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ORIGINAL ARTICLE

Night Eating Syndrome among patients with major depressive disorder

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Background Eating disorders (ED), are serious illnesses that often have a variety of medical complications and have significant psychiatric comorbidity. The Night Eating Syndrome (NES) is one of these eating disorders. It was first described in 1955 by Stunkard as a disorder consisting of morning anorexia, evening hyperphagia, and insomnia. It is strongly correlated with both obesity and depression in a bidirectional relationship with each one. Data regarding these relationships are scarce and conflicting.

Objective This study was undertaken to estimate the prevalence rate of night eating syndrome among patients with depression and its relation to antidepressant drugs.

Method The present study was conducted on 400 psychiatric outpatients aged 18-60 years and of both sexes in Alexandria Main University Hospitals over the period from February 2013 to July 2013. Patients were divided into two equal groups (200 patients each). These two groups were one depressed patients without treatment and the other depressed patents on treatment for at least 2 months. All subjects were subjected to psychiatric clinical interview by the researcher for complete history taking, assessment and diagnosis of major depressive disorder based on Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition criteria (DSM-IV-TR), anthropometric measurements and diagnosis of NES based on the proposed diagnostic criteria of NES and night eating questionnaire.

Results NES subjects constituted 26.5% among group A, while among group B subjects constituted 9%. Among group A, NES +ve cases were associated with higher BMI with positive correlation between NEQ scores and BMI. Where among group B, NES +ve cases were associated with higher BMI with positive correlation between NEQ scores and BMI, There was statistical difference between the +ve and –ve cases of NES concerning type of antidepressant received. This was found with mirtazapine, trazodone and clomipramine with the highest NEQ scores. On comparing +v cases in both groups, BMI was lower significantly among group B than group A.

Conclusions Night eating syndrome is common with MDD, also, it is one of the causes of overweight and obesity. There is a major role of some antidepressants in treating NES as well as there is a partial role of others in inducing NES.

Keywords Antidepressant, Depression, Night-eating
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INTRODUCTION

The night eating syndrome (NES) was first described in Stunkard *et al.,* (1955) as a disorder consisting ofmorning anorexia, evening hyperphagia, and insomnia. The presence of nocturnal ingestions (awakening to eat) was added to these criteria later (Stunkard *et al.,* 1955).

The prevalence of NES is estimated at 1.5% in the general population (Geliebter, 2002), 6% (Ceru-Bjork *et al.,* 2001) to 14% in obesity clinics, and 8% to 42% in preoperative bariatric surgery obese patients in the United States. Such a wide range of estimates is most certainly

influenced by varying assessment methods (e.g. survey vs. interview) and diagnostic criteria. However, in general, these results suggest that NES is more prevalent with increasing adiposity. Because NES occurs commonly among the obese and has been reported to interfere with weight loss attempts (Gluck *et al.,* 2001), it may be a useful target for treatment in a subgroup of the overweight/obese population.

NES has been associated with life stress (Stunkard *et al.,* 1955), psychoneurotocism (Birketvedt *et al.,* 1999), depression (Adami *et al.,* 1999; Birketvedt *et al.,* 1999), low mood (Gluck *et al.,* 2001), and adverse reactions to weight loss (Stunkard *et al.,* 1996).

Birketvedt *et al.,* (1999) reported that nighttime awakenings were far more common among night eaters than among controls, and more than one-half of these awakenings were associated with food intake. The typical neuroendocrine characteristics were an attenuation of nocturnal rises in secretions of melatonin and leptin and increased diurnal secretion of cortisol. Cortisol, melatonin, and leptin are regulatory hormones with typical circadian rhythms that regulate various physiological and metabolic functions (Sinha *et al.,* 1996).

Persons with NES may have a general vulnerability to stress, perceiving life event stress as less controllable and/or predictable. As NES tends to occur during periods of life event stress, the ingestion of food may be a learned aspect of mood regulation. Perhaps NES involves a genetic or hormonal predisposition, triggered by life event stress that results in a de-synchronization of the sleep/wake and eating cycles among vulnerable individuals. There is evidence that NES may involve a genetic component (Root *et al.,* 2010), but to date, no studies have focused on specific genes involved in this syndrome. Genetic studies would greatly enhance our understanding of NES. Identification of individuals at risk for the development of NES as well as the mechanisms by which NES develops and is maintained remains important goals for future research (Vander Wal, 2012).

This study suggests a relation among depression, antidepressants (AD), obesity, and NES. As obesity might be a complication of NES, NES may be a sign or associated manifestation of depression. Moreover, NES may be improved or induced by the use of certain ADs.

