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Objectively measured daytime sleepiness predicts weight change among adults: Findings from the Wisconsin Sleep Cohort Study

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ABSTRACT

Objective: Body mass index (BMI) trajectories are associated with night-time sleep, but it is not clear how they relate to daytime sleepiness in population data. This study aimed to examine longitudinal associations between levels and changes in daytime sleepiness and BMI trajectories among men and women.
Methods: We estimated growth curve models among 827 participants in the Wisconsin Sleep Cohort Study (mean [*sd*] age = 55.2 [8.0] years at baseline). The outcome variable was BMI (kg/m^2) and the key predictor was daytime sleepiness measured by Multiple Sleep Latency Test (MSLT) scores. Covariates included demographics, health behaviors, retirement status, stimulant use, and depressive symptoms. In sensitivity analyses, we evaluated the potential effects of cardiovascular disease, shift work status, and sleep apnea on the robustness of sleepiness and BMI associations.
Results: At the between-person level, men who were sleepier had higher BMI levels. At the within-person level, age moderated the positive association between sleepiness and BMI among women. Specifically, young women who became sleepier over time gained more BMI than older women with comparable increases in sleepiness. Furthermore, while BMI tended to increase with age among women, BMI trajectories were steeper among sleepy women than among well-rested women, who experienced less increase in BMI over time.
Conclusion: The study suggested that levels and changes in daytime sleepiness as objectively measured by MSLT scores are associated with body mass among adults.

Introduction

Obesity is a major public health issue; higher levels of body mass index (BMI) are associated with heightened risks of mortality from several chronic health conditions as well as adverse outcomes from COVID-19.[1–3] In addition, prior studies have largely supported the association between obesity and sleep disturbance including daytime sleepiness,[4] which is defined as the propensity to fall asleep despite the intention to stay awake.[5] Specifically, obesity is a risk factor for incident and chronic daytime sleepiness, accounting for about 16% of the variability in sleepiness.[6,7] The impact of obesity on sleepiness is explained in part by sleep apnea[8]; many obese individuals experiencing excessive daytime sleepiness improve with effective sleep apnea treatment.[9] On the other hand, excessive daytime sleepiness is not a benign sequela of obesity. It is associated with heightened risks for motor vehicle accidents,[10] poor cardiovascular health,[11] all-cause mortality,[12] and diminished quality of life.[13]

Whereas the effect of obesity on sleepiness is well established, relatively few studies have considered a potential feedback loop where higher levels of daytime sleepiness contribute to further gains in BMI.[4] Furthermore, while apnea is a key mediator of the association between obesity and sleepiness, it does not seem to mediate or confound the association between sleepiness and obesity (i.e., obesity→apnea→sleepiness→obesity). Although less explored, some studies have nevertheless found evidence that sleepiness contributes to weight gain and obesity.[14] Interestingly, night shift work is known to increase sleepiness,[15,16] which in turn is related to BMI,

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independent of age.[17] Moreover, individuals with longer job histories of shift work tend to have higher levels of BMI.[18]

Excessive daytime sleepiness may contribute to weight gain and obesity primarily through metabolic and behavioral mechanisms and not, as noted, through sleep disruption and sleep apnea.[19,20] Possible pathways include altered dietary preferences (e.g., increased craving for high-fat foods), insufficient physical activity and poor physical health, reduced metabolism (e.g., insulin resistance), and depression.[8,21–23] Additionally, sleepiness and fatigue may interact with stress regulation and cytokines so that disturbances associated with activation of the hypothalamic-pituitary-adrenal axis and elevation of proinflammatory cytokines may also contribute to weight gain and obesity.[19]

Although some prior studies have investigated the feedback loop between sleepiness and obesity, there are limitations in the literature. First, obesity prevalence, BMI trajectories, and sleep characteristics (including daytime sleepiness) differ between men and women.[24–29] Few studies, however, have examined potential gender differences in daytime sleepiness-BMI associations, which could have important health implications as we age.[30,31] Second, most prior studies have utilized cross-sectional designs to explore sleepiness-obesity associations. Cross-sectional data can capture age differences but cannot estimate varying trajectories of change in sleep health or BMI over time in the course of aging.[32–35] Furthermore, cross-sectional studies are not able to demonstrate clearly that excessive daytime sleepiness leads to weight gain and higher BMI. Population-based longitudinal studies are necessary to examine how between-person differences and within-person changes in daytime sleepiness are associated with body mass. Third, the measurement of excessive daytime sleepiness has mostly relied on subjective reports. For example, although the Epworth Sleepiness Scale is a valid measure for self-reporting risks of dozing off, it does not directly measure instances of falling asleep during daytime hours.[13] Thus, physiological sleepiness measures with the Multiple Sleep Latency Test (MSLT) can offer a more objective evaluation of levels of sleepiness during the day.

The present study

Our study examined longitudinal associations between levels and changes in daytime sleepiness and BMI trajectories. This longitudinal design has the potential to better inform causality while utilizing objective measures of daytime sleepiness and weight changes based on the MSLT scores and BMI, respectively. At the between-person level, we hypothesized that both men and women who are sleepier have higher BMI levels (*H1*). At the within-person level, we hypothesized that increasing sleepiness is associated with higher BMI levels (*H2*) and steeper increases in BMI over time (*H3*) for both men and women. We also considered participant characteristics that are commonly associated with sleep health and weight as covariates in the full models. These covariates included age, education, retirement status, exercise, stimulant use, and depressive symptoms.

