BRIEF REPORT

Emotional Eating Moderates the Relationship of Night Eating with Binge Eating and Body Mass

Adrian Meule[1]*, Kelly C. Allison[2] & Petra Platte[1]

[1]Department of Psychology I, University of Würzburg, Würzburg, Germany

[2]Department of Psychiatry, Center for Weight and Eating Disorders, University of Pennsylvania, Philadelphia, PA, USA

Abstract

Night eating syndrome is marked by substantial evening or nocturnal food intake, insomnia, morning anorexia, and depressed mood. Night eating severity has been positively associated with body mass index (BMI), binge eating frequency, and emotional eating tendencies. We conducted an online questionnaire study among students ($N = 729$) and explored possible interactive effects between those variables. Night eating severity, binge eating frequency, BMI and emotional eating were all positively correlated with each other. Regression analyses showed that night eating severity was particularly related to more frequent binge episodes and higher BMI at high levels of emotional eating but unrelated to those variables at low levels of emotional eating. Thus, eating as a means of emotion regulation appears to be an important moderator of the relationship between night eating and both binge eating and BMI. Copyright © 2013 John Wiley & Sons, Ltd and Eating Disorders Association.

Keywords

night eating; emotional eating; binge eating; body mass index

***Correspondence**

Adrian Meule, Department of Psychology I, University of Würzburg, Marcusstr. 9-11, 97070 Würzburg, Germany. Tel: +49 931 31-808 34; Fax: +49 931 31-824 24. Email: adrian.meule@uni-wuerzburg.de

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Introduction and Aims

Night eating syndrome (NES) was first recognized in 1955 as a pattern of night eating behaviours among obese individuals (Stunkard *et al.*, 1955). Although there is a positive relationship between night eating severity and body mass index (BMI), it has been shown that night eating also occurs in nonobese individuals, and it has been hypothesized that NES may precede weight gain (Marshall *et al.*, 2004; Vander Wal, 2012).

Night eating syndrome has not been included as a stand-alone diagnosis in diagnostic manuals, and it is not very widely known by eating disorder professionals yet (Goncalves *et al.*, 2009; Vandereycken, 2011). However, it is now listed in the *Otherwise Specified Feeding and Eating Disorders* section of the fifth revision of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2013). Proposed research diagnostic criteria involve the following: (i) consumption of at least 25% of daily food intake after the evening meal or ≥2 episodes of nocturnal eating per week; (ii) awareness of those eating episodes; and (iii) at least three of the following: morning anorexia, a strong urge to eat between dinner and sleep or at night, insomnia, a belief that one must eat in order to initiate or return to sleep, or a worsening of mood in the evening (Allison *et al.*, 2010).

Night eating syndrome appears to be a distinct eating disorder, but there is also a moderate overlap with binge eating disorder (BED) and bulimia nervosa (Fischer *et al.*, 2012; Vander Wal, 2012). Root *et al.* (2010) reported that the heritability of night eating for women was .35 and for men was .44, while the heritability of binge eating was .70 for women and .74 for males (both designations were based on brief screening questions). The genetic overlap between persons reporting binge eating and night eating behaviours was moderate at 0.66 (95% CI [0.48, 0.96]), suggesting considerable but not complete overlap between the two behaviours. These findings suggest that there are additional genetic factors that are unique to night eating that are not shared by binge eating and vice versa. More research is needed to determine what these factors may be.

Following these genetic findings, NES and BED severity are positively correlated but not perfectly at $r = 0.47$ (Harb *et al.*, 2012). Furthermore, recent studies have shown that NES severity is also positively associated with emotional eating (EE; Nolan & Geliebter, 2012). Emotional eating (EE) refers to food intake in response to specific emotions or more diffuse mood states (e.g. anger, sadness, boredom, or stress) and has also been found to be associated with binge eating behaviours and increased BMI (Ganley, 1989; Macht, 2008; Macht & Simons, 2011).

