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ORIGINAL PAPER

Sleep duration and mortality among breast cancer survivors in the Western New York Exposures and Breast Cancer (WEB) Study

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Abstract

Purpose There is increasing evidence that sleep duration may affect breast cancer survival through effects on circadian function, influencing disease progression. However, further investigation of this association is needed.

Methods In a population-based, prospective cohort study of women from the Western New York Exposures and Breast Cancer Study, we examined mortality outcomes with invasive breast cancer identified using the National Death Index. Cox proportion hazards ratios with 95% confidence intervals were used to estimate risk of all-cause (AC) and breast cancer-specific (BC) mortality associated with self-reported usual sleep duration with adjustment for age, race/ethnicity, years of education, body mass index (BMI), menopausal status, pack-years of smoking, tumor stage, and estrogen-receptor (ER) status. We further examined associations within strata of BMI, tumor stage, menopausal status, and ER status.

Results A sample of 817 patients with breast cancer were followed for a median of 18.7 years, during which 339 deaths were reported, including 132 breast cancer-specific deaths. Those who reported shorter or longer sleep tended to have a slightly higher BMI, to be less proportionately non-Hispanic White, to report a previous history of benign breast disease, and to have consumed more alcohol during their lifetime. We found no significant associations between sleep duration and AC or BC mortality, including within stratified analyses.

Conclusion Sleep duration was not associated with either AC or BC mortality including within strata of BMI, tumor stage, menopausal status, or ER status.

Keywords Breast cancer mortality · Epidemiology · Sleep

Introduction

There is well-established evidence of associations for shorter and longer sleep duration and several different chronic diseases and conditions in adulthood, including Type 2 diabetes, obesity, hypertension, and cardiovascular disease [1–3]. There has been a growing focus in understanding whether sleep plays a role in the progression of cancer, particularly breast cancer. In previous studies, numerous aspects of sleep have been studied in relation to breast cancer etiology. As a result, there is evidence that nightshift work and sleep deficiency are risk factors [4, 5]. Shorter sleep duration has been hypothesized to explain these observed associations [6]. Furthermore, investigations directly assessing sleep duration in relation to breast cancer incidence have yielded some supporting evidence of an association [7–9].

Given these findings, it is of importance to further investigate whether sleep duration affects breast cancer prognosis. Currently, there is evidence indicating an association between sleep duration and breast cancer mortality, however, these studies have largely involved measures of sleep postdiagnosis [10–13]. The assessment of post-diagnostic sleep measures must be interpreted with caution as it may reflect certain biases compared to pre-diagnostic measures. In particular, post-diagnostic sleep duration may be influenced by effects of treatment or the diagnosis itself [10, 14]. In some studies, the development of sleep difficulties has been suggested to be a response to menopausal symptoms

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initiated by chemotherapy or endocrine therapy [10, 15], as well as from mental stress and pain following cancer treatment [10, 13, 16, 17]. To avoid such evidence of potential reverse causality, it is critical to understand whether sleep is predictive of prognostic outcomes by assessing patterns prior to diagnosis. There have been few studies focused on pre-diagnostic sleep duration in relation to breast cancer outcomes [18, 19]. Furthermore, the direction of association between sleep duration and mortality remains poorly understood, with some studies reporting higher mortality risk for both shorter and longer sleep, while other evidence reflects associations for one or the other [10, 18, 19].

Pre-diagnostic shorter and longer sleep duration could affect mortality among women with breast cancer because of circadian rhythm disturbances that initiate disruption of both circadian clock gene function and physiological homeostasis, promoting disease progression [20, 21]. The International Agency for Research on Cancer (IARC) categorized such circadian disruption in relation to nightshift work as a likely human carcinogen [22]. Such effects may be modified by body mass composition, which has been previously associated with both sleep disturbance and breast cancer mortality [23, 24]. In some studies, inadequate sleep has been hypothesized to promote cancer by altering metabolic pathways related to obesity [25].

