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Sleep, Mood, and Binge Eating: Evidence from a Student Sample

Introduction

Binge eating (BE) is a transdiagnostic symptom, characterised by overconsumption and loss of control (Frank & Berner, 2020). It is central to several eating disorders (EDs; American Psychiatric Association, 2022) and often associated with medical (Kessler et al., 2013), social and functional impairment (Wilfley, Wilson, & Agras, 2003). BE occurs along a continuum and is common in the general population (Striegel-Moore et al., 2000). Unfortunately, its prevalence has increased over time (Hay et al., 2008; Pike & Dunne, 2015), and it is currently estimated at 20% among young females (Yan et al., 2023), with most not seeking help nor treatment (e.g., Boher et al., 2017). Nevertheless, research has mostly focused on diagnosed populations, limiting our understanding of BE more broadly. This is problematic because individuals with formal diagnoses may represent a biased subset of those experiencing ED symptoms (e.g., Dapelo et al., 2020). Understanding the mechanisms contributing to BE in undiagnosed groups is therefore critical for both fully comprehending the phenomenon, and informing public health intervention.

Recent research has shed light on the impact of sleep on emotional and psychological well-being, with sleep disturbances increasingly recognised as drivers of mental health disorders (Freeman et al., 2020). In EDs, up to 57% of individuals report sleep disturbances (Kim et al., 2010), with evidence indicating that sleep and eating are closely interconnected through shared biological and psychological mechanisms (Gomes et al., 2023). Clinically, insomnia at intake has been shown to predict ED treatment outcome in outpatient samples (Lombardo et al., 2015; Christensen Pacella et al., 2025) and to persist in about 50% of the cases (Christensen Pacella et al., 2025). It is unclear if similar sleep difficulties occur in undiagnosed individuals with BE.

The relationship between sleep and BE may be bidirectional (Linnaranta et al., 2020). Specifically, BE, which often occurs in the evening (Schreiber-Gregory et al., 2013), may delay sleep onset or reduce sleep quality through arousal, digestion, or fullness (Christensen & Short, 2021). Compensatory behaviours, such as excessive exercise, may further disrupt sleep (Stutz et al., 2019). Conversely, poor sleep has been shown to alter hunger and satiety regulating hormones (Motivala et al., 2009), increase cravings (Greere et al., 2013), caloric

intake (Chaput, 2014), and consumption of rich foods (De Leon & Hanlon, 2020), all of which accompany BE episodes.

A specific way sleep and BE may be linked is through emotional regulation difficulties. Indeed, BE is used to elevate mood (Lacey, 1986), avoid unpleasant feelings (Heatherton & Baumeister, 1991) or achieve numbing (Lev-ari et al., 2021). In EDs groups, maladaptive coping mechanisms, such as worry, are common (Startup et al., 2013) and exacerbate negative emotions (Newman et al., 2019). In healthy individuals, disrupted sleep increases negative affect (Tempesta et al., 2018), potentially heightening susceptibility to worry as a coping strategy (Diefenbach et al., 2001). Negative emotions (Kenny et al., 2018) and worry (Pillai et al., 2014) can, in turn, worsen insomnia and delay sleep onset in EDs.

Research on sleep and BE is limited and typically focuses on diagnostic groups rather than transdiagnostic mechanisms. A meta-analysis of 31 studies concluded that people with BE exhibit poorer sleep quality, greater daytime sleepiness, insomnia and difficulty falling asleep compared to people without BE (da Luz et al., 2023). However, most studies focused on clinical samples (da Luz et al., 2023), leaving sleep disturbance in undiagnosed community populations largely unexplored. The few studies that investigated BE in non-clinical groups (Kauffman et al., 2018; Bottera et al., 2025) relied exclusively on subjective sleep measures (da Luz et al., 2023). This is problematic given the well-documented discrepancies between objective and subjective sleep assessments (Rezaie et al., 2018), indicating the need for complementary assessments, especially in groups with mental health difficulties (Choi et al., 2010).

The current study aimed to investigate sleep and BE severity in an undiagnosed student sample, assessing sleep quality, duration, efficiency, fragmentation and night to night variability using both objective (i.e., actigraphy) and subjective (i.e., validated self-reports) measures. We predicted poorer sleep in people with moderate-to-high, compared to low, BE severity. A secondary aim examined the role of emotional regulation difficulties, with the prediction that low mood and worry would mediate the effect of sleep on BE severity.

