Night-eating, Overweight/obesity and Sleep Quality

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Research Article

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Abstract

Purpose

Impaired sleep has been reported to be a consequence of overweight/obesity. However, sleep-disrupting behaviour that tend to coexist with overweight/obesity are also independent risk factors for impaired sleep such as night-eating, alcohol use, insufficient physical activity (PA), electronic device use and stress/affective distress. Thus, it is unclear whether night-eating and measures of body fatness will still predict sleep quality once concurrent behaviour and affective state are taken into account.

Methods

Online questionnaires asked participants about sleep quality, night-eating, alcohol use, electronic device use and stress/affective distress at T1 (baseline) and T2 (3-months later). Height, weight and waist and hip circumference was measured at T1 and objective physical activity (PA) was assessed over 24-hours (using actigraphy) in 161 participants at T1 and T2.

Results

At T1, night-eating was related to poorer subjective sleep quality, longer sleep onset latency, lower sleep efficacy, more sleep disturbances and daytime dysfunction and obesity category was related to daytime dysfunction after controlling demographics and covariates. At T2, high BMI predicted lower sleep efficacy after controlling T1 sleep components, demographics and covariates.

Conclusion

Night-eating and obesity category were associated with multiple impairments in sleep quality, but only high BMI predicted sleep quality at T2. Thus, night-eating and measures of body fatness predicted sleep quality components at T1 and T2 even after co-existing behaviour and emotional states were taken into account.

Level of Evidence

Level III, evidence obtained from well-designed cohort.

Introduction

Overweight/obesity (e.g. high body mass index [BMI]) has been shown to be associated with impaired sleep in adults [1, 2] using objective and subjective measures of sleep. Relative to normal weight controls, obese patients have significantly longer awake time after sleep onset, longer total awake time, and lower percentage of sleep time during the night (i.e. sleep efficiency), and they are sleepier during the day [3]. Overweight/obesity and high BMI have also been linked to shorter self-reported sleep duration but the measure was deemed to be a marker of emotional stress rather than a reflection of true sleep loss [4].

That is, obesity and impaired sleep tend to co-occur with emotional *stress* and *affective distress* (e.g. depression) [5] which can independently impact on sleep. Specifically, depression and anxiety are reported to be related to shorter sleep duration and poor quality sleep [6] and depression and insomnia are bi-directionally related [7, 8], at least when subjective assessments of sleep are studied. Thus, it is unclear if a person's co-existing obesity and/or their stress/distress will impact on sleep the most.

High BMI is also related to excessive daytime sleepiness (EDS) irrespective of whether the sleepiness is measured objectively or subjectively or independent of the presence of sleep apnoea [5]. In a large study of obesity, EDS, and sleep disordered breathing, high BMI, metabolic disturbances (i.e. diabetes, insulin resistance) and sleep apnoea all predicted disturbed or inadequate sleep, but so did low physical activity (PA) levels and depression [9]. Thus, a sedentary lifestyle and *insufficient PA* are associated with impaired sleep. Participants who do not meet PA guidelines (150 minutes of moderate-intensity, or 75 minutes of vigorous-intensity PA, or a combination of both) are more likely to report daytime sleepiness than those who do meet them [10] whereas regular PA reduces sleep onset latency (SOL), increases total sleep time, and effectively manages insomnia [11]. Overweight/obese people tend to report lower PA levels than normal weight people [12]. Thus, it is unclear which of the co-existing risk factors, PA or overweight/obesity interfere with sleep the most.

Similarly, excessive *night-eating* is associated with impairments in sleep including a subjective difficulty in initiating and maintaining sleep [13] and it can interfere with objective sleep, resulting in poor sleep efficacy (SE), long SOL and changes in rapid eye movement (REM) latency, amount of REM sleep and Stage 2 sleep, and sleep arousals [14]. In turn, the sleep impairments are linked to an increase in appetite, low leptin levels (i.e. hormone produced by adipose tissue that suppresses appetite) and high ghrelin levels (i.e. peptide that stimulates appetite)[15],[16]. Night-eating is also associated with overweight/obesity: BMI is highly correlated ($r = 0.72$) with Night-Eating Questionnaire (NEQ) score as is obesity category ($r = 0.77$) [17]. NEQ can detect the presence of Night Eating Syndrome (NES) and night-eating in people without NES (e.g. university students)[18], especially in high stress contexts [19]. Criteria for NES include: consuming > = 50% of daily energy intake after the evening meal, eating after waking from sleep and morning anorexia [20].

Further, heavy alcohol use can interfere with the induction and maintenance of sleep, results in changes in sleep architecture (e.g. short REM sleep) [21], and is related to weight gain and overweight/obesity [22]. Electronic device use can also interfere with sleep [23], including excessive texting (i.e. poor sleep quality) [23] and nocturnal use of electronic readers (i.e. longer SOL, more sleep disturbances [SD], more daytime dysfunction [DD]) [24], and it has been linked to overweight/ obesity [25]. That is, overweight/obese people are more likely to engage in certain sleep-disrupting behaviour (i.e. night-eating, low PA, alcohol use, nocturnal electronic device use) and they report greater stress/distress than normal-weight people [26]. Thus, it is unclear if obesity still predicts impaired sleep once concurrent behaviour and emotional state is taken into account.

Finally, demographics can confound the relationship between the risk factors and sleep quality. Older age is related to a difficulty in initiating and maintaining sleep [27], and it is correlated with BMI [28], less PA [29], less night-eating [30] and less electronic device use [31]. Further, women are more likely to experience poor sleep quality than men [32] and they are less likely to exercise than men [33], whereas male gender is strongly linked to obesity [34]. Further, less educational attainment and unemployment are linked to longer SOL, shorter sleep duration, and overweight/ obesity [35], whereas married people tend to report the fewest sleep complaints [36]. Thus, only by controlling for demographics can a study draw conclusions about the causes that drive impaired sleep quality.

