groups.
 - a. Explicitly measure culture if there is a cultural hypothesis.
 - b. Collect information on ancestry, migration history, and language.
4. Acknowledge the association between race and social class in this country, an association perpetuated by institutionalized racism.
 - a. Explicitly measure social class if there is a social class hypothesis.
 - b. Broaden the conceptualization of social class to include issues of accumulated wealth, neighborhood characteristics, and measures of social class over the life span.
 - c. Develop valid and reliable social class measures that could be included on all birth and death certificates, as well as on other data reported to federal and state agencies.
5. Acknowledge the present-day existence and impacts of racism.
 - a. Develop explicit measures of institutionalized, personally mediated, and internalized racism.
 - b. Examine the role of racism in causing race-associated differences in health outcomes.
 - c. Examine the role of racism in diminishing the health of the entire population, not just the health of the stigmatized races.
6. Continue to collect data by race as long as there are race-associated differences in health outcomes. It is important to monitor disparities as we aim to eliminate them. However, be precise when using the word "race."
 - a. Specify why information on race was collected (e.g., because of a previously documented disparity).
 - b. Describe how race was measured (observer-coded vs. self-reported, the number and names of categories, whether multiple responses were allowed).
 - c. Collect enough other data, including measures of racism, social class, culture, and genes, so that the basis of observed differences can be determined.
7. Train persons from stigmatized backgrounds as epidemiologists (22). In this way, our understanding of the determinants of the health of populations will be enhanced.
 - a. These scientists will bring new perspectives to the questions we have already asked.
 - b. They will also raise new questions.
8. Partner with communities to raise questions, generate hypotheses, and share findings.
 - a. Recognize and respect the capacity within communities to bring important insight to scientific research.
 - b. Return information to communities so that they can mobilize and advocate for change.

The distribution of risks and protective factors that are differentially distributed by side of the city in Metropolis can be identified and addressed. In the same way, the structures that govern the distribution of risks and protective factors by race must be identified and addressed. I urge our profession to pay

focused attention to understanding the root causes of race-associated differences in health outcomes. We will need to raise new questions with renewed energy. We will need to understand that these racial disparities represent opportunities to increase our scientific understanding of many disease processes, to succeed in primary prevention rather than just screening and treating vulnerable populations, and to combat ideas of biologic determinism that shape public attitudes about the possibility of change. We need to participate in a growing national conversation on racism and to provide the scientific basis for truly understanding how to eliminate racial and ethnic disparities in health by the year 2010 (27, 28).

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