AIM

This work aimed to estimate the point prevalence of NES in a sample of patients with major depressive disorder (MDD) and its relation to the used AD drug..

PATIENTS

The present study was carried out among psychiatric outpatients in Alexandria Main University Hospitals over a period of 6 months from February 2013. Patients were divided into two groups:

1- Group A: 200 newly diagnosed patients with major depressive disorder according to *Diagnostic and statistical manual of mental disorders*, 4th ed., text revision (DSM-IV-TR) criteria and did not receive ADs yet.

2- Group B: patients diagnosed as having depression according to DSM-IV-TR criteria and received AD therapy for at least 2 months.

Inclusion criteria were as follows: (a) patients diagnosed as having depression according to DSMIV- TR criteria; (b) patients aged from 18 to 60 years, (c) both sexes were included, and (d) patient's acceptance by signing a written informed consent. Exclusion criteria were as follows: (a) presence of organic illness that contributes to either obesity or depression, for example, thyroid dysfunction; (b) refusal of participation (not providing consent); (c) depression with psychotic features; (d) patients receiving more than one AD; (e) other axis I or II disorders according to DSM-IV-TR; and (f) presence of sleep-related eating disorder.

After approval of Ethical Committee of Faculty of Medicine Alexandria University, a written informed consent was obtained from all patients after explanation of the aim and method of the study.

METHODS

This study was carried out using a cross-sectional approach, and the entire studied sample was subjected to the following tools:

1- Predesigned structured interview, including sociodemographic data, medical history, psychiatric history, and drug history including the type of AD in use.

2- Clinical psychiatric assessment and diagnosis, based on DSM-IV-TR (American Psychiatric Association, 2000) for diagnosis of major depressive disorder and exclusion of other axis I disorders.

3- Anthropometric measurements for the patients (Jelliffe *et al.,* 1989), including the weight in kilograms, and height was measured in centimeter scale. The BMI was calculated using the following formula: BMI= weight (kg)/ height^2 (m^2).

Weight status was classified according to the WHO Consultation Report on Obesity (160) (161) that BMI less than 18.5 kg/m^2 is classified as underweight, BMI 18.5–24.9 kg/m^2 is considered as normal weight, BMI 25–29.9 kg/m^2 is considered as overweight, BMI 30–34.9 kg/m^2 is considered as obesity grade I, BMI 35–39.9 kg/m^2 is considered as obesity grade II, and BMI more than or equal to 40 kg/m is considered as obesity grade III.

4- Diagnosis of NES cases based on the proposed diagnostic criteria for NES and night eating questionnaire (NEQ) (Allison *et al.,* 2010). The NEQ is a measure of NES symptom severity, with scores ranging from 0 to 52

(Allison *et al.,* 2008a, 2008b). Its reliability and validity have been established, and it has a positive predictive value of 62% using a cutoff score of 25 in a psychiatric population (Allison *et al.,* 2010). The scale can measure morning anorexia (questions 1–2), evening hyperphagia (questions 3–5), mood and sleep habits (questions 6–9), and nocturnal ingestion (questions 10–14). Items are scored on 0–4 Likert scale, and items 1, 4, and 14 are reverse scored so that the higher values reflect the greater symptomatology. The total score provides a range from 0 to 52 points. The cutoff point score used in this study was 25 (Allison *et al.,* 2008a, 2008b).

Statistical analysis (Allison *et al.*, 2008a, 2008b)

Data were fed to the computer and analyzed using IBM Statistical Package for the Social Sciences software package, version 20.0. Qualitative data were described using number and percent. Quantitative data were described using range (minimum and maximum) mean, SD, and median. Comparison between different groups regarding categorical variables was tested using c^2 test. When more than 20% of the cells have expected count less than 5, correction for c^2 was conducted using Fisher's exact test or Monte Carlo correction. The distributions of quantitative variables were tested for normality using Kolmogorov–Smirnov test, Shapiro–Wilk test, and D'Agstino test, and also Histogram and QQ plot were used for vision test. If it reveals normal data distribution, parametric tests was applied. If the data were abnormally distributed, non-parametric tests were used.

For normally distributed data, comparison between the two studied groups was done using independent t test. However, for abnormally distributed data, comparison between two studied groups were done using Mann–Whitney test. Kruskal–Wallis test was used to compare between different groups, and pair-wise comparison was assessed using Mann–Whitney test. Correlations between two quantitative variables were assessed using Spearman coefficient. Significance of the obtained results was judged at the 5% level.

