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Multidimensional sleep health is not cross-sectionally or longitudinally associated with adiposity in the Study of Women's Health Across the Nation (SWAN)

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ABSTRACT

Objectives: The association between sleep and adiposity (indexed by body mass index or waist-to-hip ratio) has typically been evaluated using a single dimension of self-reported sleep. However, other dimensions and behavioral measures of sleep may also be associated with adiposity. This study evaluated whether multidimensional sleep health calculated from actigraphy and self-report was longitudinally associated with adiposity in a sample of midlife women who have a high prevalence of sleep disturbances and adiposity.

Design: Longitudinal study with 11–14 years of follow-up time between the sleep health assessment and body mass index / waist-to-hip ratio measurements.

Participants: Two hundred and twenty-one midlife women enrolled in the Study of Women's Health Across the Nation Sleep Study.

Measurements: Multidimensional sleep health was quantified using actigraphy ($M[SD] = 29.1[7.2]$ nights) measures of sleep efficiency, midpoint, duration, regularity, and self-report measures of alertness and satisfaction. Each component was dichotomized and summed; higher values indicated better sleep health. Height, body weight, and waist and hip circumference were measured at the sleep study and at follow-up. Linear regression models were used to assess associations between sleep health and adiposity, adjusting for demographic and menopausal covariates.

Results: There was no substantial within-person change in adiposity over time. Better sleep health was cross-sectionally and longitudinally associated with lower adiposity in unadjusted, but not in adjusted, models. Individual sleep health components were not associated with adiposity after adjustment.

Conclusion: We did not observe cross-sectional or longitudinal associations between multidimensional sleep health and adiposity. The sleep-adiposity link may be weaker in midlife adults than in other age groups.

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Introduction

Obesity affects 40% of the adult population in the United States,¹ and increases risk for type 2 diabetes, cancers, cardiovascular disease,² and premature mortality.³ Because obesity is associated with a wide array of adverse health outcomes, identifying its biobehavioral

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determinants is critical for prevention and intervention strategies. Disturbed sleep is one possible determinant of obesity, and offers opportunities for tailoring and optimizing existing weight loss interventions.⁴ Meta-analytic evidence suggests a significant cross-sectional association of short self-reported sleep duration (variously defined) with higher body mass index (BMI).⁵ Though most studies evaluate self-reported sleep duration, it may be that other dimensions of sleep—independently and jointly—are more strongly associated with adiposity.

Cross-sectional data suggest that indices of sleep beyond short or long sleep duration are associated with BMI. Lower sleep continuity,⁶ poorer sleep quality,⁷ poorer daytime alertness,⁸ advanced⁹ or delayed¹⁰ sleep timing, and variability in sleep duration^{6,11} have been cross-sectionally and individually linked to higher BMI. Sleep health, a positively valenced multidimensional construct of sleep, captures the 24-hour experience of sleep-wake patterns by considering sleep duration together with other sleep and circadian factors (continuity, quality, alertness, timing, and regularity).¹² Multidimensional sleep health may be a stronger predictor of health¹³ and mortality¹⁴ than individual sleep measures alone. Testing the cross-sectional and longitudinal associations between multidimensional sleep health and adiposity may expand our understanding of the sleep-adiposity relationship.

Evaluating the longitudinal association of sleep disturbances with adiposity is critical for clarifying issues of causality and intervention opportunities. Evidence regarding longitudinal relationships between self-reported sleep duration and weight gain is mixed. A systematic review showed consistent evidence for a longitudinal association between short sleep duration and increased weight or adiposity in children.¹⁵ However, the association is less consistent for adults, and may weaken with age.¹⁵ A meta-analysis of 14 longitudinal studies with 197,906 adult participants reported a significant overall association between self-reported short sleep duration and weight gain.¹⁶ However, moderately large heterogeneity across studies suggests that study characteristics may modify the effect sizes reported.¹⁶ This variability may be attributed to not assessing other salient sleep characteristics (i.e., other components of sleep health) and/or relying on self-reported measures.

Self-reported measures are susceptible to bias, and individuals who are obese are more likely to underestimate their sleep duration, relative to actigraphy, than individuals who are not obese.¹⁷ Wrist actigraphy provides a reliable and validated assessment of habitual behavioral rest-activity patterns.¹⁸ Only two longitudinal studies have assessed the association between actigraphy-assessed sleep duration and BMI in adults. Both studies found that actigraphy-assessed sleep duration was not significantly associated with BMI at 5-year follow-up after adjustment for baseline BMI.^{19,20} These data may mean that behaviorally assessed sleep duration is not associated with weight gain. The extent to which multidimensional sleep health influences the longitudinal link between sleep and BMI is unknown.