Methods

Study population and sample

The sample was obtained from the population-based longitudinal Wisconsin Sleep Cohort Study (WSCS). Beginning in 1988, the WSCS randomly sampled adults aged 30-60 from the payroll files of Wisconsin state employees. Over the last 3 decades, the WSCS has collected data on body habitus, sleep health, and other measures of behavior and health at approximately 4-year intervals. The analytic sample for the current study consisted of 827 participants who completed the MSLT from September 1989 through September 2011. The sampling design and complete details for WSCS protocols have been previously described in ref.[36] All study participants provided written informed consent. All WSCS procedures and informed consent documents were reviewed and approved by the Health Sciences Institutional Review Board at the University of Wisconsin-Madison.

Outcome and key predictor variables

BMI

At each sleep study visit at the University of Wisconsin-Madison Hospital and Clinics, trained technicians measured the height (to the nearest centimeter without shoes) and weight (to the nearest 0.5 kg) of all participants on a mechanical beam scale (or on the digital platform scale if body weight was over 300 lbs) using standardized protocols.[37] We used height and weight data to calculate body mass index (BMI in kg/m^2) for each participant at each point of data collection, which is the outcome variable in our study. Given the nature of data collection, there are no missing BMI assessments.

Daytime sleepiness

We calculated the daytime sleepiness score based on the actual minutes participants took to fall into asleep during the MSLT. Daytime sleepiness was evaluated as the mean sleep onset latency time across the nap trials of the MSLT. As research tools evolved over time, 2 different MSLT protocols were used over the duration of the study: a research protocol (from September 1989 to September 2003; 1616 studies in total) and a clinical protocol (from August 2001 to August 2011; 998 studies in total). In the years 2001-2003, when both types of protocols—research and clinical—were being performed, study subjects only participated in one or the other protocol, not both. Most, 62%, of the studies in this project were research MSLTs. Protocols were conducted according to published standards[38,39] with minor modifications to fit the WSCS protocols, including conducting the MSLT even if sleep duration the prior night was less than 6 hours and continuing medications prior to and during the test. The protocol included 4 or 5 nap opportunities at 2-hour intervals. In the research protocol, naps started at 10:00 AM and participants were awoken immediately after sleep onset and the nap trial ended. In the clinical protocol, the first nap study started 1.5 hours after waking from the overnight in-laboratory sleep study, and participants were allowed to sleep for 15 minutes after sleep onset in each nap study. Sleep onset latency for each nap was defined as the time from the start of the nap trial to the first 30-second epoch of scored sleep. Mean sleep onset latency (in minutes) was calculated across the 4 or 5 nap trials. We used both the baseline MSLT scores (time-invariant) and all MSLT scores from follow-up data collections (time-varying) as our main predictor variables.

Covariates

To account for any potentially different effects of MSLT assessment modes on the outcomes, we included an indicator of the type of MSLT exam (clinical or research) as a time-invariant and time-varying covariate in the hypothesized models. Additionally, the following covariates on participant demographics and characteristics measured at the baseline WSCS survey (except for age) were also included in the hypothesized full models. Age was coded as both within- and between-person variables, which were centered around the person-specific versus sample means, respectively. We measured education as an ordinal variable ranging from 0-3 (0 = *≤ high school*; 1 = *some college*; 2 = *college graduate*; 3 = *post-graduate*). We measured exercise by self-reported hours of leisure-time physical

activity per week, using the Paffenbarger Physical Activity Questionnaire.[40] We measured alcohol consumption as a self-reported number of drinks per week. We measured retirement status, current smoking, and sedative and stimulant medication use as binary variables, where 1 = *yes* and 0 = *no use*. Sleep apnea severity, characterized by the apnea-hypopnea index (AHI, breathing events per hour of sleep), was assessed by overnight in-laboratory polysomnography; detailed methods described previously.[41]

We measured depressive symptoms (1 = *yes*; 0 = *no*) based on the Zung score[42] and prescription medications as determined by a review of all self-reported prescription medications by the medical director on the WSCS team. Participants received a score of "1" if (1) the Zung score was greater than 50, or (2) participants were currently taking anti-depression medication. The Zung depression scale[42] is a 20-item survey used to assess self-reported depressive symptoms. Each item is scored 1 through 4 with higher scores indicating more severe symptoms. The twenty items are summed to get a final Zung scale score, with scores lower than 50 indicating "*normal*" and scores of 50 or greater indicating mild-to-severe depression symptoms. However, there are 2 questions on the Zung scale that could create a compulsory link between depression symptoms and sleepiness, namely, "*I have trouble sleeping through the night*" and "*I get tired for no reason*." For the current study, these questions were excluded from the score and the final scores were rescaled (by a factor of 20/18 = 1.1) to retain the original range.

