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Original Article

Racial disparities in sleep: the role of neighborhood disadvantage☆

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ABSTRACT

Objective: Disparities in sleep duration and efficiency between Black/African American (AA) and White/European American (EA) adults are well-documented. The objective of this study was to examine neighborhood disadvantage as an explanation for race differences in objectively measured sleep.
Methods: Data were from 133 AA and 293 EA adults who participated in the sleep assessment protocol of the Midlife in the United States (MIDUS) study (57% female; Mean Age = 56.8 years, SD = 11.4). Sleep minutes, onset latency, and waking after sleep onset (WASO) were assessed over seven nights using wrist actigraphy. Neighborhood characteristics were assessed by linking home addresses to tract-level socioeconomic data from the 2000 US Census. Multilevel models estimated associations between neighborhood disadvantage and sleep, and the degree to which neighborhood disadvantage mediated race differences in sleep controlling for family socioeconomic position and demographic variables.
Results: AAs had shorter sleep duration, greater onset latency, and higher WASO than EAs (*ps* < 0.001). Neighborhood disadvantage was significantly associated with WASO (B = 3.54, $p = 0.028$), but not sleep minutes (B = -2.21, $p = 0.60$) or latency (B = 1.55, $p = 0.38$). Furthermore, race was indirectly associated with WASO via neighborhood disadvantage (B = 4.63, $p = 0.035$), which explained 24% of the race difference. When measures of depression, health behaviors, and obesity were added to the model, the association between neighborhood disadvantage and WASO was attenuated by 11% but remained significant.
Conclusion: Findings suggest that neighborhood disadvantage mediates a portion of race differences in WASO, an important indicator of sleep efficiency.

Sleep is a fundamental biological process that plays a critical role in the maintenance of mental and physical health. Insufficient or low quality sleep has been consistently associated with adverse health outcomes including pre-disease markers of physiologic dysregulation such as inflammation [63], glucose metabolism [57], and hypothalamic-pituitary-adrenal axis functioning [83]. Sleep problems have also been linked with increased disease risk, including for cancer [89], diabetes [37], and heart disease [8], as well as life expectancy and mortality [24]. Furthermore, mounting evidence suggests that sleep is a consequential mediator of established associations between social/physical environment factors and health [90]. With respect to mental health and psychosocial functioning, insufficient sleep has been heavily associated with negative mood states [56], decreased ability to regulate thoughts and behaviors [80], and diminished productivity and learning [3]. All told, sleep problems are a significant impediment to health and well-being, which collectively, in the United States alone, carries an economic burden of hundreds of billions of dollars each year [32].

Recent meta-analyses and reviews of the literature reveal that Black/African American (AA) adults show consistently poorer sleep

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than Whites/European Americans (EAs) [27,64,74]. In particular, studies indicate fewer total sleep minutes, poorer sleep efficiency, greater onset latency, and worse overall sleep quality among AAs relative to EAs.

These differences have been documented using self-reported data and studies using objective measures, which have generally found larger racial disparities [27,64]. Furthermore, prior findings indicate that race differences in sleep are partially accounted for, but endure after measures of socioeconomic status (SES) are controlled [26,27,93].

Beyond SES, the mechanisms for race differences in sleep are not well understood and analyses considering life course determinants of group differences are profoundly needed [27,42,64]. Possible candidate mechanisms include discrimination [22,82], chronic stress [13], health behaviors [84], and exposure to environmental toxins [39]. Neighborhood contexts represent an additional candidate mechanism, likely to operate through a range of individual-level variables [14,28,31]. For example, living in a disadvantaged neighborhood with higher crime rates, less access to healthy food, and greater exposure to toxins has been associated with higher rates of obesity, stress, and physiologic dysregulation [15]; all of these conditions have been associated with low sleep quality [34,54,85]. Furthermore, more ambient noise in disadvantaged neighborhoods has also been linked to obesity, psychological distress, and sleep problems [7,29]. Neighborhood location and, therefore, availability of health-promoting resources such as parks and community centers, may influence physical activity and psychosocial factors related to sleep quality [40].

A large number of studies have indicated that, on the whole, AAs live in far less advantaged neighborhoods than EAs [23,60,72,73]. The historical factors underlying race differences in neighborhood quality (eg, discriminatory policies) are well understood [12], as are the related processes of stigma and racism that have played a role in creating and sustaining neighborhood segregation [52]. Of note, racial differences in neighborhood quality are substantial even after accounting for individual-level socioeconomic variables [73,79]. One recent study found that AA families making upwards of $100,000 typically live in the kinds of neighborhoods inhabited by EA families making $30,000 or less [79]. Although neighborhood factors generally account for a portion of the association between family socioeconomic status and health outcomes, each of these variables have been found to exert independent effects on health and often account for unique variance in racial health disparities [10,23,70,73].