The current study evaluated cross-sectional and longitudinal associations at 11–14-year follow-up between multidimensional sleep health and BMI in a sample of midlife women, among whom sleep disturbances²¹ and adiposity²² are highly prevalent. Additionally, we evaluated the associations between sleep health and waist-to-hip ratio (WHR) given the health consequences of the redistribution of adipose tissue during the menopausal transition.²² Cross-sectional evidence suggests that self-reported short sleep duration is associated with higher WHR,²³ but similar findings have not been shown in actigraphy studies.²⁴ To our knowledge there have not been any longitudinal studies of the association between actigraphy-assessed sleep and WHR, nor have there been any longitudinal studies examining the association of actigraphy-assessed sleep dimensions (beyond sleep duration) and BMI. In the current study, we hypothesized that better sleep health (optimal sleep continuity, quantity,

quality, alertness, timing, and regularity) would be associated cross-sectionally and longitudinally with lower BMI and WHR.

Methods

The current study used data from the multi-site Study of Women's Health Across the Nation (SWAN; hereafter referred to as the core SWAN study), a longitudinal study designed to assess correlates of the menopausal transition in women living in the United States. Women were eligible at baseline if they were 42–52 years of age, reported a menstrual period within the past 3 months, had an intact uterus, and at least one ovary. Women were ineligible if they were pregnant, breastfeeding or lactating.

During one of the follow-up visits 5–8 of the core SWAN study, participants at four sites (Pittsburgh, PA; Chicago, IL; Detroit, MI; and Oakland, CA) enrolled in the ancillary SWAN Sleep Study. Exclusion criteria for the SWAN Sleep Study were noncompliance with core SWAN procedures, current oral corticosteroid use, current chemotherapy or radiation, regular shift work, diagnosis of sleep apnea, or consumption of more than four alcoholic drinks per day. During the sleep study, in-home polysomnography assessments (Temec Technologies, Vitaport 3) were conducted for three nights; on the first night, the participant's apnea-hypopnea index (AHI) was assessed. The montage for assessing AHI included a nasal pressure cannula, oral nasal thermistor, inductance plethysmography of abdominal and thoracic respiratory effort, and fingertip oximetry. Diary- and actigraphy-assessed sleep were assessed over a 35-day period, or the length of the participant's self-reported menstrual cycle, whichever was shorter. The Epworth Sleepiness Scale²⁵ was administered on the last day of the sleep study. BMI and WHR were assessed at each core SWAN visit (approximately yearly) from the SWAN Sleep Study (2001–2006) until 2015–2016. We will use the general term “adiposity” for BMI and WHR when we refer to both measures.

Participants

Participants for the current study were 221 women who participated in the SWAN Sleep Study and had full data for analyses assessing the cross-sectional and longitudinal associations among sleep health and adiposity (i.e., no missing data). We removed participants from analyses with fewer than four nights of actigraphy ($n = 42$), missing Epworth Sleepiness Scale ($n = 9$), missing adiposity data at T1 or T2 ($n = 86$), and missing AHI data (the only covariate with missing data; [$n = 12$]; see Fig. 1). Analyses (chi-squared test for categorical variables, one-way analysis of variance for continuous variables) indicated that there were differences between those participants from the SWAN Sleep Study who were included ($n = 221$) compared to those who were excluded ($n = 149$). Specifically, women who were excluded were more likely to have a higher BMI, be African American, and have a lower household income. There were no differences between groups on WHR or study covariates.

Measures

Sleep health

Sleep health was assessed during the SWAN Sleep Study using wrist actigraphy-assessed sleep duration, efficiency, timing, and regularity, and self-reported alertness and satisfaction. Actigraphy (Actiwatch-64, MiniMitter; Respironics, Bend, OR) was processed in 1-min epochs (Actiware version 5.04 software) and scored as sleep when the weighted sum of activity counts in that epoch (weight = 1), immediately adjacent epochs (weight = 0.20) and the next two adjacent epochs (weight = 0.04) was ≤ 40 (medium threshold). Event markers and daily diary data for bedtime and wake time were used to identify the sleep period from actigraphy data, and weighting was used for weekdays (weight = 5/7) and weekends (weight = 2/7).