Analytical strategy

We conducted preliminary analyses to examine descriptive statistics for key variables and participant characteristics for the overall sample and for men and women separately, given known disparities in BMI by age and gender.[43,44] To examine *H1* on the between-person association between levels of daytime sleepiness and BMI, we fit multilevel growth curve models with longitudinal BMI as the outcome variables, and baseline MSLT daytime sleepiness score as the key predictor. We took a hierarchical modeling approach in this set of analyses, where we included time variables only in *Model 1* (i.e., linear and quadratic within-person age, while controlling for average age). Then, we added in baseline MSLT scores as an additional predictor in *Model 2*, and adjusted for all covariates in the full *Model 3*.

To examine *H2* and *H3* on the within-person associations, we fit multilevel growth curve models with longitudinal MSLT daytime sleepiness scores as the key predictors. We took a similar hierarchical modeling approach in this set of analyses by having a time-variable-only model (*Model 1*), then adding in the MSLT scores as predictors (in *Model 2*), and expanding Model 2 to build the full model with covariates (*Model 3*). Specifically, we built *Models 2* and *3* sequentially by adding within-person MSLT scores and between-person MSLT scores as main effects, then adding interactions between the within-person and between-person age variables. Between-person continuous variables were centered on the sample mean, and within-person continuous variables were centered on the person mean. Between-person categorical covariates for the time-varying models were coded as 0 = *Always 0 (no)*, 1 = *Transitioned between 0/1*, and 2 = *Always 1 (yes)*.

For all hypothesized models, we specified random intercepts and random slopes and used the same set of covariates. We used Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) to evaluate model fit during the model-building process. Specifically, if a simpler model had lower AIC and BIC values, by omitting a non-significant main or interaction fixed effect, or by omitting non-significant random age linear slope or age quadratic slope random effect, then we would trim the non-significant effects to aid in model parsimony. We conducted all analyses in SAS software version 9.4 using Proc Mixed with maximum likelihood estimation methods to handle potential missing data (Copyright 2023 SAS Institute Inc).

Sensitivity analyses

We conducted sensitivity analyses by considering 3 additional participant characteristics that are likely to relate to both weight and sleep health, namely self-reported incident cardiovascular events, shift work status, and severity of sleep apnea. First, excessive daytime sleepiness is associated with cardiovascular disease[45] (CVD); obesity also contributes directly to incident cardiovascular events via multiple pathways, including sleep disorders.[46] As obesity is primarily a risk factor for (rather than an outcome of) CVD, sensitivity analyses conservatively examined models that adjusted for incident CVD events.

Further, shift work may induce sleep deprivation and circadian disturbances, which are known to be related to excessive sleepiness during the day[47] and increased risk for obesity.[48] Since our hypothesized models included shift workers, we performed additional sensitively analyses that (1) estimated the effect of ever being a shift worker as a covariate (1 = *yes*; 0 = *no*), and (2) estimated the same models while excluding 171 participants and 549 observations from those who ever reported doing shift work in these analyses.

Finally, sleep apnea is an established consequence of weight gain and elevated BMI,[49–51] which is the outcome variable in the current study. Therefore, we have decided to exclude the severity of sleep apnea in our final models since adjusting for downstream sequela of outcome variables can introduce bias, possibly severe.[52] Nevertheless, in sensitivity analyses we examined the inclusion of sleep apnea severity—characterized by the AHI—as a covariate as a robustness check of our findings.

Results

Findings from preliminary analysis

Sample characteristics and descriptive statistics for key variables at baseline are presented in Table 1, for all participants and separately for men and women. The participants (n = 827) were well educated (75% had at least some college education) with a mean age of 50.4 (sd = 8.0) years; 47% were female (n = 385), 8% were retired. Participants contributed to a total of 2614 valid MSLT sleep study observations, based on an average of about 3 sleep study visits (m = 3.2, sd = 0.9, ranging from 2-6 study visits per person). Participants were followed for an average of 9.8 years (sd = 4.1), ranging from 1.8-21.2 years. The mean BMI was 30.7 (sd = 6.6) at baseline; T-test (P = .014) showed that women had higher BMI (m = 31.6, sd = 7.7) than men (m = 29.9, sd = 5.4). Additionally, preliminary growth curve models for BMI with only gender and age as covariates confirmed gender differences in both levels (β = 1.91 kg/m^2/year, se = 0.47, P < .001) and linear trend of age in BMI (β = 0.11 kg/m^2/year, se = 0.02, P < .001). To account for these important differences in BMI between men and women, we stratified all subsequent growth curve models by gender.

Findings from hypothesis testing

We present parameter estimates from hypothesized models on between-person (Tables 2ab) and within-person associations (Tables 3ab) between daytime sleepiness and BMI for men and women. We illustrate the patterns of within-person association at a mean age of 45 for men and women (Fig. 1); we highlight these growth curves because associations between sleepiness and BMI were more pronounced at this age than at older ages. Partially supporting H1, the findings from *Model 3* in Table 2a showed that men who were sleepier at baseline (i.e., less MSLT