Thus, in this study, we evaluate night-eating and measures of body fatness (i.e. BMI, waist-to-hip ratio [WHR], obesity category) as predictors of sleep quality. The Pittsburgh Sleep Quality Index (PSQI) [37] was used to measure sleep quality and components of sleep quality (see Methods for details). Further, we treated objective and subjective measures of PA, alcohol use, nocturnal use of electronic devices, and stress, anxiety and depression as covariates in the analyses. Consistent with the prior literature, we evaluated if NEQ score and measures of body fatness predicted total PSQI score and the PSQI sleep quality components at baseline (T1) and 3-months later (T2), after controlling for the effects of demographics and covariates. Specifically, we examined whether:

H1. More night-eating and body fatness measures at T1 will predict poorer sleep quality (high PSQI score) at T1 and T2; and,

H2. More night-eating will predict poor SSQ, longer SOL, shorter sleep duration, and lower SE; and obesity category and high BMI will predict poor SSQ and more SD at T1 and T2, after controlling for the effects of the aforementioned variables. Specific hypothesis for WHR were not examined as it has rarely been evaluated with regard to sleep quality.

Methods

Participants

The study was granted full approval by the Australian National University (ANU) Human Research Ethics Committee (protocol #2015/013). Participants were recruited via social networking websites (e.g., Facebook, Gumtree), ANU Psychology Research Participation Scheme, sleep centres (e.g. Canberra Sleep Clinic), local magazines (e.g. Canberra Weekly), and email snowballing. Criteria for study inclusion were: age of 18–65 years, resident in Canberra, Australia, and BMI ≥ 18.5 (BMI = weight (kg) / height (m) squared; World Health Organisation [WHO]) [38], that is, participants were normal weight, overweight or obese. An *a priori* power analysis using G*power (version 3.0.10) estimated that 139 people were required to detect a medium effect size (f^2 = 0.15) with alpha set at 0.05, power of 0.8 and using up to 10 predictors in the analyses.

165 people clicked on the URL embedded in the advertisement and 162 of them completed the T1 questionnaire (participation rate = 98%). All but one (n = 161) completed the actigraphy phase of the study of whom 67 were male and 94 (58%) were female, with a mean age of 26.8 years (range: 18–65 years, SD = 9.45). Nearly two-thirds were single (62%, N = 100), 24% (N = 38) were married and the rest were divorced/separated (N = 5), lived with someone (N = 12) or did not want to say (N = 6). More than one-half had completed an undergraduate (29%, N = 47) or postgraduate (24%, N = 39) degree and the rest had completed a diploma (N = 9) or Year 12 (39%, N = 63) or Year 10 or below at high school (N = 3). More than one-half were students (60%, N = 96), 20% worked full-time (N = 32) and the rest worked part-time/casual (16%, N = 26), completed home duties (N = 3), were unemployed (N = 1) or were permanently unable to work or ill (N = 2).

At follow-up 3-months later (T2), most participants (n = 155) completed the T2 questionnaire and second actigraphy phase, resulting in an attrition rate of 3.7% (6/161). Repeated-measures Analysis of Variance (ANOVA) showed that age did not vary significantly between T1 and T2 (M = 26.8 vs. 26.9 years, SD = 9.5 vs. 9.6, $F_{33, 121}$ = 0.98, p = 0.52). *Chi*-square tests showed that the sample at T1 and T2 did not differ in terms of gender (χ^2_1 = 0.49, p = 0.48), marital status (χ^2_5 = 4.42, p = 0.49), education (χ^2_4 = 3.05, p = 0.55) or employment (χ^2_6 = 2.57, p = 0.86).

Apparatus

Participants' height and weight were measured by the researcher (SE) to calculate their *BMI* which was used in the study analyses. *Physical activity* (total number of steps) was assessed over 24-hours at T1 and T2 using Actical® accelerometers (Phillips, Respironics) worn on the wrist. The devices tolerate normal daily activities such as showering. Devices recorded activity using a 1-minute epoch length. Data held on the devices was downloaded using ActiReader® communications interface software via a wireless link. They have been used to monitor physiological [39, 40] and behavioural parameters [41] in humans and they show moderate convergent validity with other objective measures of PA [42].

Materials

Participants were *screened* by asking them to provide their height (metres) and weight (kg) to calculate their BMI. Participants with self-reported BMI < 18.5 were redirected to the end of the survey and thanked for their time. People with BMI ≥ 18.5 were asked to complete the two questionnaires 3-months apart which asked about demographics (age, gender, education, marital status, employment), sleep quality, consumption behaviour (night-eating, alcohol use), use of electronic devices, PA, stress, anxiety and depression in the same order as specified below.

Sleep quality was assessed using the PSQI (19-item) which assesses subjective sleep quality (SSQ), sleep onset latency (SOL), sleep duration, habitual sleep efficiency (SE), sleep disturbances (SD), sleep medication use (SM) and daytime dysfunction (DD). Participants were asked to rate their agreement with items on 4-point scales ranging from 0 (*not in the past month*) to 3 (*three or four times a month*), with high scores indicating poor sleep. PSQI is a valid measure of sleep quality in clinical and non-clinical

samples: it distinguishes between good and poor sleepers and has moderate convergent validity with objective measures of sleep [37] and information collated in sleep diaries [43]. It is reported to have high internal consistency with Cronbach's α (CA) of 0.80 [44]. In this study, PSQI score had adequate internal consistency at T1 with CA of 0.72 and SD (9-item) had a CA of 0.63. Internal consistency was not calculated for other PSQI subscales as they were comprised of only one or two items.

Physical activity (PA) was assessed using the Rapid Assessment of PA (RAPA; 9-item) [45] which asks about PA intensity, using yes/no responses. Participants were provided with a definition of PA and examples of light, moderate and vigorous PA to assist in rating their current activity on two scales: PA and strength/flexibility. RAPA has adequate test-retest reliability (r = 0.65) [46] and is a valid measure of PA distinguishing between people who do/do not undertake moderate exercise [47]. In this study, RAPA had adequate test-retest reliability (r = 0.72).

PA also was assessed using the Global PA Questionnaire-Version 2 (GPAQv2) [48, 49] that collects PA information across three domains: occupational, transportation and leisure-time PA. GPAQ asks if people have engaged in moderate or vigorous-intensity PA in each activity domain. If they answered yes, they were asked how many days they engaged in the activities in a typical week and how long they spent doing the activities on a typical day. GPAQ data was cleaned and screened using GPAQ guidelines [50] and sub-scores were calculated for each activity domain based on the average number of days, hours and minutes spent doing the activity each week. A measure of total PA (GPAQ score) taking into account the intensity of the activities (by way of metabolic equivalents [METs]) was calculated. GPAQ score has adequate test-retest reliability (r = 0.67) [51] and is a valid measure of PA [49]. In this study, GPAQ score had adequate test-retest reliability (r = 0.64).

Night-eating was assessed using the Night-Eating Questionnaire (NEQ, 14-item) that detects the presence of Night Eating Syndrome (NES) in adults on four subscales: morning anorexia, evening hyperphagia, nocturnal ingestions and mood/sleep problems. Night-eating severity was assessed using 5-point scales ranging from 0 (*not at all*) to 4 (*very often*), with high scores indicating more night-eating. NEQ score ≥ 25 indicates possible NES. It has adequate internal consistency with CA of 0.7 and good convergent validity [52]. In this study, internal consistency of NEQ score was adequate with CA of 0.66 at T1.

Other behaviour that can potentially disrupt sleep was examined including alcohol use and electronic device use (including mobile phone & TV use). Participants were asked if they had consumed alcohol or engaged in the activity (yes/no), and if so, the amount (1–5 to > 20 drinks), frequency (every day, 3–4 times per week, twice a week, once a week, once a fortnight, once a month, <once a month, never) and time of day (morning, afternoon, evening, late night) of the use/behaviour. Participants listed all the electronic devices they had used in a typical day along with the number of hours each day and the time/s of day of use.

Stress/distress was assessed using the Depression Anxiety Stress Scales – 21 (DASS-21; 21-item) that assesses the presence and severity of depression, anxiety, and stress. Participants were asked to rate each item using 4-point scales ranging from 0 (*did not apply to me at all*) to 3 (*applied to me very*

much/most of the time), with high scores reflecting more distress. The scale has high internal consistency with CAs of 0.90 for stress, 0.84 for anxiety and 0.91 for depression [53] in non-clinical [54] and clinical samples [55]. In this study, its internal consistency was high with CAs of 0.88 (depression), 0.81 (anxiety) and 0.86 (stress).

Procedure

Interested individuals clicked on the URL in the advertisement, read the study information page, indicated their consent to participate and completed the T1 questionnaire that asked about sleep, consumption behaviour, PA and affective distress. Then they met the researcher (SE) who measured their height and weight to calculate their BMI. For each participant, the Actical® device was set up using the Actical® reader. Each person's ID number, age, gender, height and weight was entered, the device was fitted to the wrist (left or right) with a disposable sterile wristband and they wore the device continuously for 24-hours, after which they returned it to the researcher. Three-months later, they were each sent a URL and asked to complete the T2 questionnaire, after which they met the researcher to have the Actical® device fitted again. They wore the device for 24-hours and then returned it to the researcher. No participant removed the accelerometer during either of the two 24-hour monitoring periods.

Statistical Analyses

Routine statistical analyses were performed using the statistical analysis program SPSS (Version 24). ANOVA and *chi*-square tests evaluated if demographics and independent variables (IVs) differed between T1 and T2. Hierarchical multiple regression analysis examined the predictors of PSQI total and sub-scores (SSQ, SOL, sleep duration, SE, SD, SM, DD) at T1 and T2 after controlling demographics correlated with the dependent variables (DV) at step 1 and covariates correlated with the weight measures (e.g. low PA) at step 2. IVs (i.e. night-eating, BMI, WHR, obesity category) were entered at step 3 of the analysis. Due to the large number of IVs and covariates, only variables that were significantly correlated with the DVs were included in the analyses, with significance set at $p < 0.05$. GPAQ score was not included in some analyses due to multicollinearity between it and work PA.

Results

Means, standard deviations and correlations between the variables are presented in Table 1 and Tables 1a-1i (see supplementary file for Tables 1a-1i). At T1, 83% of participants ($N = 134$) had poor sleep quality (PSQI score ≥ 5).[37] One-half (54%, $N = 87$) had SOL > 30 minutes; 18% ($N = 29$) had sleep duration < 7 hours; 42% ($N = 68$) had SE < 85%; 98% had SD ($N = 157$); 83% had moderate DD ($N = 134$); and 17% ($N = 28$) used SM. Over one-half (56%, $N = 90$) were normal weight (BMI: 18.5–24.9), 22% ($N = 35$) were overweight (BMI: 25-29.9) and 22% ($N = 36$) were obese (BMI ≥ 30).[38] Using RAPA, 29% ($N = 47$) were sufficiently active (≥ 150-min moderate-intensity PA or 75-min vigorous-intensity PA/week) [56]. Using GPAQ, 70% ($N = 113$) met CDC requirements for optimal PA which best corresponded to actigraphy-derived PA data (80% had moderate PA) [57]. RAPA and GPAQ scores were moderately correlated ($r = .32$-$.47$) but poorly correlated with objective PA measures (see supplementary file). Some participants

(14%, *N* = 22) had substantial night-eating (NEQ score > 25) [58]. One-fifth (19%, *N* = 30) had severe depression (DASS-D > 21), 32% (*N* = 52) had severe anxiety (DASS-A > 15) and 19% (*N* = 30) had severe stress (DASS-S > 26) using DASS-21 cut-offs [53].

Table 1
Means and Standard Deviations of Key Study Variables at T1 and T2 (N = 161)

	T1 (N = 161)	**T2 (N = 155)**	**T1 vs T2**
	M (SD)	M(SD)	
Subjective sleep quality^	1.53 (.73)	1.18 (.68)	$F(1, 154) = 33.23$, $p < .001$**
Sleep Latency (min)	1.67 (1.02)	1.42 (.98)	$F(1, 154) = 9.66$, $p = .002$**
Sleep duration (hrs)^	.96 (.76)	.83 (.75)	$F(1, 154) = 4.56$, $p = .03$*
Habitual sleep efficacy^	.66 (.91)	.73 (1.02)	$F(1, 154) = .72$, $p = .40$
Sleep disturbances (number)	1.43 (.54)	1.21 (.48)	$F(1, 154) = 29.80$, $p < .001$**
Sleep medication use	.30 (.76)	.33 (.77)	$F(1, 154) = .05$, $p = .82$
Daytime dysfunction	1.45 (.77)	1.04 (.73)	$F(1, 154) = 33.74$, $p < .001$**
PSQI^	7.99 (3.42)	6.63 (3.28)	$F(1, 154) = 32.64$, $p < .001$**
NEQ	17.30 (6.67)	15.84 (6.52)	$F(1, 154) = 7.26$, $p = .01$*
Depression	13.18 (9.57)	11.69 (10.49)	$F(1, 154) = 5.17$, $p = .02$*
Anxiety	11.79 (8.92)	9.90 (8.32)	$F(1, 154) = 9.87$, $p = .002$**
Stress	15.35 (9.81)	12.77 (10.02)	$F(1, 154) = 17.54$, $p < .001$**
RAPA: Activity level	4.37 (1.87)	4.44 (1.92)	$F(1, 154) = .08$, $p = .78$
RAPA: Strength/Flexibility	1.05 (1.13)	1.12 (1.22)	$F(1, 154) = .59$, $p = .44$
GPAQ Total	2407.60 (3274.67)	2159.68 (2970.70)	$F(1, 154) = 1.32$, $p = .25$
Alcohol Intake (# drinks)	3.48 (2.69)	.59 (.99)	$F(1, 154) = 224.9$, $p < .001$**
TV time (hrs)	1.57 (.82)	2.67 (.92)	$F(1, 154) = 393$, $p = .001$**
Note. ^ Variable inversely coded. *p < .05. **p < .005			

Between T1 and T2, SSQ, SOL, sleep duration, SD, DD, night-eating, stress, anxiety and depression slightly improved, TV viewing increased, and alcohol use decreased.

Predictors of Total PSQI score

Cross-sectional predictors of sleep quality were examined at T1. At step 1, 6.1% of its variance was predicted, $F_{2,158} = 5.13$, $p = .007$. At step 2, an additional 28.8% of its variance was predicted increasing it to 34.9%, R^2_{change}: $F_{7,153} = 11.71$, $p < .001$. At step 3, an additional 6.1% of its variance was predicted increasing it to 41%, R^2_{change}: $F_{8,152} = 13.2$, $p < .001$. High PSQI score was related to more night-eating, see Table 2.

Table 2
Predictors of PSQI at T1 (N = 161) and *T2 (N = 155)*

	Variable	B	SE	B	t	p
T1 Step1						
	Education	-1.073	.554	− .157	-1.936	.055
	Marital status	− .964	.526	− .148	-1.832	.069
T1 Step2						
	Education	.007	.49	.001	.015	.988
	Marital status	-0.915	.454	− .141	-2.014	.046*
	Depression	.085	.033	.239	2.605	.01*
	Anxiety	.094	.039	.244	2.385	.018*
	Stress	.034	.038	.098	.9	.37
	Alcohol intake	.182	.086	.143	2.118	.036*
	Work PA	.000	.000	.082	1.184	.238
T1 Step3						
	Education	− .154	.47	− .022	− .327	.744
	Marital status	− .69	.438	− .106	-1.575	.117
	Depression	.083	.031	.233	2.655	.009*
	Anxiety	.073	.038	.191	1.94	.054
	Stress	.013	.037	.039	.369	.713
	Alcohol intake	.157	.082	.124	1.911	.058
	Work PA	.00	.00	.108	1.62	.107
	NEQ	.138	.035	.27	3.965	.001**
T2 Step1						
	PSQI T1	.591	.06	.624	9.869	.001**
T2 Step2						
	PSQI T1	.561	.073	.591	7.655	.001**
	Depression	.006	.031	.019	.207	.836

Note. *p < .05. **p < .005

	Variable	B	SE	B	t	p
	Anxiety	− .004	.036	− .01	− .101	.92
	Stress	.004	.035	.013	.122	.903
	Work PA	.000	.000	.096	1.425	.156
T2 Step3						
	PSQI T1	.542	.077	.572	7.073	.001**
	Depression	.006	.031	.018	.202	.84
	Anxiety	− .006	.036	− .016	− .159	.874
	Stress	.000	.035	− .001	− .005	.996
	Work PA	.000	.000	.103	1.523	.13
	NEQ	.032	.038	.063	.848	.398
Note. *p < .05. **p < .005						

Longitudinal predictors of PSQI score were examined at T2. At step 1, 38.9% of its variance was predicted, $F_{1,153}$ = 97.4, p < .001. At step 2, an additional 1% of its variance was predicted increasing it to 39.9%, R^2_{change}: $F_{5,149}$ = 19.75, p < .001. At step 3, an additional 2% of its variance was predicted increasing it to 40.1%, R^2_{change}: $F_{6,148}$ = 16.55, p < .001. After controlling T1 PSQI, demographics and covariates, no factors predicted PSQI score at T2, see Table 2.

Predictors of Subjective Sleep Quality

Predictors of SSQ were examined at T1. At step 1, 8.3% of its variance was predicted, $F_{3,157}$ = 4.72, p = .003. At step 2, an additional 20% of its variance was predicted increasing it to 28.3%, R^2_{change}: $F_{8,152}$ = 7.49, p < .001. At step 3, an additional 3.3% of its variance was predicted increasing it to 31.6%, R^2_{change}: $F_{10,150}$ = 6.93, p < .001. Poor SSQ was related to more night-eating, see Table 3.

Table 3

Predictors of Subjective Sleep quality and Sleep Onset Latency at T1 (N = 161) and *T2 (N = 155)*

Variable	B	SE	B	t	p
Subjective Sleep Quality					
T1 Step1					
Age	− .002	.008	− .02	− .186	.853
Education	− .26	.132	− .178	-1.973	.05*
Marital status	− .224	.132	− .161	-1.701	.091
T1 Step2					
Age	− .003	.008	− .037	− .372	.711
Education	− .064	.123	− .044	− .525	.601
Marital status	− .2	.12	− .144	-1.67	.097
Depression	.021	.007	.268	2.763	.006*
Anxiety	.007	.009	.083	.77	.442
Stress	.007	.009	.099	.865	.389
Work PA	.00	.00	.091	1.226	.222
Alcohol intake	.044	.02	.163	2.266	.025*
T1 Step3					
Age	− .001	.008	− .007	− .064	.949
Education	− .105	.124	− .072	− .853	.395
Marital status	− .17	.12	− .122	-1.423	.157
Depression	.02	.007	.256	2.663	.009*
Anxiety	.003	.009	.04	.374	.709
Stress	.006	.009	.074	.642	.522
Work PA	.00	.00	.101	1.367	.174
Alcohol intake	.04	.019	.145	2.052	.042*
WHR	− .711	.716	− .077	− .993	.322

Note. *$p < .05$. **$p < .005$

	Variable	B	SE	B	t	p
	NEQ	.019	.008	.172	2.302	.023*
T2 Step1						
	Subjective sleep Quality1	.416	.067	.448	6.196	.001**
T2 Step2						
	Subjective sleep Quality1	.347	.077	.374	4.52	.001**
	Depression	.008	.007	.11	1.072	.286
	Anxiety	− .006	.009	− .082	− .729	.467
	Stress	.004	.008	.06	.52	.604
	Alcohol intake	.04	.019	.157	2.134	.035*
	Steps	.00	.00	.121	1.638	.104
T2 Step3						
	Subjective sleep Quality1	.331	.077	.356	4.274	.001**
	Depression	.007	.007	.104	1.016	.311
	Anxiety	− .007	.009	− .096	− .858	.392
	Stress	.003	.008	.038	.325	.746
	Alcohol intake	.04	.019	.157	2.139	.034*
	Steps	.00	.00	.126	1.71	.089
	NEQ	.012	.009	.108	1.357	.177
Sleep Latency						
T1 Step1						
	Age	− .007	.01	− .063	− .655	.513
	Marital status	− .315	.186	− .163	-1.695	.092
T1 Step2						
	Age	.001	.01	.01	.103	.918
	Marital status	− .317	.18	− .164	-1.763	.08
	Depression	.005	.011	.051	.491	.624
	Anxiety	.016	.013	.14	1.201	.232
Note. *$p < .05$. **$p < .005$						

	Variable	B	SE	B	t	p
	Stress	.015	.013	.142	1.168	.245
	Alcohol intake	.064	.039	.171	1.64	.103
	Alcohol Frequency	.014	.115	.013	.123	.902
T1 Step3						
	Age	− .001	.01	− .007	− .079	.937
	Marital status	− .239	.175	− .124	-1.367	.174
	Depression	.005	.011	.044	.437	.663
	Anxiety	.01	.013	.088	.778	.438
	Stress	.01	.012	.095	.807	.421
	Alcohol intake	.07	.038	.187	1.858	.065
	Alcohol Frequency	− .039	.112	− .035	− .35	.727
	NEQ	.042	.012	.278	3.548	.001**
T2 Step1						
	Sleep onset latencyT1	.588	.061	.616	9.675	.001**
	Age	− .005	.007	− .046	− .676	.5
	Employment	− .127	.135	− .063	− .939	.349
T2 Step2						
	Sleep onset latencyT1	.567	.065	.593	8.7	.001**
	Age	− .005	.007	− .047	− .667	.506
	Employment	− .129	.137	− .064	− .941	.348
	Depression	.005	.009	.049	.565	.573
	Anxiety	.007	.011	.064	.653	.514
	Stress	− .006	.01	− .063	− .621	.536
	Alcohol intake	− .03	.032	− .082	− .932	.353
	Alcohol Frequency	.188	.093	.172	2.016	.046*
T2 Step3						
	Sleep onset latencyT1	.576	.067	.603	8.559	.001**
Note. *p < .05. **p < .005						

Variable	B	SE	B	t	p
Age	− .005	.007	− .046	− .652	.516
Employment	− .129	.137	− .064	− .938	.35
Depression	.005	.009	.053	.601	.549
Anxiety	.008	.011	.07	.709	.48
Stress	− .006	.01	− .057	− .552	.582
Alcohol intake	− .031	.032	− .086	− .971	.333
Alcohol Frequency	.193	.094	.177	2.059	.041*
NEQ	− .006	.011	− .042	− .578	.564
Note. *$p < .05$. **$p < .005$					

T1 predictors of SSQ were examined at T2. At step 1, 20.1% of its variance was predicted, $F_{1,153} = 38.39$, $p < .001$. At step 2, an additional 5.5% of its variance was predicted increasing it to 25.6%, R^2_{change}: $F_{6,148} = 8.49$, $p < .001$. At step 3, an additional 0.9% of its variance was predicted increasing it to 26.5%, R^2_{change}: $F_{7,147} = 7.58$, $p < .001$. Poor SSQ at T2 was not predicted by any IV after controlling T1 SSQ, demographics and covariates, see Table 3.

Predictors of Sleep Onset Latency

Predictors of SOL were examined at T1. At step 1, 4.3% of its variance was predicted, $F_{2,158} = 3.53$, $p = .032$. At step 2, an additional 11.6% of its variance was predicted increasing it to 15.9%, R^2_{change}: $F_{7,153} = 4.14$, $p < .001$. At step 3, an additional 6.5% of its variance was predicted increasing it to 22.4%, R^2_{change}: $F_{8,152} = 5.47$, $p < .001$. Long SOL was related to more night-eating, see Table 3.

Predictors of SOL were examined at T2. At step 1, 40.8% of its variance was predicted, $F_{3,151} = 34.65$, $p < .001$. At step 2, an additional 2.1% of its variance was predicted increasing it to 42.9%, R^2_{change}: $F_{8,146} = 13.7$, $p < .001$. At step 3, an additional 0.1% of its variance was predicted increasing it to 43%, R^2_{change}: $F_{9,145} = 12.16$, $p < .000$. No IVs predicted SOL at T1 after controlling T1 SOL, demographics and covariates, see Table 3.

Predictors of Sleep Duration

Predictors of sleep duration were examined at T1. Only one covariate was correlated with sleep duration. At step 1, 2.7% of its variance was predicted, $F_{1,159} = 4.48$, $p = .04$ indicating that shorter sleep duration was related to high anxiety, see Table 4.

Table 4

Predictors of Sleep Duration, Sleep Efficacy, and Sleep disturbances at T1 (N = 161) and T2 (N = 155)

	Variable	B	SE	B	t	p
Sleep Duration						
T1 Step1						
	Anxiety	.014	.007	.165	2.115	.04*
T2 Step1						
	Sleep duration T1	.432	.07	.435	6.134	.001**
	Age	.012	.006	.158	2.24	.027*
	Gender	.198	.108	.13	1.836	.068
T2 Step2		− .415	.267		-1.552	.123
	Sleep duration T1	.416	.071	.419	5.854	.001**
	Age	.013	.006	.164	2.319	.022*
	Gender	.184	.108	.121	1.707	.09
	NEQ	.012	.008	.104	1.452	.149
Sleep Efficacy						
T1 Step1						
	Education	− .131	.056	− .183	-2.35	.02*
T1 Step2						
	Education	− .069	.056	− .096	-1.219	.225
	Depression	.007	.01	.072	.686	.494
	Anxiety	.022	.012	.22	1.871	.063
	Stress	.001	.011	.013	.102	.919
	Work PA	.00	.00	.091	1.154	.25
T1 Step3						
	Education	− .071	.055	− .1	-1.293	.198
	Depression	.006	.01	.067	.647	.518
	Anxiety	.018	.012	.179	1.536	.127

Note. *$p < .05$. **$p < .005$

	Variable	B	SE	B	t	p
	Stress	− .002	.011	− .027	− .218	.828
	Work PA	.00	.00	.11	1.403	.163
	NEQ	.027	.011	.201	2.523	.013*
T2 Step1						
	Sleep EfficacyT1	.44	.081	.391	5.408	.001**
	Gender	.438	.149	.212	2.937	.004**
T2 Step2						
	Sleep EfficacyT1	.42	.081	.374	5.211	.001**
	Gender	.419	.146	.203	2.865	.005**
	Work PA	.00	.00	.064	.884	0.378
	Device use	− .219	.084	− .186	-2.606	.01*
T2 Step3						
	Sleep EfficacyT1	.409	.079	.364	5.194	.001**
	Gender	.405	.143	.196	2.84	.005**
	Work PA	.00	.00	.071	1.007	.316
	Device use	− .17	.084	− .144	-2.025	.045*
	BMI	.031	.01	.207	2.956	.004**
Sleep Disturbances						
T1 Step1						
	Employment	.18	.088	0.159	2.037	.043*
T1 Step2						
	Employment	.171	.08	.151	2.121	.036*
	Depression	.013	.006	.227	2.3	.023*
	Anxiety	.005	.007	.084	.768	.443
	Stress	.01	.006	.18	1.572	.118
T1 Step3						
	Employment	.149	.078	.132	1.899	.059
Note. *$p < .05$. **$p < .005$						

	Variable	B	SE	B	t	p
	Depression	.013	.005	.22	2.317	.022*
	Anxiety	.002	.006	.03	.282	.778
	Stress	.007	.006	.128	1.158	.249
	BMI	.011	.006	.135	1.939	.054
	NEQ	.021	.006	.256	3.498	.001**
T2 Step1						
	Sleep disturbancesT1	.466	.06	.53	7.737	.001**
T2 Step2						
	Sleep disturbancesT1	.445	.069	.506	6.478	.001**
	Depression	.002	.005	.034	.35	.727
	Anxiety	.002	.006	.044	.419	.676
	Stress	− .002	.006	− .048	− .42	.675
	Work PA	.00	.00	.099	1.362	.175
T2 Step3						
	Sleep disturbancesT1	.44	.071	.501	6.178	.001**
	Depression	.002	.005	.034	.344	.732
	Anxiety	.002	.006	.041	.385	.701
	Stress	− .003	.006	− .052	− .446	.656
	Work PA	.00	.00	.101	1.376	.171
	NEQ	.001	.006	.019	.244	.807
Note. *p < .05. **p < .005						

T1 predictors of sleep duration were examined at T2. At step 1, 24.8% of its variance was predicted, $F_{3,151}$ = 16.64, p < .001. At step 2, an additional 1.1% of its variance was predicted increasing it to 25.9%, R^2_{change}: $F_{4,150}$ = 13.1, p < .001. At step 3, no IVs predicted sleep duration at T2, but older age predicted shorter sleep duration at T2 at step 2, see Table 4.

Predictors of Habitual Sleep Efficacy

Predictors of SE were examined at T1. At step 1, 3.4% of its variance was predicted, $F_{1,159}$ = 5.52, p = .02. At step 2, an additional 9.2% of its variance was predicted increasing it to 12.6%, R^2_{change}: $F_{5,155}$ = 4.46, p

= .001. At step 3, an additional 3.4% of its variance was predicted increasing it to 16%, R^2_{change}: $F_{6,154}$ = 4.9, p < .001. Low SE was related to more night-eating, see Table 4.

T1 predictors of SE were examined at T2. At step 1, 21.9% of its variance was predicted, $F_{2,152}$ = 21.30, p < .001. At step 2, an additional 4.2% of its variance was predicted increasing it to 26.1%, R^2_{change}: $F_{4,150}$ = 13.24, p < .001. At step 3, an additional 4.1% of its variance was predicted increasing it to 30.2%, R^2_{change}: $F_{5,149}$ = 13.24, p < .001. Low SE was predicted by high BMI after controlling T1 SE, demographics and covariates, see Table 4.

Predictors of Sleep Disturbances

Predictors of SD were examined at T1. At step 1, 2.5% of its variance was predicted, $F_{1,159}$ = 4.15, p = .043. At step 2, an additional 19.2% of its variance was predicted increasing it to 21.7%, R^2_{change}: $F_{4,156}$ = 10.82, p < .001. At step 3, an additional 7% of its variance was predicted increasing it to 28.7%, R^2_{change}: $F_{6,154}$ = 10.36, p < .001. SD at T1 was related to more night-eating, see Table 4.

T1 predictors of SD were examined at T2. At step 1, 28.1% of its variance was predicted, $F_{1,153}$ = 59.87, p < .001. At step 2, an additional 1.2% of its variance was predicted increasing it to 29.3%, R^2_{change}: $F_{5,149}$ = 12.33, p < .001. At step 3, IVs did not increase its explained variance (R^2 = 29.3%), R^2_{change}: $F_{6,148}$ = 10.22, p < .001 and no IVs predicted it after controlling T1 SD, demographics and covariates, see Table 4.

Predictors of Sleep Medication Use

Predictors of SM were examined at T1. No demographics were correlated with it so the covariates were entered at step 1. At step 1, 13.5% of its variance was predicted, $F_{5,155}$ = 4.86, p < .001. At step 2, an additional 2.5% of its variance was predicted increasing it to 16%, R^2_{change}: $F_{7,153}$ = 4.16, p < .001. SM use was only related to less electronic device use, see Table 5.

Table 5

Predictors of Sleep Medication Use and Daytime Dysfunction at T1 (N = 161) and *T2 (N = 155)*

	Variable	B	SE	B	t	p
Sleep Medication Use						
T1 Step1						
	Depression	.007	.008	.091	.873	.384
	Anxiety	.013	.01	.158	1.376	.171
	Stress	.002	.01	.031	.25	.803
	Work PA	.00	.00	.13	1.638	.103
	Device use	− .156	.067	− .179	-2.339	.021*
T1 Step2						
	Depression	.007	.008	.092	.885	.378
	Anxiety	.012	.01	.141	1.22	.224
	Stress	.00	.01	− .003	− .022	.982
	Work PA	.00	.00	.138	1.74	.084
	Device use	− .136	.068	− .155	-1.991	.048*
	Obesity category	.193	.118	.126	1.642	.103
	NEQ	.012	.009	.104	1.308	.193
T2 Step1						
	use of sleeping medicationT1	.581	.065	.579	8.888	.001**
	Age	.01	.005	.121	1.859	.065
T2 Step2						
	use of sleeping medicationT1	.547	.069	.545	7.896	.001**
	Age	.01	.006	.12	1.663	.098
	Depression	.006	.005	.08	1.176	.242
	Work PA	.00	.00	.064	.937	.351
	Device use	− .008	.066	− .009	− .128	.898
T2 Step3						

Note. *$p < .05$. **$p < .005$

	Variable	B	SE	B	t	p
	use of sleeping medicationT1	.543	.07	.541	7.725	.001**
	Age	.009	.006	.107	1.381	.169
	Depression	.006	.006	.079	1.162	.247
	Work PA	.00	.00	.064	.942	.348
	Device use	− .007	.066	− .008	− .105	.916
	Obesity category	.049	.114	.032	.433	.666
Daytime Dysfunction						
T1 Step1						
	Age	− .007	.008	− .083	− .808	.42
	Education	− .148	.054	− .246	-2.732	.007*
	Marital status	− .067	.136	− .046	− .491	.624
T1 Step2						
	Age	− .006	.008	− .078	− .834	.405
	Education	− .064	.051	− .106	-1.259	.21
	Marital status	− .06	.124	− .041	− .485	.629
	Depression	.023	.008	.288	3.014	.003**
	Anxiety	.013	.009	.152	1.426	.156
	Stress	.003	.009	.044	.401	.689
	Alcohol intake	.047	.02	.164	2.338	.021*
T1 Step3						
	Age	− .001	.008	− .018	− .172	.864
	Education	− .083	.05	− .138	-1.656	.1
	Marital status	− .018	.123	− .012	− .144	.886
	Depression	.022	.008	.274	2.919	.004**
	Anxiety	.011	.009	.131	1.232	.22
	Stress	.002	.009	.026	.233	.816
	Alcohol intake	.039	.02	.138	1.984	.049*
Note. *$p < .05$. **$p < .005$						

	Variable	B	SE	B	t	p
	Obesity category	− .253	.123	− .163	-2.055	.042*
	WHR	.151	.763	.016	.198	.843
	NEQ	.019	.008	.164	2.232	.027*
T2 Step1						
	Daytime dysfunctionT1	.264	.078	.267	3.381	.001**
	Education	− .253	.115	− .174	-2.198	.029*
T2 Step2						
	Daytime dysfunctionT1	.134	.086	.136	1.566	.119
	Education	− .18	.117	− .124	-1.534	.127
	Depression	.006	.008	.08	.75	.454
	Anxiety	.004	.01	.048	.407	.685
	Stress	.014	.009	.193	1.578	.117
	Physical activity (RAPA)	.019	.037	.048	.511	.61
	Steps	.00	.00	.108	1.341	.182
	Work PA	.00	.00	.035	.439	.661
	Leisure-time PA	.00	.00	.036	.387	.7
T2 Step3						
	Daytime dysfunctionT1	.131	.087	.133	1.517	.131
	Education	− .181	.118	− .124	-1.534	.127
	Depression	.006	.008	.078	.734	.464
	Anxiety	.004	.01	.044	.37	.712
	Stress	.014	.009	.187	1.508	.134
	Physical activity (RAPA)	.02	.037	.051	.537	.592
	Steps	.00	.00	.108	1.339	.183
	Work PA	.00	.00	.037	.454	.65
	Leisure-time PA	.00	.00	.036	.385	.7
	NEQ	.003	.009	.025	.309	.758
Note. *$p < .05$. **$p < .005$						

Predictors of SM were examined at T2. At step 1, 35.9% of its variance was predicted, $F_{2,152} = 42.48$, $p < .001$. At step 2, an additional 1.1% of its variance was predicted increasing it to 37%, R^2_{change}: $F_{5,149} = 17.50$, $p < .001$. At step 3, obesity status predicted an additional 0.1% of its variance increasing it to 37.1%, R^2_{change}: $F_{6,148} = 14.53$, $p < .001$ after controlling T1 SM, demographics and covariates, see Table 5.

Predictors of Daytime Dysfunction

Predictors of DD were examined at T1. At step 1, 10.5% of its variance was predicted, $F_{3,157} = 6.13$, $p = .001$. At step 2, an additional 19.2% of its variance was predicted increasing it to 29.7%, R^2_{change}: $F_{7,153} = 9.23$, $p < .001$. At step 3, an additional 4.1% of its variance was predicted increasing it to 33.8%, R^2_{change}: $F_{10,150} = 7.65$, $p < .001$. More DD was related to more night-eating, see Table 5.

Predictors of DD were examined at T2. At step 1, 12.9% of its variance was predicted, $F_{2,152} = 11.23$, $p < .001$. At step 2, an additional 10.2% of its variance was predicted increasing it to 23.1%, R^2_{change}: $F_{9,145} = 4.84$, $p < .001$. At step 3, an additional 0.1% of its variance was predicted increasing it to 23.2%, R^2_{change}: $F_{10,144} = 4.34$, $p < .001$ but no IVs predicted it after controlling T1 DD, demographics and covariates, see Table 5.

Discussion

Night-eating and overweight/obesity are reported to be risk factors for poor sleep quality [5, 9]. However, overweight/obesity often co-exists with insufficient PA, use of alcohol and electronic devices and stress/distress, all of which can independently interfere with sleep [14, 24, 59–61]. Thus, it is difficult to disentangle the effects of night-eating and weight from the effects of co-existing behaviour and emotional states on sleep. Thus, we evaluated if night-eating (NEQ score) and measures of weight (BMI, WHR, obesity category) predicted components of sleep quality, after controlling demographics, concurrent behaviour (e.g. low PA) and emotional states.