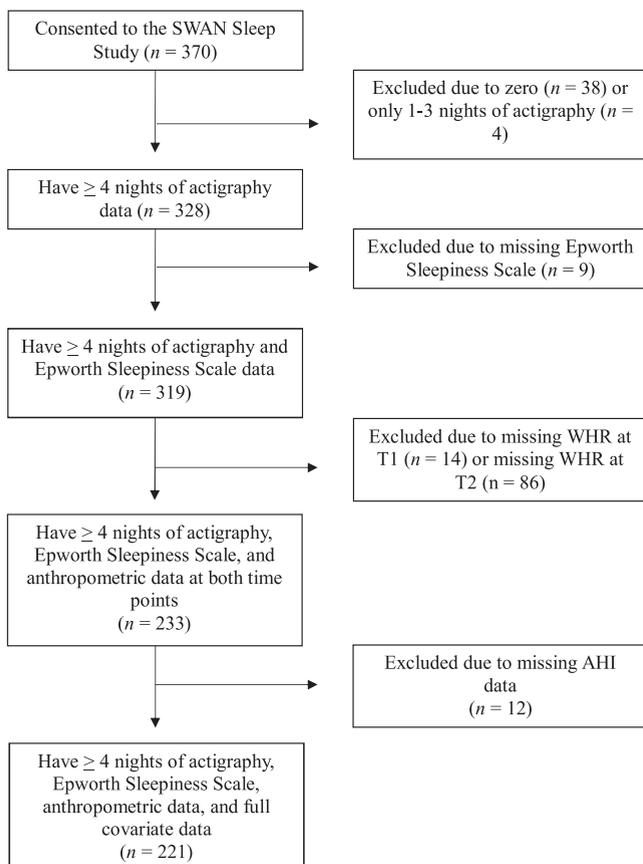


Fig. 1. Data available for analyses

Duration was defined as the average total minutes of sleep. *Efficiency* was defined as the average total minutes of sleep following sleep onset (first 1-minute epoch scored as sleep) divided by time in bed, multiplied by 100. *Timing* was defined as the average midpoint of sleep, calculated as the difference between sleep onset and wake time divided by two, and adding this value to the time of sleep onset (sleep onset + [(wake time – sleep onset)/2]). *Regularity* was defined as the standard deviation of the individual's sleep midpoint. Regularity values could plausibly relate to the number of nights assessed. However, we found that these values were unchanged when we restricted the sample to participants with at least seven or 14 nights of actigraphy data; to maximize power, we maintained our inclusion criteria of at least four nights of actigraphy data. *Satisfaction* was defined as the average diary-assessed "restedness," with scores ranging from 0 ("not at all") to 4 ("extremely") rested. *Alertness* was defined by the eight-item Epworth Sleepiness Scale, with scores ranging from 0 ("would never doze" for all questions) to 24 ("high chance of dozing" for all questions).²⁵ Each continuous sleep health variable was dichotomized using the cut-points detailed in Table 1,

with 0 indicating poor and 1 indicating good sleep health, and these variables were summed to create a composite score ranging from 0 (poor) to 6 (good). The cut-offs for each sleep health variable were created based on empirical literature.^{11,26-29} Our method of creating a sleep health score (i.e., dichotomization) allows it to be a clinically useful construct¹¹ and is comparable to previously published sleep health operationalizations.^{30,31}

Adiposity

SWAN study staff measured height (meters) and weight (kilograms), with participants in light clothing and no shoes. BMI was calculated as kilograms divided by meters squared (kg/m²). SWAN study staff measured waist and hip circumference to the nearest 0.1 cm. Using a measuring tape, waist circumference was assessed horizontally around the narrowest part of the participant's torso, and hip circumference was assessed at the level of the iliac crest. Measurements were taken over light clothing at the end of a normal exhalation. WHR was derived by dividing waist circumference by hip circumference.