Method

Participants

Participants were 69 individuals (59 females) aged 19-35 ($M = 21$; $SD = 3.19$). The sample was relatively diverse, comprising primarily individuals identifying as Asian (40.6%) and White (37.7%). Recruitment used convenience sampling via social media, word of mouth, and a university participant pool. Most participants were students (95.65%) receiving course credits.

Eligibility criteria included age 18-40 (to ensure informed consent, minimise parental influences on eating, and reduce age-related variability in sleep), low or moderate-to-high BE severity, no medical or physical condition interfering with sleep, no sleep disorder as obstructive sleep apnoea, restless leg syndrome, periodic limb movement, or parasomnias, no regular alterations to sleep routines, and no recreational drug use. Individuals with a concurrent ED diagnosis or receiving treatment for weight management or disordered eating were excluded. The sample size provided adequate power to detect a medium effect size for the primary hypotheses ($\alpha = 0.05$, $1 - \beta = 0.80$, $f^2(V) = 0.15$, required $N = 64$). Ethical approval was granted by Kingston University Research Ethics Committee.

Procedure

Participants accessed the study via a Qualtrics link, where they reviewed the information sheet, provided consent, and completed a screening survey (see below) to determine eligibility. Eligible participants attended a laboratory visit to receive an actigraphy watch, sleep diary, and instructions. During the 7-day protocol, they completed an additional survey (see below). At the end, participants returned the watch and were debriefed.

Materials

The Binge Eating Scale (BES; Gormally et al., 1982) is a 16-item self-report measure of BE severity used in clinical and general populations (Duarte et al., 2015). Scores range from 0-46, with higher scores indicating greater severity. In line with the literature (e.g., Grupski et al., 2013; Kops et al., 2020), the BES was used at screening to classify participants as ‘Low Binge Eaters’ (LBE; <17) or ‘Moderate-High Binge Eaters’ (M-HBE; >17). The BES demonstrates good psychometric properties (Duarte et al., 2015), and Cronbach’s α in the current sample was .915.

The Global Sleep Assessment Questionnaire (GSAQ; Roth et al., 2002) is an 11-item tool used to screen for sleep disorders in the general population. It assesses factors including mood, life activities, medical issues, and symptoms of insomnia, sleep apnoea, restless leg

syndrome and parasomnias over the last 4 weeks. Items are rated along a 4-point scale, with higher scores indicating greater disturbance. The GSAQ shows good construct validity and acceptable internal consistency (Roth et al., 2002); Cronbach's α in the current sample was 0.802. In this study, the measure screened out participants with significant sleep disorders, with symptom frequency and functional impairment used to determine the cut-offs as per original scoring.

The Beck Depression Inventory (BDI-II; Beck et al., 1996) is a 21-item self-report measure assessing depression severity, widely used in both clinical and non-clinical samples (Wang & Gorenstein, 2013). Scores range from 0-63, with scores ≥ 14.5 indicating potential need for medical treatment (von Glischinski et al., 2021). The BDI-II has shown good to excellent psychometric properties (Wang & Gorenstein, 2013); Cronbach's α in the current sample was 0.922. The measure was here employed to test our secondary hypothesis.

The Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990) is a 16-item self-report measure of worry tendency, intensity and uncontrollability. Scores range from 16-80, with higher scores indicating greater trait worry. The PSWQ has consistently shown good psychometric properties (e.g., Stoeber & Bittencourt, 1998); Cronbach's α in the current sample was 0.750. The measure was here used to test our secondary hypothesis.

The Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994) is a 28-item self-report measure adapted from the semi-structured Eating Disorder Examination interview (Fairburn & Cooper, 1993). It assesses ED psychopathology across four subscales (Restraint, Eating Concern, Shape Concern, and Weight Concern), from which a global score is then calculated (Fairburn & Beglin, 2008). The EDE-Q demonstrates good psychometric properties in clinical (Luce & Crowther, 1999) and non-clinical samples (Peterson et al., 2007); Cronbach's α in the current sample was .932. In this study, the measure was used to characterise the sample, following standard ED research practice.