The current study builds upon our previous published work, in which we found associations of sleep disturbance with incidence of aggressive tumor subtypes among patients with breast cancer in the Western New York Exposures and Breast Cancer (WEB) Study [6]. In this previous study, we formulated a sleep disturbance score using multiple questions from a questionnaire detailing sleep difficulties and dysfunction. Associations of sleep disturbance and mortality were observed. Here we focus on pre-diagnostic sleep duration, examining the association with mortality among patients with invasive breast cancer in the same study cohort, with additional examination for effect modification by body mass index and tumor stage.

Methods

Data collection

The WEB Study is a population-based case–control study, conducted during 1996–2001, in Erie and Niagara counties in New York State [6, 26, 27]. Briefly, included in this prospective analysis were women aged 35–79 years old diagnosed with primary, histologically confirmed, incident invasive breast cancer. Patients with breast cancer were ascertained by nurse case-finders in hospitals within the Erie and Niagara County region. Patients were interviewed within 1 year of breast cancer diagnosis, with a 72% participation rate ($n = 1{,}170$). The average time between diagnosis and sleep assessment was 6.1 months. All study participants provided informed consent and the study protocol and procedures used were approved by the Institutional Review Boards (IRB) of both the University at Buffalo's Health Sciences and all participating hospitals. Given that some modifiable lifestyle behaviors, such as sleep, may have less direct impact on the prognosis of individuals with advanced stage breast cancer, we excluded all stage 4 cases ($n = 36$) from our analyses.

Information on demographics (age at interview, race/ethnicity, years of education), lifestyle factors, pregnancy (parity, age at first full-term live birth), menopause (pre/post-menopause, age at menarche, age at menopause, hormone therapy use), health behaviors (pack-years of smoking and lifetime alcohol consumption), personal health history (previous benign breast disease) and family health history (family history of breast cancer) were obtained through an in-person interview.

The age of the patients at interview, at menarche, and at menopause were assessed as continuous covariates. Age at first full-term live birth was assessed categorically. Race/ethnicity was assessed dichotomously (non-Hispanic White vs. all others). Years of education were determined based on years of education completed and assessed as a continuous covariate. Parity was the number of live births reported. Hormone therapy use was assessed dichotomously (ever vs. never) and limited to postmenopausal women only.

Pack-years of smoking was determined among those who reported yes to smoking at least 100 cigarettes during their lifetime. This variable was assessed as a continuous covariate. Lifetime alcohol consumption was determined as the total ounces of alcohol consumed from birth until 2 years prior to the patient's diagnosis/interview, and was also assessed as a continuous covariate. Previous benign breast disease and family history of breast cancer was determined by interview and assessed dichotomously (yes/no). Family history of breast cancer referred to history among first-degree relatives (mother, sister and/or daughter) of the patient and was assessed dichotomously (yes/no).

Physical measurements were recorded at the time of interview including height and weight by trained study personnel using a standardized protocol. Body mass index (BMI) was calculated as weight in kilograms (kg) divided the square of height in meters (m). In the analysis, BMI was adjusted for as a continuous covariate. For analyses stratified by BMI, values were split at the median, since statistical power was limited when using clinically relevant cut-off points such as underweight, normal weight, overweight, and obese BMI values.

Information on tumor characteristics, such as tumor stage, were obtained from medical records reviewed by trained nurses. Tumor stage was originally categorized as stages 0,

1, 2a, 2b, 3a, 3b, and 4. These analyses were restricted to patients with invasive breast cancer only. Hormone receptor status (estrogen receptor, ER and progesterone receptor, PR status) was determined by a single breast pathologist through immunohistochemistry. ER and PR positive tumors were defined as an Allred score of ≥ 3 [6, 28].

For the exposure assessment, participants were asked via a self-reported questionnaire about sleep duration during a usual 24-hour (h) period in a typical month 1 year before the interview. Responses were originally categorized as: less than 4 h, 4–5 h, 6–7 h, 8 h, 9–10 h, more than 10 h, don't know or refused. For our analysis, the sleep duration variable was modified into three categories [short (≤ 5 h), normal (6–8 h), long (≥ 9 h)]. There were 817 patients with invasive breast cancer with available sleep data.