Table 1
Multiple sleep latency test/BMI baseline characteristics

	Overall (*n* = 827) Mean (*sd*) or n (%)	Men (*n* = 442) Mean (*sd*) or n (%)	Women (*n* = 385) Mean (*sd*) or n (%)
# MSLT sleep study observations	2614	1424 (54%)	1190 (46%)
# MSLT sleep study visits per person	3.2 (0.9) [*range*: 2-6]	3.2 (1.0) [*range*: 2-6]	3.1 (0.9) [*range*: 2-6]
Follow-up years	9.8 (4.1) [*range*: 1.8, 21.2]	10.0 (4.2) [*range*: 1.8, 21.2]	9.6 (4.0) [*range*: 2.9, 20.2]
Dependent Variable			
Body mass index (BMI) (kg/m^2)	30.7 (6.6) [*range*: 17.5-60.2]	29.9 (5.4) [*range*: 19.9, 56.5]	31.6 (7.7) [*range*: 17.5-60.2]
Demographic Characteristics			
Age (years)	50.4 (8.0) [*range*: 32.6, 74.0]	50.7 (8.0) [*range*: 32.8, 71.2]	49.9 (8.0) [*range*: 32.6, 74.0]
Female gender	385 (46.6%)	-	385 (100%)
Education[a]:			
High school or less	205 (24.8%)	83 (18.8%)	122 (31.7%)
Some college	258 (31.2%)	131 (29.6%)	127 (33.0%)
College graduate	168 (20.3%)	98 (22.2%)	70 (18.2%)
Post-graduate study	196 (23.7%)	130 (29.4%)	66 (17.1%)
Retired (yes)	86 (10.4%)	48 (10.9%)	38 (9.9%)
Objective Sleepiness Variable			
Clinical MSLT[b]	36 (4%)	14 (3%)	22 (6%)
MSLT score (minutes)[c]	9.6 (5.0) [*range*: 0.7-20.0]	9.0 (4.9) [*range*: 0.7-20.0]	10.3 (5.0) [*range*: 1.8-20.0]
Health Behaviors			
Current smoker (yes)	125 (15.1%)	72 (16.3%)	53 (13.8%)
Sedative use (yes)	37 (4.5%)	12 (2.7%)	25 (6.5%)
Stimulant use (yes)	10 (1.2%)	2 (0.5%)	8 (2.1%)
Alcoholic drinks (number/week)	3.5 (6.0) [*range*: 0-98]	4.8 (7.4) [*range*: 0-98]	2.0 (3.3) [*range*: 0-25]
Exercise (hours/week)	2.3 (2.6) [*range*: 0-15]	2.4 (2.7) [*range*: 0-15]	2.2 (2.6) [*range*: 0-15]
Depressive symptoms[d]	105 (12.7%)	34 (7.7%)	71 (18.4%)

SD, standard deviation; MSLT, Multiple Sleep Latency Test.
[a] Education was analyzed as an ordinal variable, moving from (0) ≤ high school; (1) some college; (2) college graduate; (3) post-graduate.
[b] Research MSLT (1616 studies in total) were administered from September 11, 1989, through September 6, 2003 (these were the majority of *baseline* MSLTs), while clinical MSLT (998 studies in total) were administered from August 14, 2001 through September 30, 2011.
[c] Multiple Sleep Latency Test (MSLT) scores range from 0-15. Shorter MSLT scores indicate more sleepiness.
[d] Depressive symptoms: Zung Score (rescaled to exclude the sleep questions) > 50, or currently taking anti-depression medication.

sleep latency as shown by the red solid line in the upper panel in Fig. 1) also had higher BMI levels at baseline (β = −0.11 kg/m^2/minute, *se* = 0.05, *P* = .03 based on *Model 3* in Table 2a), after controlling for covariates. We did not detect a similar association among women (β = −0.03 kg/m^2/minute, *se* = 0.08, *n.s.* in *Model 3* in Table 2b).

Partially supporting *H2*, the findings from *Model 3* in Table 3b showed significant 2-way interactions between linear within-person sleepiness and average age for women only, such that more sleepiness and higher BMI levels depended on age (β = 0.006 kg/m^2/minute, *se* = 0.003, *P* = .014; *Model 3* in Table 3b). Specifically, the within-person association between sleepiness and higher BMI levels was weaker among older women but stronger among younger women, after controlling for covariates. Additionally, the findings from *Model 3* in Table 3b showed a significant 2-way interaction between average between-person sleepiness and linear within-person age for women only, such that sleepy women (i.e., women with low MSLT scores as shown by the red solid line in the lower panel in Fig. 1) tended to experience more increase in BMI over time than women with lower levels of sleepiness (i.e., greater MSLT scores as shown by the green solid line in the lower panel in Fig. 1; β = −0.013 kg/m^2/hour, *se* = 0.006, *P* = .025; *Model 3* in Table 3b). We did not find any significant interaction terms between sleepiness and age for men; thus, we trimmed these non-significant effects for men in *Model 3* and Table 3a. However, the between-person effect of MSLT sleepiness scores had the same patterns of significance for men in all hypothesized *Model 3* (i.e., β = −0.22 kg/m^2/hour, *se* = 0.06, *P* < .001 in Table 3a, and β = −0.11 kg/m^2/hour, *se* = 0.05, *P* < .05 in Table 2a).

Findings from the sensitivity analyses

We present findings from sensitivity analyses in Supplementary Tables 2-6. There were 71 incident cardiovascular disease events reported after baseline among all observations (9%); 52 of these events occurred among men (13%) and the remaining 19 occurred among women (5%). The sensitivity analyses adjusting for incident cardiovascular disease suggested the same patterns of findings with all observations as the original *Model 2*. Furthermore, while ever being a shift worker was a significant covariate of BMI over time for both men and women, it did not change the patterns of findings from the hypothesized models. Finally, while we were concerned that adjusting for a known outcome (i.e., sleep apnea) of the dependent variable (i.e., BMI) could lead to biased estimates, we nevertheless conducted a sensitivity analysis by adjusting for sleep apnea severity by including the AHI as a covariate in *Model 2*.

