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Health Inequities and Racial Disparity in Obstructive Sleep Apnea Diagnosis: A Call for Action

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The World Health Organization defines health inequities as the systematic differences in the health status or in the distribution of health resources to different groups. Striving for health equity ultimately means eliminating social determinants, including

racial bias, to improve access to good medical care (1). Eighteen years ago, the institute of medicine identified that racial minorities receive lower quality of health care than nonminorities, even when factors such as insurance status and income were considered (2). Racial disparities to health and specifically sleep health are common, prevalent worldwide, and avoidable (3). African Americans with obstructive sleep apnea (OSA), compared with matched White individuals, are often found to have severe disease (4, 5), use treatment with positive airway pressure devices less (depending on their zip code) (6, 7), and are more susceptible to uncontrolled hypertension (8).

In this issue of *AnnalsATS*, Thornton and colleagues (pp. 272–278) investigate the impact of racial disparity on the diagnosis of OSA (9). Their report was derived from a large university-based sleep lab cohort and showed that Black people, especially Black men, have more severe disease than their White counterparts and yet present with standard symptoms of daytime sleepiness or

snoring. Black males and females were 5 years younger than their White counterparts and had higher body mass indexes ($\sim 5 \text{ kg/m}^2$) and higher percentages of hypertension. Apnea severity was also worse; among Black men, mean apnea hypopnea index was 52.4 \pm 39.4 events per hour compared with 39.0 ± 28.9 events per hour in White men, 33.4 ± 32.3 events per hour in Black women, and 26.2 ± 23.8 events per hour in White women. Subjective sleepiness as measured by the Epworth Sleepiness Scale score was highest in Black men (12.12 \pm 5. 9) followed by Black women (11.2 \pm 5), White women (9.8 \pm 5.6), and White men (9.4 ± 5.2) . Thus, Black males made up the smallest percentage of the cohort but had more disease and it impacts them more severely from a sleepiness standpoint than the other groups, which raises the question, why are these patients not identified sooner?

There are three potential mechanisms leading to this finding, including *1*) limited screening for OSA in Black people at the primary care level; *2*) overlapping symptoms

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(sleepiness) with other systemic diseases of greater perceived concern to their doctor such as coronary artery disease or diabetes (both of which have high prevalence in Black people); or 3) cultural factors such as lack of trust in the medical system, as with testing or similar to vaccine hesitancy or feelings that OSA has a limited impact on overall health.

An important question remains. How do we, as a group, educate providers and patients, particularly Black males, about OSA clinical presentation, associated comorbidities, and optimal treatment? Targeted educational campaigns have been tried (10) with varying success and less so in the Black population (11). Thus, combinations of education, use of technologic advances such as telehealth, and large system-wide changes as are taking place now should help bridge the inequities gap.

Three limitations of this study are notable. One caveat is that the sleep studies performed were all done in-lab, which

overall is less reflective of the current practice of home sleep testing followed by therapy with an autotitrating positive airway pressure device (12). Differentiating the ease of participation for Black people versus White people in obtaining access to an in-lab sleep study versus a mailed home sleep test would be informative. Prior studies have shown that travel distance to sleep labs (13) or other socioeconomic factors contribute to limitations in getting a sleep study done, but these factors are not inherently known to be racially biased. A second limitation is the premise that a greater severity of illness, measured by the apnea hypopnea index at the time of presentation, presumes that there has been a delay in diagnosis of OSA. Although it is a travesty that one sex or race group is receiving disparate care, this premise suggests a mechanism of apneas begetting more apneas and thus more complications. Apnea severity has been

shown to worsen across the night as well as across circadian timing, but it remains unclear if untreated OSA severity changes over time (in adults) independent of changes in weight (14, 15) or other risk factors. Third, it is interesting that the Black male group is approximately 5 years younger than White individuals, but it is presumed that because their disease is more severe, their presentation for therapy is delayed.

In summary, it is time to bring the issues of health inequities, particularly those of racial bias and OSA, to center stage. Thornton and colleagues have taken an important step in the right direction. Learning how to prevent these inequities going forward will go a long way to improving lives for everyone.

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

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