Direct and Indirect Relationships Between Impaired Sleep, Obesity Risk Factors and Overweight and Obesity

Shireen Walid Eid (shireen.eid@anu.edu.au)
Australian National University https://orcid.org/0000-0001-8216-6415
Rhonda Francis Brown
Australian National University
Carl Laird Birmingham
The University of British Columbia
Shane k. Maloney
The University of Western Australia

Research Article

Keywords: Body Mass Index, Waist to Hip Ratio, overweight, obesity, sleep, night eating.

Posted Date: January 6th, 2022

DOI: https://doi.org/10.21203/rs.3.rs-1151859/v1

Abstract

Purpose

The relationship between impaired sleep and overweight/obesity may be explained by sleep-disrupting behaviour that are practised by overweight people (e.g. night-eating, insufficient physical activity [PA], electronic device use) and stress/affective distress. Thus, we evaluated whether sleep parameters predicted overweight/obesity after taking into account the behaviour and affective state.

Methods

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

Results

At T1, high body mass index (BMI)/waist-to-hip ratio (WHR) and obesity category were together related to more sleep disturbances (subjective) and longer awake time (objective), after controlling covariates (e.g. watching TV) and demographics (e.g. older age, male gender). At T2, high WHR was predicted by older age and male gender after controlling T1 WHR, demographics and covariates. Mediational analyses showed that sleep disturbances mediated nocturnal indigestion (NI) to BMI, poor subjective sleep quality mediated NI to WHR and high daytime dysfunction mediated NI to obesity category relationships.

Conclusion

More time spent awake during the night (experienced as more sleep disturbances) was related to overweight/obesity indices even after taking into account other obesity risk factors (e.g. night-eating, insufficient PA, affect) and demographics. Mediational results suggest that NI parsimoniously explained the impaired sleep - overweight/obesity relationship.

Level of Evidence: Level III, evidence obtained from well-designed cohort.

Introduction

Overweight/obesity (e.g. high body mass index [BMI]) in adults is reported to be a consequence of impaired sleep as indexed by objective and subjective sleep methods [1–3]. Relative to normal-weight controls, obese patients are reported to spend more time awake after sleep onset, longer total awake time, and lower percentage of sleep time during the night (i.e. lower sleep efficacy [SE]); and, as a result, they report being sleepier during the day [4]. Even in the general population, high BMI is associated with shorter self-reported sleep duration [5]. However, it is unclear exactly what causes these sleep disturbances and how they contribute to weight gain in overweight/obese individuals.

Recent research has suggested that *night-eating* may indirectly contribute to weight gain via impaired sleep [6, 7]: It is linked to objective and subjective impairments in sleep (e.g. delayed sleep onset latency [SOL]) [8] and it is more common in obese people than normal-weight people [9], and, BMI (r=0.72) and obesity category (r=0.77) are highly correlated with Night-Eating Questionnaire (NEQ) score [10]. NEQ detects the presence of Night Eating Syndrome (NES) and night-eating symptoms in people without a NES diagnosis (e.g. university students) [11]. Criteria for NES include: consuming >=50% of daily energy intake after the evening meal, eating after waking from sleep (i.e. nocturnal ingestions; NI), and morning anorexia [12], which can result in weight gain over time [13], especially if the food is rich in carbohydrates and fats (e.g. pre-packaged foods) [14, 15].

However, other obesity risk factors may also indirectly contribute to weight gain via disrupted sleep including *insufficient physical activity (PA), electronic device use* and *stress/affective distress* [16–18]: Overweight/obese people tend to engage in less PA than normal and under-weight people [19] and less PA (or a sedentary lifestyle) can impair sleep [18] potentially contributing to weight gain [20]. Similarly, electronic device use (e.g., watching TV) is related to body fatness (e.g. higher percent body fat as indexed by skinfold methods) in women [21] and it can impair sleep [16]. Finally, sleep impairments are common in affectively distressed (e.g. anxiety, depression) people [17, 22] as is obesity, as highlighted in a review of obesity indices (i.e. BMI, waist circumference, waist-to-hip-ratio (WHR), adiposity, intra-abdominal fat) [23].

Thus, several obesity risk factors may indirectly contribute to weight gain via their effects on sleep including night-eating, insufficient PA, electronic device use and stress/affective distress. However, some risk factors are highly correlated with each other; for example, time spent sitting is correlated with time spent watching TV in physically inactive men [24]. As a result, it is unclear if sleep or the risk factor/s will contribute most to obesity and if the effects are direct and/or indirect. Thus, in this study, we examined objective and subjective measures of sleep, obesity risk factors (i.e. night-eating, PA & electronic device use) and stress/affective distress as predictors of BMI, WHR and obesity category (i.e. overweight/obese vs. normal weight) at baseline (T1) and 3-months later (T2), after controlling demographics. Sleep was indexed objectively (i.e. SOL, actual sleep time, total sleep duration, awake time, sleep efficacy) and subjectively (i.e. Pittsburgh Sleep Quality Index [PSQI] [25]: subjective sleep quality [SSQ], SOL, sleep duration, sleep efficacy [SE], sleep disturbances [SD], sleep medication use [SM], daytime dysfunction [DD]); and, PA was examined objectively (i.e. actigraphy; total number of steps) and subjectively (Global PA Questionnaire [GPAQ], Rapid Assessment of PA) [RAPA] [26, 27]. Objective sleep and PA were examined for 24-hours at T1 and T2 and obesity risk factors were assessed by questionnaire at T1 and T2. Finally, since the relationship between sleep and weight can be confounded by age (i.e. obesity risk varies with age) [28] and gender (i.e. insomnia is more common in women [29] & male gender is linked to obesity [e.g. high BMI]) [30], we controlled them in the planned study analyses.

Due to the lack of comparison studies, we did not advance any hypotheses about the relative predictive strengths of (direct) relationships. Instead, we broadly expected that all of the sleep parameters and obesity risk factors will predict the body fatness measures. However, we did expect the mediational

results (i.e. indirect relationships) to support the assertion that obesity risk factors (e.g. night-eating) and impaired sleep can together contribute to weight gain [7]. Consistent with the aforementioned findings, we therefore advanced the following hypotheses:

H1. Impaired sleep (e.g. more awake time, short sleep duration, sleep disturbances, daytime sleepiness) will predict higher BMI; night-eating will predict higher BMI and obesity category; obesity risk factors (i.e. low PA, TV & electronic device use) will predict higher BMI and obesity category; and stress/affective distress (i.e. stress, anxiety & depression) will predict higher BMI, WHR and obesity category at T1 and T2; and,

H2. Impaired sleep will mediate the relationships between obesity risk factors (e.g. night-eating, NI; low PA; watching TV; electronic device use) and stress/affective distress (i.e. stress, anxiety & depression) to the body fatness measures.