Statistical analysis

To compare differences by sleep duration status, frequencies and chi-square tests were calculated for categorical variables while means/standard deviations and ANOVA tests were performed for continuous variables. Median and interquartile range were determined for lifetime alcohol consumption. Covariates in the adjusted models included age at interview, race/ethnicity, years of education, menopausal status, BMI, pack-years of smoking, tumor stage, and ER status. Covariates were selected from an a priori list of confounders based on published literature, using a directed acyclic graph, and using data-driven methods including a difference-based empirical approach of 10% or more between crude and adjusted estimates for each covariate individually.

Survival analysis

A survival analysis was conducted to evaluate whether sleep duration was associated with all-cause and breast cancer-specific mortality. Mortality outcomes were ascertained using the National Death Index. Survival time was calculated as the number of months from the date of diagnosis until date of death or the end of follow-up, 31 December 2018, whichever came first. Cox proportional hazards models were used to estimate mortality risk using hazards ratios and 95% confidence intervals. Two adjusted models were estimated: one was adjusted for age at interview, race/ethnicity, and years of education and a second model additionally adjusted for BMI, menopausal status, pack-years of smoking, tumor stage, and estrogen-receptor (ER) status. For BMI-stratified analyses, groups were categorized as below or above the median BMI value of the study sample (27.5 kg/m^2). Additional stratified analyses included by menopausal status, ER status, and stage 1 versus stage 2/3 breast tumors. In all analyses of sleep duration associations with breast cancer mortality, normal sleep (6–8 h) was used as the referent group, compared to shorter (≤ 5 h) and longer (≥ 9 h) sleep. Statistical significance was determined using two-sided $p < 0.05$ tests and all analyses were conducted using SAS 9.4 (SAS Institute, Inc., Cary, NC).

Results

Patients with breast cancer were followed for a median of 18.7 years, during which 339 deaths from all causes were confirmed, of which 132 were breast cancer-specific. A description of breast cancer risk factors categorized by sleep duration status in the final study sample are shown in Table 1. Significant differences were observed in race/ethnicity, BMI, alcohol consumption, pack-years of smoking, and previous benign breast disease. In comparison to those who reported normal sleep duration, cases who reported shorter or longer sleep were less proportionately non-Hispanic White, had a slightly higher average BMI, had a higher median consumption of alcohol during their lifetime, and were more likely to report a previous history of benign breast disease. Cases reporting longer sleep had a higher average pack-years of smoking compared to shorter and normal sleep, which were each relatively similar.

In the study sample overall and stratified by median BMI, 27.5 kg/m^2, neither short or long sleep duration was associated with all-cause or breast cancer-specific mortality, following adjustment for confounding variables (Table 2). Furthermore, there was no statistically significant interaction with BMI for either all-cause ($p = 0.60$) or breast cancer-specific mortality ($p = 0.16$). In the overall sample, we found non-significant reductions in all-cause and breast cancer-specific mortality risk for cases who reported shorter sleep compared to normal sleep, but the confidence interval included the null. Non-significant increases in all-cause and breast cancer-specific mortality risk for cases who reported longer sleep were observed, however confidence intervals again included the null. There were no significant associations in results stratified by tumor stage (1 and 2/3) (data not shown). Furthermore, there were no associations following stratification by ER status (data not shown). There were also no associations in results stratified by menopausal status, however, the sample size was relatively small for this comparison (data not shown). Furthermore, in a sensitivity analysis using 8 h of sleep as the referent group, all associations of sleep duration and mortality remained null, including for all stratified and unstratified analyses (data not shown).

Table 1 Characteristics of patients with breast cancer by sleep duration, WEB study, 1996–2001 ($n=817$)