In a recent comprehensive review of the literature on night eating, Vander Wal (2012) concluded that NES appears to be associated with obesity but that morning anorexia may also compensate evening hyperphagia or nocturnal eating. Similarly, although most studies find a positive association between NES

severity and BMI (e.g. Harb *et al.*, 2012; Moizé *et al.*, 2012; Tholin *et al.*, 2009) and that those with NES may be younger when they present for treatment as compared with BMI-matched controls (Marshall *et al.*, 2004), there have also been null findings (e.g. Allison *et al.*, 2008; Nolan & Geliebter, 2012; Striegel-Moore *et al.*, 2010; refer to Vander Wal, 2012 for a review). Furthermore, the overlap or distinctiveness of NES and BED is highly debated and a subject of ongoing attention in studies (Fischer *et al.*, 2012; Striegel-Moore *et al.*, 2010; Vander Wal, 2012). As EE is positively associated with NES, BED, and BMI, we explored if it would be a significant moderator of the relationship of NES severity with both BED and BMI. Specifically, we tested if there would be interactive effects of self-reported night and emotional eating when predicting binge eating frequency and BMI in a large student sample.

Method

Participants and procedure

Data were collected as part of an online study that included a range of questionnaires related to eating and other constructs. Only data of three questionnaires were used for the current analyses. Further details of this study will be reported elsewhere (*cf.* Meule *et al.*, 2012). Student councils of several German universities were contacted by e-mail and asked to distribute the online study's link using their mailing lists. As an incentive, three × 50 € and five × 20 € were raffled among participants who completed the entire set of questions. Participation was completely voluntary and anonymous (e.g. e-mail addresses were recorded separately and could not be ascribed to any other data). Questionnaire completion took about 20–25 minutes. Every question required a response in order to continue. The study period lasted two weeks.

A total of $N = 729$ participants (77.0% women, $n = 561$) completed the entire study. The mean age was M = 23.55 years ($SD = 3.89$; range: 18–47) and mean BMI was M = 22.59 kg/m^2 (*cf.* Table 1 for descriptive data). According to standard guidelines (World Health Organization, 2000), $n = 59$ (8.1%) participants were underweight (BMI < 18.50 kg/m^2), $n = 527$ (72.3%) were of normal weight (BMI = 18.50–24.99 kg/m^2), $n = 106$ (14.5%) were overweight (BMI = 25.00–29.99 kg/m^2), and $n = 37$ (5.1%) were obese (BMI > 30.00 kg/m^2). Most participants were students (91.4%, $n = 666$).

Measures

Night Eating Questionnaire

The Night Eating Questionnaire (NEQ) (Allison *et al.*, 2008) is a 14-item instrument for the assessment of night eating behaviours (e.g. 'How much of your daily food intake do you consume after suppertime?' or 'Do you have cravings or urges to eat snacks when you wake up at night?'). Items are scored on a five-point scale ranging from 0 (e.g. *not at all*) to 4 (e.g. *extremely*). Validity has been shown by positive relationships with sleep problems, eating disorder symptomatology, depressive symptoms and eveningness preference (e.g. Allison *et al.*, 2008; Harb *et al.*, 2012). The NEQ was translated into German for this sample. It has an acceptable internal consistency of $\alpha = .70$ (Allison *et al.*, 2008), and internal consistency was $\alpha = .71$ in the current study.

Mood Eating Scale

The Mood Eating Scale (MES) (Jackson & Hawkins, 1980) is a 20-item instrument for the assessment of eating in response to different emotions and mood states (e.g. 'When I feel inferior to someone, it makes me want to eat.' or 'When I am under pressure, I find myself eating more often.'). Items are scored on a five-point scale ranging from 0 (*strongly agree*) to 4 (*strongly disagree*). Validity has been shown by positive relationships with restrained eating, binge eating, dissatisfaction with weight-related appearance and the degree to which body weight percentage exceeds self-determined ideal weight (Jackson & Hawkins, 1980). Internal consistency was $\alpha = .88$ in both the validation study (Jackson & Hawkins, 1980) and in the current study.