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 Student 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 of Canberra, Australia, and BMI ≥ 18.5 (BMI=weight (kg) / height (m) squared; World Health Organisation [WHO]) [31]; thus, 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 in the online advertisement and 162 completed the T1 questionnaire (participation rate = 98%). All but one (*n* = 161) completed the actigraphy study phase 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 of them 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). Over one-half had completed an undergraduate (29%, *N* = 47) or postgraduate (24%, *N* = 39) degree and the rest had a diploma (*N* = 9) or completed Year 12 (39%, *N* = 63) or Year 10 or below at high school (*N* = 3). More than one-half were college students (60%, *N* = 96), 20% worked full-time (*N* = 32) and the rest completed part-time/casual work (16%, *N* = 26), home duties (*N* = 3) or were unemployed (*N* = 1), permanently unable to work or ill (*N* = 2).

After 3-months (at T2), most participants (*n* = 155) completed the T2 questionnaire and actigraphy phase (i.e. attrition rate = 3.7% [6/161]). Repeated-measures Analysis of Variance showed that participants'

mean age did not vary 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 the T1 and T2 samples did not differ in terms of gender ($\chi^2_1 = 0.49$, $p = 0.48$), marital status ($\chi^2_5 = 4.42$, $p = 0.49$), education ($\chi^2_4 = 3.05$, $p = 0.55$) or employment ($\chi^2_6 = 2.57$, $p = 0.86$).

Apparatus

Participants' height and weight were measured to calculate *BMI* and waist and hip circumferences were measured to calculate *WHR* (i.e. dependent variables [DV]). *Physical activity* (total number of steps) over 24-hours at T1 and T2 and *sleep* were assessed using Actical® movement data. The devices can tolerate normal daily activities such as showering. Devices recorded sleep and PA using a 1-minute epoch length. Data held on the devices was downloaded using ActiReader® communications interface software via a wireless link. The devices have been used to monitor physiological [32, 33] and behavioural parameters [34] such as sleep and PA in humans. Actiware® software (version 6.09) estimated SOL (minutes); actual sleep time (minutes; from sleep onset to final morning waking) on all 'sleep' epochs, excluding epochs scored as 'wake' and 'interrupted sleep'), total sleep duration (minutes; from sleep onset to final morning awaking; computed as sum of actual sleep time and interrupted sleep time); awake time (minutes; time spent awake after waking at night); and, sleep efficiency (actual sleep time divided by total time spent in bed). The reliability and validity of actigraphy data in assessing PA and sleep is well established and measures of sleep are highly correlated with polysomnography data (r=.88-.99) [[35, 36]] and moderately with other PA measures [37].

Materials

Participants were *screened* by asking them to provide their height (metres) and weight (kg) to calculate BMI. Participants with a 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 two questionnaires 3-months apart which asked about age, gender, sleep quality, sleep-disruptive behaviour (i.e. night-eating, PA & electronic device use) and stress/affective distress, in the same order as is specified below.

Sleep quality was assessed using the PSQI (19-item) which asks about SSQ, SOL, sleep duration, SE, SD, SM and DD. Participants were asked to rate their agreement with items using 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 vs. poor sleepers and it has moderate convergent validity with objective sleep measures [25] and information collated from sleep diaries [38]. PSQI score has high internal consistency with Cronbach's α (CA) of 0.80 [39]. In this study, it had adequate internal consistency at T1 with a CA of 0.72 and SD (9-item) had a CA of 0.63. Internal consistency of other PSQI subscales was not calculated as they were comprised of one or two scale items.

Night-eating was assessed using the NEQ (14-item). It detects the presence of NES in adults over four subscales: morning anorexia, evening hyperphagia, nocturnal ingestions (NI) and mood/sleep problems.

Night-eating severity was assessed via 5-point scales ranging from 0 (*not at all*) to 4 (*very often*), with high scores indicating more night-eating. NEQ score ≥ 25 suggests the possible presence of NES. NEQ has adequate internal consistency with a CA of 0.7 and good convergent validity [40]. In this study, internal consistency of NEQ score was adequate with CA of 0.66 at T1.

Physical activity (PA) was assessed using the RAPA (9-item) [41] which asks about PA intensity using yes/no responses. Participants are provided with a definition of PA and examples of light, moderate and vigorous PA to assist them in rating their current activity on two scales: PA and strength/flexibility. RAPA has adequate test-retest reliability (r = 0.65) [42] and is a valid measure of PA that distinguishes between people who do/do not undertake moderate exercise [43]. In this study, RAPA had adequate test-retest reliability (r = 0.72).

PA was assessed using the GPAQ-Version 2 [26, 27] to collect 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 them on a typical day. GPAQ data was cleaned and screened using GPAQ guidelines [44] 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) was calculated. GPAQ score has adequate test-retest reliability (r = 0.67) [45] and it is a valid measure of PA [27]. In this study, GPAQ score had adequate test-retest reliability (r = 0.64).

Electronic device use (i.e. computer, mobile phone & TV use) was examined. Participants were asked if they had engaged in the activity (yes/no), and if so, the 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. They listed all the electronic devices they had used in a typical day along with the number of hours each day and time/s of day of the use.

Stress/distress was assessed using the Depression Anxiety Stress Scales -21 (DASS-21; 21-item) to assess the presence and severity of depression, anxiety and stress symptoms. Participants were asked to rate the items 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 [46] in non-clinical [47] and clinical samples [48]. 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 asking about their sleep quality, sleep-disruptive behaviour and stress/affective distress. Then they met with the researcher (SE) who measured their height, weight, and waist and hip circumferences. Actical® devices were set up for each participant

using the Actical® reader. Their ID number, age, gender, height and weight was entered, devices were 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 sent a URL to complete the T2 questionnaire and then they met the researcher again to have the Actical® device fitted. They wore the device for 24-hours and afterwards returned it to the researcher. No participants removed the accelerometer during either of the two 24-hour monitoring periods.