Covariates

The following measures were included in the adjusted model: age, race/ethnicity, study site, follow-up time, menopausal status, vasomotor symptoms, use of medications that affect sleep, negative affect, and AHI. All covariates except menopausal status were assessed at the time of the SWAN Sleep Study. Age and race/ethnicity (Caucasian; African American; Chinese) data were based on self-report. Site indicated where participation took place (Pittsburgh, PA; Chicago, IL; Detroit, MI; and Oakland, CA). Follow-up time was calculated as the years from the SWAN Sleep Study to the follow-up assessment. Menopausal status was determined based on self-reported bleeding patterns at the core SWAN visit closest to the SWAN Sleep Study (average of 6 months between the two visits). Premenopause was defined as no change in menstrual period regularity, early perimenopause indicated that women had bleeding in the last 3 months and bleeding irregularity, late perimenopause indicated that women had bleeding in the last 12 months but not in the past 3 months, and natural postmenopause included women who had no bleeding in the 12 months prior to the visit. Women who were pre- or perimenopausal and using hormone therapy were characterized as "indeterminate" because the therapy may change women's bleeding patterns. The percentage of nights that women reported any vasomotor symptoms was assessed using daily diary, with reports of the frequency ("How many times did you experience these symptoms last night?," with categories of 0, 1, 2, 3, 4, "5 or more," and "all night") of their hot flashes, cold sweats, and night sweats. The percentage of nights that women reported taking a medication that affects sleep was based on daily diary report of any of the following medication classes identified by the World Health Organization Anatomical Therapeutic Chemical classifications: opioids (N02A), antiepileptics (N03A), anxiolytics (N05B), hypnotics and sedatives (N05C), antidepressants (N06A), and antihistamines for systemic use (R06A).³² The negative affect composite score³³ was a continuous measure based on depressive symptoms (Quick Inventory of Depressive Symptomatology with the sleep item removed),³⁴ anxious symptoms

Table 1
Operational definitions for dimensions of sleep health

| Sleep health dimensions | Operationalization | Cut-off for good sleep health |
|-------------------------|-------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------|
| Regularity | Standard deviation of calculated sleep midpoint from actigraphy | Less than 60 min |
| Satisfaction | Average self-reported sleep quality from daily sleep diary, "restedness upon awakening" (0 = not at all; 4 = extremely) | "Moderately", "quite a bit", or "extremely" rested upon awakening |
| Alertness | Total score on Epworth Sleepiness Scale (0–24) | 10 or less |
| Timing | Average calculated sleep midpoint from actigraphy | 2 am–4 am |
| Efficiency | Average sleep efficiency from actigraphy | Greater than 85% |
| Duration | Average total sleep time from actigraphy | 6–8 h |

(State-Trait Anxiety Inventory),³⁵ and perceived stress (four-item Perceived Stress Scale).³⁶ AHI was determined from one night of in-home polysomnography.

Statistical analysis plan

Change in adiposity from the SWAN Sleep Study to the follow-up visit was evaluated to determine the best strategy for modeling longitudinal associations among sleep health and adiposity. Incident adiposity was low in this sample, with fifteen (11%) participants meeting criteria for incident obesity (BMI), and 39 (28%) participants meeting criteria for incident abdominal obesity (WHR). This low incidence is not surprising, given the high prevalence of adiposity at the time of the SWAN Sleep study (38% with BMI \geq 30, 36% with WHR \geq 0.85). Thus, few women "developed" adiposity during the follow-up period, and we did not have sufficient power to test the association between sleep health and incident adiposity. We also explored the possibility of examining the association between sleep health and longitudinal patterns of adiposity change using mixed effects models. We evaluated adiposity at each annual visit subsequent to the SWAN Sleep Study and found that the intraclass correlations were high (0.92 for BMI, 0.97 for WHR). Considering the limited within-person change over time in BMI (8%) and WHR (3%), mixed effects modeling would not be informative. Therefore, we used multiple linear regression models to test our hypotheses; women who were obese or abdominally obese at the sleep study were included in the analyses.

Descriptive statistics were used to characterize the sample. Linear regression assumptions were examined, and AHI was log-transformed to reduce skewness. Cross-sectional univariate and multivariable (adjusting for covariates) analyses were used to examine sleep health in relation to adiposity at the time of the SWAN Sleep Study. In longitudinal analyses, univariate analyses of sleep health in relation to adiposity at 11–14-year follow-up were followed by multivariable models included each outcome's homolog measured at the time of the sleep study (sleep study BMI for BMI as outcome; sleep study WHR for WHR outcome), as well as the covariates used in cross-sectional analyses. In exploratory analyses, we evaluated the extent to which BMI or WHR at the time of the sleep study attenuated longitudinal associations among sleep health and BMI or WHR, respectively. We also evaluated attenuation of longitudinal sleep health-adiposity associations with other model covariates, after adjusting for each outcome's homolog at the time of the sleep study. A power analysis³⁷ indicated that we had sufficient power (82%) to test our hypotheses given our sample size ($n = 221$) and number of predictors ($k = 15$) if the expected effect size is at least a small effect ($f^2 = 0.03$). Published studies using linear regression models to examine the association of dimensions of sleep with BMI demonstrate this effect size or larger.^{9,11}