Characteristic	Sleep duration			
	≤5 h per night ($n=101$)	6–8 h ($n=677$)	≥9 h ($n=39$)	p-value*
Age at interview, years	56.4 ± 11.1[a]	58.0 ± 10.9	58.5 ± 10.4	0.39
Race/ethnicity				< 0.01
Non-Hispanic White	84 (83.2)[b]	633 (93.5)	32 (82.1)	
All others	17 (16.8)	44 (6.5)	7 (17.9)	
Education, years	13.4 ± 2.3	13.5 ± 2.6	13.2 ± 3.2	0.67
Menopausal status				0.48
Premenopausal	31 (30.7)	190 (28.1)	8 (20.5)	
Postmenopausal	70 (69.3)	487 (71.9)	31 (79.5)	
BMI, kg/m^2	29.5 ± 6.5	28.5 ± 6.3	31.1 ± 7.9	0.02
Parity (number of births)	2.3 ± 1.6	2.3 ± 1.7	2.5 ± 2.1	0.77
Age at first birth, years				0.34
Nulliparous	17 (16.8)	116 (17.1)	8 (20.5)	
≤21	30 (29.7)	194 (28.7)	12 (30.8)	
22–25	37 (36.6)	192 (28.4)	13 (33.3)	
≥26	17 (16.8)	175 (25.8)	6 (15.4)	
Lifetime alcohol consumption, in ounces[c]	958.3 (2,612)	699.8 (2,453)	829.4 (6,163)	0.03
Unknown, n (%)	2 (2.0)	8 (1.2)	1 (2.6)	
Pack years of smoking	10.1 ± 16.6	10.6 ± 16.5	19.2 ± 25.8	< 0.01
Age at menarche, years	12.9 ± 2.0	12.6 ± 1.5	12.5 ± 1.8	0.09
Age at menopause, years	48.0 ± 6.1	48.3 ± 5.4	46.8 ± 6.2	0.34
Family history of breast cancer[d]				0.46
No	75 (74.3)	490 (72.4)	26 (66.7)	
Yes	19 (18.8)	134 (19.8)	11 (28.2)	
Unknown, n (%)	7 (6.9)	53 (7.8)	2 (5.1)	
Previous benign breast disease				0.02
No	56 (55.4)	447 (66.0)	19 (48.7)	
Yes	42 (41.6)	217 (32.1)	19 (48.7)	
Unknown, n (%)	3 (3.0)	13 (1.9)	1 (2.6)	
Hormone therapy use, ever[e]				0.73
No	58 (57.4)	420 (62.0)	24 (61.5)	
Yes	41 (40.6)	250 (37.0)	15 (38.5)	
Unknown, n (%)	2 (2.0)	7 (1.0)	0 (0.0)	
Tumor stage				0.33
I	58 (57.4)	401 (59.2)	21 (53.8)	
II a/II b	43 (42.6)	253 (37.4)	17 (43.6)	
III a/III b	0 (0.0)	23 (3.4)	1 (2.6)	
Estrogen receptor (ER) status				0.25
ER−	30 (29.7)	184 (27.2)	6 (15.4)	
ER+	69 (68.3)	475 (70.2)	31 (79.5)	
Unknown, n (%)	2 (2.0)	18 (2.6)	2 (5.1)	
Progesterone receptor (PR) status				0.87
PR−	34 (33.7)	237 (35.0)	12 (30.8)	
PR+	64 (63.3)	418 (61.7)	25 (64.1)	
Unknown, n (%)	3 (3.0)	22 (3.3)	2 (5.1)	
Vital status				0.55
Alive	62 (61.4)	396 (58.5)	20 (51.3)	
Deceased	39 (38.6)	281 (41.5)	19 (48.7)	
Breast cancer-specific deaths	12 (11.9)	113 (16.7)	7 (17.9)	0.45

Table 1 (continued)

WEB, Western New York Exposures and Breast Cancer; BMI, Body Mass Index

*Chi-square tests for categorical variables and ANOVA tests for continuous variables

[a]Mean ± standard deviation (SD) estimates were calculated for all continuous variables, except lifetime alcohol consumption. For this variable, median and interquartile range (IQR) are shown

[b]Frequency and percentages were calculated for all categorical variables

[c]1 alcoholic drink is equivalent to 14 grams of ethanol

[d]Family history of breast cancer is of first-degree relatives (mother, sister, and/or daughter)

[e]The assessment of hormone therapy use was limited to postmenopausal women only

Table 2 Sleep duration and mortality among patients with breast cancer ($n = 817$), WEB study, 1996–2001