Statistical Analyses

Routine statistical analyses were computed using the statistical analysis program SPSS (Version 24). ANOVA and *chi*-square tests examined if demographics, independent variables (IVs) and DVs differed at T1 and T2. Hierarchical multiple regression and logistic regression analyses examined the predictors of body fatness measures at T1 and T2 after controlling demographics (age, gender) at step 1 of the analyses. If sleep-disruptive behaviour and stress/distress were correlated with the body fatness measures they were entered at step 2 and the IVs (i.e. objective & subjective sleep) were entered at step 3 if they were correlated with the DVs.

Mediational analyses using the PROCESS v4 (Hayes, 2022) [49] macro for SPSS examined indirect relationships between sleep, sleep-disruptive behaviour, stress/distress and body fatness measures (DVs). Sleep parameters were conceptualised as mediators and sleep-disruptive behaviour and affect were IVs. Consistent with Baron and Kenny's conceptualization [50], mediation was deemed to be present if the relationship between an IV and mediator was significant as were the relationships between the mediator and DV and IV and DV; and when a mediator is included, if it results in the IV – DV relationship becoming non-significant, it indicates full mediation whereas if it is still significant then partial mediation is demonstrated.

Significance was set at $p < 0.05$. GPAQ score was not included in some analyses due to multi-collinearity between it and RAPA-work PA score.

Results

Correlates of BMI were examined at T1. Age was correlated with it so it was controlled at step 1 of the analysis. 23.3% of variance in BMI was predicted by age at step 1 ($F_{1, 159}$=48.26, p<.001). At step 2, covariates predicted an additional 4.1% of its variance increasing it to 27.4% (R^2_{change}: $F_{4, 156}$=14.7, p<.001). At step 3, an additional 1.9% of its variance was predicted by sleep parameters increasing it to 29.3%, R^2_{change}: $F_{5, 155}$=12.82, p<.001. At step 3, high BMI was related to more SD (p<.05) after controlling demographics and covariates (i.e. strength/flexibility, nocturnal TV & device use), see Table 2.

Risk factors for high BMI were examined at T2. T1 BMI and age were correlated with it at T2 and were entered at step 1 of the analysis. At step 1, they predicted 98.1% of the variance in BMI ($F_{2, 152}$=3950, p<.001). At step 2 ($F_{5, 149}$=1554 p<.001) and step 3 ($F_{6, 148}$=1287, p<.001), the IVs and covariates did not

increase its explained variance (R^2=98.1%) and so no IVs predicted it after controlling T1 BMI, age and covariates (strength/flexibility, nocturnal TV & device use), see Table 3.

Correlates of WHR were examined at T1. Age and gender were correlated with it and so were entered at step 1, covariates correlated with it were entered at step 2 and IVs were entered at step 3. Results indicated that 41.3% of variance in WHR was predicted at step 1 ($F_{2, 158}$= 55.7, *p*<.001). At step 2, covariates predicted an additional 1% of its variance increasing it to 42.3% (R^2_{change}: $F_{4, 156}$=28.6, *p*<.001). At step 3, an additional 0.9% of its variance was predicted increasing it to 43.2%, R^2_{change}: $F_{6, 154}$=19.5, *p*<.001. WHR was related to male gender and older age (*p*-value <.005), but not measures of sleep, see Table 4.

Risk factors for high WHR were examined at T2. WHR T1, age and gender were entered at step 1, covariates at step 2 and IVs at step 3, if they were correlated with WHR at T2. At step 1, they predicted 81.1% of its variance ($F_{3, 151}$ =216.5, *p*<.001). At step 2, covariates predicted an additional 0.1% of its variance increasing it to 81.2% (R^2_{change}: $F_{4, 150}$=162.2, *p*<.001). At step 3, an additional 0.1% of its variance was predicted increasing it to 81.3%, R^2_{change}: $F_{5, 149}$=130.0, *p*<.001. Higher WHR at T2 was predicted by male gender and older age (*p*<.005) after controlling for T1 WHR, see Table 5.

Binary logistic regression examined predictors of obesity category (i.e. overweight/obesity vs. normal weight) at T1. Age was included at step 1, covariates were entered at step 2 and IVs were entered at step 3 of the analysis. At step 1, age predicted 17% (Cox & Snell R^2) of its variance, $\chi^2(1)$ (*N*=161)=30.07, *p*<.001; model correctly classified 73.3% of cases. At step 2, covariates predicted an additional 1% of its variance increasing it to 18%, $\chi^2(4)$ (*N*=161)= 31.9, *p*<.001; model correctly classified 73.9% of cases. At step 3 ($\chi^2(6)$ (*N*=161)=47.3, *p*<.001), IVs predicted an additional 7.4% of its variance increasing it to 25.4%; model correctly classified 73.9% of cases. Only longer awake time during sleep was related to obesity category after controlling age and the covariates, see Table 6. Older age was related to 11% increased odds of being overweight/obese whereas awake time was linked to 2% increased odds of overweight/obesity.

Binary logistic regression analysis examined risk factors for obesity category at T2. T1 obesity category and age were entered at step 1, covariates were entered at step 2 and IVs correlated with it were entered at step 3 of the analysis. At step 1, the factors predicted 71.2% (Cox & Snell R^2) of its variance, $\chi^2(2)$ (*N*=155)= 192.7, *p*<.001; model correctly classified 98.7% of cases. At step 2, covariates predicted an additional 0.9% of its variance increasing it to 72.1%, $\chi^2(4)$ (*N*=155)= 197.9, *p*<.001; model correctly classified 98.1% of cases. At step 3 ($\chi^2(6)$ (*N*=155)=209.4, *p*<.001), IVs predicted an additional 2% of its variance increasing it to 74.1% ; model correctly classified all cases, see Table 7. None of the IVs predicted T2 obesity category after controlling T1 obesity category, demographics and covariates.