Objective binge episodes

The number of days with objective binge episodes (OBEs) was assessed with item #15 ('Over the past 28 days, on how many days have such episodes of overeating occurred [i.e. you have eaten an unusually large amount of food and have had a sense of loss of control at the time)?'] of the *Eating Disorder Examination—Questionnaire* (Fairburn & Beglin, 1994; Hilbert & Tuschen-Caffier, 2006). Validity has been shown by a high level of agreement with the *Eating Disorder Examination—Interview* (Berg *et al.*, 2012; Hilbert *et al.*, 2007).

Data analyses

Scores on the NEQ, MES, and OBE were positively skewed. However, log-transformation (ln) of those variables did not essentially alter results, and thus, raw data were used in the following

Table 1 Descriptive statistics and correlations between study variables

$N = 729$	M	*SD*	Range	(1)	(2)	(3)	(4)
(1) Body mass index (kg/m^2)	22.59	4.24	15.62–68.04	—	.14	.21	.18
(2) Days with objective binge episodes[†]	1.48	3.91	0–28	.14	—	.42	.34
(3) Mood Eating Scale	25.41	13.57	0–68	.21	.42	—	.32
(4) Night Eating Questionnaire	11.58	4.41	2–34	.18	.34	.32	—

Notes: All correlations are significant ($p < .001$).
[†]Within the past 28 days (item #15 of the *Eating Disorder Examination—Questionnaire*).

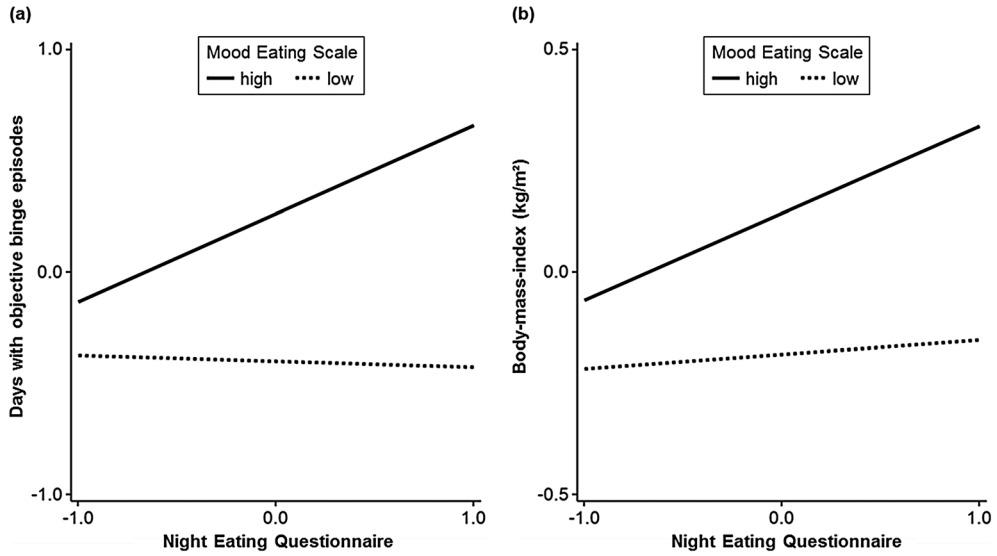

Figure 1. (a) Predicted number of days with objective binge episodes and (b) body mass index as a function of scores on the Night Eating Questionnaire and the Mood Eating Scale (all variables are *z*-standardized). High and low values represent ±1 standard deviation from the respective mean

analyses. Before computing regression analyses, all study variables were *z*-standardized. Linear regression analyses were performed using OBE and BMI as dependent variables and scores on the NEQ, MES, and the interaction NEQ × MES as predictor variables (*cf.* guidelines by Baron & Kenny, 1986, for testing moderation effects with two continuous variables). To examine the nature of interaction effects, we computed simple slopes for the regressions of NEQ on OBE and BMI for individuals with low and high scores on the MES (one standard deviation below and above the mean, respectively; Aiken & West, 1991). Regression analyses were performed with *Interaction!* Version 1.7.2211 (Freeware available at http://www.danielsoper.com/interaction). All *p*-values are reported two tailed.

