



Invited Commentary: Social Mechanisms, Race, and Social Epidemiology

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In the leading article in this issue of the *Journal*, Kaufman and Cooper (1) attempt to elucidate why social epidemiology has allegedly failed to provide explanations for the associations between social factors and disease. Their main argument is that the assumptions of counterfactual models routinely used in observational studies are not adequate for testing social factors. The authors illustrate their argument with the concept of race, on the grounds that using race in counterfactual models requires the assumption that a Black person could be thought of as being White—which the authors argue is not conceivable. In this commentary, I argue that the main reason why social epidemiology has not provided better explanations is lack of *social* theory development, due mainly to the reluctance of epidemiologists to think about social mechanisms (e.g., racial exploitation). I support this point of view with an analysis of the role of counterfactual models in social science, an illustration of the fundamental similarities between epidemiology and other sociocultural sciences, and the failure of contemporary epidemiology to generate hypotheses about social mechanisms. In particular, I point to the notion of racial social systems, the concept of Whiteness, and its measurement implications to highlight the lack of attention to social mechanisms in social epidemiology. However, I do not examine the concept of race understood as a collective response to the experience of racial ideology, oppression, and exploitation.

Counterfactual models can be useful in social science

The integration between theory and methods is an important and often neglected aspect of social science

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(2). One of the founders of quantitative methodology anticipated the lack of progress that would result from the rush to apply new statistical methods without attention to social mechanisms (3). Kaufman and Cooper (1) convincingly argue that social epidemiology, like most observational epidemiology, relies too much on the counterfactual model of causation, a model with stronger assumptions than are permissible in its conventional application to many epidemiologic problems. They also maintain that counterfactual models are ill-suited to social epidemiology because social concepts such as race make counterfactual assumptions implausible. While implausible counterfactual arguments might lead to misleading inferences, counterfactual models have been successfully applied in social sciences that are substantively close to epidemiology. Counterfactual conditionals are used in social science to express what would have occurred if something else had happened, when in fact it did not—such as hypothesizing about historical events that were possible but never took place. For example, counterfactual history has been used in social policy to estimate demographic histories under the assumption that certain events such as migrations or wars did not happen. Even if counterfactual models require imagining events that did not take place, they have been widely used to plan policies with regard to migration (4).

More to the point, counterfactual models have also been productively applied in the study of racial inequality, precisely the domain chosen by Kaufman and Cooper to exemplify their contention that counterfactual models are inadequate for social epidemiology. Calculation of counterfactual proportions has been used to study economic discrimination, as in the assessment of the “Black-White” gap in unemployment rates, where counterfactual proportions are interpreted as the proportion of Blacks who would have been in each employment group if they had been treated like Whites (5). Thus, in spite of their limitations, counterfactual models are useful in social sciences where experiments are seldom possible (6), because they propel creative social hypotheses and inform the construction of social policies, such as those envisioning social systems without racial or class stratification.

Are social epidemiology and mainstream epidemiology really so different?

A second question arising from Kaufman and Cooper's article is why *social* epidemiology was singled out for criticism when lack of causal explanations is a common criticism directed toward the discipline of epidemiology as a whole (7). In many areas of epidemiology, the most basic association of the discipline, the relation between age and disease, remains devoid of good explanations. For example, the relation between depression and age remains largely unexplained in psychiatric epidemiology (8).

In addition to the limits of social epidemiology's ability to provide explanations, Kaufman and Cooper also point to several common violations of assumptions incurred by social epidemiologists during the application of counterfactual models, such as the assumptions of stable exposure effects and independence of outcomes. As the authors themselves point out, this problem is hardly unique to social epidemiology. The near impossibility of assuming that exposure must be identical for every unit, and the assumption that outcome for any given unit is independent of the outcome for other units, are shared features of observational studies in epidemiology at large. So is the fallacy of attempting to isolate independent effects in standard regression models with multiple covariates in observational studies, a common problem in social science as well. Public health researchers have in fact been borrowing methods from social sciences that deal with some of these problems (e.g., the use of latent variables for measurement error, path analysis for modeling effects between explanatory variables, and multilevel modeling for unit and outcome interdependence) (9–12). Even if these problems are general to observational epidemiology, the insightful recommendation that data analysis should begin with quantifying the joint distributions of exposures and outcomes, rather than predicting outcomes under unrealistic assumptions, should be taken seriously by social epidemiologists. However, that important and convincing recommendation does not address what the authors identify as the central problem of social epidemiology, namely the lack of causal explanations, because causal explanations start with hypotheses and theory rather than with methods of observation and data analysis (13).

Thus, if overuse of counterfactual models is not a unique feature of social epidemiology, why is this discipline singled out? One answer is that despite its current increase in recognition (14), social epidemiology is a contested discipline, as the relevance of studying social facts within the field of epidemiology is still being intensely debated (15–21). Epidemiologists still

clash over their discipline's status as a *socionatural* science. This is a long-standing issue; the tension between the social and natural sciences within epidemiology has been a characteristic of its history, from studies of the social epidemiology of pellagra (22) to attempts to redefine public health as clinical preventive medicine during a period in which the institutional ties between medicine and public health are being reinforced (23). Therefore, the uncertain status of social epidemiology as a subdiscipline within epidemiology (24) makes it an easier target for criticisms that could be addressed to other areas of epidemiology as well.