Mediational analyses examined whether impaired sleep mediated the relationships between the measures of behaviour and body fatness. Of all the potential IVs, only NEQ-nocturnal ingestions (NI) met

criterion as an IV and only three sleep symptoms (i.e. SSQ, SD, DD) met criteria as mediators. That is, SSQ, SD and DD were correlated with NEQ-NI and in turn they were correlated with at least one body fatness measure at T1, see correlations in the supplementary file. Thus, only they were tested as potential mediators. Results showed that high NI was related to high SD, B = .07, SE = .02, 95% CI [.03,.10], β = .28, p < .001, and high SD predicted high BMI, B = 2.11, SE = 1.02, 95%CI[.11,4.12], β = .17, p = .04, supporting *Hypothesis 2*. NI no longer predicted BMI after controlling for the effects of the mediator, SD, B = .10, SE = .24, 95%CI[-.39,.58], β = .03, p = .70, thus, SD fully mediated the NI-BMI relationship accounting for about 3.3% of the variance in BMI (R^2 = .033). Indirect effects were tested using a percentile bootstrap estimation approach with 5,000 samples and the PROCESS macro-Version 4 (Hayes, 2022). Results showed that the indirect coefficient was significant, B = .14, SE = .08, 95% CI [.02,.32], standardized β = .05 suggesting that more NI may have contributed to high BMI via more SD in some participants.

Mediational analysis examined if SSQ mediated the NI - WHR relationship. Results showed that high NI was related to poor SSQ, B = .08, SE = .03, 95%CI[.03,.13], β = .24, p = .002, and SSQ predicted high WHR, B = -.02, SE = .01, 95%CI[-.04,-.002], β = -.17, p = .03, supporting *Hypothesis 2*. NI no longer predicted WHR after entering the mediator, SSQ, B = -.002, SE = -.003, 95%CI[-.008,.004], β = -.05, p = .50, suggesting that poor SSQ fully mediated the NI – WHR relationship, predicting about 1% of the variance in BMI (R^2 = .01). Indirect effects were tested using a percentile bootstrap estimation approach with 5,000 samples and the PROCESS macro-Version 4 (Hayes, 2022). Results showed that the indirect coefficient was significant, B = -.002, SE = .001, 95%CI [-.003, -.0001], standardized β = .04, suggesting that more NI may have contributed to greater WHR via a drop in SSQ.

Mediational analysis examined if DD mediated the NI - obesity category relationship. Results showed that high NI was related to high DD, B = .07, SE = .03, 95%CI[.02,.12], p = .01, and DD predicted obesity category, B = -.63, SE = .23, 95%CI[-1.08,-.17], p = .007, supporting *Hypothesis 2*. NI no longer predicted obesity category after entering the mediator DD, B = .14, SE = .08, 95%CI[-.002,.30], p = .053, suggesting that high DD fully mediated the high NI – obesity category relationship, predicting about 5.9% of its variance (CoxSnell R^2 = .059). Indirect effects were tested using a percentile bootstrap estimation approach with 5,000 samples and the PROCESS macro-Version 4 (Hayes, 2022). Results showed that the indirect coefficient was significant, B = -.04, SE = .03, 95%CI [-.1, -.01] suggesting that more NI may have contributed to an increase in DD which in turn may have contributed to more participants being categorised as overweight/obese.

Discussion

In this study, we examined if objective and subjective sleep parameters predicted measures of body fatness (i.e. BMI, WHR, obesity category) after taking into account the effects of obesity risk factors that tend to co-occur in overweight/obese people (i.e. night-eating, low PA, electronic device use, stress/affective distress) [4, 9, 16–18] and demographics (i.e. age, gender). Further, we explored whether indirect mediational paths could parsimoniously explain the tendency of obesity risk factors to contribute to impaired sleep and then weight gain [1–3], in addition to their direct effects on weight.

In the sample, more than one-half was normal weight and one-fifth each were overweight or obese, being less overweight than Australian adults (63% overweight/obese) [51]. Their mean BMI and obesity category did not change significantly over 3-months. Most of them reported poor sleep quality (75.8%) in the past month more often than in other community samples (e.g. 20-35%) [52]. 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. Few participants (13.7%) reported substantial night-eating compared to other community samples [53], but more of them reported at least mild affective distress (i.e. anxiety, depression) relative to Australian prevalence estimates [54]. Over 3-months (i.e. T1 to T2), their mean sleep quality, night-eating, stress, anxiety and depression slightly improved and TV viewing increased. These small changes were unlikely due to a change in demographics as only six people dropped out of the study before T2. Instead, they are more likely due to normal fluctuations in symptoms and behaviour that are linked to seasonal changes [55, 56] or changing life circumstances (e.g., life-event stress) [57].

Regarding *hypothesis 1*, no variables predicted BMI and obesity category at T2 and only older age and male gender predicted WHR at T2 due to non-significant changes in the body fatness measures over 3-months. At T1, the variables predicted 29% of the variance in BMI, 43% of variance in WHR, and 25% of variance in obesity category. Specifically, high BMI was related to more SD and obesity category was related to longer awake time, but WHR was unrelated to sleep. Results are consistent with prior research showing that obesity indices are related to impaired sleep (e.g. longer awake time after sleep onset, longer total awake time) [58, 59], high BMI is related to PSQI score [60], and obese patients experience longer awake time during sleep [5]. However, we did not find that BMI, WHR and obesity category were related to night-eating [9], depression [23], anxiety, stress [61] or subjective (RAPA, GPAQ) or objective (number of steps) measures of PA. Non-significant results are likely due to the: (a) testing of multiple potential predictors in the same analyses; (b) relatively short 3-month interval between T1 and T2 in which body fatness could change; (c) improved sleep, affect and night-eating between T1 and T2; (d) less than expected proportion of overweight/obese people in the sample (44.1%, *N*=71) (AIHW, 2016); and/or, (e) substantial variance in the T2 obesity measures being predicted by T1 measures.

Taken together, the results show that objective sleep impairment (i.e. longer awake time during the night) - experienced as more self-reported sleep disturbances - explained significant variance in the body fatness measures even after taking into account the effects of other concurrent obesity risk factors (i.e. covariates; e.g. night-eating) [16–18] and demographics, although we only found cross-sectional evidence of sleep-weight relationships. Further, the nature of the sleep-weight relationship is unclear; for example, are the effects of sleep on a person's weight direct or are they explained by the indirect effects of the obesity risk factors on sleep and weight [16–18]?

To address this hypothesis (*Hypothesis 2*), obesity risk factors were examined as IVs, body fatness indices were DVs and objective and subjective sleep were potential mediators. Of all potential IVs, only NEQ-nocturnal ingestions (NI) met criteria as an IV and only three sleep symptoms (SSQ, SD, DD) met criteria as potential mediators. Collectively, the results showed that SD fully mediated the NI-BMI relationship, poor SSQ fully mediated the NI – WHR relationship; and, high DD fully mediated the high NI

– obesity category relationship. Together, the results suggest that NI can parsimoniously explain the relationships between: high BMI and SD; high WHR and low SSQ; and, obesity category and DD.

