Invited Commentary


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Racial and ethnic inequalities in blood pressure and hypertension have been well documented, but their causes remain unclear, making efforts to reduce these inequalities challenging. In this issue of the Journal, Basu et al. (1) address this gap in our knowledge by using an econometric approach to examine the role of 4 conventional risk factors for hypertension. Their results suggest that targeting certain risk factors will reduce racial inequalities in the prevalence of hypertension. However, racial differences in modifiable risk factors are enmeshed within disparate socioenvironmental contexts which are in turn determined by inequalities in the distribution of social, economic, and political resources and constraints. A small but growing body of literature suggests that targeting the intermediate risk factors that link racial group membership to hypertension, rather than the context or the inequalities in the distribution of resources and constraints, will ultimately result in little change in hypertension inequalities, increase these inequalities, or even create inequalities in poor mental health.

Abbreviation: BMI, body mass index.

Racial and ethnic inequalities in blood pressure and hypertension have been well documented; however, their causes remain unclear, which makes efforts to reduce these inequalities challenging. In an article in the present issue of the American Journal of Epidemiology, Basu et al. (1) address this gap in our knowledge by using an econometric approach to examine the role of 4 conventional risk factors for hypertension (smoking, alcohol use, sodium intake, and body mass index (BMI)) in blood pressure distribution inequalities between black and white participants and between Mexican-American and white participants. The authors reported that accounting for these 4 risk factors together did little to reduce the disparities between the systolic blood pressure distributions of black and white participants and between Mexican-American and white participants. The authors reported that accounting for these 4 risk factors together did little to reduce the disparities between the systolic blood pressure distributions of black and white men because of the opposite effects of sodium intake and smoking. The authors also reported that accounting for these risk factors reduced the blood pressure disparities in women, particularly at the upper end of the distribution. This result was primarily due to adjustment for BMI, because sodium intake and smoking also operated in opposite directions. When focusing on these specific risk factors, the results imply that public health interventions target racial inequalities in BMI (for women) and sodium intake without affecting the distribution of any of the other risk factors, particularly smoking. In the present commentary, I will integrate the results of Basu et al. into the larger literature on racial inequalities in health and discuss the challenges of targeting specific risk factors that link racial group membership to health (hypertension in this case) in an effort to eliminate health inequalities. Because of space limitations and the different social contexts and histories of black and Mexican-American persons, I will focus on comparisons of black and white subjects.

Racial differences in modifiable risk factors are enmeshed within disparate socioenvironmental contexts that are in turn determined by inequalities in the distribution of the balance of social, economic, and political resources and constraints (2, 3). A small but growing body of literature suggests that reductions in risk factors without concomitant improvements in the contextual circumstances that drive these risk factors will result in the development of new racial inequalities in
poor mental health (3, 4). Similarly, there is accumulating evidence to suggest that without addressing the fundamental inequalities in the distribution of resources and constraints, new risk factors will emerge that will maintain the original inequalities in health (2, 5).

A growing literature indicates that modifiable risk factors are contextually embedded. Perhaps the best known context research in epidemiology is the growing literature on the importance of neighborhoods to health (6–8). Through neighborhood racial segregation (i.e., the sorting of racial groups into different types of neighborhoods through discriminatory policies and practices), black and white individuals experience unequal social and physical neighborhood environments (9–12). Segregation is thought to be an important mechanism by which racial inequalities in health are produced and maintained through differential access to economic opportunities, differential access to health-related resources (e.g., pharmacies and hospitals), increased psychosocial stress, and greater exposure to environmental hazards (e.g., hazardous waste and air pollution) (9, 12–16).

Black-white disparities in health outcomes, including obesity and hypertension, are greater in areas in which there is greater racial segregation (17–21). Conversely, researchers recently reported that in a racially integrated neighborhood with racial income parity, there were no racial differences in obesity rates among women or in hypertension overall (18, 22). The role of segregation likely involves a complex web of causal factors, including health-related resources and constraints. For example, neighborhoods with greater proportions of black residents have higher densities of fast food outlets (23), which is associated with greater fast food consumption and lower healthy food consumption (24). Neighborhoods with more healthy food resources, fewer fast food outlets, and greater walkability also have a lower prevalence of hypertension than do more resource-poor neighborhoods (25). Indeed, after accounting for neighborhood environment, researchers reported no racial inequalities in hypertension (26).