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) is a self-report measure assessing sleep quality and disturbance over one month. It comprises 19 items grouped into seven scores: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction – combined to produce a global score ranging from 0-21, with higher scores indicating poorer sleep quality. The PSQI typically demonstrates acceptable to good internal consistency (Buysse et al., 1989). In the current sample, Cronbach's α for the global score was 0.59,

below the conventional threshold for acceptable internal consistency. Thus, to test our hypotheses, we selected the Sleep Disturbance subscale, due to its higher internal consistency (Cronbach's $\alpha = 0.69$) and broad measure of perceived sleep disturbance. This subscale has also been shown to provide more specific insight than the global score in several studies (e.g., Telford et al., 2018; Zitser et al., 2022). Note that only 2 subscales, Sleep Disturbance and Daytime Sleepiness, had at least 3 items required for Cronbach's α calculations (Rammstedt & Beierlein, 2014). We decided not to focus on Daytime Sleepiness, as it is only marginally relevant to sleep quality and overlaps with the Epworth Sleepiness Scale (see below). While the manuscript reports analyses based on the Sleep Disturbance subscale, corresponding analyses using the global score are provided in the Supplementary Materials for completeness. The pattern of findings remains unchanged.

The Epworth Sleepiness Scale (ESS; Johns, 1991) is an 8-item self-report measure assessing persistent daytime sleepiness. Participants rate the likelihood of dozing off or fall asleep in 8 situations, with scores 0-24, and scores > 10 reflecting excessive daytime sleepiness. The ESS demonstrates high internal and good test-retest reliability in clinical and non-clinical samples (Johns, 1992). In the current sample, Cronbach's α was 0.839. The measure was used to characterise the sample and examine group differences in sleepiness, following standard practice in sleep research.

The reduced Morningness-Eveningness Questionnaire (rMEQ; Adan & Almirall, 1991) is a 5 item self-report scale adapted from the original 19-item MEQ (Horne & Östberg, 1976). Scores range from 4-26, with higher scores indicating stronger morning preference. Both versions are commonly used to assess diurnal preferences and show good external and construct validity (e.g., Di Milia et al., 2013), and high agreement between the scales (Adan & Almirall, 1991). In the current sample, Cronbach's α value was 0.639. The measure was used to characterise the sample and examine group differences in chronotype, following standard practice in sleep research.

Actigraphy data were collected using the Motion Watch 8 (MW8; CamNtech Ltd., Cambridge, UK), a wrist-worn triaxial accelerometer for monitoring sleep and circadian patterns. Participants wore the device on their non-dominant wrist continuously for 7 days, and pressed the marker button each night before asleep and each morning upon waking. The device recorded activity counts in 30-second epochs. Sleep periods were manually identified by two separate scorers using a combination of activity count, event markers, and the

National Sleep Foundation Sleep Diary (National Sleep Foundation, 2021). The sleep diary was completed daily upon waking and before bedtime and was used to support the identification of bedtimes and wake times, to verify event marker entries, and to assist scoring when markers were missing or ambiguous. Sensitivity threshold was set to high as this is the validated threshold (Camntech, 2024). To assess the reliability across the two scorers, a third reviewer independently scored 20% of the actigraphy data. Intraclass correlation coefficients (ICC) for sleep efficiency was 0.88, 95% CI [0.65, 0.96], indicating good agreement. The following parameters were extracted using manufacturer's algorithm: Total Sleep Time (the total time spent asleep according to the epoch-by-epoch sleep/wake categorisation), Sleep Efficiency (i.e., actual sleep time expressed as a percentage of the assumed in bed time), and Fragmentation Index (an indication of the degree of fragmentation of the sleep period). Night-to-night variability in sleep fragmentation and total sleep time was calculated following Jahng et al. (2008) and Sánchez-Ortuño et al. (2010) by averaging the squared differences between consecutive nights, capturing both nightly fluctuations and temporal dependencies.

The demographic questionnaire consisted of questions regarding participant age, ethnicity, gender, educational background, and body weight and height for BMI calculations. Questions were also asked about previous and current physical and mental health diagnoses, sleep routine, and drug use, as per study eligibility criteria.