Clinically, the results reflect that NI likely interferes with sleep *and* contributes to overweight/obesity [6, 10] whereas other obesity risk factors (e.g. low PA, electronic device use, mood/affect) may not. However, due to the cross-sectional nature of the results, it is unclear whether sleep disturbances contributed to NI, NI interfered with sleep or both, but it is appreciated that NI is often practiced by overweight/obese people and it may disrupt sleep [6] and lead to weight gain over time [7, 10].

However, the results should 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, the NEQ had barely adequate internal consistency suggesting that the results for night-eating should be interpreted with caution. Third, the RAPA showed that a higher proportion of people exercised to an adequate degree than GPAQ score (29% vs 70%). Finally, the use of online survey platforms to deliver surveys can attract younger and better educated adults as participants [62] which may have reduced the generalisability of the results to older and less well educated people. However, we did assess sleep and physical activity via subjective and objective methods, and more broadly, the scales showed good psychometric properties in this study [25].

Conclusion

Results suggest that impairment in objective sleep (i.e. longer awake time during the night) and subjective sleep (i.e. more nocturnal sleep disturbances) can explain significant variance in obesity indices even after taking into account the effects of other obesity risk factors (i.e. covariates; e.g. night-eating) [16–18] and demographics. Mediational results suggested that nocturnal indigestion (NI) may interfere with sleep (i.e. more SD, poor SSQ, DD) and together they may result in an increase in weight (i.e. high BMI & WHR, obesity category). Taken together, the results suggest that eating after waking from sleep is potentially obesogenic especially if it interferes with sleep. It may exert a greater effect on a person's weight than other aspects of night-eating (e.g. evening hyperphagia) or other obesity risk factors (e.g. insufficient PA, electronic device use, mood/affect), despite being highly correlated with measures of sleep and body fatness [16–18].

Abbreviations

BMI
body mass index
WHR
waist to hip ratio
NES
night eating syndrome
NEQ

Night-Eating Questionnaire
SSQ
subjective sleep quality
SOL
sleep onset latency
SE
sleep efficacy
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 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.

Acknowledgments

This work was conducted at the Australian National University, Research School of Psychology. No funds, grants, or other support was received.

Conflict of Interest

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 & weight Disorders is rendered.

References

1. Knutson, K.L., *Sociodemographic and cultural determinants of sleep deficiency: implications for cardiometabolic disease risk.* Social science & medicine, 2013. 79: p. 7-15. https://doi.org/10.1016/j.socscimed.2012.05.002
2. Rosas, L.G., et al., *Effect of an Intervention for obesity and depression on patient-centered outcomes: an RCT.* American journal of preventive medicine, 2020. 58(4): p. 496-505. https://doi.org/10.1016/j.amepre.2019.11.005
3. Doo, M., *The association between sleep duration and 25-hydroxyvitamin D concentration with obesity in an elderly Korean population: a cross-sectional study.* Nutrients, 2018. 10(5): p. 575. https://doi.org/10.3390/nu10050575
4. Vgontzas, A.N., et al., *Obesity without sleep apnea is associated with daytime sleepiness.* Archives of internal medicine, 1998. 158(12): p. 1333-1337. doi:10.1001/archinte.158.12.1333
5. Punjabi, N.M., K. Bandeen-Roche, and T. Young, *Predictors of objective sleep tendency in the general population.* Sleep, 2003. 26(6): p. 678-683. https://doi.org/10.1093/sleep/26.6.678
6. Crispim, C.A., et al., *Relationship between food intake and sleep pattern in healthy individuals.* Journal of Clinical Sleep Medicine, 2011. 7(06): p. 659-664. https://doi.org/10.5664/jcsm.1476
7. Brown R, Thorsteinsson E. (Eds.). (2020). *Comorbidity: Symptoms, Conditions, Behavior and Treatments*. Palgrave Macmillan: Switzerland AG. DOI: 10.1007/978-3-030-32545-9
8. Uçar, C., T. Özgöçer, and S. Yıldız, *Effects of late-night eating of easily—or slowly—digestible meals on sleep, hypothalamo-pituitary-adrenal axis, and autonomic nervous system in healthy young males.* Stress and Health, 2021. https://doi.org/10.1002/smi.3025
9. Stunkard, *Eating patterns and obesity.* Psychiatric Quarterly, 1959. 33(2): p. 284-295. https://doi.org/10.1007/BF01575455
10. Meule, A., et al., *The association between night eating and body mass depends on age.* Eating behaviors, 2014. 15(4): p. 683-685. https://doi.org/10.1016/j.eatbeh.2014.10.003
11. Yeh, S.-S.S. and R.F. Brown, *Disordered eating partly mediates the relationship between poor sleep quality and high body mass index.* Eating behaviors, 2014. 15(2): p. 291-297. https://doi.org/10.1016/j.eatbeh.2014.03.014
12. O'Reardon, J.P., A. Peshek, and K.C. Allison, *Night Eating Syndrome.* CNS drugs, 2005. 19(12): p. 997-1008. https://doi.org/10.2165/00023210-200519120-00003
13. Gallant, A., J. Lundgren, and V. Drapeau, *The night-eating syndrome and obesity.* Obesity reviews, 2012. 13(6): p. 528-536. https://doi.org/10.1111/j.1467-789X.2011.00975.x