In a series of secondary analyses, the six continuous dimensions of sleep health were separately evaluated for cross-sectional and longitudinal associations with adiposity. Quadratic terms were included in the model for sleep duration and timing, as "good" sleep health for these measures is neither low nor high. These analyses help to evaluate which component(s) may be driving observed associations with the multidimensional sleep health measure. In exploratory analyses, we evaluated which covariates were responsible for the attenuation of the longitudinal association between sleep health and adiposity. In a sensitivity analysis, we excluded women whose menopausal status was indeterminate ($n = 16$) to evaluate the extent to which hormone therapy in women who were not yet menopausal may have influenced study results.

Results

Participant characteristics

The sample included 67 African American, 44 Chinese, and 110 White women with an average age of 52.1 ± 2.1 years. At the time of

Table 2
Sample characteristics ($n = 221$)

| | M (SD) | N (%) |
|--------------------------------------------|---------------|---------------|
| Body mass index, at time of sleep study | 29.2 (7.5) | |
| Body mass index, at follow-up | 29.4 (7.2) | |
| Average change in body mass index | 0.25(4.17) | |
| Waist-to-hip ratio, at time of sleep study | 0.83 (0.07) | |
| Waist-to-hip ratio, at follow-up | 0.84 (0.07) | |
| Average change in waist-to-hip ratio | 0.02(0.06) | |
| Age | 52.2 (2.1) | |
| Race/ethnicity | | |
| White | | 110 (49.8) |
| African American | | 67 (30.3) |
| Chinese | | 44 (19.9) |
| Study site | | |
| Pittsburgh, PA | | 58 (26.2) |
| Detroit area, MI | | 43 (19.5) |
| Chicago, IL | | 37 (16.7) |
| Oakland, CA | | 83 (37.6) |
| Menopausal status | | |
| Pre-/early perimenopausal | | 134 (60.6) |
| Late perimenopausal | | 44 (19.9) |
| Postmenopausal | | 27 (12.2) |
| Indeterminate | | 16 (7.2) |
| Vasomotor symptoms, % of nights | 31.2 (33.9) | |
| Sleep medications, % of nights | 23.1 (40.9) | |
| Apnea-hypopnea index | 9.7 (13.7) | |
| Sleep health dimensions | | "Good", N (%) |
| Regularity | 42 min (18) | 184 (83.3) |
| Satisfaction | 2.0 (0.6) | 117 (52.9) |
| Alertness | 8.0 (4.4) | 161 (72.9) |
| Timing | 3:20am (0:31) | 201 (91.0) |
| Efficiency | 78.9% (9.8) | 62 (28.1) |
| Duration | 6.0 h (0.8) | 111 (50.2) |

Note. "Good" indicates the number of participants with "good" sleep health based on the cut-points for each dimension of sleep health.

the sleep study, 60.6% were premenopausal or early perimenopausal, 19.9% were late perimenopausal, and 12.2% were postmenopausal (Table 2). The average length of follow-up from the SWAN Sleep Study to the assessment of adiposity was 12.1 years (SD = 0.6, range = 10.8–13.9 years). This range in follow-up resulted from the SWAN Sleep Study being conducted at one time point after core SWAN follow-up visits 5–8. Two participants participated in the Sleep Study after follow-up visit 5, 61 participated after visit 6, 113 participated after visit 7, and 45 participated after visit 8.

Table 2 shows sleep health characteristics, both as continuous variables and based on empirically derived dichotomous cut-offs (defined in Table 1). Fig. 2 shows the distribution of composite sleep health scores (range 0–6). The sleep health score distribution was not significantly skewed, as assessed by tests of skewness and kurtosis (z -scores less than 1.96), and visual inspection of the Q-Q plot.