Data Analysis

Statistical analyses were conducted using the SPSS (29.2). We used multivariate analysis of variance (MANOVA) to test for group differences across our main variables. The assumptions for MANOVA were checked and met. No multicollinearity was detected across the dependent variables, as all bivariate correlations had $r < 0.90$. The Box's M test indicated that the assumption of homogeneity of covariance matrices was met ($p = 0.191$). We did not test for violations of normality as MANOVA is generally robust when sample size > 30 (Timm, 2002). Linearity was assessed using scatterplot matrices of the dependent variables within each group. Visual inspection indicated approximately linear relationships between all pairs of dependent variables, satisfying the assumption of linearity. PROCESS V5 (Hayes, 2022) Model 4 with the bootstrap method based on 5000 samples and a 95% confidence interval was used to calculate the mediating relationships of the hypothesised causal model (Hayes, 2018), namely whether low mood and worry mediated the relationship between sleep

and BE. The PROCESS macro was chosen because it provides sufficient statistical power and consistency when testing the effects in smaller sample sizes (Collier, 2020).

Results

Demographics

Table 1 summarises self-reported demographic and questionnaire-based characteristics. There were no significant group differences for age ($t(67) = -0.829, p = 0.410$), biological sex ($\chi^2(1, 69) = 2.304, p = 0.129$), ethnicity ($\chi^2(4, 69) = 1.878, p = 0.758$) or education ($\chi^2(6, 69) = 7.877, p = 0.247$). The LBE group had a BMI within the healthy range, while the M-HBE group's mean BMI fell at the lower end of the overweight range (WHO, 1995) - this difference did not reach statistical significance, ($t(65) = -1.934, p = .057$).

Eating disorders symptomatology

Consistent with our recruitment strategy, the M-HBE group scored higher on both the BES ($t(67) = -13.818, p < 0.001$) and EDE-Q ($t(69) = -7.582, p < 0.001$). While the LBE group scored comparably to community samples (Keane et al., 2017; Machado et al., 2014), the M-HBE group scored in line with clinical samples (Jennings & Phillips, 2017) – although variability exists across diagnoses (Dalhgren et al., 2017). On the BES, the LBE group scored in line with averages in community samples (Duarte et al., 2015), while the M-HBE group scored between obese (Hood et al., 2013) and ED samples (Karapatsia et al., 2024).

Sleep and circadian related findings

Regarding sleep quality, as measured by the PSQI global score, the LBE group scored in line with general population samples (Hinz et al., 2017), whereas the M-HBE group scored comparably to EDs samples (Tzischinsky et al., 2021). The rMEQ indicated that both groups had an evening chronotype (Adan and Almirall, 1991), and the ESS that neither experienced excessive daytime sleepiness (Sander et al., 2016).

Table 2 reports objective sleep measures. The MANOVA revealed a significant global effect of BE group on our dependent variables (Wilk's Lambda = 0.774, $F(1, 67) = 2.961, p = 0.013$). There were no significant BE group differences on any of the actigraphy sleep parameters (Total Sleep Time: $F(1, 65) = 0.453, p = 0.503$; Sleep Efficiency: $F(1, 65) = 0.785, p = 0.379$; Fragmentation Index: $F(1, 65) = 0.479, p = 0.491$; Night to Night

Variability Fragmentation Index: $F(1, 65) = 0.896, p = 0.347$; Night to Night Variability Total Sleep Time: $F(1, 65) = 0.589, p = 0.445$). However, when sleep was assessed via subjective measures, the M-HBE group, exhibited reduced sleep quality with both the PSQI Sleep Disturbance subscale ($F(1, 65) = 579.757, p < 0.001$) and the GSAQ ($F(1, 65) = 17.631, p < 0.001$), compared to the LBE group. Chronotype (r MEQ: $F(1, 65) = 0.185, p = 0.669$), and the daytime sleepiness (ESS: $F(1, 65) = 2.107, p = 0.151$), were not different across groups. Correlational analyses between BES and sleep measures confirmed the group-level findings, with only the PSQI showing a significant association with BES ($r = 0.414, p < 0.001$). The findings are replicated when using the PSQI global score – see Supplementary Materials.

Cognitive and emotional regulation

The LBE group scored similarly to community samples on depressed mood (Whisman & Richardson, 2015), while the M-HBE group scored similarly to EDs groups (Tanaka et al., 2015) and above the moderate depression cut-off (Dolle et al., 2012). Worry in the LBE group was near healthy norms and slightly below clinical anxiety or depression in the M-HBE group (Oliveira et al., 2023).