A higher AHI score suggested more severe sleep apnea and higher AHI scores were significantly associated with overall BMI levels. However, in models with longitudinal MSLT sleepiness scores (i.e., testing for *H2* and *H3*), all significant findings on sleepiness-BMI trajectories held, with only small alterations to effect sizes of MSLT sleepiness scores. Thus, we had the same patterns of findings on longitudinal sleepiness-BMI associations for both men and women, regardless of the severity of sleep apnea. Further, in models with baseline MSLT sleepiness scores for men (i.e., testing for *H1*), the effect size of MSLT became smaller and non-significant; in the women's model, it remained non-significant as in the model without adjusting for AHI scores. Thus, men's findings on baseline sleepiness-BMI association was different depending on whether the model adjusted for the severity of sleep apnea.

Discussion

The primary aim of our investigation was to examine longitudinal associations between objective measures of daytime sleepiness and BMI. Adjusting for many potential confounders of sleepiness-BMI

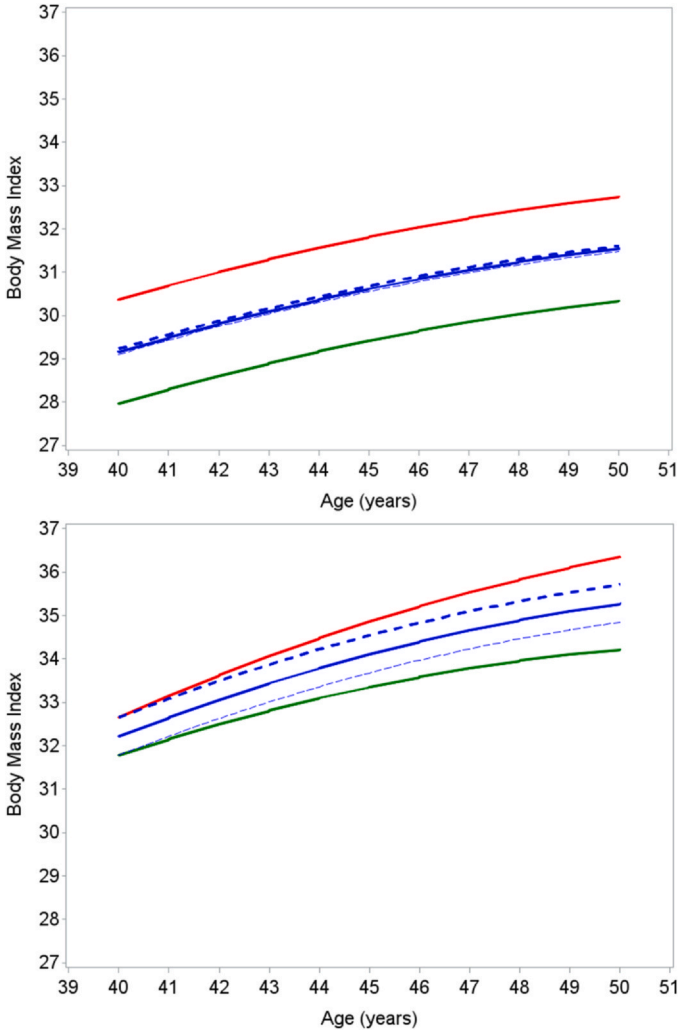

Fig. 1. Effects of within-person and between-person daytime sleepiness (measured by MSLT scores) on BMI trajectories at age 45 for men (upper panel) and women (lower panel). We illustrate growth curves during midlife (mean age 45) because sleepiness-BMI associations were more pronounced at this age than at older ages. *Red solid line*: Between-person MSLT effect = −5 and within-person effect = 0; *Green solid line*: between-person MSLT effect = 5 and within-person effect = 0; *Blue solid line*: Between-person MSLT effect = 0 and within-person effect = 0; *Thick blue dash line*: Between-person MSLT effect = 0 and within-person effect = −5; *Thin blue dash line*: between-person MSLT effect = 0 and within-person effect = 5. *Upper panel*: Men who were sleepier at baseline (i.e., lower MSLT scores as shown by the red solid line) also had higher BMI levels at baseline. *Lower panel*: Sleepy women (i.e., lower MSLT scores as shown by the red solid line) tended to experience more increase in BMI over time than women with lower levels of sleepiness (i.e., higher MSLT scores as shown by the green solid line).

Table 2a
Within- and between-person effects of baseline Multiple Sleep Latency Test on BMI among men in the WSCS (n = 1424 sleep study observations)

Fixed effects	Model 1 Beta (se)	Model 2 Beta (se)	Model 3 Beta (se)
Intercept	30.72 (0.27)***	30.73 (0.27)***	31.26 (0.30)***
Within-Person (WP) Predictors[a]			
Age	0.11 (0.01)***	0.11 (0.02)***	0.11 (0.02)***
Age^2	-0.01 (0.00)***	-0.01 (0.00)***	-0.01 (0.00)***
Between-Person (BP) Predictors[a]			
Age	-0.01 (0.03)	-0.00 (0.03)	0.02 (0.03)
Clinical MSLT[b]		-0.03 (0.15)	-0.03 (0.15)
MSLT		-0.14 (0.05)**	-0.11 (0.05)*
Education[c]			-1.19 (0.22)***
Retired			-1.00 (0.88)
Exercise			-0.30(0.09)**
Current Smoker			-1.45 (0.66)*
Sedative medication use			-3.60 (1.53)*
Stimulant medication use			7.00 (3.75)
Alcohol consumption			-0.01 (0.03)
Depression			-1.64 (0.93)
Two-Way Interactions			
Age (WP) × Age (BP)	-0.01 (0.00)***	-0.01 (0.00)***	-0.01 (0.00)***
Random effects			
Intercept variance	30.00 (2.06)***	29.61 (2.03)***	26.27 (1.81)***
Age variance	0.03 (0.00)***	0.03 (0.00)***	0.03 (0.00)***
Covariance	0.33 (0.07)***	0.34 (0.07)***	0.35 (0.06)***
Residual	1.67 (0.09)***	1.68 (0.09)***	1.68 (0.09)***
Model fit			
−2 Log-likelihood	6803.36	6796.5	6738.6

Notes. P-values * < .05, ** < .01, *** < .001.
SE, standard error; WP, within person; BP, between person; MSLT, Multiple Sleep Latency Test.
[a] WP predictors used individual observations centered on the person mean; BP predictors used the person mean centered on the sample mean. Except for age (measured in *years*), the other continuous between-person effects were centered on the baseline value.
[b] Types of MSLT (measured in *minutes*) were coded as a dummy variable with *1* = clinical MSLT and *0* = research MSLT.
[c] Education was analyzed as an ordinal variable, moving from (0) ≤ high school; (1) some college; (2) college graduate; (3) post-graduate, with computed means based on this ordering.

associations, we found that (1) higher levels of sleepiness were associated with higher levels of BMI among men and (2) increases in sleepiness over time led to quicker BMI gains among women, especially younger women. Our study has several important strengths: First, whereas prior studies have relied primarily on subjective self-reports of sleepiness, which may have low test-retest reliability,[53] we used the performance-based MSLT—an objective indicator of how long it took participants in the study to fall asleep during daytime hours. Banks and Dinges[54] highlighted striking differences between an objective indicator of vigilance and subjective reports of sleepiness under the same condition of sleep restriction; consequently, utilizing an objective indicator of sleepiness can help minimize bias when estimating its association with weight change over time.

Second, rather than focusing on global associations between daytime sleepiness and BMI, we separated its effects into between- and within-person components based on longitudinal data.[4] This approach allowed us to examine how average levels of daytime sleepiness affect BMI, while also estimating how changes in sleepiness affect BMI trajectories. The positive association between sleepiness and higher levels of and quicker gains in BMI observed in the current study is consistent with existing literature. For example, Muaidi and colleagues[55] reported a positive correlation between sleepiness and BMI among both men and women in their cross-sectional study.

In addition, our approach of modeling daytime sleepiness-BMI associations separately for men and women was necessary and consistent with extant literature. For example, prior studies have reported gender differences in excessive daytime sleepiness measured by the Epworth Sleepiness Scale,[56] which seemed to be uniquely associated with lack of regular exercise for men only.[57] A study focusing on older community-dwelling adults also reported gender-specific levels of fatigue measured by SF-36 and a unique association with depressive symptoms among older men but not women.[58] A more recent study reported that women had higher daytime sleepiness scores measured by the Karolinska Sleepiness Scale than men.[59] Gender differences in BMI levels and trajectories as well as MSLT nap latency scores observed in the current study

Table 2b
Within- and between-person effects of baseline Multiple Sleep Latency Test on BMI among women in the WSCS (n = 1190 sleep study observations)

Fixed effects	Model 1 Beta (SE)	Model 2 Beta (SE)	Model 3 Beta (SE)
Intercept	32.84 (0.41)***	32.84 (0.42)***	33.36 (0.50)***
Within-Person (WP) Predictors[a]			
Age	0.18 (0.02)***	0.18 (0.03)***	0.18 (0.03)***
Age^2	-0.01 (0.00)***	-0.01 (0.00)***	-0.01 (0.00)***
Between-Person (BP) Predictors[a]			
Age	-0.14 (0.05)*	-0.14 (0.05)**	-0.10 (0.06)
Clinical MSLT[b]		0.02 (0.24)	0.03 (0.24)
MSLT		-0.09 (0.08)	-0.03 (0.08)
Education level[c]			-1.04 (0.36)**
Retired			-0.82 (1.47)
Exercise			-0.56 (0.15)***
Current Smoker			-1.75 (1.11)
Sedative medication use			-2.83 (1.63)
Stimulant medication use			-1.20 (2.69)
Alcohol consumption			-0.32 (0.12)**
Depression			0.03 (1.05)
Two-Way Interactions			
Age (WP) × Age (BP)	-0.01 (0.00)***	-0.01 (0.00)***	-0.01 (0.00)***
Random effects			
Intercept variance	63.4 (4.77)***	63.11 (4.75)***	57.48 (4.35)***
Age variance	0.09 (0.01)***	0.09 (0.01)***	0.09 (0.01)***
Age^2 variance	0.00 (0.00)**	0.00 (0.00)**	0.00 (0.00)**
Intercept/Age Covariance	0.36 (0.17)*	0.39 (0.17)*	0.39 (0.17)*
Intercept/Age^2 Covariance	-0.09 (0.03)**	-0.09 (0.03)**	-0.10 (0.03)**
Age /Age^2 Covariance	0.00 (0.00)*	0.00 (0.00)	0.00 (0.00)
Residual	3.23 (0.26)***	3.30 (0.27)***	3.29 (0.26)***
Model fit			
−2 Log-likelihood	6693.1	6705.4	6666.8

Notes. P-values * < .05, ** < .01, *** < .001.
SE, standard error; WP, within person; BP, between person; MSLT, Multiple Sleep Latency Test.
[a] WP predictors used individual observations centered on the person mean; BP predictors used the person mean centered on the sample mean. Except for age (measured in *years*), the other continuous between-person effects were centered on the baseline value.
[b] Types of MSLT (measured in *minutes*) were coded as a dummy variable with *1* = clinical MSLT and *0* = research MSLT.
[c] Education was analyzed as an ordinal variable, moving from (0) ≤ high school; (1) some college; (2) college graduate; (3) post-graduate, with computed means based on this ordering.

Table 3a
Within- and between-person effects of Multiple Sleep Latency Test over time on BMI among men in the WSCS (n = 1424 sleep study observations)

Fixed effects	Model 1 Beta (se)	Model 2 Beta (se)	Model 3 Beta (se)
Intercept	30.72 (0.27)***	30.73 (0.27)***	31.13 (0.42)***
Within-Person (WP) Predictors[a]			
Age	0.11 (0.01)***	0.11 (0.02)***	0.11 (0.02)***
Age^2	-0.01 (0.00)***	-0.01 (0.00)***	-0.01 (0.00)***
Clinical MSLT[b]		-0.03 (0.15)	-0.03 (0.15)
Between-Person (BP) Predictors[a]			
Age	-0.01 (0.03)	0.01 (0.03)	0.01 (0.04)
MSLT		-0.24 (0.07)***	-0.22 (0.06)***
Education[c]			-1.30 (0.22)***
Retired			-0.00 (0.54)
Exercise			-0.16 (0.09)
Current Smoker			-0.91 (0.43)*
Sedative medication use			-1.89 (0.70)**
Stimulant medication use			0.42 (1.57)
Alcohol consumption			-0.07 (0.04)
Depression			0.22 (0.52)
Two-Way Interactions			
Age (WP) × Age (BP)	-0.01 (0.00)***	-0.01 (0.00)***	-0.01 (0.00)***
Random effects			
Intercept variance	30.00 (2.06)***	29.24 (2.01)***	26.23 (1.81)***
Age variance	0.03 (0.00)***	0.03 (0.00)***	0.03 (0.00)***
Covariance	0.33 (0.07)***	0.34 (0.07)***	0.34 (0.06)***
Residual	1.67 (0.09)***	1.68 (0.09)***	1.68 (0.09)***
Model fit			
−2 Log-likelihood	6803.36	6790.5	6740.2

Notes. P-values * < .05, ** < .01, *** < .001.
SE, standard error; WP, within person; BP, between person; MSLT, Multiple Sleep Latency Test.
[a] WP predictors used individual observations centered on the person mean; BP predictors used the person mean centered on the sample mean. Age was measured in *years*. The BP categorical covariates were coded as: *0* = always 0 (or no); *1* = transitions between 0 and 1; *2* = Always 1 (or yes).
[b] Types of MSLT (measured in *minutes*) were coded as a dummy variable with *1* = clinical MSLT and *0* = research MSLT.
[c] Education was analyzed as an ordinal variable, moving from (0) ≤ high school; (1) some college; (2) college graduate; (3) post-graduate, with computed means based on this ordering.

may have contributed to the different patterns of sleepiness-BMI association for men versus women.

Third, in addition to providing objective measures of sleepiness and BMI, our data source (the WSCS) is population-based and has followed participants for many years, providing important advantages in duration of follow-up, statistical power, and generalizability. Additionally, the findings have practical implications for lifestyles prone to sleep restriction; it is important for such individuals to take measures to alleviate daytime sleepiness, as it has the potential to protect against being overweight and associated risks of morbidity and mortality.

Limitations

Our investigation has some limitations. First, although our main predictor (daytime sleepiness) and outcome variable (BMI) were derived through objective assessments, covariates such as leisure-time physical activity were gathered through subjective self-reports. Although survey-based methods are often valid and reliable tools for gathering epidemiologic data, they are more prone to measurement error than objective measures.[60] For example, self-reporting of height and weight can introduce biases and random error into estimates of BMI.[61] To the extent that measurement error is present in self-reported measures of potential confounders, it could lead to incomplete adjustment for their influence on sleepiness-BMI associations. Thus, we need to use caution in interpreting these findings. Second, aside from incident CVD and sleep apnea severity in sensitivity analyses, we did not include chronic health conditions as potential confounders in our models. The primary reason we excluded such variables is that obesity is a contributor to many chronic health issues. Including variables that are causal descendants (a particularly salient example here is sleep apnea severity) of primary outcomes (BMI, in our study) is likely to introduce biases into estimated associations in regression-based models.[62] Further, experiencing menopause transitions and aging from pre-, peri-, and post-menopausal status has been associated with poor sleep quality and sleep disorders.[63,64] It would be interesting to consider the effect of menopause on the association between daytime sleepiness and BMI. Due to limitations in the current sample and data, however, we were not able to include menopause status as a covariate. A third limitation of our study is the lack of racial/ethnic diversity in the WSCS. However, prior research in more diverse adult populations has found that sleep problems have deleterious effects on body mass,[65–67] suggesting that our findings are likely generalizable to other populations. Finally, we stratified our analyses by gender to estimate different sleepiness-BMI associations for men and women but did not include formal interaction effects to test for gender differences, as this would have stretched our statistical power and created highly complex models that would be difficult for future studies to replicate.

Table 3b
Within- and between-person effects of the Multiple Sleep Latency Test over time on BMI among women in the WSCS (n = 1190 sleep study observations)

Fixed effects	Model 1 Beta (SE)	Model 2 Beta (SE)	Model 3 Beta (SE)
Intercept	32.84 (0.41)***	32.81 (0.42)***	30.10 (0.65)***
Within-Person (WP) Predictors[a]			
Age	0.18 (0.02)***	0.18 (0.03)***	0.18 (0.03)***
Age^2	-0.01 (0.00)***	-0.01 (0.00)***	-0.01 (0.00)***
Clinical MSLT[b]		0.08 (0.24)	0.08 (0.24)
MSLT		-0.02 (0.02)	-0.02 (0.02)
Between-Person (BP) Predictors[a]			
Age	-0.14 (0.05)*	-0.13 (0.05)*	-0.10 (0.06)
MSLT		-0.15 (0.10)	-0.11 (0.10)
Education[c]			-0.84 (0.36)
Retired			0.10 (0.81)
Exercise			-0.65 (0.15)***
Current Smoker			-0.53 (0.65)
Sedative medication use			-0.95 (0.82)
Stimulant medication use			3.89 (1.62)*
Alcohol consumption			-0.43 (0.12)***
Depression			-0.42 (0.57)
Two-Way Interactions			
MSLT (WP) × Age (BP)		0.01 (0.00)*	0.01 (0.00)*
MSLT (BP) × Age (WP)		-0.01 (0.01)*	-0.01 (0.01)*
Age (WP) × Age (BP)	-0.01 (0.00)***	-0.01 (0.00)***	-0.01 (0.00)***
Random effects			
Intercept variance	63.4 (4.77)***	63.07 (4.74)***	54.91 (4.16)***
Age variance	0.09 (0.01)***	0.09 (0.01)***	0.09 (0.01)***
Age^2 variance	0.00 (0.00)**	0.00 (0.00)**	0.00 (0.00)**
Intercept/Age Covariance	0.36 (0.17)*	0.36 (0.17)*	0.33 (0.16)*
Intercept/Age^2 Covariance	-0.09 (0.03)**	-0.09 (0.03)**	-0.09 (0.03)**
Age /Age^2 Covariance	0.00 (0.00)*	0.00 (0.00)*	0.00 (0.00)*
Residual	3.23 (0.26)***	3.23 (0.26)***	3.22 (0.26)***
Model fit			
−2 Log-likelihood	6693.1	6693.0	6640.9

Notes. P-values * < .05, ** < .01, *** < .001.
SE, standard error; WP, within person; BP, between person; MSLT, Multiple Sleep Latency Test.
[a] WP predictors used individual observations centered on the person mean; BP predictors used the person mean centered on the sample mean. Age was measured in *years*. The BP categorical covariates were coded as: 0 = always 0 (or no); 1 = transitions between 0 and 1; 2 = Always 1 (or yes).
[b] Types of MSLT (measured in *minutes*) were coded as a dummy variable with 1 = clinical MSLT and 0 = research MSLT.
[c] Education was analyzed as an ordinal variable, moving from (0) ≤ high school; (1) some college; (2) college graduate; (3) post-graduate, with computed means based on this ordering.

Conclusions

Using data collected from a population-based sample, we estimated associations between daytime sleepiness and BMI spanning the period from mid-to-late life. We found significant associations between daytime sleepiness and BMI among both men and women. Among men, higher mean levels of sleepiness were associated with higher mean levels of BMI. Among women, increases in sleepiness over time were associated with faster gains in BMI; this association was especially strong among young women. Our investigation contributes to the sleep-BMI literature by demonstrating that both levels and changes in objective measures of daytime sleepiness contribute to higher BMI, highlighting the importance of sleep health to weight maintenance in adulthood. Future studies should further explore mechanisms that may account for associations between daytime sleepiness and BMI, and try to replicate our findings using longitudinal data in a more diverse population of adults.

Declaration of Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in the current paper.

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CRediT authorship contribution statement

ENR and YL conceptualized and designed the study. **JHB, EWH, PEP** collected data. **YL and JHB** conducted the analysis and interpreted the data. **YL** drafted the manuscript; all co-authors revised the manuscript critically for important intellectual content.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.sleh.2024.03.002.

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