RESULTS

There was no statistically significant difference between the two groups regarding sex (P= 0.825). There was a statistically significant difference between the two groups regarding age (P= 0.012). Patients of group A were significantly older than group B (Table 1).

There was a statistically significant difference between the two groups regarding BMI (P <0.001). Moreover, the difference between overweight and obese patients between both groups was statistically significant (83 and 57%, respectively) ($P \leq 0.001$) (Table 2).

There was a statistically significant difference between the two groups regarding the number of cases fulfilling the criteria for NES according to the NEQ scores (P <0.001) (Table 3).

The table reveals no significant difference betweenNES cases of both groups concerning age and sex.Concerning the sex, in both groups, females were a risk target for NES. In group A, females constitute 67.9% compared with 32.1% males. The same finding was in group B, as females were 77.8% compared with 22.2% males.

Patients with comorbid MDD and NES had significantly higher BMI than those without NES in both naive patients with MDD (group A) and patients with MDD with history of AD use (group B) (P <0.031) (Table 4).

There was a statistical difference between the two groups concerning type of AD received. This was found with mirtazapine (P= 0.002), trazodone (P= 0.008), and clomipramine (P= 0.041). Multivariate analysis showed that diagnosis of NES was significantly correlated with being younger in age in the onset of MDD in drug-naive patients (t= 2.231, P= 0.034) and being obese with BMI more than 30 (t= 3.108, P= −0.002) and those diagnosed with atypical depression (χ^2= 5.658, P= 0.017) in both groups. The presence of NES in patients withMDD already on AD (group B) was correlated with the type of AD being significantly higher in those receiving mirtazapine, trazodone, or clomipramine.

DISCUSSION

The reciprocal links associating obesity and depression have been reported in many clinical and epidemiological studies and seem to correlate more in adulthood, especially among women (Pattern *et al.,* 2009). Among their interlinking relations, some literature studies view obesity to be a clinical manifestation of a subtype of depression similar to that of atypical depression, whereas others view that they are separate constructs, having an influencing effect on each other, even being a risk factor to the development of one another (Rosmond, 2004; Blaine, 2008). Recently, NES had entered this vicious circle by means of being strongly correlated with both obesity and depression in a bidirectional relationship with each one. NES was first noted among obese patients. The association between NES and obesity has also been supported, with rate estimates suggesting that NES is more common among obese persons (6–16%) (Ceru-Bjork *et al.,* 2001) compared with the general population (1.5%).

Regarding NES relation with depression, it is not clear whether depression is a cause, an effect, or a feature of NES. In such a context, this descriptive study was conducted to compare the NES in patients diagnosed as major depression with and without treatment.

Table 1: Patient's demographic and Anthropometric data:

	Group A (*n*= 200)		Group B (*n*= 200)		Test of sig.	*p*
	No.	%	No.	%		
Sex						
Male	56	28.0	58	29.0	χ^2= 0.049	0.825
Female	144	72.0	142	71.0		
Age (years)						
Min.–Max.	20.0–58.0		22.0–56.0			
Mean±SD	39.58±9.61		37.37±7.86		*t*= 2.517*	0.012*
Median	40.50		38.0			
BMI (kg/m²)						
Under weight (<18.8)	0	0.0	0	0.0		
Normal (18.5 - <25)	34	17.0	86	43.0		
Over weight(25 - <30)	57	28.5	74	37.0	χ^2=57.492*	<0.001*
Obese (I) (30 - <35)	73	36.5	24	12.0		
Obsess (II) (35 - <40)	24	12.0	12	6.0		
Obese (III) (≥ 40)	12	6.0	4	2.0		
Min.–Max.	22.80-42.60		21.60-42.60			
Mean±SD	30.55±4.87		27.27±4.38		*t*= 7.092*	<0.001*
Median	30.10		26.20			

χ2: Chi square test. *t*: student *t* test. *p: p* value for comparing between the studied groups. *: Statistically significant at $p \leq 0.05$.

Table 2: Prevalence of night eating syndrome in both studied according NEQ score:

	Group A (*n*= 200)		Group B (*n*= 200)		χ^2	*P*
	No.	%	No.	%		
NEQ score						
Without NES (score <25)	147	73.5	182	91.0	20.977*	<0.001*
With NES (score ≥25)	53	26.5	18	9.0		

χ2: Chi square test. *p: p* value for comparing between the studied groups. *: Statistically significant at $p \leq 0.05$.