After assuming that social epidemiology is firmly established within the field of epidemiology, Kaufman and Cooper argue that the epidemiologic method is ill-suited for considering social risk factors. This emphasis on the distinctiveness of the "epidemiologic method" eschews the fundamental unity of methods used in the biosocial sciences (e.g., in such fields as demography, human geography, the sociology of health and illness, and health psychology), including their recourse to counterfactual arguments (6). An emphasis on the uniqueness of the epidemiologic method contributes to: 1) narrowing of the scope of epidemiology to methodology (25, 26); 2) concealing the fundamental unity of the scientific method (i.e., background, problem, and hypothesis, in addition to testing, evaluation of the hypothesis, and dynamic revision based on any of the previous steps) in favor of a multiplicity of methods tied to epidemiology (24); and 3) isolating epidemiology from other disciplines that generate explanations for social facts such as class and race (25–28). For example, demography, often thought of as a branch of sociology, does not encounter the same need to justify its inquiry into social facts (29). Both epidemiology and demography are *socionatural* sciences that share many characteristics, among them a research community (e.g., in schools of public health), a set of research techniques such as surveys and statistical models, and a domain of biosocial concepts such as mortality rates, life expectancy, and birth rates.

Whiteness as exposure: the health effects of racial ideology, exclusion, and discrimination

In general, epidemiologists have been holding to a biologic theory of race that has been rejected in adjacent disciplines (30, 31). Even when race is not explicitly used as a biologic concept (32), racial categories that are not informed by social mechanisms leave room for multiple interpretations, including biologic or cultural notions of race as an essential and unchanging constituent of a person (30, 33–36). The claim that counterfactual models are inadequate for the study of

race because race is an “unalterable characteristic” falls within this tradition, because race is defined as a constituent attribute of organisms rather than as a social fact. Therefore, Kaufman and Cooper are correct when they argue that the use of race as an exposure in counterfactual models is implausible because “if the exposure is an attribute of u , we cannot contemplate the same unit in the unexposed state” (1, p. 115). However, contrary to the authors’ claim, what renders implausible the use of race as an exposure in counterfactual models is not a logical contradiction (i.e., “a Black person who is not Black cannot be considered to be the same person” (1, p. 115)) brought about by counterfactual models’ inability to incorporate social facts, it is the authors’ definition of race as a nonmodifiable attribute of organisms.

On the other hand, when we consider race as a social fact (37, 38), the use of counterfactual arguments becomes plausible: A person with dark skin can be exposed (or not) to Whiteness, that is, to the set of exposures involving racial ideology, political exclusion, and economic discrimination that is fostered, mostly indirectly, by Whites. If race is a social relation, “Blackness” is not an inalterable feature of an organism but a contingent outcome of changing social relations. Therefore, the use of “Blackness” (or exposure to Whiteness) in epidemiologic counterfactual models might be appropriate. A person can indeed change “Blackness” position and its associated cultural, political, and economic exposures in a lifetime, depending on the social system in which he or she is located. For example, an individual with both Black and White color features (i.e., *parda*) who could be classified as White (i.e., *branca*) in Brazil could become Black after migrating to the United States, where the category White is restricted to people with white skin color (39). As social relations change with time and place, the degree of exposure to Whiteness can vary across countries and periods. Less Black exposure to racial ideology, exclusion, and discrimination could account for the lack of a Black-White gap in preterm birth observed in Cuba several years ago (40). Individuals’ long term exposure to different racial social systems might determine the long term effect of “race” on health. The confluence of migration and racial social systems seems to induce multiple racial identities among people who, on the basis of skin color alone, would be categorized simply as “Black” or “White” (41).

Conceptualizing race as an unalterable attribute also has important measurement consequences. A young Dogon who migrates from the Bandiagara escarpment (Mali) to New York would not have been exposed to Whiteness; therefore, simply categorizing him or her as Black upon arrival in the United States would pro-

duce measurement error. Moreover, the sharp distinction in epidemiology between race as an unchangeable attribute and social class as a modifiable one (42) does not consider the fact that people can change racial systems while remaining in similar class positions (43) or that there is substantial class immobility in Western industrialized countries (44).

When race is approached as a social relation, the category “Black” stops being a mysterious black box of potential biologic vulnerabilities or unmeasurable exposures leading to indeterminism (i.e., the view that the problem is too complex to be understood) or speculation. Suggestions of innate racial predispositions in epidemiologic studies are the consequence not of an inappropriate use of counterfactual arguments but of the lack of attention to social theory (27) and the persistence of implicit notions of race that have been rejected in adjacent disciplines (30). The associations between “race” and health might be viewed as reflections of cumulative exposures to racial ideology, economic discrimination, and political exclusion. Cumulative exposures to racial social systems might appear incommensurable, as when there are “myriad ways, reflecting the complex pathways... thought to affect or determine exposures” (1, p. 113). Nevertheless, it is precisely the task of social epidemiologists to generate hypotheses on the social mechanisms whereby racial systems affect health (e.g., the lifetime institutional discrimination in promotions enforced by managers through differential weighting of negative nonracial characteristics among Blacks).

