



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.

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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 sub-study. 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 as 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

compute sleep-related outcomes. Ninety-six percent of participants had at least six nights of actigraphy data with all remaining participants having three or more nights of data. Consistent with current research, sleep indicators were considered separately in all analyses. Correlations between sleep measures were as follows: sleep minutes and sleep onset latency ($r = -0.35$), sleep minutes and wake after sleep onset ($r = -0.09$), sleep onset latency and WASO ($r = 0.38$).

Actigraphy is a well-established method for assessing a variety of sleep parameters, including sleep minutes, onset latency, and WASO, each of which has good correspondence with polysomnography [51,86]. Although actigraphy has been shown to overestimate sleep (ie, low specificity to detect wakefulness), the inclusion of several nights of data leads to more reliable estimation of sleep parameters [88]. Further, actigraphy has good validity and reliability in community-based samples [75] and is generally considered a reasonable estimation method for several sleep characteristics.

1.2.2. Sleep aids

Participants reported each morning whether any medication or supplements were taken to help with sleep. The use of sleep aids (prescription, over-the-counter, or supplement) was coded as a dummy variable to control for possible sleep disorders such as insomnia.

1.2.3. Neighborhood economic disadvantage

Neighborhood characteristics were geocoded by linking home addresses at the time of participation to tract-level data from the 2000 US Census. An aggregate index of neighborhood disadvantage was then created as the mean of five standardized neighborhood level economic characteristics: percentage of residents below poverty line, percentage on public assistance, percentage with highest level of education less than high school graduation, median household income (reverse-coded), percent with highest level of

education of a four-year college degree or more (reverse-coded). These indicators were highly correlated (r 's between 0.43 and 0.82) and created a reliable composite scale ($\alpha = 0.91$). Comparable indexes of neighborhood disadvantage have been used in prior research [6,61,62,71].

1.2.4. Population density

Using established methods, population density was calculated from census data as the population per square mile at the tract level [77]. This variable was then coded on a 20-point scale (using quantiles) ranging from less than 21 to greater than 10600 persons per square mile. The reason for coding into 20 quantiles was to meet requirements for protecting participant confidentiality while also maintaining meaningful variance in the data. Population density was included as a covariate in all models.

1.2.5. Health behaviors

Cigarette smoking, alcohol consumption, and physical activity were assessed via self-report. Smoker status was assessed in the MIDUS II survey and was dichotomously coded (1 = current smoker, 0 = nonsmoker). Alcohol consumption and physical activity were reported each night in daily sleep diaries. Alcohol consumption was measured as, "How many alcoholic drinks did you have today?" with information for participants that one drink is equivalent to one bottle of beer or wine cooler, one glass of wine, or one shot of liquor. Due to known dose-response associations between alcohol use and sleep parameters [16], alcohol consumption was coded as the average number of drinks per day across the seven-day sleep study. Physical activity was assessed using the following item, "How many minutes of moderate or vigorous exercise did you get today?", and coded as the average number of minutes reported per day. Although using a single item offers a coarse measure of physical activity, it is likely to have high validity due to the short recall period and repeated measurement [76].

Table 1
Sample characteristics.