Table 3: Comparison between NES cases in both groups according to demographic and Anthropometric characteristics:

	NES cases in Group A (*n*= 53)		NES cases in Group B (*n*= 18)		Test of sig.	*p*
	No.	%	No.	%		
Sex						
Male	17	32.1	4	22.2	χ^2= 0.626	0.429
Female	36	67.9	14	77.8		
Age (years)						
Min.–Max.	20.0–54.0		22.0–44.0			
Mean±SD	37.19±8.82		33.89±7.17		*t*= 1.433	0.156
Median	39.0		35.0			
BMI					χ^2	*P*
18.5 - <25 Normal weight	2	3.8	0	0.0	0.699	1.000
25 - <30 Over weight	13	24.5	10	55.6	5.906*	0.015*
30 - <35 Obese GI	22	41.5	2	11.1	5.549*	0.018*
35 - <40 Obese GII	16	30.2	6	33.3	0.062	0.803
χ^2(^{MC}p)	8.015*(0.031*)					

χ2: Chi square test, MC: Monte Carlo; *t:* student *t* test. *p: p* value for comparing between the studied groups. *: Statistically significant at $p \leq 0.05$.

Table 4: Patient's demographic and Anthropometric data:

Group B	NES diagnosis				χ^2	*P*
	Cases without NES (*n*= 182)		Cases with NES (*n*= 18)			
	No.	%	No.	%		
Type of antidepressant						
Citalopram	48	26.4	2	11.1	2.035	^{FE}p = 0.252
Sertraline	64	35.2	4	22.2	1.223	0.269
Fluoxetine	10	5.5	0	0.0	1.041	^{FE}p = 0.604
Paroxetine	26	14.3	2	11.1	0.137	^{FE}p = 1.000
Clomipramine	2	1.1	2	11.1	8.378*	^{FE}p =0.041*
Amitriptyline HCL	4	2.2	0	0.0	0.404	^{FE}p =1.000
Trazodone	0	0.0	2	11.1	20.426*	^{FE}p =0.008*
Mirtazapine	12	6.6	6	33.3	14.300*	^{FE}p = 0.002*
Venlafaxine	10	5.5	0	0.0	1.041	^{FE}p = 0.604
Bupropion	4	2.2	0	0.0	0.404	^{FE}p =1.000
Fluvoxamine	2	1.1	0	0.0	0.200	^{FE}p =1.000
χ^2(^{MC}p)	27.256* (<0.001*)					

χ^2: Chi square test. FE: Fisher Exact test. MC: Monte Carlo test. *: Statistically significant at $p \leq 0.05$.

In the current study sample, 26.5% of the patients with depression who did not take AD (group A) met the proposed diagnostic criteria for NES and scored more than or equal to 25 at NEQ, which was significantly higher than the patients on ADs at 9% (group B). The explanation proposed behind the relatively high percentage among group A includes stress, sleep disturbance, and the lack of medications taken by the patients at that time which proved to be effective in treating NES. Although AD may have a role in precipitating NES in some cases (9%) as shown in the current study, at the same time, they had a major role in decreasing the percentage as such either by treating depression with consequently decreasing stress, improving sleep and eating pattern, or by its direct effect on NES independent from depression treatment.

In accordance with our findings, a study described 17 persons having NES who were treated with sertraline and reported that an AD (sertraline) improved the NES, which includes the presence of depression, and thus raised the question of whether it did more than relieve the depression. The low correlation between improvement in depression and NES (*r*= 0.28, *P*= 0.40) suggested that sertraline improved the NES independent of its effect on depression (O'Reardon *et al.,* 2006; Stunkard *et al.,* 2006).

Focusing on the prevalence of NES in depressed patients, our results revealed 26.5% of the cases among group A (naive) and 9% among group B (on treatment). A comparable study of Orhan *et al.,* (2011) reported that the prevalence of NES was reported to be significantly higher in patients with depression (35.2%) than in a healthy control group (19.2%) (Orhan *et al.,* 2011). The difference was that their inclusion criteria were depressed patients (weather taking ADs or not), whereas in our study, we divided the patients into two groups.

Patients with NES were reported to be more likely to meet the criteria for major depressive disorders in a study of De Zwaan *et al.,* (2006) with percentage of 56%. Comparisons of patients with and without NES showed that patients with NES scored significantly higher than controls in the Zung Depression Scale (Gluck *et al.,* 2001) and Beck Depression Inventory (Lundgren *et al.,* 2008).