More participants reported poor sleep quality in the past month than in other community samples (e.g. 20–35%) [62]. Most reported significant SD and moderate DD and one-half had poor SSQ, SOL > 30-min and SE < 85% but few slept < 7-hours or used SM. Over one-half were normal weight and one-fifth each were overweight and obese showing they were less overweight than the Australian adult population (i.e. 63% overweight/obese) [63]. A smaller proportion of them had substantial night-eating relative to other community samples [64], but most had at least mild affective distress (i.e. anxiety, depression), more than prevalence estimates for the Australian population [65]. Over 3-months (i.e. T1 to T2), their mean sleep quality, night-eating, stress, anxiety and depression slightly improved, alcohol intake decreased, and TV viewing increased. The small changes were not likely due to a change in demographics as only six people dropped out before T2. It is likely that the changes were due to normal fluctuations in symptoms

and behaviour that are linked to seasonal changes [66, 67] or changing life circumstances (e.g., life-event stress) [60, 68].

Hypothesis 1

examined night-eating and the weight measures as predictors of sleep quality (PSQI score) after controlling for PA (objective & subjective), use of alcohol and electronic devices, stress/affective distress, and demographics. At T1, more night-eating (but not weight measures) was related to high PSQI, somewhat supporting the hypothesis. However, no IVs predicted PSQI score at T2 after controlling T1 PSQI, demographics and covariates.

Hypothesis 2

examined night-eating and weight measures as predictors of sleep quality components (SSQ, SOL, sleep duration, SE, SD, DD, SM) at T1 and T2. At T1, night-eating was related to five of seven sleep components (i.e. SSQ, SOL, SE, SD, DD) and obesity category (i.e. normal-weight, overweight or obese) was related to more DD, but not other weight measures (i.e. BMI, WHR), somewhat supporting the hypothesis that night-eating will predict poor SSQ, long SOL, shorter sleep duration and lower SE and obesity category and high BMI will predict poor SSQ and SD at T1 and T2. Of the covariates, depression was related to SSQ, SD, and DD; anxiety was related to short sleep duration; alcohol use was related to poor SSQ and more DD; and electronic device use was related to less SM use. Stress and PA measures were unrelated to the sleep quality components.

At T2, night-eating did not predict the sleep quality components. High BMI predicted less SE but not the other weight measures (WHR, obesity category) after controlling for T1 sleep components, covariates and demographics. Of the covariates, more electronic device use predicted less SE and more alcohol use predicted poor SSQ and SOL. No IVs predicted SD, SM or sleep duration at T2. The lack of significant results at T2 were likely due to observed improvements in sleep quality and night-eating but it may also be due to testing multiple potential predictors in the analyses or fewer than expected overweight/obese people in the sample [69].

Our results are consistent with other research showing that impaired sleep was related to high BMI or obesity (i.e. shorter sleep duration, EDS) [4]‑[15, 70] and more night-eating (i.e. long SOL, less SE, sleep maintenance) [13, 14, 18]‑[14], and that showing it was linked to alcohol [68] and electronic device use [71] and anxiety [72] and depression [7, 8]. We did not find any association between sleep quality and PA [73] or stress, as have other studies [74]. In our published study using the same participants as in this study, we found [75] that night-eating and the same weight measures did not predict *objective* sleep (SOL, sleep duration, awake time, SE) whereas alcohol use, TV use, objective and subjective PA and depression did. In the current study, we also found that alcohol and electronic device use and depression predicted the sleep quality components at T1 and/or T2. Taken together, the results suggest that subjective reporting of sleep does not necessarily provide reliable information about objective sleep [76], and that a different profile of behaviour may interfere with objective and subjective sleep. That is, night-eating (and

overweight/obesity) may interfere with subjective sleep whereas other sleep-disrupting behaviour (e.g. alcohol use) may interfere with objective and subjective sleep.

Nonetheless, our results need to be interpreted in light of several *study limitations*. First, the sample was relatively small but an *a priori* power analysis showed that it was sufficient to detect medium effect sizes. Second, sleep was assessed via self-report scale rather than objective sleep tests, which can result in over or under-reporting [77], but PSQI has good psychometric properties [37]. Third, the NEQ had barely adequate internal consistency suggesting the results for night-eating should be interpreted with caution. Fourth, RAPA indicated that a higher proportion of people exercised to an adequate degree than GPAQ score (29% vs 70%). Finally, the use of an online survey platform to deliver questionnaires can attract younger and better educated adults as participants [78] which may reduce the generalisability of the results to older and less educated people.

Conclusions

Night-eating and body fatness (i.e. BMI, WHR, obesity category) were examined as predictors of sleep quality at two time-points 3-months apart (T1 & T2) in a community sample that included normal-weight and overweight/obese adults. Covariates included objective and subjective PA, alcohol use, electronic device use and stress/distress symptoms, all of which can independently impact on sleep and co-exist with overweight/obesity. Results showed that night-eating (but not weight measures) was related to high PSQI at T1 but no factors predicted it at T2. Night-eating was also related to most PSQI components (SSQ, SOL, SE, SD, DD), obesity category was related to high DD at T1 and high BMI predicted SE at T2. Results suggest that a person's body fatness (and possibly nocturnal eating behaviour) may independently contribute to impaired sleep quality, at least in the short-term, even after taking into account the effects of co-existing sleep-disrupting behaviour and stress/distress.

Abbreviations

BMI: body mass index

WHR: waist to hip ratio

REM: rapid eye movement

NES: night eating syndrome

NEQ: Night-Eating Questionnaire

SSQ: subjective sleep quality

SOL: sleep onset latency

SE: sleep efficiency

SD: sleep disturbances

SM: sleep medication use

DD: daytime dysfunction

Declarations

Declaration of interest statement

All authors declare that they have no conflict of interest. This manuscript has not been previously published, and is not presently under consideration by another journal, and will not be submitted to another journal before a final editorial decision from Eating and Weight Disorders is rendered. No funds, grants, or other support was received.

Data availability statement

The data that support the findings of this study are available from the corresponding author, [S.E], upon reasonable request.

Ethics approval

The questionnaire and methodology for this study was approved by the Human Research Ethics committee of the Australian National University (Ethics approval number: #2015/013.

Consent to participate and Consent to publish

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