14. Wurtman, R.J. and J.J. Wurtman, *Brain serotonin, carbohydrate-craving, obesity and depression.* Obesity, 1995. 3(S4). https://doi.org/10.1002/j.1550-8528.1995.tb00215.x
15. Swinburn, B.A., et al., *Diet, nutrition and the prevention of excess weight gain and obesity.* Public health nutrition, 2004. 7(1a): p. 123-146. https://doi.org/10.1079/PHN2003585
16. Chahal, H., et al., *Availability and night-time use of electronic entertainment and communication devices are associated with short sleep duration and obesity among C anadian children.* Pediatric obesity, 2013. 8(1): p. 42-51. https://doi.org/10.1111/j.2047-6310.2012.00085.x
17. Petersen, H., et al., *Stress vulnerability and the effects of moderate daily stress on sleep polysomnography and subjective sleepiness.* Journal of sleep research, 2013. 22(1): p. 50-57. https://doi.org/10.1111/j.1365-2869.2012.01034.x
18. Kimura, N., et al., *Association between objectively measured walking steps and sleep in community-dwelling older adults: A prospective cohort study.* Plos one, 2020. 15(12): p. e0243910. https://doi.org/10.1371/journal.pone.0243910
19. Weinsier, et al., *The etiology of obesity: relative contribution of metabolic factors, diet, and physical activity.* The American journal of medicine, 1998. 105(2): p. 145-150. https://doi.org/10.1016/S0002-9343(98)00190-9
20. Fogelholm, M. and K. Kukkonen-Harjula, *Does physical activity prevent weight gain – a systematic review.* Obesity Reviews, 2000. 1(2): p. 95-111. https://doi.org/10.1046/j.1467-789x.2000.00016.x
21. Tucker, L.A. and M. Bagwell, *Television viewing and obesity in adult females.* American Journal of Public Health, 1991. 81(7): p. 908-911. https://doi.org/10.2105/AJPH.81.7.908
22. Spoormaker, V.I. and J. van den Bout, *Depression and anxiety complaints; relations with sleep disturbances.* European psychiatry, 2undefined
23. Luppino, F.S., et al., *Overweight, obesity, and depression: A systematic review and meta-analysis of longitudinal studies.* Archives of General Psychiatry, 2010. 67(3): p. 220-229. doi:10.1001/archgenpsychiatry.2010.2
24. Katzmarzyk, P.T., *Physical activity, sedentary behavior, and health: paradigm paralysis or paradigm shift?* Diabetes, 2010. 59(11): p. 2717-2725. https://doi.org/10.2337/db10-0822
25. Buysse, D.J., et al., *The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research.* Psychiatry Research, 1989. 28(2): p. 193-213. https://doi.org/10.1016/0165-1781(89)90047-4
26. WHO. *Global Physical Activity Questionnaire version 2*. 2012 April 2018]; Available from: http://www.who.int/chp/steps/GPAQ. .
27. Armstrong, T. and F. Bull, *Development of the world health organization global physical activity questionnaire (GPAQ).* Journal of Public Health, 2006. 14(2): p. 66-70. https://doi.org/10.1007/s10389-006-0024-x
28. Olds, T.S., et al., *Trends in the prevalence of childhood overweight and obesity in Australia between 1985 and 2008.* International journal of obesity, 2010. 34(1): p. 57. https://doi.org/10.1038/ijo.2009.211

29. Li, R., et al., *Gender differences in insomnia—a study in the Hong Kong Chinese population.* Journal of psychosomatic research, 2002. 53(1): p. 601-609. https://doi.org/10.1016/S0022-3999(02)00437-3
30. Kanter, R. and B. Caballero, *Global gender disparities in obesity: a review.* Advances in nutrition, 2012. 3(4): p. 491-498. https://doi.org/10.3945/an.112.002063
31. WHO. *BMI Classification*. 2013 [cited 2018 08 April]; Available from: http://apps.who.int/bmi/index.jsp?introPage=intro_3.html.
32. Jones, D.J. Hare, and K. Evershed, *Actigraphic assessment of circadian activity and sleep patterns in bipolar disorder.* Bipolar disorders, 2005. 7(2): p. 176-186. https://doi.org/10.1111/j.1399-5618.2005.00187.x
33. Yang, C.-C. and Y.-L. Hsu, *A review of accelerometry-based wearable motion detectors for physical activity monitoring.* Sensors, 2010. 10(8): p. 7772-7788. https://doi.org/10.3390/s100807772
34. Kaplan, K.A., et al., *Evaluating sleep in bipolar disorder: comparison between actigraphy, polysomnography, and sleep diary.* Bipolar disorders, 2012. 14(8): p. 870-879. https://doi.org/10.1111/bdi.12021
35. Walsh, J.K., et al., *Sleepiness/alertness on a simulated night shift following sleep at home with triazolam.* Sleep, 1991. 14(2): p. 140-146. DOI:10.1093/sleep/14.2.140
36. Ancoli-Israel, S., et al., *The role of actigraphy in the study of sleep and circadian rhythms.* Sleep, 2003. 26(3): p. 342-392. http://aasm.org/resources/practicereviews/cpr_actigraphy.pdf
37. Adam Noah, et al., *Comparison of steps and energy expenditure assessment in adults of Fitbit Tracker and Ultra to the Actical and indirect calorimetry.* Journal of medical engineering & technology, 2013. 37(7): p. 456-462. https://doi.org/10.3109/03091902.2013.831135
38. Buysse, D.J., et al., *Relationships Between the Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), and Clinical/Polysomnographic Measures in a Community Sample*. Journal of Clinical Sleep Medicine : JCSM : Official Publication of the American Academy of Sleep Medicine, 2008. 4(6): p. 563-571. https://doi.org/10.5664/jcsm.27351
39. Carpenter, J.S. and M.A. Andrykowski, *Psychometric evaluation of the pittsburgh sleep quality index.* Journal of Psychosomatic Research, 1998. 45(1): p. 5-13. https://doi.org/10.1016/S0022-3999(97)00298-5
40. Allison, et al., *The Night Eating Questionnaire (NEQ): psychometric properties of a measure of severity of the Night Eating Syndrome.* Eating behaviors, 2008. 9(1): p. 62-72. https://doi.org/10.1016/j.eatbeh.2007.03.007
41. Health Promotion Research Centre. *RAPA; 9-item*. 2006; Available from: https://depts.washington.edu/hprc/resources/products-tools/rapa/.
42. Vega-López, S., et al., *Validity and reliability of two brief physical activity questionnaires among Spanish-speaking individuals of Mexican descent.* BMC research notes, 2014. 7(1): p. 29. https://doi.org/10.1186/1756-0500-7-29

