Published in final edited form as:
Int J Eat Disord. 2012 July ; 45(5): 695–703. doi:10.1002/eat.22003.

Sleep Problems are Associated with Binge Eating in Women

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Abstract

Objective—We examined the association among current self-reported sleep problems, lifetime binge eating, and current obesity in women from the Swedish Twin study of Adults: Genes and Environment study

Method—Logistic regression analyses were used to evaluate these associations in 3,790 women aged 20-47 years.

Results—Binge eating was reported by 244 (6.4%) women and was positively associated with not getting enough sleep ($p < .015$), sleeping poorly ($p < .001$), problems falling asleep ($p < .001$), feeling sleepy during work or free time ($p < .001$), and disturbed sleep ($p < .001$). These same sleep variables, as well as napping and being a night person, were also significantly associated with obesity. The associations between binge eating and sleep remained after accounting for obesity.

Discussion—This investigation offers empirical support for an independent association between sleep problems and binge eating, which is likely due to complex psychological, biological, neuroendocrine, and metabolic factors.

Sleep problems and inadequate sleep are associated with a variety of physical and psychological problems[1] including neuroendocrine abnormalities[2,3] and depressive symptomatology.[4,5] Both cross-sectional[6,7] and prospective studies[8-10] suggest that short sleep may also be associated with higher body mass index (BMI) and obesity. Over the past half-century there has been a steady and rapid decline in time spent sleeping, which has paralleled the rapid rise in the prevalence of overweight and obesity.[11] Between 1988 and 1994, the age-adjusted prevalence of obesity was 22.9%, which rose to 30.5% between 1999 and 2000.[12] During this same time, Americans reported an average of 1.5 to 2.0 hour decrease in amount of sleep per night.[13,14] Complex factors are believed to underlie a possible association between sleep curtailment and increased weight including changes in endocrine function and metabolic pathways, which may result in decreased satiety and energy expenditure, and increased hunger and food cravings.[11,15-18]

Sleep problems may also be associated with binge eating (BE),[19,20] a disordered eating behavior that is also associated with obesity.[21] The behavior of BE[22] is characterized by the consumption of an unusually large amount of food in a discrete period of time, combined

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Disclosures: All authors reported no biomedical financial interests or potential conflicts of interest.

with a feeling of loss of control over eating. BE is a key symptom of several Diagnostic and Statistical Manual of Mental Disorders 4th ed., text rev. (DSM-IV-R)[23] eating disorder diagnoses, including bulimia nervosa, anorexia nervosa binge-purge subtype, and the provisional diagnosis of binge eating disorder (BED). In the United States, the lifetime prevalence of BE at a frequency of twice per week for a duration of three months is estimated to be 4.5% for adults.[24]

Similar to sleep problems, BE is associated with physical and psychological comorbidity including neuroendocrine abnormalities,[25,26] depressive symptomatology,[27,28] and obesity.[29-33] Approximately 25%[29] to 52%[30] of adult individuals seeking treatment for obesity report BE.[31-33] Despite the common physical and psychological symptoms shared by sleep problems and BE, this association has not been thoroughly investigated

The aims of this study were: 1) to investigate the association between current self-reported sleep problems (during the six months prior to interview) and lifetime BE in a population-based sample of female twins; 2) to confirm the well-supported association between sleep problems and obesity in this sample; and 3) to determine whether any associations between sleep and BE in aims 1 and 2 are accounted for by obesity status. We hypothesized that sleep problems would be positively associated with both BE and obesity independent of age, current cohabiting status and lifetime depression diagnosis. In addition, we hypothesized that obesity status would only partially account for the association between sleep problems and BE and that an independent association between sleep problems and BE would exist.

METHOD

Participants

Participants were from the Swedish Twin study of Twin Adults: Genes and Environment (STAGE; http://ki.se/ki/jsp/polopoly.jsp?d=9610&l=en), a large population-based study that is a subsample of the Swedish Twin Registry (STR; http://ki.se/twinreg).[34,35] Data for the STAGE study were collected in 2005 and over 43,000 male and female twins born between 1959 and 1985 were eligible to participate.

Using web-based questionnaires (or computer assisted telephone interviews for those who preferred this manner of response), participants provided information on a variety of health and sociodemographic measures as well as habits and behaviors. The response rate was 59.6% (43.1% web-based questionnaire only; 16.5% telephone interview only). One hundred twins were recontacted to assess test-retest reliability and to compare data collection methods (computer-based vs. telephone interview). For the eating disorders section there was adequate agreement between the web-based questionnaire and telephone interviews (kappa = .76).[34,35]

STAGE is in accord with the Declaration of Helsinki and was approved by the Regional Ethics Committee at the Karolinska Institutet and the Biomedical Institutional Review Board at the University of North Carolina at Chapel Hill. All participants provided informed consent prior to participation. A detailed description of the study design can be found elsewhere.[34,35]

Eating Assessment

Lifetime history of BE was assessed using an expanded Structured Clinical Interview for *DSM-IV* based instrument.[36] A dichotomous variable for lifetime BE was created from the following questions: “Have you ever had eating binges when you ate what most people would regard as an unusually large amount of food in a short period of time?” with response options *yes, no*, and *don’t know/refuse* and “When you were having eating binges, did you

feel your eating was out of control?" with response options not at all, slightly, somewhat, very much, extremely, and *don't know/refuse*. Individuals were placed in the BE group if they answered *yes* to the first question and *slightly, somewhat, very much*, or *extremely* to the second question. If respondents answered *no* to the first question or *not at all* to the second question, they were categorized as NoBE.

In the eating assessment, self-report current height and weight were collected from which current BMI was calculated. Individuals reporting a current BMI greater than or equal to 30 kg/m^2 were considered obese and participants reporting BMIs less than 30 kg/m^2 were considered non-obese.

Sleep Assessment

A subsample of participants was asked about current sleep habits and problems. Items included: (a) Do you usually take a nap at least every second day? (*yes, no*); (b) Do you think you get enough sleep? (yes, definitely enough; yes, mostly enough; no, a bit too little; no, clearly not enough; no, far from enough); (c) How do you think you sleep on the whole? (very well, pretty well, neither poorly nor well, pretty poorly, very poorly); (d) Try to determine to what degree you are a morning person or a night person (definitely a morning person, to some degree a morning person, to some degree a night person, definitely a night person). In addition, participants were asked if they had the following complaints during the last three months: Problems falling asleep? and Sleepy during work or free time? They were also asked if they had any of the following complaints during the last six months: Waking up too early and not being able to sleep again? Feeling of not having enough sleep on awakening? and Disturbed or uneasy sleep? Response options for items assessing sleep complaints during the last three and last six months were *never, seldom, sometimes, usually*, and *always*.

Covariate Selection

Age at interview, current cohabiting status, and lifetime diagnosis of major depression were evaluated for inclusion as covariates in the models. For cohabitation, participants who indicated that they were currently married or cohabiting were characterized as cohabiting. Those who indicated that they were divorced, separated, widowed, living alone, or living apart were characterized as not cohabiting. The presence of depression was based on the criteria for major depressive disorder according to the American Psychiatric Association *DSM-IV-TR*.[23] Individuals were scored positive for lifetime depression if they endorsed at least five symptoms of depression associated with a change of functioning (criterion A) and that the symptoms caused significant distress or impairment (criterion C).

Statistical Analyses

All analyses were performed using SAS/STAT software, Version 9.2 of the SAS System for Windows.[37] Frequencies were calculated to estimate the prevalence of lifetime BE and current obesity status for each sleep item.

A series of logistic regression models using PROC GENMOD were applied to assess whether individual sleep items were associated with lifetime BE and current obesity. In the first set of models (Analysis 1), the potential covariates of age at interview and current cohabiting were included. The second set of models (Analysis 2) was then applied to evaluate the associations between the sleep variables and BE and obesity accounting for lifetime depression diagnosis. Additional models (Analysis 3) were then applied to evaluate the association between BE and the various sleep measures accounting for lifetime depression diagnosis and obesity status. Generalized estimating equations were applied to all models to account for non-independence of the data due to the inclusion of twin pairs.

RESULTS

A total of 4,021 women completed the sleep questionnaire and were eligible to be included in this study. Participants were excluded if they were missing information on any of the following variables: BE ($n = 24$), current BMI ($n = 72$), age at interview ($n = 2$), lifetime depression diagnosis ($n = 102$), and current cohabiting ($n = 49$). In total, 231 women who completed the sleep questionnaire were removed due to missing data. Men were excluded in the current study due to low prevalence of BE ($n = 33$, 1.2%), precluding meaningful analysis. The sample size for the current study was 3,790 women.

A total of 244 (6.4%) women endorsed BE. Individuals reporting BE were significantly younger with a mean (SD) age = 31.0 (7.4) years than individuals reporting NoBE with a mean (SD) age = 33.4 (7.7; $t = 4.68$, $p < .001$). Significantly fewer women endorsing BE reported cohabiting at the time of the interview (BE: 54.9%; NoBE: 66.4%; $\chi^2 = 13.24$, $p < .001$). In addition, significantly more women with BE endorsed a lifetime history of depression (BE: 57.0%; NoBE: 25.1%; $\chi^2 = 116.94$, $p < .001$) than those with NoBE. There were 228 (6.0%) women who had a current BMI ≥ 30 kg/m^2. Obese participants were significantly older with a mean (SD) age = 35.6 (7.2) years than participants who were not obese with a mean (SD) age = 33.1 (7.7; $t = -4.81$, $p < .001$). However, no differences were found for current cohabiting (obese: 70.6%; not obese: 65.3%; $\chi^2 = 2.68$, $p < .11$). Similar to BE, significantly more obese women endorsed a lifetime history of depression (obese: 36.4%; not obese: 26.6%; $\chi^2 = 10.44$, $p < .002$) than non-obese women. Significantly more women with lifetime BE were obese compared with women who did not report BE (BE: 9.4%; NoBE: 5.8%; $\chi^2 = 5.37$, $p < .03$). Endorsement frequencies for the sleep items for individuals with BE and NoBE and for obese and non-obese participants are presented in Table 1.

Women who reported not getting enough sleep, sleeping poorly, problems falling asleep, feeling sleepy during work or free time, and disturbed sleep were significantly more likely to report lifetime BE after accounting for age at interview, current cohabiting status and lifetime depression diagnosis (Table 2). Women who reported waking too early and those reporting not getting enough rest were more likely to report BE; however, when these models were adjusted for lifetime depression diagnosis, the associations were no longer significant. A similar pattern of results was observed for obese women (Table 3): after accounting for age at interview, women were significantly more likely to report napping every second day, not getting enough sleep, sleeping poorly, that they were night people, problems falling asleep, feeling sleepy during work or free time, waking too early, not enough rest, and disturbed sleep. However, after adjusting for lifetime depression diagnosis, waking too early and not getting enough rest were no longer significantly associated with obesity status. Adjusting for obesity status in the models did not change the findings: significant associations between self-reported sleep problems and lifetime BE remained significant (Table 2).

DISCUSSION

Significant associations were found between sleep problems (reports of not getting enough sleep, sleeping poorly, problems falling asleep, feeling sleepy during work or free time, and disturbed sleep) and BE after accounting for age at interview, current cohabiting status, lifetime depression diagnosis, and obesity status. Changes in the odds ratios associated with the addition of various confounders into the models indicate that lifetime depression diagnosis accounts for some (i.e., early awakening and not getting enough rest), but not all facets of the relation between sleep problems and BE and between sleep problems and obesity. Numerous prior investigations have reported an association between sleep

curtailment and increased weight[3,6,11,38] and a recent investigation from our group[39] reported that BED before or during pregnancy was associated with sleep problems during pregnancy and dissatisfaction with sleep 18 months after childbirth.

Potential Mechanisms of Association

The nature of the association between sleep problems and BE is likely to be complex. Sleep problems may lead to BE, BE may lead to sleep problems, or both sleep problems and BE may be the result of a third variable or variables, such as environmental stressors, depression, or underlying biological mechanisms. The reported association between sleep curtailment and weight status is of considerable public health relevance given the rising obesity trends[40,41] and underscore the relevance of the biological relation between sleep and appetite regulation.[3,11,42,43] One compelling hypothesis is that shared endocrine and metabolic pathways proposed to underlie the association between sleep curtailment and increased weight status may also be operative in the association between sleep problems and BE.

Specifically, the appetite hormones leptin and ghrelin may be implicated in the association between sleep problems and BE. Leptin and ghrelin are hypothesized to "represent the 'yin-yang' of one regulatory system that has developed to inform the brain about the current energy balance state"(p. 249).[44] Leptin, which is released by adipocytes, is positively correlated with increased fat mass and reduces appetite,[45,46] whereas ghrelin, a peptide produced predominantly by the stomach, stimulates appetite and decreases energy expenditure.[44,47,48] Both acute and chronic sleep deprivation have been associated with decreased leptin and elevated ghrelin,[3,15,18,49] consistent with a hormonal profile increasing appetite and decreasing energy expenditure.[45-47] Spiegel et al.[18] found that two consecutive nights of sleep deprivation (four hours of sleep per night) compared with sleep extension (10 hours of sleep per night) induced an 18% decrease in leptin and a 28% increase in ghrelin. Interestingly, a similar hormonal profile, characterized by decreased leptin and elevated ghrelin, has also been observed in response to food restriction in an animal model[50] and weight loss in humans.[51-53] Thus, it is plausible that insufficient sleep may mirror a calorically restricted hormonal state, whereby an individual is biologically driven to increase food intake, which may enhance susceptibility to BE.

Stress may also be a common factor underlying both sleep problems and BE. Stress plays a critical role in both human and animal models of BE[54-57] and sleep problems.[58,59] The key hormonal pathway that regulates the endocrine response to stress is the hypothalamic-pituitary-adrenal (HPA) axis.[60] Cortisol secretion, a major component of the stress response regulated by the HPA-axis, is positively associated with food intake in women[61,62] and with BE severity in obese individuals with BED.[63] Increases in cortisol and abnormalities of the HPA-axis have also been associated with sleep curtailment.[64] Thus, one hypothesis is that insufficient sleep may result in increased cortisol levels, which may enhance hunger and feeding behaviors and increase susceptibility to BE. Cortisol has also been found to dampen the appetite-suppressive effects of leptin[65] and to increase plasma ghrelin levels,[66,67] suggesting that multiple neuroendocrine factors acting in concert likely underlie the association between sleep problems and BE.

Sleep deprivation is associated with numerous physiological changes, which may also influence energy homeostasis and contribute to BE. For example, Spiegel, Leproult, and Van Cauter[68] found that, compared to baseline, sleep curtailment to 4 hours for 6 days reduced glucose tolerance by 40% and glucose disposal by 30%. A similar investigation[69] found that nocturnal wakefulness increased not only morning postprandial plasma glucose concentrations, but also daytime concentrations of thyreotropin, cortisone, and norepinephrine. In addition, one night of sleep deprivation acutely reduced energy

expenditure in healthy men, further reiterating the complex nature of the interactions between sleep deprivation, neuroendocrine changes, and energy homeostasis.

Limitations

The findings and implications of this investigation must be evaluated in concert with its limitations. First, the population evaluated included only women from Sweden and results might differ for men or for individuals from different ancestry groups. Second, this study used a cross-sectional epidemiological data set and the time frame across variables was not consistent. For example, BE and depression were assessed over the lifetime, whereas the sleep variables, cohabiting, and obesity reflected current status. Although lifetime history of BE was assessed, BED, which is characterized by BE, tends to be a chronic and stable disorder.[24,70] Thus, these results might be suggestive of causal associations; however, longitudinal investigations including contemporaneous assessments of both sleep problems and BE are needed to provide definitive information regarding causality. Third, data were largely collected using computer-administered self-report assessments. Although participants might be more forthcoming with sensitive information using computer-based assessments, these can be less precise than interview-based assessments conducted by a clinician.[71] Lastly, this investigation assessed only subjective sleep disturbances without objective validation though laboratory investigations and did not account for use of sleep medications. Future longitudinal investigations incorporating psychological, biological, and neuroendocrine factors, as well as contemporaneous assessment of a wide range of sleep problems (voluntary sleep curtailment vs. insomnia), are needed to address clinical questions related to why sleep problems and BE co-occur, and how they may affect one another.

Conclusion

This study provides empirical support for an association between sleep problems and lifetime BE. Although definitive conclusions regarding the pathways underlying the association between sleep problems and BE cannot be drawn, future interventions targeting the association between sleep problems and BE may interrupt a vicious hormonal and metabolic cycle, having significant implications both at an individual and a public health level. It is not premature for health care professionals to screen for both sleep problems and BE in clinical settings where individuals struggle with either problem and optimally efficacious obesity interventions should address BE in addition to sleep hygiene.

Acknowledgments

Drs. Trace and Runfola were supported by National Institute of Health grant T32MH076694 (PI: Bulik). Dr. Thornton was supported by National Institute of Health grant 1K01AA 18719-01A1 (PI: Root). This study was also supported by grants CA-085739 (PI: Sullivan) and AI-056014 (PI: Sullivan) from the National Institutes of Health. The Swedish Twin Registry is supported by grants from the Swedish Department of Higher Education the Swedish Research Council. We thank all participants for their time and effort.

References

1. Grandner MA, Patel NP, Gehrman PR, Perlis ML, Pack AI. Problems associated with short sleep: Bridging the gap between laboratory and epidemiological studies. Sleep Med Reviews. 2010; 14:239–247.
2. Steiger A. Sleep and endocrinology. J Intern Med. 2003; 254:13–22. [PubMed: 12823639]
3. Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. PLoS Med. 2004; 1:201–217.
4. Argyropoulos SV, Wilson SJ. Sleep disturbances in depression and the effects of antidepressants. Int Rev Psychiatry. 2005; 17:237–245. [PubMed: 16194795]

5. Benca RM, Peterson MJ. Insomnia and depression. Sleep Med. 2008; (Suppl 1):S3–S9. [PubMed: 18929317]
6. Fogelholm M, Kronholm E, Kukkonen-Harjula K, Partonen T, Partinen M, Harma M. Sleep-related disturbances and physical inactivity are independently associated with obesity in adults. Int J Obes. 2007; 31:1713–1721.
7. Ko GT, Chan JC, Chan AW, Wong PT, Hui SS, Tong SD, et al. Association between sleeping hours, working hours and obesity in Hong Kong Chinese: The 'better health for better Hong Kong' health promotion campaign. Int J Obes. 2007; 31:254–260.
8. Chaput JP, Després JP, Bouchard C, Tremblay A. The association between sleep duration and weight gain in adults: A 6-year prospective study from the Quebec Family Study. Sleep. 2008; 31:517–523. [PubMed: 18457239]
9. Hasler G, Buysse DJ, Klaghofer R, Gamma A, Ajdacic V, Eich D, et al. The association between short sleep duration and obesity in young adults: A 13-year prospective study. Sleep. 2004; 27:661–666. [PubMed: 15283000]
10. Patel SR, Malhotra A, White DP, Gottlieb DJ, Hu FB. Association between reduced sleep and weight gain in women. Am J Epidemiol. 2006; 164:947–954. [PubMed: 16914506]
11. Knutson KL, Spiegel K, Penev P, Van Cauter E. The metabolic consequences of sleep deprivation. Sleep Med Rev. 2007; 11:163–178. [PubMed: 17442599]
12. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999-2000. JAMA. 2002; 288:1723–1727. [PubMed: 12365955]
13. The Gallup Organization. Sleep in America: 1995 Gallup Poll. conducted for the National Sleep Foundation. Retrieved July 25, 2011, from http://www.stanford.edu/~dement/95poll.html
14. National Center for Health Statistics. Morbidity and Mortality Weekly Report. 2005. Quick Stats: Percentage of adults who report an average of ≤6h of sleep per 24-hour period, by sex and age group-United States, 1985 and 2004.
15. Chaput JP, Després JP, Bouchard C, Tremblay A. Short sleep duration is associated with reduced leptin levels and increased adiposity: Results from the Quebec family study. Obesity. 2007; 15:253–261. [PubMed: 17228054]
16. Chaput JP, Klingenberg L, Sjódin A. Do all sedentary activities lead to weight gain: Sleep does not. Curr Opin Clin Nutr Metab Care. 2010; 13:601–607. [PubMed: 20823775]
17. Mullington JM, Chan JL, Van Dongen HP, Szuba MP, Samaras J, Price NJ, et al. Sleep loss reduces diurnal rhythm amplitude of leptin in healthy men. J Neuroendocrinol. 2003; 15:851–854. [PubMed: 12899679]
18. Spiegel K, Tasali E, Penev P, Van Cauter E. Brief communication: Sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. Ann Intern Med. 2004; 141:846–50. [PubMed: 15583226]
19. Tzischinsky O, Latzer Y, Epstein R, Tov N. Sleep-wake cycles in women with binge eating disorder. Int J Eat Disord. 2000; 27:43–48. [PubMed: 10590448]
20. Vardar E, Caityurt O, Arikan E, Tuglu. Sleep quality and psychopathological features in obese binge eaters. Stress and Health. 2004; 20:35–41.
21. Bulik CM, Reichborn-Kjennerud T. Medical morbidity in binge eating disorder. Int J Eat Disord. 2003; 34:S39–S46. [PubMed: 12900985]
22. Stunkard AJ. Eating patterns and obesity. Psychiatr Q. 1959; 33:284–295. [PubMed: 13835451]
23. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. American Psychiatric Press; Washington, DC: 2000. Fourth Edition Text Revision
24. Hudson JI, Hiripi E, Pope HG Jr. Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. Biol Psychiatry. 2007; 61:348–358. [PubMed: 16815322]
25. Klump KL, Keel PK, Culbert KM, Edler C. Ovarian hormones and binge eating: Exploring associations in community samples. Psychol Med. 2008; 38:1749–1757. [PubMed: 18307829]
26. Monteleone P, Di Lieto A, Tortorella A, Longobardi N, Maj M. Circulating leptin in patients with anorexia nervosa, bulimia nervosa or binge-eating disorder: Relationship to body weight, eating patterns, psychopathology and endocrine changes. Psychiatry Res. 2000; 94:121–129. [PubMed: 10808037]

27. Telch CF, Agras WS. Obesity, binge eating, and psychopathology: Are they related? Int J Eat Disord. 1994; 15:53–61. [PubMed: 8124327]
28. Azarbad L, Corsica J, Hall B, Hood M. Psychosocial correlates of binge eating in Hispanic, African American, and Caucasian women presenting for bariatric surgery. Eat Behav. 2010; 11:79–84. [PubMed: 20188290]
29. Hsu LKG, Mulliken B, McDonagh B, Krupa Das S, Rand W, Fairburn CG, Rolls B, McCrory MA, Saltzman E, Shikora S, Dwyer J, Roberts S. Binge eating disorder in extreme obesity. Int J Obes Relat Metab Disord. 2002; 26:1398–1403. [PubMed: 12355338]
30. Powers PS, Perez A, Boyd F, Rosemurgy A. Eating pathology before and after bariatric surgery: A prospective study. Int J eat disord. 1999; 25:293–300. [PubMed: 10191994]
31. de Zwaan M. Binge eating disorder and obesity. Int J Obes Relat Metab Disord. 2001; 25:S51–S55. [PubMed: 11466589]
32. Adami GF, Gandolfo P, Bauer B, Scopinaro N. Binge eating in massively obese patients undergoing bariatric surgery. Int J Eat Disord. 1995; 17:45–55. [PubMed: 7894452]
33. Kalarchian MA, Wilson GT, Brolin RE, Bradley L. Binge eating in bariatric surgery patients. Int J Eat Disord. 1998; 23:89–92. [PubMed: 9429923]
34. Furberg H, Lichtenstein P, Pedersen NL, Thornton L, Bulik CM, Lerman C, et al. The STAGE cohort: A prospective study of tobacco use among Swedish twins. Nicotine Tob Res. 2008; 10:1727–1735. [PubMed: 18988069]
35. Lichtenstein P, Sullivan PF, Cnattingius S, Gatz M, Johansson S, Carlstrom E, et al. The Swedish Twin Registry in the third millennium: An update. Twin Res Hum Genet. 2006; 9:875–882. [PubMed: 17254424]
36. First, M.; Gibbon, M.; Williams, J.; Spitzer, P.; Staff, M. SCID Screen Patient Questionnaire (SSPQ) and SCID SCREEN Patient Questionnaire-Extended (SSPQ-X), Computer Program for Windows TM, Software Manual. American Psychiatric Press, Inc.; Washington, D.C.: 1999.
37. SAS Institute Inc.. SAS/STAT® Software: Version 9. SAS Institute, Inc; Cary, NC: 2004.
38. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and long-term weight gain in women and men. N Engl J Med. 2011; 364:2392–2404. [PubMed: 21696306]
39. Ulman TF, Von Holle A, Torgersen L, Stoltenberg C, Reichborn-Kjennerud T, Bulik CM. Sleep disturbances in binge eating disorder during and after pregnancy. Manuscript submitted for publication (copy on file with author).
40. Chaput JP, Després JP, Bouchard C, Tremblay A. Longer sleep duration associates with lower adiposity gain in adult short sleepers. Int J Obesity. 2011; 34:1291–1297.
41. Reiter RJ, Tan DX, Korkmaz A, Ma S. Obesity and metabolic syndrome: Association with chronodisruption, sleep deprivation, and melatonin suppression. Ann Intern Med. in press. Epub 2011 Jun 13.
42. Brondel L, Romer MA, Nougues PM, Touyarou P, Davenne D. Acute partial sleep deprivation increases food intake in healthy men. Am J Clin Nutr. 2010; 91:1550–1559. [PubMed: 20357041]
43. Omisade A, Buxton OM, Rusak B. Impact of acute sleep restriction on cortisol and leptin levels in young women. Physiol Behav. 2010; 99:651–656. [PubMed: 20138072]
44. Muccioli G, Tschop M, Papotti M, Deghenghi R, Heiman M, Ghigo E. Neuroendocrine and peripheral activities of ghrelin: Implications in metabolism and obesity. Eur J Pharmacol. 2002; 440:235–254. [PubMed: 12007539]
45. Ahima RS, Saper CB, Flier JS, Elmquist JK. Leptin regulation of neuroendocrine systems. Front Neuroendocrinol. 2000; 21:263–307. [PubMed: 10882542]
46. Chin-Chance C, Polonsky KS, Schoeller DA. Twenty-four-hour leptin levels respond to cumulative short-term energy imbalance and predict subsequent intake. Jour Clin Endocrinol Metab. 2000; 85:2685–2691. [PubMed: 10946866]
47. Wren AM, Seal LJ, Cohen MA, Brynes AE, Frost GS, Murphy KG, et al. Ghrelin enhances appetite and increases food intake in humans. J Clin Endocrinol Metab. 2001; 86:5992. [PubMed: 11739476]
48. Nakazato M, Murakami N, Date Y, Kojima M, Matsuo H, Kangawa K, et al. A role for ghrelin in the central regulation of feeding. Nature. 2001; 409:194–198. [PubMed: 11196643]

49. Schmid SM, Hallschmid M, Jauch-Chara K, Born J, Schultes B. A single night of sleep deprivation increases ghrelin levels and feelings of hunger in normal-weight healthy men. J Sleep Res. 2008; 17:331–334. [PubMed: 18564298]
50. Bagnasco M, Kalra PS, Kalra SP. Ghrelin and leptin pulse discharge in fed and fasted rats. Endocrinology. 2002; 143:726–729. [PubMed: 11796530]
51. Janas-Kozik M, Stachowicz M, Krupka-Matuszczyk I, Szymszal J, Krysta K, Janas A, et al. Plasma levels of leptin and orexin A in the restrictive type of anorexia nervosa. Regul Pept. 2011; 168:5–9. [PubMed: 21338627]
52. Scheid JL, De Souza MJ, Leidy HJ, Williams NI. Ghrelin but not PYY is related to change in body weight and energy availability. Med Sci Sports Exerc. 2011; 43:2063–2071. [PubMed: 21502892]
53. Wolfe BE, Jimerson DC, Orlova C, Mantzoros CS. Effect of dieting on plasma leptin, soluble leptin receptor, adiponectin and resistin levels in healthy volunteers. Clin Endocrinol. 2004; 61:332–338.
54. Artiga AI, Viana JB, Maldonado CR, Chandler-Laney PC, Oswald KD, Boggiano MM. Body composition and endocrine status of long-term stress-induced binge-eating rats. Physiol Behav. 2007; 91:424–431. [PubMed: 17498757]
55. Freeman LM, Gil KM. Daily stress, coping, and dietary restraint in binge eating. Int J Eat Disord. 2004; 36:204–212. [PubMed: 15282690]
56. Hagan MM, Wauford PK, Chandler PC, Jarrett LA, Rybak RJ, Blackburn K. A new animal model of binge eating: Key synergistic role of past caloric restriction and stress. Physiol Behav. 2002; 77:45–54. [PubMed: 12213501]
57. Wolff GE, Crosby RD, Roberts JA, Wittrock DA. Differences in daily stress, mood, coping, and eating behavior in binge eating and nonbinge eating college women. Addict Behav. 2000; 25:205–216. [PubMed: 10795945]
58. Kristiansen J, Persson R, Bjork J, Albin M, Jakobsson K, Ostergren PO, et al. Work stress, worries, and pain interact synergistically with modelled traffic noise on cross-sectional associations with self-reported sleep problems. Int Arch Occup Environ Health. 2011; 84:211–224. [PubMed: 20697733]
59. Vgontzas AN, Lin HM, Papaliaga M, Calhoun S, Vela-Bueno A, Chrousos GP, et al. Short sleep duration and obesity: The role of emotional stress and sleep disturbances. Int J Obesity. 2008; 32:801–809.
60. Tsigos C, Chrousos GP. Hypothalamic-pituitary-adrenal axis, neuroendocrine factors and stress. J Psychosomat Res. 2002; 53:865–871.
61. Epel E, Lapidus R, McEwen B, Brownell K. Stress may add bite to appetite in women: A laboratory study of stress-induced cortisol and eating behavior. Psychoneuroendocrinology. 2001; 26:37–49. [PubMed: 11070333]
62. Newman E, O'Connor DB, Conner M. Daily hassles and eating behaviour: the role of cortisol reactivity status. Psychoneuroendocrinology. 2007; 32:125–132. [PubMed: 17198744]
63. Coutinho WF, Moreira RO, Spagnol C, Appolinario JC. Does binge eating disorder alter cortisol secretion in obese women? Eat Behav. 2007; 8:59–64. [PubMed: 17174852]
64. Balbo M, Leproult R, Van Cauter E. Impact of sleep and its disturbances on hypothalamo-pituitary-adrenal axis activity. Int J Endocrinol. 2010; 2010:10–16.
65. Zakrzewska KE, Cusin I, Sainsbury A, Rohner-Jeanrenaud F, Jeanrenaud B. Glucocorticoids as counterregulatory hormones of leptin: Toward an understanding of leptin resistance. Diabetes. 1997; 46:717–719. [PubMed: 9075817]
66. Lutter M, Sakata I, Osborne-Lawrence S, Rovinsky SA, Anderson JG, Jung S, et al. The orexigenic hormone ghrelin defends against depressive symptoms of chronic stress. Nat Neurosci. 2008; 11:752–753. [PubMed: 18552842]
67. Rouach V, Bloch M, Rosenberg N, Gilad S, Limor R, Stern N, et al. The acute ghrelin response to a psychological stress challenge does not predict the post-stress urge to eat. Psychoneuroendocrinology. 2007; 32:693–702. [PubMed: 17560728]
68. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. Lancet. 1999; 354:1435–1439. [PubMed: 10543671]

69. Benedict C, Hallschmid M, Lassen A, Mahnke C, Schultes B, Schioth HB, et al. Acute sleep deprivation reduces energy expenditure in healthy men. Am J Clin Nutr. 2011; 93:1229–1236. [PubMed: 21471283]

70. Pope HG Jr. Lalonde JK, Pindyck LJ, Walsh T, Bulik CM, Crow SJ, et al. Binge eating disorder: A stable syndrome. Am J Psychiatry. 2006; 163:2181–2183. [PubMed: 17151172]

71. Tourangeau, R.; Smith, T., editors. Collecting Sensitive Information with Different Modes of Data Collection. Wiley; New York: 1998.

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Table 1

N (%) of responses to the sleep items for individuals with binge eating and no binge eating and for obese and not obese participants

Interview Question	Possible Response	BE n = 244	NoBE n = 3546	Obese n = 228	Not Obese n = 3562
Do you usually nap at least every second day?	No	197 (80.7)	2857 (81.0)	155 (68.3)	2899 (81.8)
	Yes	47 (19.3)	669 (19.0)	72 (31.7)	644 (18.2)
Do you think you sleep enough?	Definitely enough	27 (11.1)	371 (10.5)	18 (7.9)	380 (10.7)
	Mostly enough	87 (35.7)	1697 (48.1)	90 (30.6)	1694 (47.8)
	A bit too little	83 (34.0)	1073 (30.4)	75 (33.0)	1081 (30.5)
	Clearly not enough	29 (11.9)	286 (8.1)	25 (11.0)	290 (8.2)
	Far from enough	18 (7.4)	100 (2.8)	19 (8.4)	99 (2.8)
How well do you sleep on the whole?	Very well	28 (11.6)	877 (24.9)	33 (14.5)	872 (24.7)
	Pretty well	102 (42.2)	1641 (46.6)	96 (42.3)	1647 (46.6)
	Neither poorly nor well	43 (17.8)	507 (14.4)	32 (14.1)	518 (14.5)
	Pretty poorly	47 (19.4)	411 (11.7)	46 (20.3)	412 (11.6)
	Very poorly	22 (9.1)	84 (2.4)	20 (8.8)	86 (2.4)
Try to determine if you are a morning or night person with the following response options:	Morning person, definitely	38 (16.0)	656 (19.0)	37 (16.9)	657 (18.9)
	Morning person, to some degree	63 (26.6)	1108 (32.1)	54 (24.7)	1117 (32.2)
	Night person, to some degree	66 (27.8)	972 (28.2)	50 (22.8)	988 (28.5)
	Night person, definitely	70 (29.5)	715 (20.7)	78 (35.6)	707 (20.4)
Problems falling asleep	Never	26 (10.7)	760 (21.6)	39 (17.2)	747 (21.1)
	Seldom	57 (23.5)	1207 (34.3)	63 (27.8)	1201 (33.9)
	Sometimes	97 (39.9)	1177 (33.4)	77 (33.9)	1197 (33.8)
	Usually	43 (17.7)	304 (8.6)	28 (12.3)	319 (9.0)
	Always	20 (8.2)	75 (2.1)	20 (8.8)	75 (2.1)
Sleepy during work or free time	Never	7 (2.9)	135 (3.8)	8 (3.5)	134 (3.8)
	Seldom	32 (13.1)	865 (24.6)	33 (14.5)	864 (24.2)
	Sometimes	113 (46.3)	1891 (53.7)	125 (55.1)	1879 (53.1)
	Usually	66 (27.0)	513 (14.6)	39 (17.2)	540 (15.2)
	Always	26 (10.7)	120 (3.4)	22 (9.7)	124 (3.5)

Interview Question	Possible Response	BE n = 244	NoBE n = 3546	Obese n = 228	Not Obese n = 3562
Waking too early	Never	43 (17.6)	975 (27.7)	46 (20.4)	972 (27.5)
	Seldom	82 (33.6)	1204 (34.2)	67 (29.6)	1219 (34.5)
	Sometimes	87 (35.7)	1045 (29.7)	84 (37.2)	1048 (29.7)
	Usually	24 (9.8)	246 (7.0)	22 (9.7)	248 (7.0)
	Always	8 (3.3)	45 (1.3)	7 (3.1)	46 (1.3)
Not enough rest	Never	6 (2.5)	134 (3.8)	10 (4.4)	130 (3.7)
	Seldom	43 (17.6)	877 (25.0)	44 (19.5)	876 (24.8)
	Sometimes	101 (41.4)	1533 (43.6)	90 (39.8)	1544 (43.7)
	Usually	64 (26.2)	811 (23.1)	59 (26.1)	816 (23.1)
	Always	30 (12.3)	159 (4.5)	23 (10.2)	166 (4.7)
Disturbed sleep	Never	12 (4.9)	446 (12.7)	22 (9.8)	436 (12.3)
	Seldom	55 (22.5)	1244 (35.4)	56 (24.9)	1243 (35.2)
	Sometimes	112 (45.9)	1270 (36.1)	81 (36.0)	1301 (36.8)
	Usually	44 (18.0)	439 (12.5)	44 (19.6)	439 (12.4)
	Always	21 (8.6)	117 (3.3)	22 (9.8)	116 (3.3)

Table 2

Results from logistic regression analyses predicting lifetime binge eating from current sleep problems in women. Analysis 1 includes age and current cohabiting status as covariates. Analysis 2 includes age and current cohabiting status as covariates and adjusts for lifetime depression diagnosis. Analysis 3 includes age and current cohabiting status as covariates and adjusts for lifetime depression diagnosis and current obesity status.

Sleep Variables (Referent Response for the Odds Ratios)	Response Comparison	Analysis 1		Analysis 2		Analysis 3	
		Odds Ratio (95% CI)	χ^2 (p-value)	Odds Ratio (95% CI)	χ^2 (p-value)	Odds Ratio (95% CI)	χ^2 (p-value)
Usually nap at least every second day (No)	Yes	1.01 (0.72, 1.40)	0.00 (.97)	0.85 (0.61, 1.20)	0.89 (.35)	0.82 (0.58, 1.16)	1.29 (.26)
Sleep enough (Definitely enough)	Mostly enough	0.79 (0.51, 1.24)	19.39 (.001)	0.78 (0.49, 1.22)	12.36 (.015)	0.78 (0.68, 1.22)	11.81 (.019)
	A bit too little	1.21 (0.77, 1.91)		1.08 (0.68, 1.70)		1.07 (0.68, 1.70)	
	Clearly not enough	1.58 (0.89, 2.79)		1.23 (0.69, 2.20)		1.23 (0.69, 2.19)	
	Far from enough	3.22 (1.69, 6.11)		2.36 (1.22, 4.54)		2.24 (1.15, 4.34)	
How well you sleep (Very well)	Pretty well	1.92 (1.25, 2.95)	44.00 (.001)	1.71 (1.11, 2.63)	24.94 (.001)	1.70 (1.10, 2.62)	23.90 (.001)
	Neither poorly nor well	2.64 (1.62, 4.33)		2.08 (1.25, 3.44)		2.06 (1.25, 3.41)	
	Pretty poorly	3.69 (2.25, 6.03)		2.52 (1.52, 4.21)		2.48 (1.49, 4.13)	
	Very poorly	9.01 (4.87, 16.67)		5.80 (3.01, 11.16)		5.50 (2.85, 10.61)	
Morning/night person (Morning person, definitely)	Morning person, to some degree	0.90 (0.58, 1.37)	6.73 (.09)	0.85 (0.55, 1.29)	3.57 (.32)	0.85 (0.55, 1.30)	3.02 (.39)
	Night person, to some degree	1.06 (0.70, 1.61)		0.96 (0.63, 1.46)		0.96 (0.63, 1.46)	
	Night person, definitely	1.45 (0.95, 2.20)		1.20 (0.79, 1.84)		1.17 (0.77, 1.80)	
Problems falling asleep (Never)	Seldom	1.34 (0.84, 2.14)	42.85 (.001)	1.28 (0.80, 2.07)	23.06 (.001)	1.28 (0.80, 2.06)	22.23 (.001)
	Sometimes	2.23 (1.43, 3.49)		1.86 (1.18, 2.91)		1.85 (1.18, 2.90)	
	Usually	3.82 (2.31, 6.34)		2.51 (1.49, 4.23)		2.49 (1.48, 4.19)	

Sleep Variables (Referent Response for the Odds Ratios)	Response Comparison	Analysis 1		Analysis 2		Analysis 3	
		Odds Ratio (95% CI)	χ^2 (p-value)	Odds Ratio (95% CI)	χ^2 (p-value)	Odds Ratio (95% CI)	χ^2 (p-value)
	Always	7.15 (3.84, 13.30)		4.43 (2.30, 8.54)		4.13 (2.12, 8.03)	
Sleepy during work or free time (Never)	Seldom	0.69 (0.31, 1.54)	36.54 (.001)	0.63 (0.28, 1.42)	19.49 (.001)	0.63 (0.28, 1.43)	18.33 (.001)
	Sometimes	1.07 (0.50, 2.29)		0.85 (0.40, 1.82)		0.85 (0.39, 1.82)	
	Usually	2.05 (0.94, 4.52)		1.36 (0.61, 3.01)		1.35 (0.61, 3.00)	
	Always	3.91 (1.67, 9.11)		2.05 (0.85, 4.92)		1.97 (0.82, 4.75)	
Waking too early (Never)	Seldom	1.58 (1.08, 2.30)	20.17 (.001)	1.44 (0.98, 2.12)	9.20 (.06)	1.44 (0.98, 2.12)	8.85 (.07)
	Sometimes	2.01 (1.37, 2.94)		1.57 (1.06, 2.33)		1.57 (1.05, 2.32)	
	Usually	2.30 (1.33, 3.97)		1.63 (0.93, 2.87)		1.62 (0.92, 2.86)	
	Always	4.43 (1.95, 10.08)		3.35 (1.47, 7.63)		3.19 1.37, 7.36)	
Not enough rest (Never)	Seldom	1.11 (0.47, 2.60)	17.82 (.002)	1.07 (0.45, 2.56)	9.12 (.06)	1.09 (0.45, 2.63)	8.53 (.08)
	Sometimes	1.41 (0.62, 3.22)		1.21 (0.52, 2.82)		1.23 (0.53, 2.90)	
	Usually	1.67 (0.71, 3.89)		1.27 (0.53, 3.02)		1.28 (0.54, 3.07)	
	Always	4.14 (1.71, 10.07)		2.61 (1.05, 6.51)		2.57 (1.02, 6.44)	
Disturbed sleep (Never)	Seldom	1.68 (0.90, 3.15)	50.77 .001)	1.62 (0.86, 3.04)	29.03 .001)	1.62 (0.86, 3.06)	28.35 .001)
	Sometimes	3.45 (1.90, 6.27)		2.80 (1.53, 5.13)		2.80 (1.53, 5.13)	
	Usually	3.95 (2.06, 7.55)		2.69 (1.40, 5.17)		2.64 (1.37, 5.09)	
	Always	7.72 (3.70, 16.09)		5.07 (2.37, 10.85)		4.84 (2.26, 10.35	

Int J Eat Disord. Author manuscript; available in PMC 2013 July 01.

Table 3

Results from logistic regression analyses predicting current obesity from current sleep problems in women. Analysis 1 includes age as a covariate. Analysis 2 includes age as a covariate and adjusts for lifetime depression diagnosis.

Sleep Variables (Referent Response for the Odds Ratios)	Response Comparison	Analysis 1		Analysis 2	
		Odds Ratio 95% CI)	χ^2 p-value)	Odds Ratio 95% CI)	χ^2 p-value)
Usually nap at least every second day (No)	Yes	2.03 (1.51, 2.73)	15.53 (.001)	1.94 (1.45, 2.63)	14.07 (.001)
Sleep enough Definitely enough)	Mostly enough	1.13 (0.66, 1.93)	12.02 (.018)	1.13 (0.66, 1.93)	10.45 (.034)
	A bit too little	1.40 (0.81, 2.40)		1.36 (0.79, 2.35)	
	Clearly not enough	1.63 (0.84, 4.25)		1.53 (0.79, 2.96)	
	Far from enough	3.66 (1.82, 7.36)		3.41 (1.69, 6.89)	
How well you sleep (Very well)	Pretty well	1.53 (1.03, 2.82)	27.30 (.001)	1.49 (1.00, 2.23)	23.62 (.001)
	Neither poorly nor well	1.57 (0.96, 2.56)		1.48 (0.90, 2.42)	
	Pretty poorly	2.73 (1.72, 4.32)		2.53 (1.58, 4.04)	
	Very poorly	5.66 (3.09, 10.37)		5.17 (2.79, 9.59)	
Morning/night person (Morning person, definitely)	Morning person, to some degree	0.96 (0.62, 1.47)	21.29 (.001)	0.94 (0,61, 1.45)	19.75 (.001)
	Night person, to some degree	1.03 (0.66, 1.60)		1.01 (0.65, 1.57)	
	Night person, definitely	2.26 (1.49, 3.43)		2.16 (1.42, 3.29)	
Problems falling asleep Never)	Seldom	1.04 (0.69, 1.55)	16.39 (.003)	1.03 (0.69, 1.54)	14.19 (.007)
	Sometimes	1.26 (0.86, 1.86)		1.20 (0.81, 1.78)	
	Usually	1.71 (1.03, 2.83)		1.56 (0.92, 2.63)	
	Always	5.12 (2.76, 9.50)		4.57 (2.45, 8.50)	
Sleepy during work or free time Never)	Seldom	0.74 (0.32, 1.71)	21.40 (.001)	0.73 (0.31, 1.69)	18.09 (.002)
	Sometimes	1.30 (0.58, 2.89)		1.24 (0.55, 2.77)	
	Usually	1.49 (0.64, 3.50)		1.37 (0.58, 3.23)	
	Always	3.46 (1.38, 8.69)		3.04 (1.20, 7.73)	
Waking too early (Never)	Seldom	1.09 (0.74, 1.61)	10.04 (.040)	1.07 (0.73, 1.58)	7.39 (.12)
	Sometimes	1.52 (1.05, 2.19)		1.42 (0.98, 2.07)	
	Usually	1.70 (1.02, 2.85)		1.56 (0.92, 2.64)	

Sleep Variables (Referent Response for the Odds Ratios)	Response Comparison	Analysis 1		Analysis 2	
		Odds Ratio 95% CI)	χ^2 p-value)	Odds Ratio 95% CI)	χ^2 p-value)
	Always	2.81 (1.22, 6.47)		2.57 (1.12, 5.86)	
Not enough rest Never)	Seldom	0.62 (0.30, 1.26)	10.90 (.028)	0.61 (0.30, 1.25)	9.26 (.06)
	Sometimes	0.73 (0.37, 1.47)		0.71 (0.36, 1.41)	
	Usually	0.90 (0.44, 1.82)		0.84 (0.41, 1.71)	
	Always	1.72 (0.78, 3.81)		1.53 (0.69, 3.42)	
Disturbed sleep Never)	Seldom	0.84 (0.51, 1.38)	19.98 (.001)	0.83 (0.50, 1.37)	17.26 (.001)
	Sometimes	1.14 (0.71, 1.84)		1.09 (0.68, 1.77)	
	Usually	1.73 (1.02, 2.93)		1.60 (0.94, 2.72)	
	Always	3.32 (1.78, 6.20)		3.05 (1.62. 5.73)	