UC Irvine

Title

Prospective associations among objectively and subjectively assessed sleep and the metabolic syndrome.

Permalink

https://escholarship.org/uc/item/3786x1x3

Authors

Bowman, Marissa
Duggan, Katherine
Brindle, Ryan
et al.

Publication Date

DOI

Peer reviewed

Prospective associations among objectively and subjectively assessed sleep and the metabolic syndrome

Marissa A. Bowman[1], **Katherine A. Duggan**[2], **Ryan C. Brindle**[3], **Christopher E. Kline**[4], **Robert T. Krafty**[5], **Julian F. Thayer**[6], **Martica H. Hall**[2,*]

[1]Department of Psychology, University of Pittsburgh, Pittsburgh, PA

[2]Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA

[3]Department of Cognitive and Behavioral Science & Neuroscience Program, Washington and Lee University, Lexington, VA

[4]Department of Health and Physical Activity, University of Pittsburgh, Pittsburgh, PA

[5]Department of Biostatistics, University of Pittsburgh, Pittsburgh, PA

[6]Department of Psychology, Ohio State University, Columbus, OH

Abstract

Objective: Subjective sleep disturbances have been associated with greater risk for concurrent and incident metabolic syndrome (MetS). Previous studies have not examined prospective associations among polysomnography-assessed sleep and the MetS, despite knowledge that self-reported sleep is subject to reporting bias, and that subjectively and objectively assessed sleep are weakly correlated.

Method: In the current study, objectively-assessed (polysomnography) and subjectively-assessed (Pittsburgh Sleep Quality Index) sleep was measured in 145 adults at two timepoints, separated by 12–30 years. A continuous measure of the MetS was assessed at the second time point. Statistical analyses were adjusted for age, sex, lifetime history of major depressive disorder, follow-up time, and apnea-hypopnea index.

Results: Polysomnography-assessed sleep duration, latency, efficiency, and slow wave sleep were not significantly prospectively associated with the MetS ($ps \geq .16$). Self-reported longer sleep latency was prospectively associated with higher MetS scores in unadjusted ($\beta = 0.29$, $p = .002$) and adjusted models ($\beta = 0.25$, $p = .009$). Longer sleep latency was associated with higher fasting glucose levels ($\beta = 0.47$, $p < .001$).

Conclusion: Our study provides evidence that subjective and objective measures of sleep may differ in their ability to prospectively predict MetS.

*Corresponding author: 3811 O'Hara Street, Rm: E-1131, Pittsburgh, PA 15213, USA. Telephone: +1-412-246-6413; Fax: +1-412-246-5300; hallmh@upmc.edu.

Keywords

sleep; polysomnography; Pittsburgh Sleep Quality Index; metabolic syndrome; cardiometabolic health; glucose

---

1. Introduction

Poor sleepers are at increased risk for all-cause mortality (Cappuccio, D'Elia, Strazzullo, & Miller, 2010a). This association may be driven by relationships between sleep and intermediate risk factors that substantially affect mortality. One such intermediate risk factor is the metabolic syndrome (MetS), a modifiable cluster of symptoms (high blood pressure, triglycerides, and glucose, large waist circumference, and low levels of high density lipoproteins) known to increase risk for cardiovascular disease, stroke, myocardial infarction, and all-cause mortality (Mottillo et al., 2010). Several studies have reported significant cross-sectional associations among dimensions of self-reported poor sleep and the MetS including short sleep duration (meta-analysis of 18 studies; Iftikhar et al., 2015), difficulty initiating and maintaining sleep (Lin et al., 2016), excessive daytime sleepiness (Akbaraly et al., 2015), and poor sleep quality (Jennings, Muldoon, Hall, Buysse, & Manuck, 2007). Only one cross-sectional study has used polysomnography (PSG) to evaluate associations among objectively-assessed sleep and the MetS. Hall and colleagues (2012) reported that lower PSG-assessed sleep efficiency, greater sleep disordered breathing, and greater cortical arousal during sleep were associated with greater odds of prevalent MetS. However, prospective data are critical to clarify temporal associations among disturbed sleep and the MetS.

Several studies have shown prospective associations between self-reported sleep disturbances and the MetS. While one study showed that short sleep duration was associated with increased odds of incident MetS at three-year follow-up (Kim et al., 2015), another study demonstrated that both short and long sleep duration were associated with increased risk for MetS at four-year follow-up (Li et al., 2015). A third study reported increased incident MetS in individuals who were persistent short sleepers or those whose sleep duration decreased by more than two hours at a four-year follow-up (Song et al., 2016). Troxel and colleagues (2010) reported that self-reported difficulty initiating sleep, unrefreshing sleep, and loud snoring predicted incident MetS at a three-year follow-up. To our knowledge, no prospective study has tested the association between PSG-assessed sleep and the MetS.