In addition to serving as the source of these health-related resources and constraints, neighborhoods also serve as the source of social stressors, a fact that challenges us to view these modifiable risk factors through the lens of stress and coping. Social stress is related to behaviors such as smoking, physical inactivity, and high intake of saturated fat (27, 28). In fact, biological evidence indicates that the mood-enhancing properties of risk factors such as consumption of “comfort food” might explain its relation with stress (29, 30). Although there are healthy behaviors (e.g., exercise) that have these mood-enhancing properties, it is important to note that behaviors are contextually embedded. In other words, the choice of coping behavior depends on the socioeconomic resources available. Because of the disparate environments experienced by black and white adults, it may be that black adults are more likely to engage in coping health behaviors that preserve mental health at the expense of physical health. For example, it has been shown that stress is positively related to depression in black adults. However, this association is modified by the number of these modifiable risk factors. For those black adults who did not report these risk factors, the association between stress and depression was strong and positive. Conversely, for those black adults who did report at least 2 of these risk factors, the association between stress and depression was negative (4). This suggests that without addressing the social factors that drive these risk factors, we might actually increase racial inequalities in poor mental health.

Although these modifiable risk factors are embedded within the social environment, this environment itself is driven by larger social forces that determine the distribution of resources and constraints, whether these are directly health-related (e.g., fast food outlets) or not (e.g., employment opportunities). Research has suggested that the nature of the population distribution of these risk factors is dynamic, that it responds to new scientific knowledge and is ultimately driven by social and economic advantage (i.e., power, prestige, and resources). For example, before the mid- to late-1960s, there was a positive association between socioeconomic status and mortality from lung cancer such that those of the lowest socioeconomic status had the lowest levels of death from lung cancer. As new information on the harmful effects of smoking became known, this association flipped such that there was (and is) an inverse association between socioeconomic status and death from lung cancer (31). Furthermore, when examining adult mortality due to conditions of high (e.g., cardiovascular disease) versus low (e.g., brain cancer) preventability, large socioeconomic status gradients were present only in deaths due to highly preventable conditions (2, 32). These studies suggest that targeting the intervening risk factors without targeting the balance of resources and constraints that drive the distribution of these risk factors will ultimately result in little change or even an increase in health inequalities as socially advantaged groups (e.g., white Americans) are able to adopt new knowledge and reduce disease risk (2, 5) to a greater extent than socially disadvantaged groups (e.g., black Americans). Although tests related directly to racial inequalities in hypertension have yet to be performed, there is growing support for this theory, which has been tested with racial inequalities in infant mortality and other conditions (33, 34).

Therefore, I would suggest that caution is warranted when determining the public health implications of these results. For example, to say that “. . . black women’s [systolic blood pressure] would be better (lower) if black women had the white women’s levels of BMI” (Web Figure 1D legend in Basu et al. (1)) does not account for this larger literature on racial health inequalities because it is based on the assumption that each of these risk factors occurs in a vacuum and is static over time. Rather, a growing body of evidence suggests that targeting BMI specifically, for example, will result in little change in the racial inequality in hypertension because the fundamental causes of health inequalities—the balance of resources and constraints that enneth the factors that result in inequalities in BMI—have not been addressed. Furthermore, reducing BMI in black women without reducing the social stressors that may be driving BMI might result in the emergence of inequalities in new outcomes, such as depression.

Additionally, by viewing disparities in modifiable risk factors within their contextual causal web, we can see that white adults, despite having greater prevalence of certain poor health behaviors such as smoking, are an advantaged group compared with black or Mexican-American adults because of
their robust social, economic, and political advantages. By focusing on each risk factor, targeting the “advantaged” group, and then targeting public health interventions toward that factor within that group, we cannot hope to eliminate racial inequalities in health and might actually increase them.

One additional note is related to the smoking measure used because it may affect the results reported. Basu et al. used the smoking variable of “smoked at least 100 cigarettes in their lifetime.” Although white adults have a greater prevalence of smoking 100 cigarettes in one’s lifetime, they have a lower prevalence of current smoking (35), lower cotinine levels (35, 36), and higher cessation rates (37) than do black adults, which is consistent with the literature on social context and stress discussed above. Furthermore, it could be that these alternative measures are more relevant to hypertension prevalence and that using these measures would yield results that are more consistent with those of the other risk factors.

The authors conclude by saying that “[t]he approach allows us to identify how modifications that may be achieved through public health interventions could affect different aspects of disparities . . .” (1, p. 352). I add to their contribution by placing the results from their study within the larger literature on racial health inequalities. Targeting each of these modifiable risk factors alone (or even all of them together) will not reduce racial health inequalities, and evidence suggests that it may actually increase inequalities in other conditions, such as poor mental health. Rather, by placing these modifiable risk factors in context, as has been done with the growing epidemiologic literature on neighborhoods and health and the social science literature on structural racism and health, we can begin to clarify the social conditions that produce and reproduce these racial health inequalities.

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