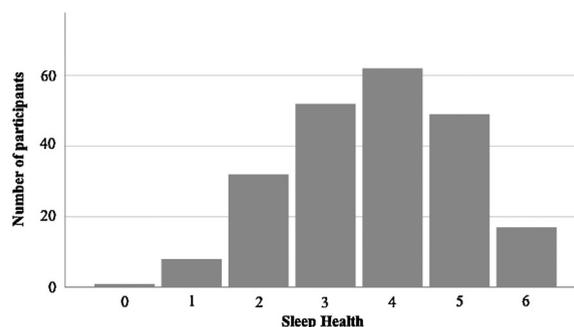


Fig. 2. Distribution of sleep health scores

Table 3
Cross-sectional and longitudinal associations among sleep health and adiposity

| | Unadjusted | | Adjusted ^{a,b} | |
|------------------------|------------|------|-------------------------|------|
| | β | P | β | P |
| <i>Cross-sectional</i> | | | | |
| Body mass index | -0.17 | 0.01 | -0.02 | 0.78 |
| Waist-to-hip ratio | -0.12 | 0.08 | 0.05 | 0.48 |
| <i>Longitudinal</i> | | | | |
| Body mass index | -0.14 | 0.04 | 0.02 | 0.60 |
| Waist-to-hip ratio | -0.14 | 0.04 | -0.05 | 0.50 |

^a Adjusted for age, race, study site, menopausal status, vasomotor symptoms, apnea-hypopnea index, negative affect.

^b Longitudinal models include homologs of each outcome measured at the time of the sleep study (BMI for BMI, WHR for WHR), follow-up time, and covariates included in cross-sectional models.

Cross-sectional associations of sleep health with adiposity

Better sleep health was associated with lower BMI in unadjusted ($\beta = -0.17$, $P = 0.01$) but not adjusted regression models (Table 3). Sleep health was not significantly associated with WHR in unadjusted or adjusted models (P values ≥ 0.08).

Evaluating each continuous sleep health component separately, better regularity ($\beta = 0.20$, $P = 0.003$) and higher sleep efficiency ($\beta = -0.15$, $P = 0.02$) were significantly associated with lower BMI in the unadjusted, but not in the adjusted, model (Table 4). Higher sleep efficiency was associated with lower WHR in the unadjusted ($\beta = -0.15$, $P = 0.02$), but not the adjusted, model. Better self-reported alertness was significantly associated with higher WHR in the

adjusted ($\beta = -0.15$, $P = 0.03$), but not the unadjusted, model. None of the other dimensions of sleep health were cross-sectionally associated with BMI or WHR in unadjusted or adjusted models.

Longitudinal associations of sleep health with adiposity

Better sleep health was associated with lower BMI ($\beta = -0.14$, $P = 0.05$) and lower WHR ($\beta = -0.14$, $P = 0.04$) measured 11–14 years later in the unadjusted model. These associations were not significant after adjustment for covariates and the homologous adiposity measure at the time of the sleep study (Table 3).

Better regularity and higher sleep efficiency were longitudinally associated with lower BMI ($\beta = 0.15$, $P = 0.03$; $\beta = -0.16$, $P = 0.02$, respectively) and lower WHR ($\beta = 0.13$, $P = 0.05$; $\beta = -0.20$, $P = 0.003$, respectively) in unadjusted models (Table 5). These associations were no longer significant in the adjusted model. None of the other dimensions of sleep health were associated with BMI or WHR in unadjusted or adjusted models.

In exploratory analyses, we evaluated the extent to which covariates were responsible for the attenuation of longitudinal associations among sleep health and adiposity. BMI and WHR measured at the time of the sleep study were significantly associated with follow-up adiposity (P values < 0.001), and sleep health was no longer significant in either model (P values ≥ 0.21). None of the other model covariates were significantly associated with follow-up adiposity after adjusting for adiposity at the time of the sleep study.

Sensitivity analyses indicated that excluding participants with unknown menopausal status due to hormone therapy use ($n = 16$) did not substantially modify coefficients or statistical significance for the associations of sleep health and adiposity.

Table 4
Cross-sectional associations between continuous dimensions of sleep health and indices of adiposity

| | BMI, unadjusted | | BMI, adjusted ^a | | WHR, unadjusted | | WHR, adjusted ^a | |
|-----------------------|-----------------|-------|----------------------------|------|-----------------|-------|----------------------------|------|
| | β | P | β | P | β | P | β | P |
| Regularity | 0.20 | 0.003 | 0.08 | 0.19 | 0.11 | 0.09 | 0.04 | 0.53 |
| Satisfaction | -0.06 | 0.39 | 0.01 | 0.88 | -0.11 | 0.11 | -0.07 | 0.35 |
| Alertness | 0.05 | 0.47 | 0.05 | 0.44 | -0.06 | 0.42 | -0.15 | 0.03 |
| Timing ^b | 0.33 | 0.45 | 0.01 | 0.99 | 0.06 | 0.89 | -0.22 | 0.59 |
| Efficiency | -0.15 | 0.02 | 0.08 | 0.20 | -0.22 | 0.001 | -0.07 | 0.38 |
| Duration ^b | -0.22 | 0.75 | -0.53 | 0.28 | 0.77 | 0.27 | 0.57 | 0.38 |