In line with the literature, we found lower mood ($F(1, 65) = 42.409, p < 0.001$) and higher worry ($F(1, 65) = 7.299, p = 0.009$) in the M-HBE, compared to the LBE, group. To test our secondary hypothesis, we examined whether low mood, as measured by the BDI-II, and worry, as measured by the PSWQ, mediated the relationship between sleep (our IV in the model) and BE scores (our dependent variable in the model). The PSQI Sleep Disturbance subscale was chosen as the sleep quality mediator, as it discriminated between the groups. This analysis (Graph 1) revealed that sleep quality was significantly associated with low mood ($B = 7.135, t = 2.766, p = 0.007, 95\% \text{ CI } [1.987, 12.283]$) and marginally associated with worry ($B = 5.923, t = 1.892, p = 0.063, 95\% \text{ CI } [-0.327, 12.173]$). When both mediators were entered simultaneously, low mood was significantly associated with BE ($B = 0.489, t = 5.292, p < 0.001, 95\% \text{ CI } [0.305, 0.674]$), whereas worry was not ($B = 0.110, t = 1.448, p = 0.153, 95\% \text{ CI } [-0.042, 0.262]$). Bootstrap analyses indicated a significant indirect effect of sleep quality on BE through low mood ($B = 3.490, \text{bootstrap } 95\% \text{ CI } [1.016, 6.220]$) but not through worry ($B = 0.653, \text{bootstrap } 95\% \text{ CI } [-0.429, 2.309]$). These results indicate that low mood, but not worry, statistically mediated the association between sleep quality and BE. The findings are replicated when using the PSQI global score – see Supplementary Materials.

Discussion

Our screening approach was effective, as the groups differed in both BE and general EDs symptomatology. Consistent with high comorbidity between EDs, depression and anxiety (Meng & D'Arcy, 2015), the M-HBE group also showed higher levels of worry and low mood. Contrary to our predictions, no significant group difference emerged in actigraphy-derived sleep parameters, contrasting with prior actigraphy and polysomnography findings in EDs (Degasperi et al., 2024). One possible interpretation is that the M-HBE group did not experience the degree of objective sleep disturbance observed in clinically diagnosed EDs. Although the M-HBE group scored comparably to clinical samples on the EDE-Q, and between obese and EDs samples on the BES, it is possible that additional clinical features – such as illness chronicity, compensatory behaviours, nutritional instability, or other – are necessary for objective sleep difficulties to emerge. Evidence linking specific clinical features to sleep disturbances remains limited. A recent meta-analysis (Degasperi et al., 2024) reported significant sleep disturbances in anorexia nervosa but insufficient evidence for bulimia nervosa and BE disorder – with the latter represented by one study only.

Subjective sleep quality differed significantly across groups, consistent with EDs samples showing reduced sleep quality on self-report measures (Degasperi et al., 2024). Discrepancies between objective and subjective sleep measures are common (Van Den Berg et al., 2008), with low mood, anxiety, and cognitive-emotional arousal more strongly associated with perceived sleep quality than actigraphy-derived parameters (Herbert et al., 2017). In our study, individuals with elevated BE may experience their sleep as disturbed due to these heightened processes. However, this discrepancy may also reflect limitations of actigraphy in capturing relevant aspects of sleep disturbance, including those linked to affective states and arousal. Future studies could explore this using biological stress markers, such as cortisol, associated with sleep quality in non-EDs samples (Laufer et al., 2025), and measures of sleep architecture and circadian patterns to clarify links between sleep, BE and affective states.

Our secondary aim was to examine whether emotion regulation difficulties – specifically, low mood and maladaptive worry tendencies – mediate the relationship between sleep and BE. Although groups differed on both, only low mood significantly mediated this relationship. Previous research has highlighted a mediating role of low mood in sleep and EDs (Lampe et al., 2024), emotional eating (Akkus et al., 2024) and disordered eating

(Lombardo et al., 2014). To our knowledge, this is the first evidence of such mediation in an undiagnosed BE sample. However, it is important to note that cross-sectional designs do not allow for conclusions about causality or temporal ordering (Weems, 2025), thus further experimental or longitudinal research is needed.