Recent literature on neighborhoods and sleep has shown that self-reports of neighborhood safety and quality are associated with several sleep outcomes, including duration [14], efficiency [2], daytime sleepiness [33], and overall sleep quality [28,31]. However, very few studies have considered objective measures of neighborhood disadvantage. Two such studies found evidence of neighborhood effects on sleep duration [62,91], and another showed links to sleep efficiency [2]. Whether neighborhood disadvantage might account for racial disparities in sleep between AAs and EAs has not, to our knowledge, been previously examined. In addition, the degree to which mental health, health behaviors, and obesity might account for neighborhood effects on sleep have rarely been considered. This omission is notable, given established links between these health factors and neighborhood context [4,35,48,68] and sleep [9,25,53].

The overarching hypotheses of this study were that (1) AAs would obtain shorter and more disrupted sleep (measured objectively) than EAs, (2) neighborhood disadvantage (measured objectively) would be associated with sleep parameters net of family-level socioeconomic status and other demographic factors, and (3) neighborhood disadvantage would partially account for racial disparities in sleep. Lastly (4), we hypothesized that neighborhood effects on sleep would be partially attenuated after adjusting for measures of health behaviors, obesity, and depression.

1. Methods

1.1. Data and sample

Analyses draw on data from the Midlife in the United States (MIDUS) Study [5,47]. MIDUS is a national study of health and aging begun in 1995 with 7000 non-institutionalized adults from the 48 contiguous states [5,66]. The second wave (MIDUS 2) began in 2004, with 75% of surviving MIDUS 1 respondents participating. An oversample of AAs from Milwaukee, WI ($N = 592$) was added at MIDUS 2 to increase representation of AAs and facilitate analysis of racial disparities in health [81]. Participants in the biomarker project visited one of three data collection sites: University of Wisconsin (UW), University of California, Los Angeles, and Georgetown University (see Ref. [47] for a detailed description of the biomarker protocol). Travel arrangements and costs were provided, with assignments to the specific site based on approximate geography.

All subjects who participated in the biomarker project at the UW site (2004–2009) were also invited to take part in a sleep substudy. This study involved seven nights of home-based actigraphy sleep assessment, and an accompanying daily sleep survey. A total of 441 participants (83% of UW biomarker sample) were included in the sleep study. Due to our focus on Black- White disparities in sleep, 15 individuals not categorized at AA or EA were excluded from analyses. The final analytic sample (N = 426; 31% AA, 69% EA) included a wide range of ages (35–85 years; $M = 56.8$; $SD = 11.4$) and was 61% female. Sample characteristics for each racial/ethnic group are presented in Table 1. All data collection and analysis was approved by an Institutional Review Board, and all participants provided written, informed consent.

1.2. Measures

1.2.1. Sleep assessment

Beginning on the Tuesday following their biomarker data collection visit, participants were asked to wear a Mini Mitter Actiwatch®-64 water-resistant activity monitor (Respironics, Inc., Bend, OR) for 7 nights. Measurements of nighttime sleep minutes, onset latency, and waking after sleep onset (WASO) were derived from actigraphy data using Actiware® Software (Version 5.0) [44,55]. Detailed information about the scoring protocol used to compute sleep and wake periods is publicly available at http://www.midus.wisc.edu/midus2/project4/. In brief, actigraphy devices were configured to collect data over 30 s epochs, a time-scale that has been validated against polysomnography and used in other large studies [43,45]. Epochs were scored as wake based on a threshold of 40 activity counts or greater using a weighted average of the epoch in question and those surrounding it, with epochs below this threshold being scored as sleep. To supplement actigraphy measurements, MIDUS participants completed diaries each night and within ten minutes of waking up to report bedtime, time out of bed and other sleep-related items. Consistent with best practices for the coding of actigraphy data [46], bedtimes and rise times were used to define sleep periods from which calculations of sleep onset latency, WASO, and sleep minutes (defined as total sleep minutes between sleep onset and wake time) were determined. Summary statistics that aggregate nightly sleep and wake epochs were produced by Actiware software, and daily summary statistics were averaged across all nights of available data to