	African American (n = 133)		European American (n = 293)		Race difference
	Mean \pm SD	%	Mean \pm SD	%	P value
Level 1 sleep outcomes:					
Sleep time (minutes)	340.6 \pm 73.9		381.3 \pm 62.9		<0.001
Onset latency (minutes)	43.8 \pm 33.9		24.8 \pm 21.7		<0.001
Waking after sleep onset (minutes)	59.0 \pm 26.8		44.4 \pm 19.7		<0.001
Level 1 predictors:					
Age (years)	54.9 \pm 10.2		57.6 \pm 11.8		0.016
Female		72.9		54.9	<0.001
Partnered (married or cohabiting)		43.2		75.8	<0.001
Sleep aids		3.9		6.8	0.20
Education					
High school diploma or less		44.4		26.0	<0.001
Some college attendance		32.3		27.7	0.34
Four year college degree or more		23.3		46.2	<0.001
Income (in thousands)	39.4 \pm 33.3		74.1 \pm 56.1		<0.001
Body mass index (kg/m ²)	33.1 \pm 8.1		29.4 \pm 5.8		<0.001
Depressive symptoms	11.82 \pm 9.56		6.34 \pm 6.26		<0.001
Cigarette smoker		24.8		10.9	0.001
Alcohol consumption (drinks per day)	0.44 \pm 0.97		0.51 \pm 0.85		0.50
Physical activity (minutes per day)	35.5 \pm 65.2		39.6 \pm 46.0		0.47
Level 2 neighborhood predictors:					
Population density	17.5 \pm 2.7		10.0 \pm 5.5		<0.001
Neighborhood disadvantage	0.96 \pm 0.95		-0.18 \pm 0.64		<0.001
Median income (in thousands)	30.9 \pm 10.2		48.8 \pm 17.1		<0.001
Poverty rate (%)	24.3 \pm 16.2		8.0 \pm 6.6		<0.001
Household public assistance (%)	6.0 \pm 4.5		2.3 \pm 2.3		<0.001
Less than high school degree (%)	26.4 \pm 12.5		13.9 \pm 8.2		<0.001
Four-year degree or higher (%)	13.7 \pm 8.1		26.6 \pm 16.7		<0.001

1.2.6. Body mass index

Nursing staff collected measurements of weight and height as part of the biomarker protocol, the week prior to the sleep assessment. Body mass index (BMI) was calculated using the standard formula: weight (kg)/height (m)².

1.2.7. Depression

Depressive symptoms were assessed using the Center for Epidemiologic Studies Depression Scale (CES-D [67]); during the biomarker protocol visit. To avoid overlap with sleep measures, one item relating to restless sleep was omitted when computing the total score. This adaptation from the original twenty-item scale did not affect the reliability of the scale ($\alpha = 0.89$). Depression is robustly associated with sleep parameters [87], and thus is important to consider in sleep research.

1.2.8. Sociodemographic covariates

Controls included sex (coded as 0 = female, 1 = male), age (in years), race (0 = White, 1 = Black and/or African American), cohabitation status (1 = married or living with partner, 0 = else), household income, and educational attainment. Total household income was calculated from a series of items that assessed wages, pension, social security, and government assistance for all household members [21]. Education was reported using a twelve-item categorical scale that was coded into dichotomous variables representing high school degree or less (reference category), some college attendance, and four-year college degree or greater.

1.3. Analysis plan

Multilevel models were estimated in Mplus Version 7.4 to examine neighborhood influences on sleep [59]. The use of multilevel modeling is necessary to account for clustering of individuals within neighborhoods and to correctly estimate standard errors of neighborhood variables. Consistent with established guidelines [69], two-level models were used in which individuals (level 1) were nested within neighborhood census tracts (level 2). Participants were from 307 tracts (AAs from 23 and EAs from 284). Due to the city-specific recruitment of AA participants in Milwaukee, AAs were clustered in more densely populated neighborhoods, while EAs were distributed more broadly across the Midwestern region in both metro and non-metro areas. This reflects the MIDUS sampling strategy, but is also largely consistent with the racial demographics of the region [58]. Missing data on predictor variables were handled using Full Information Maximum Likelihood (FIML) estimation, a robust missing data approach which uses all available data while also providing unbiased standard errors [19]. Of the 426 individuals included in analyses, three percent had missing data for physical activity, two percent for alcohol use, and less than one percent for all other variables considered.