All cases ($n = 817$)	All-cause mortality			Breast cancer mortality		
	Events (n)	Model A HR (95% CI)[a]	Model B HR (95% CI)[b]	Events (n)	Model A HR (95% CI)[a]	Model B HR (95% CI)[b]
Short sleep[c] ($n = 101$)	39	0.90 (0.64–1.27)	0.92 (0.65–1.30)	12	0.65 (0.36–1.19)	0.71 (0.39–1.30)
Normal sleep[d] ($n = 677$)	281	Reference	Reference	113	Reference	Reference
Long sleep[e] ($n = 39$)	19	1.10 (0.68–1.78)	1.01 (0.61–1.69)	7	0.98 (0.45–2.14)	1.16 (0.53–2.52)
BMI ≤ median (27.5 kg/m^2) ($n = 408$)						
Short Sleep[c] ($n = 43$)	14	0.93 (0.53–1.66)	1.01 (0.56–1.80)	7	1.12 (0.51–2.49)	1.38 (0.62–3.08)
Normal sleep[d] ($n = 348$)	123	Reference	Reference	50	Reference	Reference
Long sleep[e] ($n = 17$)	7	1.40 (0.65–3.01)	1.21 (0.53–2.76)	3	1.31 (0.41–4.24)	1.26 (0.39–4.09)
BMI > median (27.5 kg/m^2) ($n = 409$)						
Short sleep[c] ($n = 58$)	25	0.83 (0.54–1.28)	0.85 (0.54–1.32)	5	0.39 (0.15–0.98)	0.42 (0.16–1.07)
Normal sleep[d] ($n = 329$)	158	Reference	Reference	63	Reference	Reference
Long sleep[e] ($n = 22$)	12	0.86 (0.46–1.62)	0.93 (0.48–1.79)	4	0.72 (0.25–2.07)	1.10 (0.39–3.12)
p interaction			0.60			0.16

WEB, Western New York Exposures and Breast Cancer; BMI, Body Mass Index; HR, Hazards Ratio; CI, Confidence Interval

[a]Adjusted for age at interview, race/ethnicity, and years of education

[b]Adjusted for age at interview, race/ethnicity, years of education, menopausal status, pack-years of smoking, tumor stage, and estrogen-receptor status; additionally adjusted for BMI in the analyses of all cases

[c]Shorter sleep refers to ≤ 5 h of sleep per night

[d]Normal sleep refers to 6–8 h of sleep per night

[e]Longer sleep refers to ≥ 9 h of sleep per night

Discussion

In this population-based prospective study of women with invasive breast cancer, we found sleep duration to be significantly associated with several known breast cancer risk factors including race/ethnicity, BMI, lifetime alcohol consumption, pack-years of smoking, and history of previous benign breast disease. In comparison to those who reported a relatively normal sleep duration, those reporting shorter or longer sleep were less proportionately non-Hispanic White, had a slightly higher BMI, and higher median lifetime consumption of alcohol. However, we did not find any associations of self-reported pre-diagnostic sleep duration with all-cause or breast cancer-specific mortality. In our previous published work, we examined sleep disturbance and mortality. Using 4 questions from a sleep questionnaire, we devised an index score assessing the frequencies in: (1) trouble falling asleep, (2) waking up at night, (3) trouble staying asleep, and (4) waking up feeling tired and worn out, during the 12–24 month-period prior to the breast cancer diagnosis. We found no associations with all-cause mortality [6]. However, sleep disturbance was associated with a lower risk of breast cancer-specific mortality among postmenopausal women and higher risk among premenopausal women [6]. Similar to our previous findings of no associations between sleep disturbance and tumor stage, reported sleep duration was not associated with tumor stage.