There are documented links between experimental sleep restriction or deprivation and components of the MetS. Experimental sleep restriction has been associated with increased blood concentrations of free fatty acids (Broussard et a., 2015) while total sleep deprivation has been associated with increased diastolic blood pressure (Ogawa et al., 2003). Moreover, experimental suppression of slow wave sleep (SWS) has been linked to acute decreases in glucose tolerance (Tasali et al., 2008). Although none of these experimental studies evaluated all components of the MetS, these data are consistent with the hypothesis that sleep and circadian disturbances may be causally associated with MetS risk.

In the present study, we examined the prospective association between multiple indices of PSG and self-reported sleep with the MetS, measured 12–30 years later (M_{years} = 19.1, SD = 4.7). Indices of sleep duration and continuity were assessed both subjectively, using self-reports, and objectively, as measured by PSG. In contrast, sleep quality was assessed by self-report and SWS was measured by PSG due to the absence of objective and subjective homologues, respectively. It was hypothesized that (1) shorter sleep duration; (2) longer sleep latency; (3) lower sleep efficiency; (4) lower sleep quality; and (5) lower percent SWS would prospectively predict greater MetS prevalence. Similar predictions were made for cross-sectional associations among sleep and MetS, using sleep data collected concurrently with the MetS. Additionally, we examined two possible effect modifiers of associations: depression and follow-up time. Previous meta-analytic evidence has demonstrated that individuals with depression are more likely to have sleep disturbances (Bao et al., 2017) and the MetS (Pan et al., 2012). Follow-up time was examined as a potential moderator of associations between sleep and the MetS to examine whether greater follow-up time weakens associations.

2. Methods

2.1 Participants

Participants were recruited from previous studies evaluating sleep in individuals with major depressive disorder ($n = 508$) and healthy controls ($n = 204$) at the University of Pittsburgh from 1982 to 1999 (Time 1; T1). Participants had neither significant medical illness nor the presence of any other clinician-assessed Axis 1 disorder at Time 1. From 2010 to 2014, 339 participants from the T1 studies were contacted to participate in a study of sleep and cardiovascular disease risk. Of these, 177 participants provided written informed consent to the follow-up study (Time 2; T2). A priori exclusion criteria were unstable medical illness ($n = 2$) or development of major depressive disorder with psychotic features or bipolar disorder ($n = 9$). We excluded an additional 21 people for the following reasons: withdrawal from the study prior to data collection ($n = 8$), lived out of state and could not visit the lab for cardiovascular assessments ($n = 5$), or were missing polysomnography and/or waist circumference data ($n = 8$). The current report includes data from the remaining 145 participants (88 participants with current or past major depressive disorder and 57 control participants). Compared to those included in the analyses ($N = 145$), participants who provided some data but were excluded from analyses ($N = 21$) did not differ on age, sex, history of depression, T2 apnea-hypopnea index, or any sleep variable measured at T1 or T2 (ps .06). All participants had full PSG data at T1 and T2. Time 1 subjective sleep data were missing in 37 to 38 participants, depending on the sleep component, because some T1 visits occurred before the Pittsburgh Sleep Quality Index (PSQI, Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) was developed. These participants were not included in T1 subjective sleep measure analyses but were included in all other analyses. This study was approved by the University of Pittsburgh Institutional Review Board and financial compensation was provided to study participants.

2.2   Measures

2.2.1   Objectively-assessed sleep.—Participants underwent overnight sleep studies for two nights at T1 and three nights at T2 at Western Psychiatric Institute and Clinic in Pittsburgh, Pennsylvania. Lights out and lights on corresponded to participants' self-reported habitual sleep patterns. The PSG recording montage included electroencephalography (C3, Cz, C4), electrooculography, and electromyography, with each electrode referenced to the contralateral mastoid. For both T1 and T2 sleep studies, night one served as an adaptation night. For T1, night two studies were re-scored between 2010–2014 in 30-second epochs pursuant to American Academy of Sleep Medicine (AASM) criteria (AASM, 2007). On night one for the T2 sleep study, apnea-hypopnea index (AHI) was assessed. For T2, data for nights two and three were scored using AASM criteria and sleep values were averaged across nights (Israel et al., 2012).

Sleep duration was calculated as the total minutes spent in non-rapid and rapid eye movement sleep. Sleep latency was defined as the time elapsed between lights out and the first epoch of N1 sleep. Sleep efficiency was calculated as the total minutes of sleep divided by the total minutes of time in bed, multiplied by 100. Percent SWS was defined as the number of minutes with >50% delta waves (>75 μV), divided by sleep duration, and multiplied by 100. We did not examine other measures of sleep architecture, as we did not have a priori hypotheses. We could not evaluate quantitative EEG measures, as T1 sleep data were recorded on paper.