Initial empty models were used to estimate the proportion of variance in each sleep outcome at level 1 and level 2. A series of models was then estimated for each sleep outcome, respectively (sleep minutes, latency, WASO). Model 1 estimated the magnitude of race differences in sleep controlling for age, sex, cohabitation, income, education, and use of a sleep aid (H_1). Because AAs were more likely to live in urban areas, Model 1 and all subsequent models controlled for population density. This ensured that neighborhood effects on sleep were not confounded by population density. Model 2 added neighborhood disadvantage to consider associations with sleep and the degree to which racial disparities were attenuated (H_2 , H_3). As an illustration, the Model 2 equation used to consider neighborhood disadvantage as a predictor of WASO was as follows:

$$\text{Level 1: WASO}_{ij} = \beta_{0j} + \beta_1 (\text{age}_{ij}) + \beta_2 (\text{sex}_{ij}) + \beta_3 (\text{race}_{ij}) + \beta_4 (\text{cohabitation}_{ij}) + \beta_5 (\text{income}_{ij}) + \beta_6 (\text{some college}_{ij}) + \beta_7 (\text{four-year college degree}_{ij}) + \beta_8 (\text{sleep aid}_{ij}) + r_{ij}$$

$$\text{Level 2: } \beta_{0j} = \gamma_{00} + \gamma_{01} (\text{population density}_j) + \gamma_{02} (\text{neighborhood disadvantage}_j) + u_{0j}$$

The subscript j is for neighborhood census tracts and i is for individuals. In the level 1 equation, β_{0j} is the intercept, $\beta_1 - \beta_8$ are the level 1 regression coefficients for each of the controls, and r_{ij} is the residual error term. The level 2 equation then shows that the intercept, β_{0j} , is allowed to vary across neighborhoods, u_{0j} , and as a function of neighborhood economic deprivation, γ_{01} , and population density, γ_{02} . The combined model therefore estimates sleep minutes for individual i in neighborhood j as a function of neighborhood disadvantage, while also accounting for the effects of individual- and neighborhood-level variables and the clustering of individuals within neighborhoods.

Two further models (Models 3 and 4) were estimated to consider additional control variables expected to attenuate race differences or neighborhood effects (H_4): Model 3 adjusted for health behaviors (alcohol consumption, current smoking status, and moderate/vigorous physical activity) and BMI, and Model 4 added depressive symptoms. Sensitivity analyses were conducted to consider all model results when population density was not included as a control. Findings from these models showed the same pattern of results and inference as those reported. Unstandardized parameter estimates and standard errors are reported in Table 2 for all models, and 95% confidence intervals (CIs) and exact p -values are reported in the text.

2. Results

2.1. Preliminary analyses

Initial descriptive analyses indicated substantial race differences in sleep indicators (Table 1). Specifically, as compared to EAs, AA adults obtained 40.7 min less total sleep per night (95% CI: -55.3, -26.1; $p < 0.001$), took 19 min longer to fall asleep (95% CI: 12.7, 25.3; $p < 0.001$), and experienced an additional 14.6 min of waking after sleep onset (95% CI: 9.5, 19.7; $p < 0.001$). With respect to neighborhood environments, AAs generally came from neighborhoods characterized by greater socioeconomic disadvantage ($p < 0.001$), equivalent to 1.24 standard deviation (SD) units difference. Race differences in other sociodemographic and health characteristics were also detected, as documented in Table 1. Initial empty models indicated that the intraclass correlation coefficients (ICCs)—the proportion of the total variance in each outcome that existed across neighborhoods—was 0.13, 0.05, and 0.14 for minutes, latency, and WASO respectively.

2.2. Primary analyses

2.2.1. H_1 : sleep disparities

After adjusting for sex, age, cohabitation status, education, income, use of sleep aids, and population density, race differences in each sleep indicator remained substantial (see Model 1 in Table 2). In particular, AA adults obtained less total sleep ($B = -36.11$, $p < 0.001$, 95% CI: -54.15, -18.06), had greater sleep onset latency ($B = 16.33$, $p < 0.001$, 95% CI: 10.30, 22.36), and greater WASO ($B = 13.30$, $p < 0.001$, 95% CI: 7.26, 19.33) than EAs.