Our findings for shorter pre-diagnostic sleep were in contrast to those of the Women's Health Initiative (WHI) Study

where poorer breast cancer-specific survival was observed [18]. However, other findings for pre-diagnostic sleep were in concordance with those from the WHI. Sleep duration was not associated with all-cause nor breast cancer-specific mortality following stratification by BMI [18]. However, in addition to shorter sleep duration, frequent snoring was associated with poor breast cancer-specific survival among patients in the WHI. In the Nurses' Health Study (NHS), a post-diagnostic report of longer sleep was associated with all-cause, breast cancer-specific and non-breast cancer-specific mortality outcomes among patients with breast cancer [19]. However, no associations were observed for shorter sleep. Evidence of higher all-cause mortality risk for increased sleep duration from pre- to post-diagnosis was also observed in the NHS. Similar results were reported for non-breast cancer-specific mortality outcomes [19]. While the trend of hazards ratio estimates in the NHS reflected a *U*-shaped direction, this was in contrast to the findings in our study and the WHI. In the WHI, the trend of hazards estimates for breast cancer-specific mortality was positive for shorter sleep and inversely linked to longer sleep. Although our study shared some similar findings with the WHI and NHS, there were differences in the selection of covariates and in the categorization of sleep duration, particularly shorter sleep. Furthermore, in contrast to the WEB Study, these cohorts largely consisted of older, postmenopausal women and the NHS primarily assessed post-diagnostic sleep duration.

Shorter sleep duration and sleep deprivation have been hypothesized to contribute to cancer mortality largely through disruption of circadian rhythm, altering the functions of the suprachiasmatic nucleus, circadian cell cycle and clock gene expression [21], and affecting nocturnal release of melatonin and other endocrinal/neural outputs. There is evidence that loss of circadian clock gene expression influences tumor progression [20], altering signaling pathways that normally control cell proliferation, apoptosis and tumor suppressor pathways [21]. Both pre-and post-diagnostic sleep potentially could impact prognosis as a result of promoting systemic inflammation and accelerating tumor growth and invasiveness [10, 18].

In assessing these results, it is important to consider the strengths and limitations of the study. One limitation was potential error in recall because cases were asked after their diagnosis about their sleep prior to diagnosis; however, in this case-only analysis, this error is likely non-differential. Self-reported assessments have been shown to be a valid indicator of pre-diagnostic sleep in another study cohort [18]. Also, since this was a prospective study, error in report was not likely related to the outcome. Another limitation was the limited sample size of patients with breast cancer other than non-Hispanic Whites in this study cohort, which limits the generalizability of study results. Furthermore, there was limited statistical power in this study, particularly among patients who reported longer sleep duration. The limited power may have affected our results, particularly in the stratified analyses. Additionally, given that there has been established evidence of more aggressive breast tumor subtypes and poorer mortality outcomes among Black women [29], it would have been of importance to investigate these associations more extensively among this group. Although our study did not have the power to examine analyses stratified by race/ethnicity, all models were adjusted for this confounding variable.

Our study had numerous strengths as well. One primary strength was the assessment of sleep duration prior to diagnosis. Assessing sleep at this time point reduces the likelihood of certain biases, particularly reverse causality. Another strength of this study was the prospective, population-based design with an extensive follow-up period. The longer follow-up allowed for increased statistical power. Our access to comprehensive data from both the questionnaire and medical records was also a strength, providing extensive information on socio-demographics, smoking history, and breast tumor characteristics. This information enabled us to identify and account for various confounding variables and perform multiple stratified analyses.

In conclusion, we did not find an association between sleep duration and mortality among women with invasive breast cancer, including within strata of BMI or tumor stage. Future detailed pre- and post-diagnostic assessments of sleep patterns among a larger, racially diverse cohort would add to the understanding of a role, if any that sleep may serve in breast cancer prognosis. Further investigation of these associations using other methods to measure sleep, such as biomarkers or actigraphy, are warranted to accurately assess this exposure and better understand associations, if any. The implementation of such approaches may offer further exploration of the underlying biological mechanisms of sleep and cancer mortality.

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Author contributions JLF contributed to the study conception, design, methodology, and funding acquisition. JN contributed to data curation and software. NMN completed the formal data analysis and data interpretation. The first draft of the manuscript was written by NMN and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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Data availability The datasets generated during an/or analyzed during the current study are not publically available but are available from the principal investigators upon reasonable request.

Declarations

Conflict of interest The authors declare no conflict of interest.

Consent to participate Informed consent was obtained from all individual participants included in the study.

Ethics approval This is an observational study. The University at Buffalo Research Ethics Committee has confirmed that no ethical approval is required.

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