2.2.2   Subjectively-assessed sleep.—The PSQI is an 18-item self-report measure of individuals' habitual sleep quality (Buysse et al., 1989). The PSQI was used to derive homologous measures of sleep duration, latency, and efficiency. Sleep duration, latency, bed time, and wake time were derived from individual PSQI questions. Sleep efficiency was calculated as sleep duration divided by the interval of time between bed time and wake time, multiplied by 100; the maximum value possible for sleep efficiency was 100%. Sleep quality, which has no PSG homologue, was assessed from the question, "During the past month, how would you rate your sleep quality overall?", with "very good" (0), "fairly good" (1), "fairly bad" (2), or "very bad" (3) as response options.

2.2.3   Metabolic syndrome.—Components of the MetS were assessed at T2 in the morning while participants were fasting, in a separate visit from the in-lab sleep assessment. Blood pressure was assessed while the participant was in a supine position following a 10-minute rest period using an automated sphygmomanometer (CONTEC08A, Qinhuangdao CONTEC Medical Systems Co., Ltd., China). Systolic and diastolic blood pressure readings were averaged across three measures collected at one-minute intervals. Forearm venous blood draws were used to assess fasting plasma glucose (mg/dl), serum triglycerides (mg/dl), and high-density lipoproteins (HDL). Waist circumference was measured at the superior border of the iliac crest by a clinician, as recommended by the National Cholesterol Education Program's Adult Treatment Panel III (ATP III) report (Grundy, Brewer, Cleeman, Smith, & Lenfant, 2004).

Metabolic syndrome is often defined as present in participants who meet three or more ATP III cutoff scores for systolic blood pressure, fasting glucose, triglycerides, high density lipoprotein cholesterol, and waist circumference (Grundy et al., 2004). However, a joint statement by the American Diabetes Association and the European Association for the Study of Diabetes recommends that researchers characterize the metabolic syndrome continuously, given its linear association with cardiovascular disease (Kahn, Buse, Ferrannini, & Stern, 2005). Further, some components of the metabolic syndrome may contribute more to negative health outcomes than others, and a continuous measure allows for greater "weighting" than a dichotomous outcome (Kahn et al., 2005). Therefore, in the present report, the metabolic syndrome was characterized as the average of the z-scores of each component of the metabolic syndrome, which creates a continuous measure (i.e. summing the z-scores of systolic blood pressure, glucose, triglycerides, waist circumference, and the reverse-score of HDL, then dividing by five), with higher values reflecting higher MetS scores.

2.2.4   Covariates.—Covariates included sex, age, lifetime history of major depressive disorder, and apnea-hypopnea index (AHI). Additionally, follow-up time was adjusted for in the prospective analyses. Lifetime history of major depressive disorder was a dichotomous variable, with "yes" defined as current or past major depressive disorder per structured clinical interviews at T1 and/or T2. Obstructive sleep apnea, assessed using AHI at T2, was included as it is frequently comorbid with the MetS (for review, see Tasali & Ip, 2008); no participant was treated for obstructive sleep apnea. Follow-up time was calculated as the difference in years between T1 and T2.

2.3   Statistical Approach

Descriptive statistics were used to characterize the sample. A comparison of T1 and T2 sleep variables was conducted using paired sample t-tests. Linear regression assumptions were examined; AHI was log-transformed. Hierarchical linear regression was used to assess associations between subjectively and objectively assessed indices of sleep and the z-score of MetS. Univariate analyses for each individual sleep variable were followed by multiple linear regression models. Prospective models adjusted for T2 age, sex, lifetime depression history, T2 AHI, and follow-up time. Cross-sectional models adjusted for T2 age, sex, lifetime depression history, and T2 AHI. Age, AHI, and follow up time were continuous and centered based on the sample's mean. Sex was effects coded as −1 (females) and 1 (males). Lifetime history of depression was effects coded as −1 (no history) and 1 (history). We evaluated the association between significant sleep variables and MetS components to determine whether specific components were driving observed associations.

In analyses of effect modification, we examined whether lifetime depression history or follow-up time were moderators of the association between sleep variables and the MetS. Moderation analyses were conducted in hierarchical linear regression models, with the sleep variable entered first, then the moderator, then their interaction, and finally the covariates. We applied Bonferroni corrections to the p-values for interaction terms, as the analyses were exploratory. Finally, we conducted two sensitivity analyses to further investigate the role of depression in associations. First, we included a dichotomous variable of antidepressant use

at T2 ($n = 51$ were taking at least one antidepressant) as a covariate to evaluate whether treatment may affect our results. Second, we excluded participants with current major depressive disorder at T2 ($n = 16$) to ensure that current depression was not influencing study results. Analyses were conducted in IBM SPSS Statistics software (version 25) and all tests were two sided at a=0.05.