Effect sizes of estimated racial disparities in sleep time, onset latency, and WASO were equivalent to 0.52, 0.59, and 0.58 SD units of the sleep parameter, respectively. There was no evidence for sex

Table 2

Estimates from a series of multilevel regression models testing predictors of minutes spent awake after sleep onset in a sample of White/European American and Black/African American adults living in the Midwest ($N = 426$).

Outcome: Wake after Sleep Onset (WASO)	Model 1		Model 2		Model 3		Model 4	
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
Intercept, γ_{00}	41.37***	7.55	38.93***	7.42	25.57**	9.92	21.66**	10.17
Age, β_1	0.02	0.10	0.05	0.10	0.13	0.10	0.15	0.10
Sex (female = 0; male = 1), β_2	5.55*	2.17	5.57**	2.15	4.08	2.15	3.88	2.17
Race (White = 0; Black = 1), β_3	13.30***	3.08	10.06**	3.48	8.86**	3.31	7.56*	3.11
Cohabitation, β_4	-2.36	2.21	-2.34	2.25	-2.06	2.17	-2.03	2.14
Income, β_5	-0.42	0.26	-0.33	0.26	-0.31	0.26	-0.28	0.26
Education (reference is high school graduate)								
Some college attendance, β_6	5.70*	2.36	6.25*	2.41	5.79*	2.31	6.03*	2.33
Four-year college degree, β_7	2.33	2.52	3.67	2.61	4.36	2.56	4.54	2.56
Sleep aids, β_8	7.20	3.81	7.04	3.77	7.51*	3.57	7.64*	3.43
Body mass index, β_9					0.25	0.18	0.26	0.18
Current smoker, β_{10}					5.81	3.16	4.75	3.15
Alcohol consumption, β_{11}					2.95*	1.25	3.12*	1.24
Physical activity, β_{12}					-0.02	0.02	-0.01	0.02
Depression symptoms, β_{13}							0.28	0.17
Population density, γ_{01}	0.10	0.21	0.08	0.22	0.11	0.21	0.13	0.21
Neighborhood disadvantage, γ_{02}			3.54*	1.61	3.21*	1.57	3.16*	1.60

Note. Unstandardized parameter estimates and standard errors are shown. Units of the outcome are minutes spent awake between sleep onset and rise time. *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$.

moderation of racial disparities in sleep (all race by sex interaction $p_s \geq 0.30$).

2.2.2. $H_{2,3}$: neighborhood disadvantage as predictor of sleep and sleep disparities

In Model 2, neighborhood disadvantage was added as a predictor of each of the sleep indicators and the extent to which racial disparities in sleep were attenuated was considered. Results indicated that neighborhood disadvantage was associated with WASO ($B = 3.54$, $p = 0.028$, 95% CI: 0.39, 6.68) but not total sleep time ($B = -2.21$, $p = 0.60$, 95% CI: -10.38, 5.96) or sleep onset latency ($B = 1.55$, $p = 0.38$, 95% CI: -1.88, 4.98). Further, the disparity in WASO between AAs and EAs was attenuated by 24% when controlling for neighborhood disadvantage (Table 2). In order to formally test mediation using established methods [65], a multi-level structural equation model was also estimated to consider the indirect effect of race on WASO through neighborhood disadvantage controlling for age, sex, cohabitation, income, education, use of a sleep aid, and population density. Results of this model indicated a significant indirect effect ($B = 4.63$, $p = 0.035$, 95% CI: 0.34, 8.92), confirming that attenuation of race differences after adding neighborhood disadvantage to the model is indicative of mediation.

