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# Risk, Race, and Structural Racism

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Black women in the United States are more likely to die during childbirth than white women (1). Yet, it is a misnomer to label Black race as a "risk" for maternal mortality. Rather, Black women are at risk of risk factors for maternal mortality, such as discrimination within the health system, worse quality of care, limited access to perinatal care, and higher comorbidity burden. These are the proximal drivers of disparities in maternal mortality, and they result not from a woman's race but from how she is treated as a result of her race. In other words, Black women do not die from childbirth at higher rates because of race. They die at higher rates because of racism.

Too often, race and ethnicity are conflated with biology and genetics. Instead, race and ethnicity are social attributions associated with systemic inequities in risk and resources. Health disparities arise when social, political, and economic structures systematically expose certain groups of people to greater risk or limit their access to timely evaluation and high-quality medical care. These so-called fundamental causes of disease, including structural racism, are social in origin and biological only in outcome (2, 3).

In this issue of *AnnalsATS*, Gershengorn and colleagues (pp. 1326–1334) provide an important scientific and clinical advance by identifying variables along the causal pathway that, at least in part, demonstrate the role of racism, not race, in coronavirus disease (COVID-19) disparities (4). Although existing literature demonstrates a strong relationship between race, ethnicity, and COVID-19 (5, 6), the authors hypothesized that socioeconomic factors, such as household size, neighborhood income, and population density, are the proximal drivers of COVID-19 infection. In other words, minorities experience higher rates of COVID-19 infection partly as a result of systematically lower socioeconomic status in a profoundly unequal society.

This hypothesis is built on a solid foundation of literature on social determinants of health and their impact on COVID-19. For example, consider the relationship between these social determinants and two essential infection

control measures undertaken during the pandemic: physical distancing and COVID-19 testing. Physical distancing is essential to reducing the risk of COVID-19 infection. However, racial and ethnic minorities were more likely to face key barriers to physical distancing—being more likely to live in larger households, work at high-contact jobs in the service industry, and live in densely populated areas. Access to COVID-19 testing helped to curb infections through early identification of at-risk individuals. However, minorities are more likely to have limited access to health care, concerns about out-of-pocket costs, and work- or transportation-related limitations that could result in a higher threshold for testing. With a higher threshold, an individual must feel sicker to pursue testing when it is not readily available. Gershengorn and colleagues believed that these barriers could explain downstream healthcare events like infection, hospitalization, and mortality.

To test this hypothesis, the authors conducted a mediation analysis. In a mediation analysis, there are three relevant



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variables: exposure, mediator, and outcome. The exposure (X) may have both a direct effect on the outcome (Y) and an indirect effect on the outcome through the mediator (M). In one example from the critical care literature, Walkey and colleagues conducted a mediation analysis of a randomized trial (7), which had demonstrated worse outcomes among patients who received higher blood pressure targets for shock (8, 9). The investigators demonstrated that worse outcomes were not due to higher blood pressure directly but rather because patients in the high blood pressure arm received more vasopressors, leading to a higher rate of arrhythmias. In this example, the relationship between the exposure (high blood pressure target) and the outcome (mortality) was mediated by arrhythmias.

Analogously, Gershengorn and colleagues modeled their study to examine whether the relationship between race or ethnicity and their study's outcomes (COVID-19 test positivity, hospitalization, and mortality) were mediated by socioeconomic factors. The investigators examined a cohort of 15,768 patients tested for or hospitalized with COVID-19 at the University of Miami Hospital. Their study population was diverse—nearly three-quarters Hispanic or non-Hispanic Black. They found that Black and Hispanic patients had three times the odds of test positivity as white patients. However, the authors found that approximately one-fifth of this association was mediated by socioeconomic factors. There was no meaningful association between race and/or ethnicity and mortality.

Despite the study's many strengths, there are also important limitations to consider. First, retrospectively disentangling the relationship between socioeconomic factors and COVID-19 is complicated by poorly measured barriers to health that could both limit access to COVID-19 testing and place certain groups at higher risk of important comorbidities associated with severe disease. Second, this study uses widely available but admittedly coarse socioeconomic metrics. As a result, the authors mediate approximately a fifth of the relationship between race or ethnicity and test positivity. What other mechanisms might more intricately explain this relationship remain unclear but could include type of employment, reliance on public transportation, community activism, environmental pollution, household income, or access to health care and testing. Third, the use of neighborhood-level and census-tract–level data may diminish existing variation, possibly resulting in misestimation of true individual- or household-level socioeconomic status. Finally, although the authors examined associations between race or ethnicity and mortality, the analysis was likely underpowered to detect meaningful differences, as evidenced by the wide confidence intervals.

The lifecycle of health disparities research has been described as having three key phases: *1*) detecting, *2*) understanding, and *3*) reducing (10). Research in pulmonary and critical care has for too long been tethered to detecting health disparities. The study by Gershengorn and investigators, supported by decades of work in health disparities, is an important step away from detecting disparities and toward understanding their underlying causes and mechanisms in order for future research to develop and evaluate interventions to reduce and ultimately eliminate these disparities. Such interventions must be predicated on strong observational analyses that trace the causal pathway from patient race and/or ethnicity through differential exposures to risk and, finally, to patient outcomes. For example, this study suggests that pop-up testing and telehealth might increase healthcare access to low-income neighborhoods, areas where higher positivity rates might be attributed to more frequent exposures and greater barriers to testing.

These socioeconomic conditions cannot be improved overnight. Nonetheless, clinicians and scientists can improve healthcare services by recognizing how socioeconomic factors influence opportunities that patients have to engage in prevention, test when exposed, and link to care when infected. Gershengorn and colleagues have established that a substantial proportion of the effect of race and ethnicity on infection and hospitalization for COVID-19 is attributable to socioeconomic disadvantage. Black and Hispanic patients are more likely than white patients to live in neighborhoods with lower income, and this is not a coincidence—it is the net result of social, political, and economic structures that have systematically limited people of color for generations.

Structural racism has a real-world impact on our patients. It endangers their very lives. We, as clinicians and scientists, must recognize the existence of structural racism, study its mechanisms of action on health, and work to eliminate it. ■

**Author Disclosures** are available with the text of this article at www.atsjournals.org.

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