43. Topolski, et al., *Peer reviewed: the Rapid Assessment of Physical Activity (RAPA) among older adults.* Preventing chronic disease, 2006. 3(4). Available from: URL: http://www.cdc.gov/pcd/issues/2006/oct/06_0001.htm
44. WHO. *Global physical activity questionnaire (GPAQ) analysis guide*. 2012; Available from: http://www.who.int/ncds/surveillance/steps/resources/GPAQ_Analysis_Guide.pdf.
45. Bull, F.C., T.S. Maslin, and T. Armstrong, *Global physical activity questionnaire (GPAQ): nine country reliability and validity study.* Journal of Physical Activity and health, 2009. 6(6): p. 790-804. https://doi.org/10.1123/jpah.6.6.790
46. Lovibond, P.F. and S.H. Lovibond, *The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories.* Behaviour research and therapy, 1995. 33(3): p. 335-343. https://doi.org/10.1016/0005-7967(94)00075-U
47. Crawford, J.R. and J.D. Henry, *The Depression Anxiety Stress Scales (DASS): Normative data and latent structure in a large non-clinical sample.* British Journal of Clinical Psychology, 2003. 42(2): p. 111-131. https://doi.org/10.1348/014466503321903544
48. Page, A.C., G.R. Hooke, and D.L. Morrison, *Psychometric properties of the Depression Anxiety Stress Scales (DASS) in depressed clinical samples.* British Journal of Clinical Psychology, 2007. 46(3): p. 283-297. https://doi.org/10.1348/014466506X158996
49. Hayes, A.F., *Partial, conditional, and moderated moderated mediation: Quantification, inference, and interpretation.* Communication Monographs, 2018. 85(1): p. 4-40. https://doi.org/10.1080/03637751.2017.1352100
50. Zhao, X., J.G. Lynch, Jr., and Q. Chen, *Reconsidering Baron and Kenny: Myths and Truths about Mediation Analysis.* Journal of Consumer Research, 2010. 37(2): p. 197-206. https://doi.org/10.1086/651257
51. Australian Institute of Health & Welfare *Overweight and obesity: an interactive insight*. 2019. Cat. no. PHE 251. Canberra: AIHW. Available from: https://www.aihw.gov.au.
52. Mansfield, et al., *Sleep loss and sleep disorders.* The Medical journal of Australia, 2013. 199(8): p. S5. doi: 10.5694/mja13.11157
53. Striegel-Moore, et al., *Night eating: prevalence and demographic correlates.* Obesity, 2006. 14(1): p. 139-147. https://doi.org/10.1038/oby.2006.17
54. Australian Bureau of Statistics. *National Health Survey: First Results, 2014-15* First results 2015; Available from: http://www.abs.gov.au/ausstats/abs@.nsf/PrimaryMainFeatures/4364.0.55.001?OpenDocument.
55. Muzet, Libert, and Candas, *Ambient temperature and human sleep.* Experientia, 1984. 40(5): p. 425-429. https://doi.org/10.1007/BF01952376
56. Brobeck, J.R., *Food intake as a mechanism of temperature regulation.* The Yale journal of biology and medicine, 1948. 20(6): p. 545. PMCID: PMC2602369

57. Åkerstedt, T., *Psychosocial stress and impaired sleep.* Scandinavian journal of work, environment & health, 2006: p. 493-501. https://www.jstor.org/stable/40967601
58. Kobayashi, D., et al., *Association between weight gain, obesity, and sleep duration: a large-scale 3-year cohort study.* Sleep and Breathing, 2012. 16(3): p. 829-833. https://doi.org/10.1007/s11325-011-0583-0
59. Chaput, et al., *The Association Between Sleep Duration and Weight Gain in Adults: A 6-Year Prospective Study from the Quebec Family Study.* Sleep, 2008. 31(4): p. 517-523. https://doi.org/10.1093/sleep/31.4.517
60. Jennings, J.R., et al., *Self-reported sleep quality is associated with the metabolic syndrome.* Sleep, 2007. 30(2): p. 219-223. https://doi.org/10.1093/sleep/30.2.219
61. Dallman, M.F., et al., *Chronic stress and obesity: a new view of "comfort food".* Proceedings of the National Academy of Sciences, 2003. 100(20): p. 11696-11701. https://doi.org/10.1073/pnas.1934666100
62. Howard, Rainie, and Jones, *Days and nights on the Internet: The impact of a diffusing technology.* American Behavioral Scientist, 2001. 45(3): p. 383-404. https://doi.org/10.1177/0002764201045003003

Tables

Table 1 Means and Standard Deviations of the main study variables at T1 and T2

	T1 (N=161)	T2 (N=155)	T1 vs T2
	M (SD)	M (SD)	
BMI (kg)	25.69 (6.79)	25.88 (7.05)	$F(1, 154) = .67$, $p = .413$
WHR	0.78 (.079)	.0776 (.079)	$F(1, 154) = .08$, $p = .783$
Sleep Latency (mins)	10.37 (18.49)	10.77 (18.41)	$F(1, 154)=.524$, $p=.470$
Actual Sleep (hrs)	7.6 (1.66)	7.9 (1.96)	$F(1, 154)=3.184$, $p=.076$
Wake Time (mins)	56.52 (38.27)	54.92 (35.24)	$F(1, 154)=.21$, $p=.646$
Total Sleep (hrs)	8.5 (1.96)	8.8 (2.20)	$F(1, 154)=1.87$, $p=.173$
Sleep Efficacy	85.84 (7.25)	86.26 (6.68)	$F(1, 154)=.10$, $p=.748$
PSQI^ (total score)	7.99 (3.42)	6.63 (3.28)	$F(1, 154) = 32.64$, $p < .000$
Subjective sleep quality^	1.53 (.73)	1.18 (.68)	$F(1, 154)=33.23$, $p<.000$**
Sleep Latency (min)	1.67 (1.02)	1.42 (.98)	$F(1, 154)=9.66$, $p=.002$**
Sleep duration (hrs)^	.96 (.76)	.83 (.75)	$F(1, 154)=4.56$, $p=.034$*
Habitual sleep efficacy^	.66 (.91)	.73 (1.02)	$F(1, 154)=.72$, $p=.399$
Sleep disturbances (number)	1.43 (.54)	1.21 (.48)	$F(1, 154)=29.8$, $p<.000$**
Use of sleeping medication	.3 (.76)	.33 (.77)	$F(1, 154)=.05$, $p=.819$
Daytime dysfunction	1.45 (.77)	1.04 (.73)	$F(1, 154)=33.74$, $p<.000$**
NEQ	17.30 (6.67)	15.84 (6.52)	$F(1, 154) = 7.26$, $p = .008$
Depression	13.18 (9.57)	11.69 (10.49)	$F(1, 154) = 5.17$, $p = .024$
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)	$F(1, 154) = 17.54$, $p <.000$
RAPA: Activity level	4.37 (1.87)	4.44 (1.92)	$F(1, 154) = .08$, $p = .781$
RAPA: Strength/Flexibility	1.05 (1.13)	1.12 (1.22)	$F(1, 154) = .59$, $p = .442$
GPAQ Total	2407.60 (3274.67)	2159.68 (2970.70)	$F(1, 154) = 1.32$, $p =.253$
TV time (hrs)	1.57 (.820)	2.67 (.920)	$F(1, 154) = 393$, $p = .000$