^a Adjusted for age, race, study site, menopausal status, vasomotor symptoms, apnea-hypopnea index, z-score negative affect.

^b Indicates that a quadratic term was included, and the quadratic term coefficient is reported. Note. BMI, body mass index; WHR, waist-to-hip ratio.

Table 5
Longitudinal associations among continuous dimensions of sleep health and indices of adiposity

| | BMI, unadjusted | | BMI, adjusted ^a | | WHR, unadjusted | | WHR, adjusted ^a | |
|-----------------------|-----------------|------|----------------------------|------|-----------------|-------|----------------------------|------|
| | β | P | β | P | β | P | β | P |
| Regularity | 0.15 | 0.03 | -0.03 | 0.47 | 0.13 | 0.05 | 0.07 | 0.24 |
| Satisfaction | -0.05 | 0.45 | -0.02 | 0.69 | -0.14 | 0.04 | -0.04 | 0.52 |
| Alertness | 0.04 | 0.60 | 0.01 | 0.91 | -0.01 | 0.93 | -0.02 | 0.79 |
| Timing ^b | 0.13 | 0.77 | -0.36 | 0.14 | 0.45 | 0.30 | 0.46 | 0.19 |
| Efficiency | -0.18 | 0.01 | -0.03 | 0.53 | -0.20 | 0.003 | -0.08 | 0.21 |
| Duration ^b | -0.38 | 0.61 | -0.91 | 0.15 | 0.99 | 0.13 | 0.60 | 0.30 |

^a Adjusted for age, race, study site, menopausal status, vasomotor symptoms, apnea-hypopnea index, negative affect, follow-up time, and the adiposity value at the time of the sleep study.

^b Indicates that a quadratic term was included, and the quadratic term coefficient is reported. Note. BMI, body mass index; WHR, waist-to-hip ratio.

Discussion

The current study evaluated cross-sectional and longitudinal associations between multidimensional sleep health and adiposity. In unadjusted analyses, better sleep health was cross-sectionally and longitudinally associated with lower BMI and longitudinally associated with lower WHR (small effect sizes). These associations were not significant after adjustment for covariates. Secondary analysis of individual dimensions of sleep health showed that greater regularity and higher sleep efficiency were longitudinally associated with lower BMI and WHR in unadjusted models, although these associations were no longer significant following covariate adjustment. In particular, including baseline BMI or WHR in the model accounted for the lack of significance in adjusted models, because of the stability of adiposity in our sample.

In contrast to previous studies which have focused on individual indices of sleep in relation to adiposity, the current study evaluated multiple dimensions of sleep concurrently. Measurement of sleep health as a multidimensional construct acknowledges that individual dimensions of sleep are concurrent and inter-related. Moreover, emerging evidence suggests that self-reported multidimensional sleep may be a stronger predictor of poor self-rated health¹³ and all-cause mortality¹⁴ relative to health behaviors such as smoking, alcohol consumption, and physical activity. In direct comparisons, multidimensional sleep health is a stronger predictor of mortality than are individual dimensions of sleep.¹⁴ Consistent with the present study, DeSantis and colleagues reported that actigraphy-assessed multidimensional sleep health and its individual components were not cross-sectionally associated with BMI.³¹ The current study replicated and extended the findings of DeSantis and colleagues³¹ by demonstrating that multidimensional sleep health is neither cross-sectionally nor longitudinally associated with BMI or WHR.

Associations of sleep with adiposity may be weaker in midlife than during other phases of life. The current study's results are consistent with two previous studies that reported no significant longitudinal association between actigraphy-assessed sleep and adiposity in midlife adults²⁰ and in midlife women in the SWAN study.¹⁹ We extended the work by Appelhans and colleagues¹⁹ in the current study by assessing multiple dimensions of sleep health rather than the independent effect of sleep duration, and evaluated adiposity for a longer follow-up period. Our study, in conjunction with previous work, may suggest that the association between sleep and adiposity is weak in midlife adults. Consistent with this theory, a population-based study reported that the relationship between self-reported short sleep duration and high BMI was significant for participants aged 18–49, but not significant for participants over the age of 50.³⁸ The possibility of age differences in the sleep-adiposity link is further supported by an experimental study evaluating the metabolic consequences of concurrent sleep restriction and circadian misalignment in 20-year-olds compared to 60-year-olds.³⁹ Resting metabolic rate decreased significantly in young adults in a pattern consistent with a 12-pound per year increase in weight, but there was no significant change in older adults.³⁹ Together, this evidence suggests that the impact of sleep on adiposity may be attenuated with age and, as a result, explain the findings from the present study.

Several limitations of the current study should be noted. First, BMI and WHR showed only small changes over the follow-up period. This precluded an analysis of change in BMI or WHR over time, as well as examination of incident obesity. Previous longitudinal evidence in SWAN suggests that both chronological and reproductive aging contribute to increases in fat mass,⁴⁰ and therefore it is worth investigating whether our associations would be replicated in samples that exhibited weight gain over time. In our sample, only 8% of BMI and 3% of WHR variance was due to within-person changes. Second, the long follow-up interval may limit our ability to detect an association

given intervening changes in health status with age. To address this possibility, follow-up time was included as a covariate in longitudinal models, and this did not modify any associations. Third, although longitudinal, our study cannot be used to infer causality. Our study was observational, based on secondary data analyses, and sleep health was assessed at only one time point. Fourth, it may be that the association between sleep health and adiposity is stronger among certain subgroups of individuals. Previous research has suggested that sex¹¹ and age^{11,38} may modify the sleep-adiposity link.

Because multidimensional sleep health is an emerging construct, future studies should focus on a number of critical questions. For instance, additional studies could address optimal cut-points for dichotomizing sleep health scores across a variety of populations (e.g., varying cut-offs as women traverse the menopausal transition); whether alternative statistical methods (e.g., principal component analysis, structural equation modeling) may better capture the construct; whether subjective versus behavioral assessment of sleep health components change observed associations; whether each sleep health component should be equally or variably weighted; and whether that weighting varies by health outcome.⁴¹ Our method of operationalizing sleep health is consistent with previous studies^{30,31} and may be more easily translated to clinical settings with the creation of a questionnaire with cut-points.¹² Additionally, we evaluated the effects of individual components of sleep health for comparison with studies assessing sleep as a unidimensional construct.

On the other hand, this study has strengths in its measurement of variables. First, the study evaluates sleep using actigraphy, measuring habitual rest-activity patterns in participants' natural environment for nearly 1 month ($M = 29$ nights). Second, our study includes objective measurement of BMI and WHR by study staff at both time points. Third, we adjusted for key factors known to be associated with sleep and/or adiposity. Our inclusion of AHI as a measure of obstructive sleep apnea is critical, given the large body of evidence suggesting that adiposity is associated with obstructive sleep apnea.⁴²

Conclusions

Our study is the first to examine cross-sectional and longitudinal associations of multidimensional sleep health with BMI and WHR. This study was conducted in a large, well-characterized sample of midlife women with nearly a month of actigraphy and diary data, and a follow-up period of 11–14 years. There was no substantial within-person change in BMI or WHR over the follow-up period. We found no significant cross-sectional or longitudinal associations among sleep health and adiposity after adjustment for key covariates. Our null results should be contextualized within our sample of midlife women, as there is previous evidence that the sleep-adiposity link may be modified by age.

Disclosure

Dr. Buysse reports personal fees from BeHealth, Emmi Solutions, American Academy of Physician Assistants, Bayer, CME Institute, and Ebb Therapeutics. In addition, Dr. Buysse received licensing fees for several research instruments whose copyright is held by the University of Pittsburgh and Ryerson University. Dr. Neal-Perry reports personal fees from Ferring Pharmaceutical, Endocrine Society, and the American Board of Obstetrics and Gynecology. Dr. Joffe reports serving on the consultant/advisory board for NeRRre, Sojournix, and Eisai, and providing grant/research support to NIH, Merck, NeRRre/KaNdy, Pfizer, and QUE Oncology. Dr. Joffe's spouse is an employee for Merck, does consulting for and has equity in Arsenal Biosciences, and has equity in Tango. The other authors declare no conflicts of interest relevant to this manuscript.

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