3. Results

As shown in Table 1, 65% of the sample was female and 61% of the sample had a history of major depressive disorder. The average follow-up time between T1 and T2 was 19.1 years (range: 12.5 to 30.3 years). At T2, the average participant was middle-age (59.9 yr; range: 36.3 to 82.9 yr).

Table 2 presents T1 and T2 descriptive statistics for sleep measures. In paired sample t-tests, there was a significant within-person increase in PSG-assessed sleep latency ($p < .001$), significant within-person decreases in PSG-assessed sleep duration ($p < .001$) and PSG-assessed sleep efficiency ($p < .001$). No other sleep measures significantly changed from T1 to T2.

Prospective associations among T1 sleep and the MetS are presented in Table 3. None of the PSG-assessed indices of sleep were significantly prospectively associated with MetS (ps . 16). Subjective sleep latency was associated with higher MetS scores in unadjusted ($\beta = 0.29$, $p = .002$) and adjusted models ($\beta = 0.25$, $p = .009$). No other subjective indices of sleep were significantly prospectively associated with MetS. Among covariates, sex (p .006) and log-transformed AHI (p .002) were significantly associated with the MetS, such that being male or having higher AHI was associated with higher MetS scores. In contrast, age (p . 71), lifetime history of depression (p .12), and follow-up time (p .20) were not significant correlates of the MetS in any of the prospective models.

In cross-sectional analyses, none of the objectively- or subjectively-assessed indices of sleep were significantly associated with the MetS (Table 4). Similar to prospective analyses, sex (men had higher MetS scores; ps .001) and log-transformed AHI ($ps < .001$) were associated with the MetS in cross-sectional analyses such that being male or having higher AHI was associated with higher MetS scores, while age (ps .54) and lifetime depression history (ps .13) were not.

Associations among sleep and individual components of the MetS were examined for T1 sleep latency, as this was the only sleep measure that was significantly associated with MetS scores. Time 1 sleep latency was significantly associated with higher T2 fasting blood glucose levels ($\beta = 0.47$, $p < .001$). Sleep latency was not significantly associated with any of the other MetS components (blood pressure, triglycerides, waist circumference, and HDL; $ps > .15$). Among covariates, higher log-transformed AHI was significantly associated with higher systolic blood pressure ($p = .01$), greater waist circumference ($p = .004$), and lower HDL ($p = .01$). No other covariate was significantly associated with any of the MetS components ($ps > .05$).

3.1 Secondary Analyses

In prospective moderation analyses, lifetime history of depression was a significant moderator of associations among subjectively-assessed sleep duration and sleep quality with the MetS. In cross-sectional moderation analysis, lifetime history of depression was a significant moderator of associations among subjectively-assessed sleep latency and the MetS. None of these models were non-significant after Bonferroni correction. Similarly, moderation of prospective associations among subjectively-assessed sleep quality and MetS by follow up time was not significant after Bonferroni correction.

In sensitivity analyses, we (1) adjusted for antidepressant use at T2 and (2) restricted our sample to individuals who were not currently in a major depressive episode at T2 to evaluate the robustness of our results. A substantial change in results was defined as a change from significance to non-significance, from non-significance to significance, or a change in the standardized beta coefficient of ± 0.10. Antidepressant use was not significantly associated with the MetS ($\beta = -0.04$, $p = .65$). Including the covariate of antidepressant use did not substantially change our results. Removal of participants who were currently in a major depressive episode at T2 ($n = 16$) also did not change our results substantially.

4. Discussion

We are not aware of any published study that has examined prospective associations between both PSG- and subjectively-assessed sleep and the MetS. We report that self-reported longer sleep latency was prospectively associated with higher MetS scores. Observed associations may be due, in part, to associations between sleep latency and higher glucose levels. In contrast, PSG-assessed sleep was not prospectively associated with the MetS.

The prospective association between longer subjective sleep latency and the MetS is consistent with a previous study of self-reported sleep and MetS at 3-year follow up (Troxel et al., 2010). The present study extends these findings in that we report that longer sleep latency is associated with the MetS over a longer follow up period of 12–30 years. In analysis of the individual components of the MetS, sleep latency was strongly associated with higher glucose levels. These results are consistent with evidence that insomnia symptoms are associated with type 2 diabetes (Cappuccio et al., 2010b). Prolonged sleep latency measured by polysomnography has also been linked to all-cause mortality in older adults, after adjusting for confounding by mental and physical health (Dew et al., 2003). With respect to mechanisms that may underlie prospective associations among sleep latency and the MetS, longer sleep latency may be due to habitual sleep anxiety (Babson, Trainor, Bunaciu, & Feldner, 2008), which may result in sympathetic nervous system activation (for review, see McEwen, 2000). In addition, individuals with longer sleep latency may use sleep medications or alcohol as sleep aids, which are associated with increased mortality (Di Castelnuovo et al., 2006; Kripke, Garfinkel, Wingard, Klauber, & Marler, 2002). Prospective and experimental studies are needed to identify the pathways through which prolonged sleep latency influences MetS risk.

The absence of significant prospective associations among PSG-assessed sleep and the MetS differs both from current and previous self-reported associations (Li et al., 2015; Kim et al.,

2015; Song et al., 2016; Troxel et al., 2010), as well as our own report of cross-sectional associations among PSG-assessed indices of sleep continuity and depth with the MetS (Hall et al., 2012). Differences in self-reported and PSG-assessed sleep in relation to the MetS may be related to recognized discrepancies between objective and subjective assessments of sleep, which do not correlate highly with one another (Kaplan et al., 2017). Statistical adjustment for factors that might influence subjective assessments, such as history of depression, mitigated differences related to reporting bias. There are factors that may influence reporting bias that were not included in this study. For example, one study has reported that individuals who were higher in trait hostility had a weaker correlation between PSG-assessed sleep duration and self-reported sleep duration (Matthews et al., 2018). In another study, race/ethnicity was shown to bias self-report estimates, with Caucasian Americans more likely to overestimate their sleep duration on self-reports versus PSG-assessed sleep compared to African Americans and Chinese Americans (Jackson et al., 2018). Future studies of the association of sleep with cardiovascular health outcomes might evaluate hostility or race/ethnicity, as both factors are known to be associated with cardiovascular disease outcomes (Harper et al., 2007; Miller et al., 1996).

Differences in results across sleep measurement modalities in the current study may also be related to differences in time frame; PSG was assessed on one to two nights and self-reported sleep referenced the past month. It may be that PSG in this study represents a short-term measure of sleep, and self-report reflects a more habitual measure of sleep. Indeed, we, and others, have reported that PSG-assessed sleep continuity varies widely from night-to-night, suggesting that a night or two of PSG may not be a reliable indicator of sleep continuity (Israel et al., 2012). Although PSG-assessed indices of sleep depth are highly stable across nights (Israel et al., 2012), age-related decreases in slow-wave sleep over the long follow-up period in the present study (Ohayon et al., 2004) may have diminished its long-term impact on the MetS. In summary, in this study we demonstrated that one night of PSG-assessed sleep was not significantly related to the MetS 12–30 years later. These results do not suggest that an objective assessment of sleep is unwarranted in MetS research, but rather that future studies might measure objectively-assessed sleep for a greater number of nights to better capture habitual sleep.

There were no significant cross-sectional associations among measures of sleep and the MetS in the current study, which contrasts with significant associations reported in previous studies. One reason for this discrepancy may be our choice to model sleep dimensions as continuous variables. Previous studies of self-reported sleep duration used categorical bins based on a range of hours that participants slept. However, in a meta-analysis of 18 studies, the meta-regression was not significant due to non-linearity (Iftikhar et al., 2015), which our study was not powered to test as we had few participants with a sleep duration greater than 9 hours. Similarly, previous studies of sleep latency characterized participants based on the number of times per week it takes them longer than one hour to fall asleep (Lin et al., 2016), or how often they have difficulty falling asleep (Akbaraly et al., 2015), and have assessed sleep efficiency indirectly by asking participants how often they have difficulty maintaining sleep (Akbaraly et al., 2015). Again, these categorical measures that require participants to assess how frequently they have difficulty sleeping may be more likely to capture variability, rather than average measures. Similar to the only published study of PSG-assessed sleep and

the MetS, we show here a non-significant association for linearly modeled sleep duration and a significant association for AHI (Hall et al., 2012). In contrast to our earlier study (Hall et al., 2012), we did not find a significant association for PSG-assessed sleep efficiency. This may be because the previous study only studied female participants, and there are well-documented sex differences in both sleep (Zhang & Wing, 2006) and the MetS (Ervin, 2009). In the context of these studies, our study suggests that the way sleep is assessed and modeled is crucial for the conclusions that are drawn.

Several design considerations should be considered when evaluating the present report of secondary analyses in a study of prospective associations among sleep, depression, and cardiovascular disease. First, we were unable to evaluate incident MetS or adjust for MetS in prospective analyses, because components of the MetS were not assessed at T1. Second, we were not powered to evaluate MetS as a categorical variable. However, others have suggested that modeling the MetS as a continuous score may be preferable, as continuous scores may better capture MetS as a spectrum of risk (Kahn et al., 2005). Third, the present report is the first to evaluate prospective associations among both PSG- and self-reported indices of sleep duration, continuity, depth, and quality with the MetS. However, differences in results across measurement modalities raises additional questions about the extent to which objectively-assessed indices of habitual sleep may be prospectively associated with the MetS, including incident MetS. Finally, adjustment for confounding factors including clinical assessment of depression history, objective measurement of obstructive sleep apnea, as well as the use of similar sleep assessments at both study time points enhances the rigor of this observational study.

