



Invited Commentary | Infectious Diseases

Racism, Not Race, Drives Inequity Across the COVID-19 Continuum

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Since the early stages of the coronavirus disease 2019 (COVID-19) pandemic, significant racial and ethnic inequities have persisted across the continuum of COVID-19 morbidity, hospitalization, and mortality. The US Centers for Disease Control and Prevention have estimated that COVID-19 case and hospitalization rates are at least 2.5 and 4.5 times higher, respectively, among Black, Hispanic, and Native American populations than among White populations. Black individuals have died from COVID-19 at more than twice the rate as White individuals. Area-based studies have similarly revealed elevated COVID-19 infection and death rates in socially disadvantaged counties with larger racial and ethnic minority populations. In the context of intergenerational, structural inequalities in the United States, these trends are as devastating as they are unsurprising. The need to elucidate factors associated with COVID-19 inequity and identify tangible action steps continues. Kabarriti et al³ and Muñoz-Price et al⁴ delve deeper into the racial and ethnic disparities across the COVID-19 care continuum by presenting findings from their urban, single-center, cross-sectional studies in the Bronx, New York, and Milwaukee, Wisconsin.

Kabarriti et al³ studied a predominantly Black and Hispanic population in the setting of a COVID-19 hot spot. Their patient population experienced very high and significantly disparate test positivity rates of 68.5%, 65.3%, and 53.0% for Black, Hispanic, and White patients, respectively. They reported higher rates of hospitalization for Black and Hispanic patients (60.2% and 62.3%, respectively) compared with White patients (47.7%) but found no difference in intensive care unit admissions by race or ethnicity. Black and Hispanic patients had slightly lower unadjusted mortality rates (17.2% and 16.2%, respectively) than White patients (20.0%), which was a statistically significant (albeit relatively small) difference after accounting for follow-up time and controlling for age, sex, neighborhood socioeconomic status, and comorbidities.

Muñoz-Price et al⁴ studied a population of primarily White patients (62.3%), with 30.2% identifying as Black and 7.4% identifying as belonging to other racial groups. The authors compared the outcomes of Black vs non-Black patients (eg, combined group of White patients and those identifying as belonging to other racial groups). Most Black patients lived in poverty, and more than three-quarters resided in disadvantaged neighborhoods, while only approximately one-fifth of non-Black patients fit the same descriptors. COVID-19 positivity rates were considerably lower in the study by Muñoz-Price et al⁴ than in that by Kabarriti et al,³ with 27.8% and 8.3% Black and non-Black patients testing positive, respectively. Even after controlling for age, sex, comorbidities, poverty status, neighborhood disadvantage, and zip code-level clustering, Black patients were 5 times more likely to test positive for COVID-19 than persons identifying as belonging to other racial groups. A multilevel analysis to evaluate differences in COVID-19 test positivity found that 79% of the variance in test positivity was explained by zip code of residence. While Black race and poverty were both independently associated with hospitalization in adjusted analyses, poverty but not race was associated with intensive care unit admission, and neither were associated with mortality.

Both studies highlighted that Black and/or Hispanic patients had greater comorbidity burdens and lower socioeconomic status than White patients, but the measurement of these variables differed. Kabarriti et al³ used the Charlson Comorbidity Index as a primary measure of comorbidities, whereas Muñoz-Price et al⁴ selected specific comorbidities (ie, hypertension, diabetes, and chronic heart, lung, or kidney diseases). Kabarriti et al³ used a relative measure of socioeconomic status, based solely on neighborhood-level characteristics derived from patients' home addresses (ie, median household income, median value of housing units), whereas Muñoz-Price et al⁴ assessed

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both neighborhood and individual socioeconomic status using the Area Deprivation Index (based on 9-digit zip codes) and poverty status (based on lack of health insurance or enrollment in Medicaid). Notably, 23.4% of the cohort in the study by Kabarriti et al³ were labeled as belonging to other racial/ ethnic groups or having unknown race/ethnicity, 11.4% were missing socioeconomic data, and 14.1% were missing body mass index information, highlighting that limitations to the comprehensive collection of sociodemographic data persist even in an inpatient setting.

Kabarriti et al³ and Muñoz-Price et al⁴ concur with other recent studies⁵ that population-level disparities in COVID-19 mortality are best explained by differential disease incidence, prevalence of comorbid conditions, and socioeconomic marginalization among Black and Hispanic individuals. As these are cross-sectional studies limited by the data available in hospital records, residual confounders that differentially affect racial and ethnic groups may limit the generalizability of the findings. These could include severity rather than quantity of comorbidities; temporally relevant indicators rather than baseline measures of socioeconomic status, such as recent unemployment; and high-risk exposure history, such as residence in a long-term care facility or essential worker employment. Furthermore, solely examining mortality rates among inpatients with positive COVID-19 tests excludes individuals who did not receive a COVID-19 test, deaths prior to and following hospitalization, and increased overall excess deaths in disadvantaged communities. ^{6,7} Thus, hospital-based mortality figures may underrepresent the true level of disparity.

Limitations aside, the implications of this growing body of literature remain worth interrogation. If inpatient care does not exacerbate mortality disparities, then expanding access to care may help to ameliorate inequity by reducing deaths prior to and after hospitalization. Furthermore, the elevated incidence of COVID-19 among Black and Hispanic communities, largely attributable to social and structural vulnerabilities, seems to drive the differences in mortality among Black, Hispanic, and White populations. In short, rather than validating long-debunked hypotheses about intrinsic biological susceptibilities among non-White racial groups, the evidence to date reaffirms that structural racism is a critical driving force behind COVID-19 disparities.⁸

However, while policy measures to increase health care access may help to provide a foundation for health equity, this alone will not suffice. Incidence, hospitalization, and mortality are not the only significant and disproportionate implications of the pandemic. In urban environments like Milwaukee and New York, these 2 studies suggest that fundamental causes of COVID-19 inequity include systemically racist policies, such as historic racial segregation and their inextricable downstream effects on the differential quality and distribution of housing, transportation, economic opportunity, education, food, air quality, health care, and beyond. Each of these factors is associated with the risk of COVID-19 exposure and severity through direct (eg, work conditions, crowded housing, carceral overrepresentation) and indirect (eg, limited access to health information or insurance; increased prevalence of comorbidities; cumulative life-course exposure to discrimination, low socioeconomic status, and other health risk conditions) mechanisms. Furthermore, the associations between social inequities and COVID-19 disease outcomes are bidirectional; the concentrated harm of the pandemic within marginalized communities has exacerbated racial inequities in income, housing, education, employment, and access to quality health care. These intertwined, inequitable social and clinical differences reaffirm that we cannot treat our way out of public health crises even if outcomes are equalized within the hospital. Rather, a structurally competent set of systems-level reforms and interventions will be required. 10

As both a deontological and a consequentialist imperative, researchers must name and interrogate structural racism and its sociopolitical consequences as a root cause of the racial health disparities we observe. Short-term and long-term interventions to ameliorate health inequity must address the synergistic interactions of clinical and structural risk conditions, including compounding spillover effects on the mental, physical, and economic health of Black and Hispanic communities. Clinicians, health systems, and policy makers alike must grapple with the fundamental inequities that lie upstream of disparate COVID-19 outcomes to make tangible progress toward health justice.

ARTICLE INFORMATION

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