Why race is not considered a social fact

Race as a social fact could be defined in epidemiology as a set of social relations that are a subset of the structure of a social system: a hierarchical relation between White and Nonwhite populations that produces ill health among Nonwhites through economic, political, and cultural relations. The reluctance to adopt a social concept of race in epidemiology and medicine has multiple potential determinants, several of which I highlight below.

First, racial ideology (i.e., racism) is viewed exclusively as a psychological property of individuals, a fact that precludes investigation of race as an economic or political relation in social institutions such as firms, neighborhoods, or nations (38).

Second, racism is conceived of as an “irrational prejudice” rather than an ideology that promotes objective economic and political benefits for the dominant race, also known as the “wages of Whiteness” or “White skin privilege” (38). For example, Whites’ wages decline less than Blacks’ when they live in areas where the aggregate unemployment rate is high (45).

Third, racism is explained by reference to a remote historical past rather than in relation to the structure of current racial social systems (38). Slavery in the US South produced a strong interdependence between racial ideology and economic exploitation (46). However, the fact that slavery as an economic system has been largely eliminated in the United States does not constitute a justification for eschewing research into the health effects of modern forms of racial ideology that persevere under a different economic system (38). For example, during the 1970s and 1980s, the declining numbers of manufacturing jobs, the suburbanization of employment, and the growth of low-paying service jobs produced rising rates of poverty and income inequality that were intensified among Blacks by racial segregation (47).

Fourth, racism is considered to be static, unchanging with time (38). Thus, assuming that the prevailing notions of racial hierarchy are still based on biologic concepts (e.g., lineage, type, subspecies) precludes the examination of contemporary racial ideology, which relies on the preservation of cultural differences, appeals to abstract notions of "equality of opportunity" in the face of objective racial inequality, and nationalism (38, 48, 49).

Fifth, racial *economic* discrimination and racial *political* exclusion are viewed as overt behavior, when the evidence points toward a larger role for indirect and passive behaviors in institutional settings (38, 50, 51). For example, liberal Whites (i.e., those who endorse public policies designed to combat racism and promote racial equality) have been shown to be as likely as conservative Whites (i.e., those who do not hold those views) to discriminate against Blacks when the situation does not involve direct prejudice as the basis for their discriminatory actions (52). Social psychologists have also found that liberal Whites who hold negative attitudes about Blacks (such as discomfort, disgust, uneasiness, or fear) tend to express them indirectly or in situations where their liberal self-image is not threatened (52). Institutional racial ideology, discrimination, and exclusion are manifested in policies and norms such as law enforcement brutality, disparate sentencing, and denial of access to education by the government; housing segregation by real estate companies; denial of access to employment markets by businesses; and redlining and denial of credit by banks (47, 50, 51, 53, 54).

Conclusion: so far from social

The above arguments and examples support the view that it is not primarily the overuse of counterfactual models that lies at the core of the lack of explanations in social epidemiology. The problem lies one step prior

to that in the scientific method, the method of epidemiology and social science alike—namely, in a lack of understanding of the role of social mechanisms in the health effects of class, gender, and race. A plausible reason for the lack of explanations in social epidemiology is the attachment to an empiricist philosophy that searches for empirical generalizations (e.g., using observations to build models) while avoiding conjectures about the underlying social mechanisms that would help us understand how social systems work. This is why epidemiologic studies typically present coefficients associated with categories of race rather than with measures of exposure to economic discrimination, which is an explicit social mechanism rather than an assumed fixed attribute of organisms.

Generation of causal explanations in social epidemiology would require abandoning the Humean notion of causality (55) and adopting a realist philosophy that favors generating social theory in addition to observation (56). The role of theory is precisely to go beyond "what we can observe." For example, Skinnerian operant behaviorism is useful for gaining experimental control over behavior in applied psychology (57), but it ultimately needs unobservable constructs to develop explanations for why behavior occurs (e.g., maximizing and matching theories (58)). Similarly, no one will ever directly observe institutional economic discrimination; we can only measure indicators such as firm-level indices of wage inequality between Black and White workers. To acquire depth, any science, not only epidemiology, needs to go beyond observations, because our senses are too limited (56). In the absence of social mechanisms with which to explain the health effects of exposure to racial ideology (59), economic discrimination, and political exclusion, improvement in the way epidemiologists conduct data analysis of Black and White categories will still yield results on "racial differences" that contain implicit or explicit speculations about race, genetics, behaviors, and health. Important as the appropriateness of methods of data analysis might be to the epidemiology of race, gender, and class, it needs to be preceded by the generation and testing of hypotheses on social mechanisms. Testing of realistic social mechanisms will ultimately provide the explanations that social epidemiology needs to grow as a discipline.

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