Results

Descriptive statistics of study variables and associations with age and gender

Descriptive statistics and correlations between variables are depicted in Table 1. Using the NEQ cut-off scores of 25 and 30 (Allison *et al.*, 2008), $n = 9$ (1.2%) and $n = 2$ (0.3%) participants, respectively, were classified as having NES. Eighty-eight (12.1%) participants reported regular binge eating (≥4 OBE) within the past month.

Female participants (M = 11.76, $SD = 4.52$) had higher NEQ scores than males (M = 10.96, $SD = 4.00$, $t_{(727)} = 2.06$, $p < .05$), but this effect was negligible ($\eta^2_p = .01$). They also had higher MES scores (M = 26.69, $SD = 13.92$), reported more frequent OBE (M = 1.69 days, $SD = 4.32$) and had lower BMI (M = 22.22 kg/m^2, $SD = 4.33$) as compared with males (MES: M = 21.15, $SD = 11.35$, $t_{(727)} = 4.71$, $p < .001$; OBE: M = 0.76 days, $SD = 1.88$, $t_{(727)} = 2.72$, $p < .01$; BMI: M = 23.83 kg/m^2, $SD = 3.69$, $t_{(727)} = 4.37$, $p < .001$), but effect sizes were also small (MES: $\eta^2_p = .03$; OBE: $\eta^2_p = .01$; BMI: $\eta^2_p = .03$).

Age was unrelated to scores on the NEQ ($r = -.06$, ns) and OBE ($r = -.04$, ns) but was weakly correlated with BMI ($r = .18$, $p < .001$)[1] and MES scores ($r = -.08$, $p < .05$). Using gender or age as a covariate in the subsequent analyses did not substantially change the results.

Regression analyses

The overall model for the prediction of OBE was significant ($F_{(3,725)} = 94.23$, $p < .001$, *adj.* $R^2 = .28$). All three variables significantly predicted OBE (NEQ: $\beta = .19$, MES: $\beta = .33$, NEQ × MES: $\beta = .24$, all *p*-values < .001). Scores on NEQ positively predicted OBE when scores on MES were high ($\beta = .40$, $t_{(725)} = 9.89$, and $p < .001$) but not when scores on the MES were low ($\beta = -.03$, $t_{(725)} = -0.56$, ns; Figure 1a).

The overall model for the prediction of BMI also was significant ($F_{(3,725)} = 17.24$, $p < .001$, *adj.* $R^2 = .06$). All three variables significantly predicted BMI (NEQ: $\beta = .11$, $p < .01$, MES: $\beta = .16$, $p < .001$, NEQ × MES: $\beta = .09$, $p < .05$). Scores on the NEQ positively predicted BMI when scores on the MES were high ($\beta = .20$, $t_{(725)} = 4.27$, $p < .001$) but not when scores on the MES were low ($\beta = .03$, $t_{(725)} = 0.60$, ns; Figure 1b).

Discussion

In the current study, we found small-to-moderate positive correlations between self-reported NES severity, binge eating frequency, EE, and BMI, replicating prior studies (e.g. Ganley, 1989; Harb *et al.*, 2012; Macht & Simons, 2011; Nolan &

[1]We also explored whether age moderated the relationship between NEQ and BMI and found that the interaction NEQ × age was a marginally significant predictor of BMI ($\beta = .06$, $p = .08$). That is, the relationship between NEQ and BMI was particularly pronounced ($\beta = .25$, $t_{(725)} = 5.17$, $p < .001$) in older participants (+1 *SD*) and less pronounced ($\beta = .14$, $t_{(725)} = 2.91$, $p < .01$) in younger participants (−1 *SD*).