Note. ^ Variables inversely coded. *p<.05 **p<.005

Table 2 Predictors of BMI at T1 (N = 161)

	Variable	B	SE	B	t	p
Step1						
	Age	.347	.05	.483	6.947	.001**
Step2						
	Age	.339	.055	.472	6.192	.001**
	Strength/Flexibility (RAPA)	-.367	.421	-.061	-.871	.385
	TV night	-2.556	.902	-197	-2.832	.005*
	Device use	.333	.596	.043	.559	.577
Step3						
	Age	.334	.054	.465	6.16	.001**
	Strength/Flexibility (RAPA) T1	.313	.418	-.052	-.749	.455
	TV night	-2.473	.895	-.191	-2.764	.006*
	Device use	.321	.59	.041	.544	.587
	Sleep disturbances	1.713	.846	.137	2.024	.045*

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

Table 3 Predictors of BMI at T2 (N = 155)

	Variable	B	SE	B	t	p
Step1						
	BMI T1	1.027	.013	.997	78.36	.001**
	Age	-.01	.009	-.013	-1.034	.303
Step2						
	BMI T1	1.028	.014	.998	75.547	.001**
	Age	-.012	.01	-.016	-1.134	.259
	Strength/Flexibility (RAPA)	-.032	.073	-.005	-.445	.657
	TV night	.073	.16	.005	.458	.647
	Device use	-.032	.102	-.004	-.313	.754
Step3						
	BMI T1	1.028	.014	.997	73.979	.001**
	Age	-.012	.01	-.016	-1.123	.263
	Strength/Flexibility (RAPA)	-.031	.073	-.005	-.429	.668
	TV night	.072	.161	.005	.45	.654
	Device use	-.033	.103	-.004	-.318	.751
	Sleep disturbances	.041	.149	.003	.273	.785

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

Table 4 Predictors of WHR at T1 (N = 161)

	Variable	B	SE	B	t	P
Step1						
	Age	.003	.001	.402	6.593	.001**
	Gender	-.08	.01	-.5	-8.206	.001**
Step2						
	Age	.003	.001	.376	5.631	.001**
	Gender	-.079	.01	-.492	-8.034	.001**
	NEQ	-.001	.001	-.086	-1.395	.165
	Device use	-.004	.006	-.049	-.729	.467
Step3						
	Age	.003	.001	.352	5.121	.001**
	Gender	-.08	.01	-.499	-8.128	.001**
	NEQ	-.001	.001	-.048	-.736	.463
	Device use	-.004	.006	-.047	-.695	.488
	Subjective sleep Quality	-.007	.008	-.064	-.888	.376
	Daytime dysfunction	-.006	.007	-.057	-.794	.428

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

Table 5 Predictors of WHR at T2 (N = 155)

		B	SE	B	t	p
Step1						
	WHR T1	.728	.046	.736	15.912	.001**
	Age	.001	.001	.145	3.624	.001**
	Gender	-.028	.007	-.177	-4.17	.001**
Step2						
	WHR T1	.73	.046	.738	15.916	.001**
	Age	.001	.001	.157	3.676	.001**
	Gender	-.028	.007	-.174	-4.103	.001**
	Device use	.003	.004	0.032	.827	.41
Step3						
	WHR T1	.724	.046	.732	15.672	.001**
	Age	.001	.001	.15	3.458	.001**
	Gender	-.029	.007	-.18	-4.204	.001**
	Device use	.003	.004	.032	.816	.416
	Daytime dysfunction	-.004	.004	-.038	-1.015	.312

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

Table 6 Logistic Regression Analysis with Obesity as the Dependent Variable (absence vs. presence of overweight/obesity) and the Independent Variables at T1 (N=161)

	Variable	B	S.E.	P	OR	OR (95% C.I)	
						Lower	Upper
Step 1	Age	.1	14.7	.026	1	.001	1.105
	Strength/Flexibility	-.162	.844	.176	1	.358	.85
	Device use	-.228	.871	.245	1	.351	.796
	Wake Time	.016	9.58	.005	1	.002	1.016
	use of sleeping medication	.376	2.113	.259	1	.146	1.456
	Daytime dysfunction	-.347	1.767	.261	1	.184	.707

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

Table 7 Logistic Regression Analysis with Obesity as the Dependent Variable (absence vs. presence of overweight/obesity) and the Independent Variables at T2 (N = 155)

	Variable	B	SE	Wald	df	p
Step 1	BMI 1 Logistic (1)	-419.814	12796.66	.001	1	.974
	Age	4.905	157.306	.001	1	.975
	Strength/Flexibility	25.476	1081.34	.001	1	.981
	Device use	69.093	2236.505	.001	1	.975
	Wake Time	-.979	31.438	.001	1	.975
	Sleep disturbances	74.682	2466.24	.001	1	.976

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

Table 8 *Summary of the Mediation Analysis for each explored pathway (N=161)*

	Path *a* (XàM)	Path *b* (MàY)	Path *c'* (XàY) (Direct effect)	Path *ab* (Indirect effect)	BOOTLLCI (a*b)	BOOT ULCI (a*b)
Mediation 1	NIàSD	SDàBMI	NIàBMI	Mediation effect	.02	.32
Coeff	.07	2.11	.10	.14		
SE	.02**	1.02*	.24	.08 **		
Mediation 2	NIàSSQ	SSQàWHR	NIàWHR	Mediation effect	-.003	-.0001
Coeff	.08	-.02	-.002	-.002		
SE	.03 *	.01*	.003	.001 *		
Mediation 3	NIàDD	DDàOC	NIàOC	Mediation effect	-.1	-.01
Coeff	.07	-.63	.14	-.04		
SE	.03 *	.23 *	.08	.03 *		

Note. NI=Nocturnal Ingestion; SD=Sleep Disturbances, BMI=Body Mass Index; SSQ=Subjective Sleep Quality; WHR=Waist to Hip Ratio; DD=Daytime Dysfunction; OC=Obesity Category. *p <.05. **p <.001.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementaryFileThecorrelationMatrix.xlsx