Left, Harlan P. Jones. Right, Nicole R. Phillips. Credit: Jill Johnson, University of North Texas Health Science Center.

The embers underneath the CoV-2 pandemic: a semblance of the persistent burn of health inequities and disparities in the United States

Could we have predicted that the second deadliest pandemic encountered since the influenza pandemic of 1918 would result in the highest mortality and adverse health outcomes among minority and underserved populations in the United States? Given the abundant evidence documenting the disproportionately high burden of preventable disease, disability, and injury among these underserved groups, our answer should echo a resounding 'yes'.

COVID-19 is a glaring example of the pervasive inequities among minorities and underrepresented groups in health access that plague the United States and indeed populations worldwide. Evidence continues to emerge that links the disproportion of contagion and mortality from severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), a result of adverse social determinants of health. Epidemiological data from the Centers for Disease Control and Prevention (CDC) show that the risk of death from SARS-CoV-2 is significantly correlated with individuals with underlying chronic conditions, with devastation and mortality brought against the aged and those affected by such chronic disease. Consideration of a better understanding of the bio–psycho–social intersections through which SARS-CoV-2 orchestrates disease pathogenesis is of high relevance to reducing the gross disproportion in mortality of minority and vulnerable populations.

Throughout our existence, social determinants of health (SDH) and structural racism have defined and driven health inequities. Global accounts of health differences emerged as early as the eighteenth century and into the mid-nineteenth century, driving associations between both healthcare and non-medical (social) determinants. In 1984, the US Department of Health and Human Services officially documented the existence of marked health disparities experienced by blacks and other minority groups as compared to its citizenry. Ever since, racial and ethnic minority groups in the United States have unequally been affected by adverse social and health consequences. COVID-19 has arguably fueled the persistent flame of health inequities and disparities in the United States by raising attention to the inadequacies of access to care, economic stability, quality housing, work conditions and racial discrimination. In a study by Wilkins et al., 26% of Vanderbilt University Medical Center patients who initially tested positive for SARS-CoV-2 had limited English language proficiency, compared with 6% of patients with English as their primary language, and the disparity continues (https://catalyst.nejm.org/doi/full/10.1056/CAT.20.0374). During the initial month of the US COVID-19 pandemic vaccination response, the CDC reported a lower percentage (39.6%) of racial or ethnic minorities receiving at least one vaccination dose compared to non-Hispanic white populations. Such pervasiveness of the existing social determinants has placed racial and ethnic minority groups at higher risks for exposure to COVID-19 and less access to treatment and preventative care. One might also contend that while COVID-19 cases and mortality rates steadily fall, COVID-19 is expected to have collateral consequences: COVID-19-related influences on mental health, long-term health care, health literacy and employment stability are likely to have a long-term disproportionate effect on racial and ethnic minority groups.

Undeniably, resolving SDH-sourced influencers of risk for COVID-19 outcomes is the rate-limiting step in alleviating related health disparities, and until the societal 'elephant in the room' is addressed, we will be handicapped in our ability to study and understand the (albeit lesser) role of biological mechanisms that underlie disease risk and progression. SARS-CoV-2 disproportionally affects those with co-morbidities such as diabetes, heart disease and asthma — common chronic conditions that have an increased prevalence in minority and underserved populations relative to non-Hispanic white populations (https://www.frontiersin.org/articles/10.3389/fpubh.2020.559312/full). There are complex interactions between sociological, psychological and biological human domains, which include a role for genetic variation and epigenetic influencers linked to human disease risk (https://www.mdpi.com/1422-0067/20/5/1081). These genetic signatures, both hardwired and as environmental proxies, may offer opportunities for new breakthroughs to collectively and comprehensively address sources of risk to develop targeted interventions that function alongside existing social determinants of health in the context of COVID-19. Conceptually, psychosocial influences such as stress, anxiety and depression, along with pre-existing or co-morbid disease (for example, hypertension, type 2 diabetes, chronic respiratory), and COVID-19 may interconnect based on known and unknown facets that translate into human health outcomes and molecular modes of viral pathogenesis (https://pubmed.ncbi.nlm.nih.gov/32965660/). The recent meta-analysis from the COVID-19 Host Genetics Initiative identified variants associated with both risk and severity that have been implicated in autoimmune and inflammatory disease (https://www.nature.com/articles/s41586-021-03767-x). In addition, taking into context host pressures, such as underlying chronic inflammatory disease, one might expect SARS-CoV-2 quasispecies mutations

to arise, resulting in genetically favored viral variants. These may include more pathogenic species with the potential to escape detection by molecular and serologic screens and, importantly, production of the pathophysiologically diverse quasispecies relating to biomedically relevant phenomenon, such as immune system evasion (https://www.oatext.com/impact-of-sars-cov-2-quasi-species-on-covid-19-testing-reliability-in-the-african-american-aa-population.php).

Several epidemiological studies of the 1918 influenza pandemic suggested that the strain of influenza was extremely virulent with an unbiased infection rate across racial and socio-demographic characteristics. Yet, as the literature evolved with more sophisticated research methodologies, social determinants were central to disproportionate mortality among racial and ethnic minority and underserved groups. The novel SARS-CoV-2 virus has also proven to be highly infectious but unlike the 1918 pandemic, because of the persistent inequities in health, it did not take long to witness the unfortunate disproportionate mortality among racial and ethnic minority and underserved groups. We can only hope that experiences and the lessons learned will increase knowledge and understanding of the determinants of health to implement equitable comprehensive practices to saving US lives during and after COVID-19. ❐

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