Geliebter, 2012; Vander Wal, 2012). Most importantly, we found that an interactive effect of NES and EE predicted both binge eating frequency and BMI; that is, associations between NES and both binge eating and BMI were particularly strong at high levels of EE. Contrarily, no associations between NES and both binge eating and BMI could be found in individuals reporting low levels of EE.

Thus, the present findings highlight the fact that moderators influence the relationship between NES and both BED and BMI. Those moderators may be important to consider in the interpretation of inconsistent results (Vander Wal, 2012). It appears that some individuals exhibit night eating behaviours that are not associated with more frequent binge eating or higher BMI when they do not occur as a means of emotion regulation. On the other hand, night eating that is utilized as a form of emotion regulation strategy is likely to result in binge eating and weight gain. More specifically, it seems likely that the function of night eating may vary by EE status and may affect the expression of the behaviour. For example, persons with low EE may be able to regulate the portion size of their evening and/or nocturnal eating such that they eat only the minimum of what is necessary to achieve their desired effect, that is, to facilitate sleep onset. Conversely, those with high EE may be less likely to regulate the amount they are eating or to estimate the proper dose–response for eating in the service of achieving sleep. In this case, the eating is more similar to that described in daytime binge eating in that once eating commences, it is very difficult to stop. Clinically, the difference in presentation may be a report of eating one serving of yogurt or one piece of candy as compared with eating most of a box of cereal or a sleeve of crackers, thus resulting in weight gain over time.

Notably, the observed relationships between EE and night eating with BMI were smaller than those found with binge eating, which is likely because of the fact that BMI is influenced by many factors other than binge eating. Nevertheless, consumption of larger amounts of food in individuals scoring high in both EE and night eating is a probable contributor to higher BMI. In line with this, a recent experimental study among female students found that those who scored high on EE and reported short sleep showed elevated food consumption when under stress in the laboratory (Dweck *et al.*, 2014). Although NES severity was not assessed in this study, shorter sleep duration has also been associated with NES (Rogers *et al.*, 2006), and thus, these results may parallel findings of the current study.

One alternative hypothesis regarding the link between night eating and higher BMI could be that those with high EE engage in night eating more frequently and that energy consumed at night may be metabolized by the body less efficiently. Rodent studies suggest that when these animals eat out of phase, they gain more weight than control animals, despite no differences in caloric intake and locomotion (Fonken *et al.*, 2010; Paschos *et al.*, 2012). Human studies are few in this area, but those with randomized experimental designs have shown small increases in weight when individuals are eating more food at night as compared with a daytime eating condition over the course of about two weeks (Hibi *et al.*, 2013; Qin *et al.*, 2003). Fat oxidation has also been shown to be less efficient with increased eating in the evening (Gluck *et al.*, 2011; Hibi *et al.*, 2013). More well-controlled, longer-term studies of the impact of night eating in humans on weight and metabolism, controlling for sleep and energy intake, are clearly needed.

It is important to note, of course, that our data are cross-sectional, and thus, causal interpretations are speculative. Future studies would benefit from a longitudinal assessment of those eating behaviours and body mass, in order to determine causal relationships and long-term development of those variables. Furthermore, the current results are based on self-reports, which are potentially biased. Field or experimental studies in which actual food intake is measured are needed to replicate those subjectively gained insights. Finally, our sample comprised predominantly normal-weight students. It would be desirable to replicate and extend the present findings in more representative or clinical samples, particularly as the age of onset of NES more often occurs in the late 20s or early 30s (Allison *et al.*, 2004).

Notwithstanding those limitations, we showed that EE tendencies are an important moderator of the relationship between NES and both binge eating and BMI in a large student sample with a wide range in body mass. Future research may consider this finding when interpreting the overlap between, or discussing the dissociation of, night and binge eating. Moreover, it may be also relevant for studying the association between night eating and future weight gain. Finally, practitioners may be encouraged to make use of both (i) intervention strategies related to adjusting circadian rhythm and meal timing and (ii) intervention strategies that foster alternative emotion regulation strategies when night and emotional eating co-occur.

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