These findings should be interpreted in light of several limitations. First, while we excluded individuals with formal EDs diagnoses, we did not conduct full mental health assessment, so some participants may have met diagnostic criteria, limiting generalisability to non-clinical BE samples. Second, our short actigraphy protocol precluded circadian rhythms assessment, limiting detection of irregular or misaligned rest-activity patterns in BE (Roveda et al., 2017). Third, despite attempts at broader recruitment, all but three participants were students. Analyses were replicated excluding non-student participants, and the findings remained unchanged (see *Supplementary Materials*). Future research should examine non-university populations to obtain a better representation of sleep difficulties in BE. Notably, our sample included a relatively high proportion of Asian participants, often underrepresented in ED and sleep research (Egbert et al., 2022), potentially enhancing the generalisability of our findings. Finally, although powered for the main MANOVA, subsequent analyses may have been underpowered - negative findings should therefore be considered preliminary.

In conclusion, this study extends previous research by showing that, in a student sample with undiagnosed BE, sleep disturbances are primarily reflected in subjective rather than objective sleep measures. Individuals with moderate-to-high BE reported poorer perceived sleep alongside higher worry and low mood, despite no differences in actigraphy-derived sleep. Low mood – but not worry – significantly mediated the sleep-BE relationship, supporting the view that BE may function as a mood-regulation strategy. Together, these findings underscore the importance of considering affective processes and sleep perception, rather than objective sleep alone, when examining sleep-BE links in BE undiagnosed samples.

Table 1. Demographic and questionnaire-based characteristics of the sample

	Total Sample (<i>n</i> = 69)	LBE (<i>n</i> = 33)	M-HBE (<i>n</i> = 36)
Age <i>M</i> (<i>SD</i>)	21 (3.19)	20.67 (3.14)	21.31 (3.25)
Sex at birth <i>n</i> (%)			
Female	59 (85.5)	26 (78.8)	33 (91.7)
Male	10 (14.5)	7 (21.2)	3 (8.3)
Ethnicity <i>n</i> (%)			
Black	11 (15.9)	4 (12.1)	7 (19.4)
White	26 (37.7)	11 (33.3)	15 (41.7)
Asian	28 (40.6)	16 (48.5)	12 (33.3)
Mixed	2 (2.9)	1 (3.0)	1 (2.8)
Other	2 (2.9)	1 (3.0)	1 (2.8)
Education Level <i>n</i> (%)			
GCSE	2 (2.9)	0 (0.0)	2 (5.6)
A Level	34 (49.3)	20 (60.6)	14 (38.9)
Higher Education Certificate (CertHE)	9 (13.0)	3 (9.1)	6 (16.7)
Foundation Degree	12 (17.5)	5 (15.2)	7 (19.4)
Bachelor's	10 (14.5)	3 (9.1)	7 (19.4)
Master's	1 (1.4)	1 (3.0)	0 (0.0)
Doctorate	1 (1.4)	1 (3.0)	0 (0.0)
BMI (kg/m ²) <i>M</i> (<i>SD</i>)	23.35 (7.74)	21.54 (6.56)	25.12 (8.46)
BES <i>M</i> (<i>SD</i>)	16.42 (10.29)	7.94 (4.86)	25.53 (5.64)
GSAQ <i>M</i> (<i>SD</i>)	9.62 (4.99)	7.70 (3.85)	12.78 (5.81)
BDI-II <i>M</i> (<i>SD</i>)	17.84 (11.16)	10.67 (7.59)	24.67 (9.84)
PSWQ <i>M</i> (<i>SD</i>)	56.87 (13.45)	52.79 (11.37)	61.72 (13.61)
EDE-Q Global <i>M</i> (<i>SD</i>)	2.15 (1.56)	1.10 (0.95)	3.24 (1.34)
Restraint	1.53 (1.55)	0.78 (0.97)	2.37 (1.63)
Eating Concerns	1.52 (1.47)	0.58 (0.66)	2.51 (1.53)
Shape Concerns	2.96 (1.93)	1.68 (1.45)	4.20 (1.53)
Weight Concerns	2.59 (1.85)	1.35 (1.23)	3.87 (1.56)
PSQI Global <i>M</i> (<i>SD</i>)	6.67 (3.02)	5.23 (2.60)	8.52 (2.61)
PSQI Sleep Disturbance <i>M</i> (<i>SD</i>)	1.33 (0.50)	1.09 (0.29)	1.56 (0.56)
ESS <i>M</i> (<i>SD</i>)	8.62 (4.87)	7.58 (4.96)	9.56 (5.01)
r-MEQ <i>M</i> (<i>SD</i>)	11.38 (3.81)	12.09 (4.23)	11.77 (3.64)