5. Conclusion

In summary, self-reported prolonged sleep latency was prospectively associated with increased severity of the MetS, after adjusting for other MetS risk factors. Results were independent of depression history and follow-up time. In contrast, PSG-assessed sleep was not prospectively associated with the MetS, perhaps due to reliance on a single night of laboratory-assessed sleep. Cross-sectional associations among objectively- and subjectively-assessed sleep and severity of the MetS were not significant due to sample and measurement considerations. Taken as a whole, these results suggest that PSG-assessed and self-reported sleep may be differentially related to the MetS, although future studies are needed to evaluate more habitual indices of objectively-assessed sleep in relation to the MetS. Certainly, multi-method assessment of sleep may provide a clearer picture of the long-term impact of sleep on health.

Acknowledgements:

References

Akbaraly TN, Jaussent I, Besset A, Bertrand M, Barberger-Gateau P, Ritchie K, … Dauvilliers Y (2015). Sleep complaints and metabolic syndrome in an elderly population: The three-city study. Am J Geriatr Psychiatry, 23(8), 818–828. 10.1016/j.jagp.2014.10.001 [PubMed: 25499672]

American Academy of Sleep Medicine (2007). The AASM manual for the scoring of sleep and associated events: Rules Terminology and Technical Specifications. Westchester: AASM.

Babson KA, Trainor CD, Bunaciu L, & Feldner MT (2008). An examination of anxiety sensitivity as a moderator of the relation between sleep anticipatory anxiety and sleep onset latency. J Cogn Psychother, 22(3), 258 10.1891/0889-8391.22.3.258

Bao Y-P, Han Y, Ma J, Wang R-J, Shi L, Wang T-Y, … Lu L (2017). Cooccurrence and bidirectional prediction of sleep disturbances and depression in older adults: Meta-analysis and systematic review. Neurosci Biobehav Rev, 75, 257–273. 10.1016/j.neubiorev.2017.01.032 [PubMed: 28179129]

Baron KG, Reid KJ, Kern AS, & Zee PC (2011). Role of sleep timing in caloric intake and BMI. Obesity, 19(7), 1374–1381. 10.1038/oby.2011.100 [PubMed: 21527892]

Broussard JL, Chapotot F, Abraham V, Day A, Delebecque F, Whitmore HR, & Tasali E (2015). Sleep restriction increases free fatty acids in healthy men. Diabetologia, 58(4), 791–798. 10.1007/s00125-015-3500-4 [PubMed: 25702040]

Buysse DJ, Reynolds CF, Monk TH, Berman SR, & Kupfer DJ (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. Psychiatry Research, 28(2), 193–213. [PubMed: 2748771]

Cappuccio FP, D'Elia L, Strazzullo P, & Miller MA (2010a). Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. Sleep, 33(5), 585–592. 10.1093/sleep/33.5.585 [PubMed: 20469800]

Cappuccio FP, D'Elia L, Strazzullo P, & Miller MA (2010b). Quantity and quality of sleep and incidence of type 2 diabetes: A systematic review and meta-analysis. Diabetes Care, 33, 414–420. [PubMed: 19910503]

Dew MA, Hoch CC, Buysse DJ, Monk TH, Begley AE, Houck PR, … & Reynolds CF III (2003). Healthy older adults' sleep predicts all-cause mortality at 4 to 19 years of follow-up. Psychosom Med, 65(1), 63–73. [PubMed: 12554816]

Di Castelnuovo A, Costanzo S, Bagnardi V, Donati MB, Iacoviello L, & De Gaetano G (2006). Alcohol dosing and total mortality in men and women: an updated meta-analysis of 34 prospective studies. Arch Intern Med, 166(22), 2437–2445. 10.1001/archinte.166.22.2437 [PubMed: 17159008]

Ervin RB (2009). Prevalence of metabolic syndrome among adults 20 years of age and over, by sex, age, race and ethnicity, and body mass index: United States, 2003–2006. Natl Health Stat Report, 13, 1–7.

Grundy SM, Brewer HB, Cleeman JI, Smith SC, & Lenfant C (2004). Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association Conference on Scientific Issues Related to Definition. Circ, 109(3), 433–438. 10.1161/01.CIR.0000111245.75752.C6

Hall MH, Okun ML, Sowers M, Matthews KA, Kravitz HM, Hardin K, … Sanders MH (2012). Sleep is associated with the metabolic syndrome in a multi-ethnic cohort of midlife women: The SWAN Sleep Study. Sleep, 35(6), 783–90. 10.5665/sleep.1874 [PubMed: 22654197]

Harper S, Rushani D, & Kaufman JS (2012). Trends in the black-white life expectancy gap, 2003–2008. JAMA, 307(21), 2257–2259. [PubMed: 22706828]

Iftikhar IH, Donley MA, Mindel J, Pleister A, Soriano S, & Magalang UJ (2015). Sleep duration and metabolic syndrome: An updated dose-risk metaanalysis. Ann Am Thorac Soc, 12(9), 1364–1372. 10.1513/AnnalsATS.201504-190OC [PubMed: 26168016]

Israel B, Buysse DJ, Krafty RT, Begley A, Miewald J, & Hall M (2012). Short-term stability of sleep and heart rate variability in good sleepers and patients with insomnia: for some measures, one night is enough. Sleep, 35(9), 1285–1291. 10.5665/sleep.2088 [PubMed: 22942507]

Jackson CL, Patel SR, Jackson WB, Lutsey PL, & Redline S (2018). Agreement between self-reported and objectively measured sleep duration among white, black, Hispanic, and Chinese adults in the United States: Multi-Ethnic Study of Atherosclerosis. Sleep, 41(6), zsy057 10.1093/sleep/zsy057

Jennings JR, Muldoon MF, Hall M, Buysse DJ, & Manuck SB (2007). Self-reported sleep quality is associated with the metabolic syndrome. Sleep, 30(2), 219–223. 10.1093/sleep/30.2.219 [PubMed: 17326548]

Kahn R, Buse J, Ferrannini E, & Stern M (2005). The metabolic syndrome: Time for a critical appraisal. Diabetologia, 48(9), 16841699 10.1007/s0012500518762

Kaplan KA, Hirshman J, Hernandez B, Stefanick ML, Hoffman AR, Redline S, … Zeitzer JM (2017). When a gold standard isn't so golden: Lack of prediction of subjective sleep quality from sleep polysomnography. Biol Psychol, 123, 37–46. 10.1016/j.biopsycho.2016.11.010 [PubMed: 27889439]

Kim J-Y, Yadav D, Ahn SV, Koh S-B, Park JT, Yoon J, … Lee S-H (2015). A prospective study of total sleep duration and incident metabolic syndrome: the ARIRANG study. Sleep Med, 16(12), 1511–1515. 10.1016/j.sleep.2015.06.024 [PubMed: 26611949]

Kripke DF, Garfinkel L, Wingard DL, Klauber MR, & Marler MR (2002). Mortality associated with sleep duration and insomnia. Arch Gen Psychiatry, 59(2), 131–136. [PubMed: 11825133]

Li X, Lin L, Lv L, Pang X, Du S, Zhang W, … & Deng H (2015). U-shaped relationships between sleep duration and metabolic syndrome and metabolic syndrome components in males: a prospective cohort study. Sleep Med, 16(8), 949–954. 10.1016/j.sleep.2015.03.024 [PubMed: 26116460]

Lin S-C, Sun C-A, You S-L, Hwang L-C, Liang C-Y, Yang T, … Chou Y-C (2016). The link of self-reported insomnia symptoms and sleep duration with metabolic syndrome: A Chinese population-based study. Sleep, 39(6), 1261–6. 10.5665/sleep.5848 [PubMed: 27070137]

Matthews KA, Patel SR, Pantesco EJ, Buysse DJ, Kamarck TW, Lee L, & Hall MH (2018). Similarities and differences in estimates of sleep duration by polysomnography, actigraphy, diary, and self-reported habitual sleep in a community sample. Sleep Health, 4(1), 96–103. 10.1016/j.sleh.2017.10.011 [PubMed: 29332687]

McEwen BS (2000). Allostasis and allostatic load: implications for neuropsychopharmacology. Neuropsychopharmacology, 22(2), 108–124. 10.1016/S0893-133X(99)00129-3 [PubMed: 10649824]

Miller TQ, Smith TW, Turner CW, Guijarro ML, & Hallet AJ (1996). Meta-analytic review of research on hostility and physical health. Psychological Bulletin, 119(2), 322. [PubMed: 8851276]

Mottillo S, Filion KB, Genest J, Joseph L, Pilote L, Poirier P, … Eisenberg MJ (2010). The metabolic syndrome and cardiovascular risk: A systematic review and meta-analysis. J Am Coll Cardiol, 56(14), 1113–1132. 10.1016/j.jacc.2010.05.034 [PubMed: 20863953]

Ogawa Y, Kanbayashi T, Saito Y, Takahashi Y, Kitajima T, Takahashi K, … Shimizu T (2003). Total sleep deprivation elevates blood pressure through arterial baroreflex resetting: a study with microneurographic technique. Sleep, 26(8), 986–989. 10.1093/sleep/26.8.986 [PubMed: 14746379]

Ohayon MM, Carskadon MA, Guilleminault C, & Vitiello MV (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. Sleep, 27(7), 1255–1273. 10.1093/sleep/27.7.1255 [PubMed: 15586779]

Pan A, Keum N, Okereke OI, Sun Q, Kivimaki M, Rubin RR, & Hu FB (2012). Bidirectional association between depression and metabolic syndrome: A systematic review and meta-analysis of epidemiological studies. Diabetes Care, 35(5), 1171–1180. [PubMed: 22517938]

Song Q, Liu X, Zhou W, Wang X, & Wu S (2016). Changes in sleep duration and risk of metabolic syndrome: The Kailuan prospective study. Sci Rep, 6, 36861 10.1038/srep36861 [PubMed: 27857185]

Tasali E, & Ip MS (2008). Obstructive sleep apnea and metabolic syndrome: Alterations in glucose metabolism and inflammation. Proc Am Thorac Soc, 5(2), 207–217. doi: 10.1513/pats.200708-139MG [PubMed: 18250214]

Tasali E, Leproult R, Ehrmann DA, & Van Cauter E (2008). Slow-wave sleep and the risk of type 2 diabetes in humans. PNAS, 105(3), 1044–9. 10.1073/pnas.0706446105 [PubMed: 18172212]

Troxel WM, Buysse DJ, Matthews K. a, Kip KE, Strollo PJ, Hall M, … Reis SE (2010). Sleep symptoms predict the development of the metabolic syndrome. Sleep, 33(12), 1633–1640. 10.1210/jc.2011-3063 [PubMed: 21120125]

Zhang B, & Wing YK (2006). Sex differences in insomnia: A meta-analysis. Sleep, 29(1), 85–93. 10.1093/sleep/29.1.85 [PubMed: 16453985]

Table 1.

Sample characteristics.

	M (SD)	N (%)
Sex, female		94 (65)
Lifetime history of major depressive disorder		88 (61)
Follow-up time, years	19.1 (4.7)	
Age, years	59.9 (9.1)	
Apnea-hypopnea index	8.8 (12.6)	

Table 2.

Sleep characteristics at T1 and T2.

	PSG-assessed sleep, M (*SD*)		Subjectively-assessed sleep, M (*SD*)	
	T1	T2	T1	T2
Sleep duration, h	6.7 (0.8)	6.4 (0.9)[*]	6.7 (1.2)	7.0 (1.3)
Sleep latency, min	9.4 (9.7)	15.0 (15.9)[*]	21.6 (36.9)	16.3 (14.0)
Sleep efficiency, %	91.4 (6.3)	82.1 (9.6)[*]	89.7 (11.9)	87.8 (10.8)
Slow wave sleep, %	10.7 (8.2)	9.6 (8.2)		
Sleep quality[1]			0.9 (0.9)	0.9 (0.7)

Note.

[1] Recorded on a 0 (very good) to 3 (very poor) scale. PSG, polysomnography; T1, Time 1 data collected from 1982–1999; T2, Time 2 data collected from 2010–2014.

[*] denotes T2 sleep measure is significantly different ($p < .05$) from corresponding T1 value.

Table 3.

Prospective associations among T1 sleep and T2 metabolic syndrome.

	PSG-assessed sleep				Subjectively-assessed sleep			
	Unadjusted		Adjusted[1]		Unadjusted		Adjusted[1]	
	β	p	β	p	β	p	β	p
Sleep duration	−0.07	.41	−0.02	.83	−0.06	.51	0.01	.96
Sleep latency	0.09	.26	0.11	.16	0.29	.002	0.25	.009
Sleep efficiency	−0.05	.54	−0.02	.80	−0.08	.39	−0.08	.43
Slow wave sleep, %	−0.08	.36	−0.02	.77				
Sleep quality					0.08	.39	0.09	.46

Note.

[1]Adjusted for sex, lifetime depression history, age, log-transformed apnea-hypopnea index, and follow-up time. T1, Time 1 data collected from 1982–1999; T2, Time 2 data collected from 2010–2014; PSG, polysomnography.

Table 4.

Cross-sectional associations among T2 sleep and T2 metabolic syndrome.

	PSG-assessed sleep				Subjectively-assessed sleep			
	Unadjusted		Adjusted[1]		Unadjusted		Adjusted[1]	
	β	*p*	β	*p*	β	*p*	β	*p*
Sleep duration	−0.12	.17	−0.03	.73	−0.09	.27	−0.06	.48
Sleep latency	0.08	.32	0.08	.31	0.13	.13	0.10	.20
Sleep efficiency	−0.15	.08	−0.08	.32	−0.08	.32	−0.09	.27
Slow wave sleep, %	−0.09	.31	0.08	.33				
Sleep quality					−0.03	.73	−0.03	.71

Note.

[1] Adjusted for sex, lifetime depression history, age, log-transformed apnea-hypopnea index. T2, Time 2 data collected from 2010 to 2014; PSG, polysomnography.