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EDITORIAL

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Outcomes of COVID-19: disparities in obesity and by ethnicity/race

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COVID-19 outcome disparities are rapidly becoming apparent for people with obesity and multiple black, Asian, and minority ethnic (BAME) groups. Researchers have reported differences in COVID-19 hospitalization and mortality rates by race/ethnicity in the United Kingdom (UK) [1, 2] and United States (US) [3, 4]. Numerous minority ethnic groups in these countries live with a greater burden of obesity and other chronic diseases. This is particularly significant as obesity has emerged as a risk factor for severe COVID-19, the disease caused by novel coronavirus SARS-CoV-2 [3, 5]. We synthesize a range of potential biological, socioeconomic, behavioral, and sociological contributors to the disparate outcomes for people with obesity and minority ethnic groups in COVID-19.

Initial retrospective cohort analyses have demonstrated higher rates of hospitalization and intensive care, including invasive mechanical ventilation, for patients with obesity [5, 6]. Though these observational results do not assess mortality outcomes and adjust for few comorbidities, they signal potential biologic vulnerability for a large proportion of people worldwide living with obesity. Obesity rates are significantly higher among Hispanic and African Americans, as well as black, Bangladeshi, and Pakistani groups in the UK, than their white counterparts [1, 7]. The overlap of COVID-19 risk signals between obesity and ethnicity therefore is of consequence.

The most comprehensive epidemiology on ethnicity and COVID-19 currently available is from the UK [1, 2]. BAME individuals comprise over 30% of hospitalized, critically ill patients with COVID-19 [2]. The Office for National Statistics highlights stark ethnic disproportionalities in COVID-19 mortality odds, over four- and threefold for black and Bangladeshi/Pakistani individuals respectively compared to white individuals [1]. Indeed, the first ten UK physicians to die from COVID-19 were all of BAME background. Though US national data are limited, states and municipalities report a disproportionate burden of COVID-19 cases, hospitalizations, and deaths among Hispanic and African Americans [4, 8]. In Norway and Sweden, cases among Somalis are seven- to ten times expected based on population [9].

Ethnic differences in economic status, underlying health conditions, density of residence, and household crowding all contribute to the unequal impact of COVID-19. For example, the English Housing Survey noted household overcrowding in 30% of Bangladeshi, 16% of Pakistani, and 2% of white British households [1]. The mortality gap between the most and least deprived areas is greater for COVID-19 than that normally observed for all-cause mortality, highlighting the importance of socioeconomic status [1]. However, adjustments for age, geography, educational attainment, level of deprivation, and self-reported health only partially attenuate the higher odds ratios of COVID-19 mortality for black individuals (1.9), Bangladeshi/Pakistani men (1.8) and women (1.6) compared to white individuals in the UK [1]. Further inclusion of data on socioeconomic status and specific medical conditions such as obesity (absent from this model) will enhance our understanding of the joint and independent contributions of ethnicity, class, and preexisting comorbidities in COVID-19.

Sociodemographic patterns in COVID-19 may diverge from other viral pathogens. While the 2009 H1N1 influenza hospitalized disproportionate numbers of ethnic minorities, differences in mortality were not observed among hospitalized minority versus non-minority patients [10]. BAME disparities in COVID-19 intensive care mortality rates were similarly not observed in a recent

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historical cohort of patients critically ill with pneumonia from other viral pathogens [2].

One biologic hypothesis for observed associations between ethnicity, obesity, and worse COVID-19 outcomes is vitamin D deficiency [11]. The anti-inflammatory and anti-microbial properties of vitamin D include maintenance of tight junctions and reduced production of inflammatory cytokines [11]. Massive cytokines release ("storm") has been implicated in severe COVID-19. Vitamin D supplementation in randomized, placebo-controlled studies has demonstrated reduced risk for acute respiratory tract infection, and associations between vitamin D deficiency and acute respiratory distress syndrome have been reported [12]. Non-white ethnicity and obesity are each independently associated with hypovitaminosis D [13], offering one plausible explanation for higher COVID-19 burden in these groups.

In addition to differential rates of comorbidities such as diabetes and cardiovascular disease across ethnic groups, pathophysiologic differences in inflammation may also be significant. The chronic low-grade inflammatory state of obesity is well-defined elsewhere, but inflammatory changes may not be consistent across race/ethnicity. Hispanic and African American children have higher risk of low-grade inflammation compared to white peers, an effect only partially mediated by parental education and body mass index (BMI) [14]. During a weight loss intervention among healthy female participants with overweight (BMI 27–30 kg/m^2), fewer markers of inflammation decreased among African American compared to white patients [15]. It has been proposed that there may be a genomically influenced response to viral pathogens to explain the potential interaction of ethnicity-related factors on SARS-CoV-2 infection and subsequent outcomes [16]. In this model, one's health state, health behaviors, and social behaviors interact with factors such as comorbidity burden and control in disease outcomes. Comprehensive data which include ethnic background should be evaluated to better understand biological factors that contribute to the disproportionate burden on certain segments of the population.

We also acknowledge the complex roles of behavior and sociology. Occupational exposures contribute to ethnic disparities in SARS-CoV-2 [1]. Public health communication may not prepare minority citizens to respond to SARS-CoV-2 due to language or other structural barriers to access [9]. Similar access issues exist in health messaging for obesity and other chronic diseases. Individual choice and self-referral patterns also play a role. A holistic view does not attribute observed differences to any single factor.

Published retrospective cohort analyses of obesity and COVID-19 have not adjusted for ethnicity [3, 5], and analyses of ethnicity and COVID-19 have not adjusted for differences in obesity rates [1, 2]. Studies to date have incompletely parsed obesity from comorbidities such as diabetes and cardiovascular disease as risks for COVID-19. A critical knowledge gap remains to understand the interaction between ethnicity, obesity, and class in COVID-19 outcomes. We have presented several plausible biological, socioeconomic, and behavioral factors, which may inform the observed disproportionalities. The degree to which obesity and ethnicity are additive, multiplicative, mediators or confounders—and the degree to which obesity increases risk independent from its many comorbidities—have significant implications for our medical and public health response.

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Compliance with ethical standards

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