2.2.3. H_4 : health measures as explanatory covariates

To consider potential explanations or confounders of the association between neighborhood disadvantage and WASO, health behaviors, obesity, and depressive symptoms were added as covariates. First, after adjusting for health behaviors (smoker status, alcohol use, physical activity), and BMI (Model 3 in Table 2), the association between neighborhood disadvantage and WASO was attenuated by nine percent but remained significant ($B = 3.21$, $p = 0.041$, 95% CI: 0.13, 6.29). Examination of Model 3 parameter estimates indicated that greater alcohol use was associated with more WASO ($B = 2.95$, $p = 0.018$, 95% CI: 0.51, 5.40), and smoker status was a marginally significant predictor ($B = 5.81$, $p = 0.066$, 95% CI: -0.40, 12.01), such that those who smoke experienced more WASO. Additional analyses (not shown) indicated that neighborhood disadvantage was associated with risk of being a smoker ($B = 0.65$, $p = 0.001$, 95% CI: 0.26, 1.05), but not number of alcoholic drinks consumed ($B = 0.03$, $p = 0.39$, 95% CI: -0.04, 0.10). Model 4 further adjusted for depressive symptoms. Depressive symptoms

was marginally associated with WASO ($B = 0.28$, $p = 0.09$, 95% CI: -0.05, 0.60) yet the association between neighborhood disadvantage and WASO remained similar in magnitude ($B = 3.16$, $p = 0.049$, 95% CI: 0.02, 6.30). Specifically, the neighborhood disadvantage coefficient was attenuated only by an additional two percent when depressive symptoms were added (total of 11% drop in the neighborhood disadvantage coefficient between Models 2 and 4). Together, neighborhood disadvantage, depression, health behaviors, and obesity explained 43%, 25%, and 27% of the race difference in time awake after sleep onset, sleep minutes, and sleep onset latency, respectively. Full model results for sleep minutes and sleep onset latency are shown in an Appendix (as supplemental online material).

3. Discussion

Understanding mechanisms for racial health disparities is an essential component of broader efforts to achieve health equity and improve population health [38]. However, studies identifying social determinants of racial disparities in sleep are scarce [27,64], despite widely documented race differences in both neighborhood quality and sleep problems. The principle aim of this study was to bring together these two literature by examining associations between neighborhood disadvantage and sleep and considering the degree to which neighborhood disadvantage accounted for sleep disparities.

Results indicated that Census tract neighborhood disadvantage was significantly associated with waking after sleep onset (WASO)—an important indicator of sleep efficiency with robust links to subsequent health and well-being [41]. Furthermore, neighborhood disadvantage accounted for a substantial portion of race differences in WASO, attenuating racial disparities in this sleep parameter by 24%. To our knowledge, this is the first study to show that race differences in objectively measured sleep are partly accounted for by neighborhood disadvantage. Results therefore indicate that, with respect to race disparities in sleep, neighborhoods are a consequential factor. These findings also converge with other studies of neighborhood influences on health, to underscore that the racial differences in neighborhood environments must be viewed as essential for addressing and mitigating racial health disparities [52,78].

Limited prior research has studied objective neighborhood influences on sleep. [1]; demonstrate that neighborhood disadvantage predicts sleep efficiency among adolescents, and the current study extends these findings to show a similar association in an adult sample. Our finding that neighborhood disadvantage was not associated with sleep minutes is, however, consistent with the prior finding that objective neighborhood measures are associated with actigraphy assessments of sleep minutes among adolescents [1] and measures of sleep duration among adults [62,91]. The reason for nonsignificant effects on sleep minutes in the current study is unclear; however, because sleep duration and quality provide different information about an individual's sleep, it is not atypical to have significant findings for sleep fragmentation (low efficiency and wake episodes) and not duration (or smaller effects for duration). For example, in Ref. [2], concerns about community violence were associated with sleep efficiency and long wake episodes in expected directions, yet no effects for the full sample were observed for duration. Furthermore, other recent research has found stress exposure and psychological distress are often more strongly linked to sleep efficiency (eg, wake episodes) than sleep duration (eg, [17,18]). Additional studies are needed to further explicate the specific neighborhood-level predictors of sleep efficiency and total sleep duration, as well as the relative magnitude of effects on these different sleep parameters.

Although BMI and health behaviors accounted for a small portion of neighborhood effects (nine percent), the association between neighborhood disadvantage and waking episodes was largely independent of these variables, suggesting that other pathways relating to psychosocial stress or the physical environment may be at play. One important future direction will be to consider *how* neighborhood environments get under the skin to influence sleep and sleep disparities. Possible avenues for such inquiry include perceptions of safety, social cohesion, or exposure to toxins [20,31,36]. For example, factors related to the physical environment (eg, noise) as well as the social environment (eg, presence of crime) are specific pathways that may further elucidate the relationship between neighborhood disadvantage and sleep. In addition, in light of longstanding antagonistic relationships between law enforcement and disadvantaged communities of color—an issue which has been more fully exposed in recent years—finding ways to quantify policing practices at the neighborhood level and consider associations with sleep and other health outcomes for minority residents may be another important area of inquiry. Furthermore, understanding how racism-related stresses and subsequent hyper-vigilance may interact with neighborhood environment factors to adversely impact sleep outcomes will also be crucial.

3.1. Limitations

There are several noteworthy limitations in this study. One caveat is the use of a city-specific sample of AA participants, while the EA sample was more broadly distributed across the region. Use of different sampling strategies raises questions related to comparability and generalizability, and will also give credence to potential confounders (ie, population density). Analyses were considered with population density as a control (shown) and without population density (not shown). Findings did not support the notion that race differences in sleep were accounted for by population density, and are therefore consistent with our interpretation that neighborhood disadvantage (not population density) is responsible for some of the racial disparity in sleep. Furthermore, it should be noted that the sampling strategy ensured participation of a substantial number of AAs and reflects the demographics of the region; specifically, the majority of AAs in the region tend to reside in urban

areas, while EAs are more likely to inhabit rural and suburban locations. Nonetheless, additional research with larger and more randomly obtained samples will be needed to establish the generalizability of our findings.

Another caveat is that the broader MIDUS study did not sample by census tract, meaning there were relatively few individuals (level 1 units) per cluster (level 2 unit). Simulation studies considering the influence of clustering and sample size on parameter estimates in multilevel models indicate that level 2 variance components may be inflated with very few units per cluster [11]. However, fixed effect (β) estimates and their standard errors are robust to these variations in clustering, even with a small number of units per cluster and a sizable portion of clusters with only one unit [11,49,50]. Furthermore, in datasets with a large number of clusters (>100), confidence in parameter estimates is bolstered [49,50]. Overall, the methodological literature on multilevel modeling provides confidence with the analytic strategy chosen, particularly for the estimation of fixed effects.

A third limitation relates to the census tract measures of neighborhood disadvantage. Although the objective nature of these measures is a notable strength, census tract boundaries do not always map well onto an individuals' lived experience of their neighborhood. Furthermore, important additional information is likely to be captured by subjective self-report measures of neighborhood characteristics [92]. Future work which codes observed characteristics of neighborhoods (eg, street view data) and incorporates other neighborhood measures (eg, walkability, ambient noise, crime rates, perceptions of neighborhood safety and chaos) is therefore needed to identify additional consequential neighborhood characteristics not captured by census tract measures. Building on existing work [30], studies considering the degree to which effects of census tract neighborhood measures on sleep may operate through subjective perceptions and other objective measures of neighborhood environments are also warranted.

Despite these limitations, findings suggest that neighborhood factors contribute to and may partially explain racial disparities in waking episodes—an important indicator of sleep efficiency with strong links to subsequent health and well-being. Addressing race differences in neighborhood disadvantage and mitigating its effects on sleep are therefore likely to be important targets in efforts to reduce racial health disparities.

Conflict of interest

None.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: <http://dx.doi.org/10.1016/j.sleep.2016.10.008>.

Appendix A. Supplementary data

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.sleep.2016.10.008>.

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