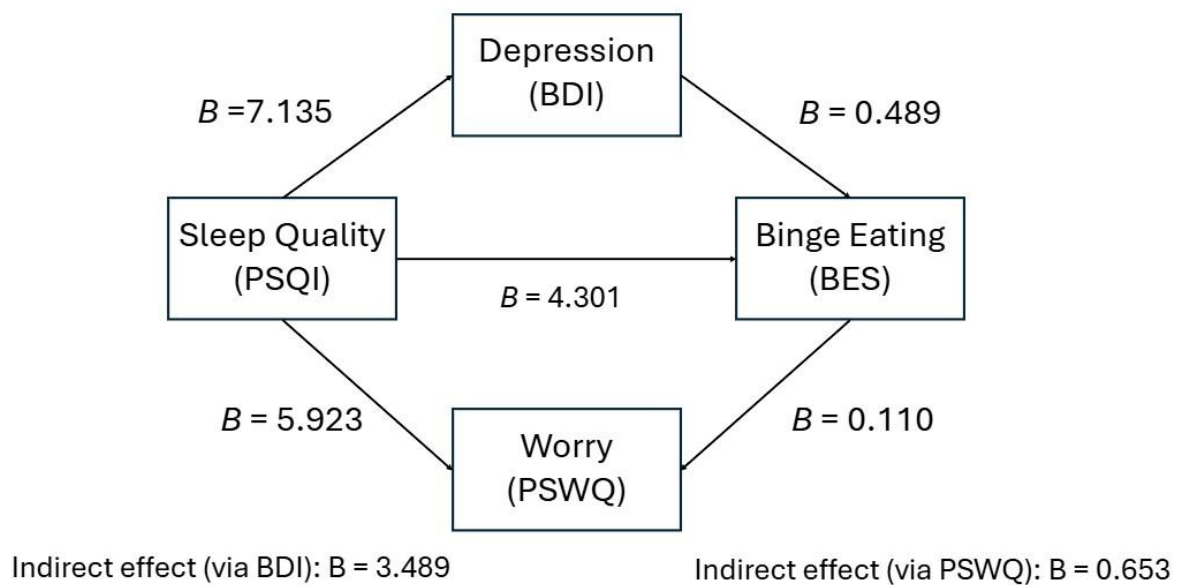
Note: LBE = Low Binge Eating; M-HBE = Moderate-to-High Binge Eating; *M* = Mean; *SD* = Standard Deviation; BMI = Body Mass Index; BES = Binge Eating Scale; GSAQ = Global Sleep Assessment Questionnaire; BDI-II = Beck Depression Inventory (Version 2); PSWQ = Penn State Worry Questionnaire; EDE-Q = Eating Disorder Examination Questionnaire; PSQI = Pittsburgh Sleep Quality Index; ESS = Epworth Sleepiness Scale; r-MEQ = reduced Morningness-Eveningness Questionnaire.

Table 2. Objective measures of sleep in the sample

	Total Sample (<i>n</i> = 69) <i>M(SD)</i>	LBE (<i>n</i> = 33) <i>M(SD)</i>	M-HBE (<i>n</i> = 36) <i>M(SD)</i>
Total Sleep Time <i>mins</i>	417.96 (58.31)	422.88 (60.16)	413.32 (56.99)
Sleep Efficiency %	84.94 (4.81)	85.48 (4.05)	84.44 (5.44)
Fragmentation Index <i>mins</i>	26.18 (8.89)	25.41(6.65)	26.91 (10.63)
Night-to-Night Variability – Total Sleep Time	5.28 (4.84)	5.86 (5.62)	4.74 (3.98)
Night-to-Night Variability – Fragmentation Index	148.96 (105.18)	138.84 (87.64)	158.49 (119.91)

Note: LBE = Low Binge Eating; M-HBE = Moderate-to-High Binge Eating; *M* = Mean; *SD* = Standard Deviation.

Graph 1: Mediation analysis



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