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Sleep duration and breast cancer risk among black and white women

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Abstract

Background—Sleep has been suggested to influence breast cancer risk, however the evidence is mixed. Black women have higher prevalence of both short (<6 hr) and long (≥9 hr) sleep duration and are more likely to develop more aggressive, hormone receptor negative breast cancer. No study has examined the relationship between sleep and breast cancer in blacks. We focused on race-specific associations among the blacks.

Methods—In the Southern Community Cohort Study (SCCS), a prospective study of which 2/3 of the population were black, we prospectively investigated self-reported sleep duration in relation to overall breast cancer risk and by estrogen (ER) and progesterone receptor (PR) status in all women and in black women alone.

Results—Sleep duration was not associated with risk of total or hormone receptor positive breast cancer. However, we found a suggestion of an inverse relationship between sleep duration and risk of ER− and PR− breast cancer among all women and black women. Compared to the reference group (8 hr), black women who reported shorter sleep had an increased risk of ER−PR− breast cancer (odds ratios (95% confidence intervals): 2.13 (1.15, 3.93), 1.66 (0.92, 3.02) and 2.22 (1.19, 4.12) for <6, 6 and 7 hr, respectively, p for trend, 0.04).

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Potential competing interests

The authors declared no conflict of interest.

Conclusion—Short sleep may be a risk factor for hormone receptor negative breast cancer among black women.

Keywords

Sleep; breast cancer; Black women; hormone receptor positive breast cancer; hormone receptor negative breast cancer

Introduction

Night time sleep, a critical component of the 24 hour circadian rhythm, has been hypothesized to play a role in breast cancer development (1). Mechanistic studies have shown that melatonin, a key hormone involved in circadian regulation, can interact with the estrogen signaling pathway and may act as a tumor suppressor (2). Moreover, sleep deficit is associated with various breast cancer risk factors, including obesity (3), metabolic dysfunction (4) and chronic inflammation (5). Previous studies have found an elevated breast cancer risk among night shift workers (6), further supporting a potential carcinogenic effect of circadian disruption.

Several epidemiologic studies have examined sleep duration in relation to breast cancer risk, but their findings are inconsistent (7–13). Two prospective studies found an inverse association, with one reporting an elevated risk among short sleepers (<6 hour) (10), and the other reporting reduced risk among long sleepers (≥9 hour) (7), comparing to women with 7–8 hours of sleep. In contrast, a case-control study suggested a positive trend of modestly increased risk with longer sleep (9). Four studies, one case-control (11) and three large cohort studies (8, 12, 13), had largely null findings.

In addition to these conflicting findings, another deficit in the literature is lack of examinations of sleep duration and breast cancer risk in black women. In the United States, blacks have the highest prevalence of both long (≥9 h) and short (<6 h) sleep durations (14), making it particularly important to understand the health consequences of insufficient and excessive sleep in this population. Moreover, black women are more likely to develop more aggressive, hormone receptor negative breast cancer than white women (15). Causes of this disparity remain unclear and may involve biological, social and lifestyle factors (16). Little is known about whether the associations between sleep duration and breast cancer differ by tumor subtypes, and whether racial differences in sleep duration contribute to racial disparities in breast cancer. To date, only two studies have examined the effect of sleep duration on breast cancer risk by estrogen receptor (ER) status and both reported null findings for ER+ and ER− subtypes (11, 13).

In this study, we investigated sleep duration in relation to breast cancer risk in the Southern Community Cohort Study (SCCS), a prospective study that enrolled a large number of blacks. We placed special emphasis on tumor subtypes by ER and progesterone receptor (PR) status and examined the race-specific associations among the blacks.

Methods

Study population

The SCCS is a prospective study initiated in 2002 focusing on racial and socioeconomic disparities in the risk of cancer and other chronic diseases (17). Between 2002 and 2009, over 85,000 men and women, aged 40 to 79, were recruited from 12 southeastern states. The majority of the participants (86%) were enrolled from 71 Community Health Centers (CHC), institutions providing basic health and preventive services mainly to low income, underinsured and uninsured persons. The rest of the study participants (14%) enrolled in 2004–2006 by responding to a mailed questionnaire sent to randomly selected residents of the same 12 states. Informed consent was obtained from each participant upon enrollment into the SCCS. Institutional Review Boards at Vanderbilt University (Nashville, TN) and Meharry Medical College (Nashville, TN) approved the study.

For this analysis, of the 48,970 women with information on sleep duration, we excluded those who reported a previous diagnosis of cancer except for nonmelanoma skin cancer (N=3,872), and those who resided outside the 11 states (Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Mississippi, North Carolina, South Carolina, Tennessee, and Virginia) where cancer registry data was available (N=2,145). The final analytic sample included 42,953 women.

Breast cancer ascertainment

Incident breast cancer cases were identified via linkage to state cancer registries. Because information on follow-up times for cancer incidence varied from state to state and censoring dates were not rigid, we used a nested case-control design. All incident invasive breast cancers were included as cases (N=518) and controls were all the other female participants who were free of breast cancer at the most recent registry linkage (N=42,435). Data on ER status and PR status were obtained from the cancer registries and supplemented by pathology reports and medical records. ER and PR status was available for 438 and 436 women, respectively (status for both ER and PR was available for 430 cases).

Assessment of sleep duration and covariates

In the baseline questionnaire, participants were asked how many hours they typically slept in a 24-hour period, on weekdays and weekends separately. In addition, we calculated a weighted average sleep duration per 24 hours ((weekday sleep duration x 5)+(weekend sleep duration x 2)/7). We grouped weekday and weekend into 5 categories: <6 hr, 6 hr, 7 hr, 8 hour and ≥9 hr. Average sleep duration was rounded and grouped into the same 5 categories. The largest group (8 hr) was considered as the reference. In analysis of tumor subtypes among white women, we combined the 8 and 9+ hour groups to form the reference group to preserve statistical power. To examine the effects of different weekday vs. weekend sleep patterns, we also categorized participants as consistently normal/long sleepers (8+ hour of sleep on both weekdays and weekends), consistently short sleepers (<8 hour of sleep on both weekdays and weekends), weekday-only short sleepers (<8 hour of sleep on weekdays but 8+ hour of sleep on weekends), and weekend-only short sleepers (<8 hour of sleep on weekends but 8+ hour of sleep on weekdays).

The baseline questionnaire also collected comprehensive information on demographic characteristics, height and weight, medical history, reproductive history, hormone therapy use, family history of cancer, and diet and other lifestyle factors such as physical activity, alcohol drinking, and smoking.

Statistical analysis

Participants in different sleep categories were compared using χ^2 statistics for categorical characteristics and the nonparametric Kruskal Wallis test for continuous variables. Logistic regression models were used to estimate odds ratios (OR) and 95% confidence intervals (CI) for incident breast cancer for each category of sleep duration, with 8 hour of sleep or consistent normal/long sleepers as the reference groups. In the multivariable models we adjusted for potential confounders and risk factors for breast cancer, including age (continuous), enrollment year (continuous), race (black, white, other, or missing), enrollment state (Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Mississippi, North Carolina, South Carolina, Tennessee, and Virginia), education (less than high school, high school or GED, some college or vocational training, college graduate or higher, or missing), marital status (single, married, separated, divorced or widowed, or missing), income (< $15,000,$ 15,000–<$25,000, $25,000–<$50,000, ≥$50,000, or missing), body-mass index (<25, 25–<30, 30+ kg/m^2 or missing), moderate-to-vigorous physical activity (quartiles, metabolic equivalent), overall sitting (quartiles, hours/day), smoking status (current, former, never or missing), pack-years of smoking (0, 0–20, 21–40, 40+, or missing), number of live births (0, 1, 2+, or missing), age at first birth (nulliparous, <20, 20–<30, 30 years, or missing), length of breast feeding (nulliparous, 0–<1 year, ≥1 year, or missing), age at menarche (≤12, >12 years of age, or missing), post-menopausal (yes, no, or missing), ever use of menopausal hormone therapy (yes, no, or missing), current use of multivitamins (yes, no, or missing), current use of aspirin (yes, no, or missing), history of diabetes (yes, no, or missing), family history of cancer among first degree relatives (yes, no, or missing), average number of alcoholic drinks consumed per day (0, >0–1, >1, or missing), and daily intake of total fat (continuous), fiber (continuous), folate (continuous) and total calories (continuous). For all dietary intakes, missing values (N=2,297) were set at mean, and were adjusted for total energy intake using the density method (dividing intake amount by total calories). Tests for linear trend were performed by modeling a numeric value (1 through 5) for each sleep category.

We conducted additional subgroup analyses stratified by race (white/black), as well as by ER and PR status. In sensitivity analyses, we also excluded cases diagnosed within 2 years after enrollment. Additionally, we tested for interactions between sleep duration and age, education, BMI, and menopausal status using the likelihood ratio test comparing models with the cross-product terms to those without. All analyses were performed using SAS (SAS 9.3; SAS Institute, Cary, North Carolina).

Results

At baseline, 28% of the women reported sleeping 8 hours per night (our analytic referent) on weekdays, 59% reported <8 hours and 13% reported ≥9 hours. On weekends, 29%, 50% and

21% reported 8, <8 and ≥9 hours of sleep, respectively. Baseline characteristics by weekday sleep duration are presented in table 1 (race-specific baseline characteristics by weekday sleep duration, supplementary table 1). Compared to women who reported 8 hours of sleep, both <6 hr and ≥9 hr groups were less likely to have a college education, or to be married, but they were more likely to be current smokers and report household income less than $15,000. Moreover, sleep duration was inversely associated with early menarche and physical activity.

We did not observe an association between overall breast cancer risk and weekday sleep duration, weekend sleep duration, or the weighted average of weekday and weekend sleep duration, in combined analyses of the entire cohort (table 2), or in analyses separately conducted among white or black women (supplementary table 2). Adjusting for potential confounders had little impact on the results, and the null associations were unchanged after we excluded cases diagnosed within the first 2 years of follow up. We also found no interaction between sleep duration and age, education, income, BMI or menopausal status on the risk of breast cancer (data not shown).

Because people who had more variation in sleep duration over a week's time may experience more circadian disruption, we examined the relationship between weekday/weekend sleep patterns and breast cancer risk. Compared to consistent normal/long sleepers (who reported sleeping 8 hour or more on both weekdays and weekends), we did not find a significant difference in overall breast cancer risk among women who were consistent short sleepers (OR (95% CI): 1.08 (0.89, 1.31)), weekday-only short sleepers (1.16 (0.87, 1.54)) or weekend-only short sleepers (0.98 (0.59, 1.62)). These results were similar for black and white women separately (data not shown).

We further examined the relationship between sleep duration and breast cancer by hormone receptor status. Among black women, we did not find an association between weekday sleep duration and ER+, PR+ and ER+PR+ breast cancers (table 3). However, we found a suggestion of elevated risks for ER−, PR− and ER−PR− breast cancers among black women who reported <8 hours of sleep on weekdays. When compared to the reference group the ORs (95% CI) for ER−PR− breast cancer were 2.13 (1.15, 3.93), 1.66 (0.92, 3.02) and 2.22 (1.19, 4.12) for <6 hr, 6 hour and 7 hours of sleep, respectively (p for trend, 0.04). In contrast, we did not observe a similar association with hormone receptor-negative breast cancer among white women, but the number of cases was small (supplementary table 3); and the results for all women were largely similar to those for black women (supplementary table 4). Finally, we observed no association between weekend sleep duration and breast cancer risk by hormone receptor status, and the results for average sleep duration were similar to those for weekday sleep.

Discussion

In this cohort comprised of predominantly black women, we did not observe an association between sleep duration and overall breast cancer risk, either in the total study population or among black or white women when examined separately. However, we found a suggestion

of an elevated risk of ER− and PR− breast cancer subtypes among the subgroup of black women who reported less than 8 hours of sleep on weekdays.

The lack of association between sleep duration and overall breast cancer risk is consistent with the findings from some previous studies, particularly the two large cohort studies, the Nurses' Health Study (NHS) (8) and the Women's Health Initiative (WHI) (13). Both studies included thousands of breast cancer cases (4,223 in NHS and 5,149 in WHI). Compared to the reference group (7 hours of sleep), the multivariate relative risk (95% CI) associated with ≤5 hour and ≥9 hour were 0.93 (0.79–1.09) and 0.95 (0.82–1.11) in the NHS and 0.95 (0.85, 1.07) and 1.03 (0.90, 1.18) in the WHI. In contrast, the two cohort studies that reported an inverse relationship between sleep duration and overall breast cancer risk were smaller (242 for the Finnish Twin cohort (7) and 143 for the Japanese Ohsaki cohort (10)). A recent meta-analysis that summarized six of the seven studies (without the WHI study) reported no evidence for a relationship between sleep duration and breast cancer risk (18).

We found a suggestion of increased risk of hormone receptor negative tumors among women reporting less than 8 hours of sleep. In contrast, previous studies that examined ER− tumors reported no association with sleep duration (11, 13). One of the key differences between our study and the other studies is that two-thirds of our study population are blacks, while the two earlier studies were of predominantly European ancestry populations. Although we had small numbers of ER− and PR− tumors and the findings could be due to chance alone, especially given the multiple comparisons made, the elevated risk among black women with short sleep may deserve further investigation. Compared to their white counterparts, black women are more likely to develop more aggressive, hormone receptor negative tumors (15), and have short sleep duration (14). Therefore it would be important to examine whether insufficient sleep contributes to the racial disparities of aggressive breast cancer. Additionally, circadian disruption has been associated with factors that may influence breast cancer risk independent of estrogen pathways, such as metabolic dysfunction (4) and chronic inflammation (5), suggesting that a link between short sleep and hormone receptor negative breast cancer is biologically plausible.

We found no association between sleep duration and hormone receptor positive tumors. An Australian case-control study and the WHI also showed no relationship between sleep duration and ER+ tumors (11, 13). These findings are contradictory to the melatonin hypothesis, which suggests that short sleep duration is associated with decreased levels of melatonin, a molecule with anti-estrogenic effects that may protect against ER+ tumors (1). Although studies have linked low levels of melatonin with increased breast cancer risk, supporting a role of melatonin in breast carcinogenesis (19–21), there is little evidence showing a link between self-reported sleep duration and melatonin level and previous studies on this topic showed mixing results (12, 22, 23). The secretion of melatonin is promoted by the absence of light at night and does not require the occurrence of sleep (1). Therefore, self-reported sleep duration may be a poor marker of nighttime light exposure and melatonin production, which may partially explain the lack of association between sleep duration and hormone receptor positive breast cancer in the literature.

Our study has several limitations. First, sleep duration was self-reported. In another population, a validation study comparing self-reported and actigraphy-measured sleep duration among adults found a moderate correlation (correlation coefficient ~0.45) between the two (24), and sleep duration reported in questionnaire tended to be longer than the measured duration, particularly among people with short sleep duration and among blacks. Therefore, such measurement error in sleep duration reporting may lead to a misclassification of short sleepers as normal/long sleepers, which would have attenuated the results in our study. Second, we only had a single report of sleep duration, and were not able to capture a long term sleep patterns among the study population. As argued by a recent paper, to better understand the relationship between sleep and cancer risk, it may be important to evaluate the cumulative exposure of short or long sleep in critical time windows of cancer development (25). Third, we lacked information on other important aspects of sleep and circadian rhythm which may also influence breast cancer risk, such as sleep quality, snoring, sleep disorder, and chronotype (2). Moreover, we did not have information on shift work, another potentially important confounder of the relationship between sleep and breast cancer risk. Lastly, we were underpowered to examine ER− and PR− tumors, thus our findings should be interpreted cautiously. Particularly, we had a small number of hormone receptor negative cases among the white women, and thus are limited in commenting on the true likelihood of effect modification by race..

Our study does have several important advantages. This is a large prospective study of a well characterized population, with sleep duration measured before the development of breast cancer. Also, we were able to perform sensitivity analyses in which cases diagnosed within two years after sleep assessment were excluded, which should reduce the likelihood of reverse causation (e.g., that undiagnosed breast cancer influenced reported sleep patterns). We controlled for a large number of potential confounders, including demographics, socioeconomic status, reproductive factors, smoking, alcohol, diet and physical activity. Finally, we had a large population of black women included in this analysis, which allowed us to conduct analyses specific to black women for the first time.

In conclusion, our findings suggest that sleep duration has no effect on hormone receptor positive breast cancers, but we could not exclude the possibility of increased risks for ER− and PR− tumors among black women with short sleep. Our study is the first to examine sleep and breast cancer risk among black women. Given the high prevalence of insufficient sleep in this population, more studies are needed to investigate its potential health consequences.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviation

BMI	body-mass index
CIs	confidence intervals
MET	metabolic equivalent
MHT	menopausal hormone therapy
MVPA	moderate-to-vigorous physical activity
OR	odds ratio
SD	standard deviation

References

1. Blask DE. Melatonin, sleep disturbance and cancer risk. Sleep Med Rev. 2009; 13(4):257–64. [PubMed: 19095474]
2. Stevens RG. Circadian disruption and breast cancer: from melatonin to clock genes. Epidemiology. 2005; 16(2):254–8. [PubMed: 15703542]
3. Van Cauter E, Knutson KL. Sleep and the epidemic of obesity in children and adults. Eur J Endocrinol. 2008; 159(Suppl 1):S59–66. [PubMed: 18719052]
4. Gangwisch JE, Heymsfield SB, Boden-Albala B, Buijs RM, Kreier F, Pickering TG, et al. Sleep duration as a risk factor for diabetes incidence in a large U.S sample. Sleep. 2007; 30(12):1667–73. [PubMed: 18246976]
5. Irwin MR, Wang M, Campomayor CO, Collado-Hidalgo A, Cole S. Sleep deprivation and activation of morning levels of cellular and genomic markers of inflammation. Arch Intern Med. 2006; 166(16):1756–62. [PubMed: 16983055]
6. Wang F, Yeung KL, Chan WC, Kwok CC, Leung SL, Wu C, et al. A meta-analysis on dose-response relationship between night shift work and the risk of breast cancer. Ann Oncol. 2013; 24(11):2724–32. [PubMed: 23975662]
7. Verkasalo PK, Lillberg K, Stevens RG, Hublin C, Partinen M, Koskenvuo M, et al. Sleep duration and breast cancer: a prospective cohort study. Cancer Res. 2005; 65(20):9595–600. [PubMed: 16230426]
8. Pinheiro SP, Schernhammer ES, Tworoger SS, Michels KB. A prospective study on habitual duration of sleep and incidence of breast cancer in a large cohort of women. Cancer Res. 2006; 66(10):5521–5. [PubMed: 16707482]
9. McElroy JA, Newcomb PA, Titus-Ernstoff L, Trentham-Dietz A, Hampton JM, Egan KM. Duration of sleep and breast cancer risk in a large population-based case-control study. J Sleep Res. 2006; 15(3):241–9. [PubMed: 16911025]
10. Kakizaki M, Kuriyama S, Sone T, Ohmori-Matsuda K, Hozawa A, Nakaya N, et al. Sleep duration and the risk of breast cancer: the Ohsaki Cohort Study. Br J Cancer. 2008; 99(9):1502–5. [PubMed: 18813313]
11. Girschik J, Heyworth J, Fritschi L. Self-reported sleep duration, sleep quality, and breast cancer risk in a population-based case-control study. Am J Epidemiol. 2013; 177(4):316–27. [PubMed: 23324334]
12. Wu AH, Stanczyk FZ, Wang R, Koh WP, Yuan JM, Yu MC. Sleep duration, spot urinary 6-sulfatoxymelatonin levels and risk of breast cancer among Chinese women in Singapore. Int J Cancer. 2013; 132(4):891–6. [PubMed: 22644618]
13. Vogtmann E, Levitan EB, Hale L, Shikany JM, Shah NA, Endeshaw Y, et al. Association between sleep and breast cancer incidence among postmenopausal women in the women's health initiative. Sleep. 2013; 36(10):1437–44. [PubMed: 24082303]

14. Adenekan B, Pandey A, McKenzie S, Zizi F, Casimir GJ, Jean-Louis G. Sleep in America: role of racial/ethnic differences. Sleep Med Rev. 2013; 17(4):255–62. [PubMed: 23348004]
15. Hausauer AK, Keegan TH, Chang ET, Clarke CA. Recent breast cancer trends among Asian/Pacific Islander, Hispanic, and African-American women in the US: changes by tumor subtype. Breast Cancer Res. 2007; 9(6):R90. [PubMed: 18162138]
16. Ademuyiwa FO, Edge SB, Erwin DO, Orom H, Ambrosone CB, Underwood W 3rd. Breast cancer racial disparities: unanswered questions. Cancer Res. 2011; 71(3):640–4. [PubMed: 21135114]
17. Signorello LB, Hargreaves MK, Steinwandel MD, Zheng W, Cai Q, Schlundt DG, et al. Southern community cohort study: establishing a cohort to investigate health disparities. J Natl Med Assoc. 2005; 97(7):972–9. [PubMed: 16080667]
18. Qin Y, Zhou Y, Zhang X, Wei X, He J. Sleep duration and breast cancer risk: A meta-analysis of observational studies. Int J Cancer. 2013
19. Schernhammer ES, Hankinson SE. Urinary melatonin levels and breast cancer risk. J Natl Cancer Inst. 2005; 97(14):1084–7. [PubMed: 16030307]
20. Schernhammer ES, Hankinson SE. Urinary melatonin levels and postmenopausal breast cancer risk in the Nurses' Health Study cohort. Cancer Epidemiol Biomarkers Prev. 2009; 18(1):74–9. [PubMed: 19124483]
21. Schernhammer ES, Berrino F, Krogh V, Secreto G, Micheli A, Venturelli E, et al. Urinary 6-Sulphatoxymelatonin levels and risk of breast cancer in premenopausal women: the ORDET cohort. Cancer Epidemiol Biomarkers Prev. 2010; 19(3):729–37. [PubMed: 20200429]
22. Wu AH, Wang R, Koh WP, Stanczyk FZ, Lee HP, Yu MC. Sleep duration, melatonin and breast cancer among Chinese women in Singapore. Carcinogenesis. 2008; 29(6):1244–8. [PubMed: 18448486]
23. Grundy A, Sanchez M, Richardson H, Tranmer J, Borugian M, Graham CH, et al. Light intensity exposure, sleep duration, physical activity, and biomarkers of melatonin among rotating shift nurses. Chronobiol Int. 2009; 26(7):1443–61. [PubMed: 19916841]
24. Lauderdale DS, Knutson KL, Yan LL, Liu K, Rathouz PJ. Self-reported and measured sleep duration: how similar are they? Epidemiology. 2008; 19(6):838–45. [PubMed: 18854708]
25. Erren TC. Sleep duration and cancer risk: time to use a "sleep-years" index? Cancer Causes Control. 2012; 23(9):1399–403. [PubMed: 22806258]

Highlights

- We did not observe an association between sleep duration and overall breast cancer risk
- We found a suggestion of an elevated risk of ER− and PR− breast cancer subtypes among the subgroup of black women who reported less than 8 hours of sleep
- Our study is the first to examine sleep and breast cancer risk among black women

Table 1

Baseline characteristics of women in the Southern Community Cohort Study, 2002–2009

	Weekday sleep duration, hr					
	<6	**6**	**7**	**8**	**≥9**	***p-value***
N (%)	7503 (17.5)	10296 (24.0)	7303 (17.1)	12072 (28.2)	5666 (13.2)	
Age, yr, mean (SD)	51.2 (8.2)	52.3 (8.8)	53.0 (9.1)	52.4 (9.1)	51.7 (8.9)	*<.0001*
BMI, kg/m2, mean (SD)	32.6 (8.4)	32.1 (8.0)	31.5 (7.8)	31.5 (7.9)	32.2 (8.5)	*<.0001*
White, non-Hispanic, %	26.4	27.6	33.0	26.2	22.0	*<.0001*
Black, non-Hispanic, %	68.6	67.6	63.0	70.1	73.9	*<.0001*
College or post college, %	9.1	14.0	17.3	12.0	8.1	*<.0001*
Married, %	28.0	33.0	36.7	32.2	29.9	*<.0001*
Household income <$15,000, %	62.1	51.5	47.1	57.0	64.3	*<.0001*
Family history of any cancer, %	23.4	23.9	23.0	23.5	20.6	*<.0001*
Current smoker, %	37.2	32.7	27.9	33.4	38.5	*<.0001*
Nulliparous, %	9.7	10.0	10.7	10.6	10.9	*0.002*
Age at menarche<=12, %	49.1	48.7	47.4	45.2	42.1	*<.0001*
Post-menopausal, %	68.0	68.5	68.6	65.5	63.2	*<.0001*
MHT use, %	27.4	30.0	32.0	25.9	24.1	*<.0001*
Diabetes, %	24.4	22.2	21.5	22.8	25.9	*<.0001*
MVPA, MET hr/d, mean (SD)	13.6 (14.4)	13.7 (13.5)	13.0 (12.1)	13.0 (12.6)	12.1 (12.5)	*<.0001*
Multivitamin use, %	37.2	41.6	45.7	39.4	36.8	*<.0001*
Alcohol consumption, 1+ drink/d, %	11.4	10.5	9.9	12.1	14.1	*<.0001*
Dietary intakes, mean (SD)						
total calories	2300 (1329)	2164 (1202)	2087 (1136)	2250 (1285)	2391 (1349)	*<.0001*
total fat, gram/kcal	38.1 (7.0)	38.2 (6.7)	38.0 (6.6)	38.0 (6.7)	38.3 (6.9)	*0.31*
total fiber, gram/kcal	9.0 (3.2)	9.2 (3.1)	9.5 (3.2)	9.2 (3.2)	8.9 (3.1)	*<.0001*
total folate, mg/kcal	208 (64)	212 (64)	218 (65)	215 (64)	209 (62)	*<.0001*

Abbreviations: MET, metabolic equivalent; MHT, menopausal hormone therapy; MVPA, moderate-to-vigorous physical activity; SD, standard deviation

Table 2

Association between baseline sleep duration and breast cancer risk

		OR (95% CI)	
Sleep duration, hr	**No. of cases/controls**	**Age adjusted**	**Multivariate [a]**
Weekday			
<6	86/7417	1.09 (0.83, 1.43)	1.16 (0.88, 1.52)
6	123/10173	1.09 (0.85, 1.39)	1.09 (0.85, 1.40)
7	102/7201	1.24 (0.96, 1.61)	1.25 (0.96, 1.63)
8	134/11938	ref	ref
≥9	70/5596	1.15 (0.86, 1.53)	1.18 (0.88, 1.58)
p for trend		*0.87*	*0.70*
Weekend			
<6	69/6541	0.93 (0.70, 1.25)	1.01 (0.75, 1.35)
6	107/8203	1.10 (0.85, 1.41)	1.11 (0.86, 1.43)
7	83/6278	1.08 (0.82, 1.41)	1.09 (0.83, 1.44)
8	146/12277	ref	ref
≥9	113/9013	1.10 (0.86, 1.41)	1.11 (0.87, 1.43)
p for trend		*0.59*	*0.89*
Weighted average			
<6	89/7739	1.04 (0.79, 1.36)	1.09 (0.83, 1.44)
6	118/10003	1.01 (0.79, 1.30)	1.01 (0.78, 1.30)
7	113/8074	1.19 (0.92, 1.53)	1.19 (0.92, 1.54)
8	127/10662	ref	ref
≥9	72/5957	1.07 (0.80, 1.43)	1.08 (0.80, 1.45)
p for trend		*0.92*	*0.88*
Weekday, excluding cases diagnosed within 2 years after baseline			
<6	64/7417	1.13 (0.82, 1.56)	1.20 (0.87, 1.66)
6	95/10173	1.17 (0.88, 1.56)	1.18 (0.89, 1.58)
7	75/7201	1.27 (0.94, 1.72)	1.30 (0.96, 1.76)
8	96/11938	ref	ref
≥9	49/5596	1.12 (0.79, 1.58)	1.14 (0.80, 1.61)
p for trend		*0.50*	*0.35*

[a] adjusted for age, enrollment year, enrollment state, race, education, income, marital status, body-mass index, moderate-to-vigorous physical activity, overall sitting, smoking status, pack-year, number of live birth, age at first birth, length of breast feeding, age at menarche, menopause, use of menopausal hormone therapy, use of multivitamin, use of aspirin, history of diabetes, family history of cancer, alcohol consumption, and dietary intakes of total fat, fiber, folate and total calories.

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Table 3

Baseline weekday sleep duration and breast cancer risk among black women, by hormone receptor status

	Weekday sleep duration, hr					
Breast cancer subtype	**<6**	**6**	**7**	**8**	**≥9**	***p for trend***
ER +						
No. of cases	33	47	34	56	31	
Multivariate OR (95% CI) [a]	1.03 (0.66, 1.59)	0.98 (0.66, 1.45)	1.05 (0.68, 1.62)	ref	1.18 (0.76, 1.85)	*0.63*
ER −						
No. of cases	24	27	24	20	15	
Multivariate OR (95% CI) [a]	2.10 (1.15, 3.83)	1.65 (0.92, 2.96)	2.30 (1.26, 4.19)	ref	1.64 (0.84, 3.23)	*0.10*
PR +						
No. of cases	29	37	24	50	26	
Multivariate OR (95% CI) [a]	0.98 (0.62–1.56)	0.82 (0.54, 1.26)	0.80 (0.49, 1.30)	ref	1.12 (0.69, 1.81)	*0.41*
PR −						
No. of cases	27	37	32	26	20	
Multivariate OR (95% CI) [a]	1.85 (1.08, 3.19)	1.78 (1.07, 2.95)	2.36 (1.40, 3.99)	ref	1.66 (0.92, 2.98)	*0.12*
ER+/PR+						
No. of cases	28	35	23	47	23	
Multivariate OR (95% CI) [a]	1.02 (0.63, 1.63)	0.83 (0.53, 1.29)	0.82 (0.49, 1.36)	ref	1.06 (0.64, 1.76)	*0.62*
ER−/PR−						
No. of cases	23	26	22	19	12	
Multivariate OR (95% CI) [a]	2.13 (1.15, 3.93)	1.66 (0.92, 3.02)	2.22 (1.19, 4.12)	ref	1.38 (0.67, 2.85)	*0.04*

[a] adjusted for age, enrollment year, enrollment state, race, education, income, marital status, body-mass index, moderate-to-vigorous physical activity, overall sitting, smoking status, pack-year, number of live birth, age at first birth, length of breast feeding, age at menarche, menopause, use of menopausal hormone therapy, use of multivitamin, use of aspirin, history of diabetes, family history of cancer, alcohol consumption, and dietary intakes of total fat, fiber, folate and total calories.