In the present work, NES was significantly correlated with being younger in age in the onset of MDD in drug-naive patients (*t*= 2.231, *P*= 0.034), being obese with BMI more than 30 (*t*= 3.108, *P*= −0.002), and those diagnosed with atypical depression (χ^2= 5.658, *P*= 0.017) in both groups. There was a study nearly matching the results of the present study, where NES appears to be more prevalent in younger age (Striegel-Moore *et al.,* 2006). However, when we compared NES-positive cases between both groups regarding age, it was found that the difference was not statistically significant (*P*= 0.156), and this confirm that NES is more common at younger age whether the patient was exposed to AD or not.

Regarding sex, the females among NES-positive cases in both groups were predominant than males, with percentages of 67.9% female compared with 32.1% males in group A and 77.8% females compared with 22.2% males in group B. However, this difference between both groups was not statistically significant (*P*= 0.429). Data regarding the relationship between sex and NES are variable. The present study was in agreement with the studies by Greeno *et al.,* (1995) and Stunkard *et al.,* (1996). Women were more likely to have NES, which implicates being female as a risk factor for NES.

Two important relations were noticed in the present work. One is the relation between depression and obesity and the other is the relation between NES and obesity.

Regarding the relation between depression and obesity, each group revealed predominance of overweight and obese patients compared with the normal weight ones (83% compared with 17% in group A and 57% compared with 43% in group B), and this difference among each group was statistically significant (P <0.001 in group A and P= 0.048 in group B). This statistical difference in both groups might be the result of strong association between depression and obesity (Rosmond, 2004; Heo *et al.,* 2006).

Obesity can be seen as an inflammatory state, as weight gain has been shown to activate inflammatory pathways (Bremmer *et al.,* 2008) and inflammation in turn has been associated with depression (Milaneschi *et al.,* 2009). Inflammation could be the mediator of the association. Moreover, the hypothalamic–pituitary–adrenal axis (HPA-axis) might play a role, because obesity might involve HPA-axis dysregulation (Pasquali and Vicennati, 2000) and HPA-axis dysregulation is well known to be involved in depression (Belanoff *et al.,* 2001; Walker, 2001).

In addition to biological mechanisms, psychological pathways should be mentioned. Body dissatisfaction and decreased self-esteem are risk factors for depression (Beesdo *et al.,* 2010). Disturbed eating patterns and eating disorders, as well as experiencing physical pain as a direct consequence of obesity, are also known to increase the risk of depression (Gadalla and Piran, 2008).

The significant decrease in BMI in the group B than group A might be the result of treatment of depression and thus, improvement of stress, sleep, some symptoms as hyperphagia, and all of the aforementioned factors.

Regarding the relation between NES and obesity, it was revealed in the present study that BMI was significantly higher in patients with NES among both groups. This statistical difference was confirmed by the positive correlation between NEQ score and BMI among both groups. This was in accordance with the study by Colles *et al.,* (2007) (χ^2= 22.7, P <0.001) and the other study by Aronoff *et al.,* (2001) (χ^2= 7.1, P= 0.008). Both showed a strong relationship between overweight, obesity, and NES. This can be described as the coherence of the behavioral and neuroendocrine patterns of persons with NES at night due to their circadian shift together with the normal low basal metabolic rate at that time. These factors combine together to result in higher BMI (Birketvedt *et al.,* 1999).

The behavioral pattern stated as patients wake up at night and eat more frequently and take higher calories nighttime snacks, which is suggested to happen to restore the disrupted sleep of the night eaters. It has been reported that this pattern of eating increases the availability of tryptophan for transport into the brain and conversion into serotonin, resulting in facilitation of sleep (Yokogoshi and Wurtman, 1986).

We found in the present work, a statistical significant association between certain types of ADs and the presence of NES. Among group B studied sample the difference between −ve and +ve cases of NES was significant with mirtazapine, trazodone, and clomipramine. No studies were found discussing the relation between NES and the type of AD used. However, there is as association between them and weight gain, where NES might be one of its possible causes.

There are various hypotheses that can explain why patients gain weight when they take ADs. One is an increased carbohydrate craving, which is attributed to their strong anti-histaminergic effect. Another postulated mechanism for weight gain is neurotransmitter modulation at the hypothalamic level, which changes the regulation of body fat stores and increases energy efficiency (Garland *et al.,* 1988; Zimmerman *et al.,* 2003).

CONCLUSION

1- The prevalence of night eating syndrome in patients with depression without AD treatment was 26.5%, whereas among patients with depression taking ADs at least for 2 months was 9%.

2- There is a positive correlation between NES and BMI. So, BMI was found to be significant risk factor for NES.

3- NES is one of the causes of overweight and obesity, and treatment of NES could treat overweight and obesity particularly in MDD.

4- As there is a relationship between NES and certain types of ADs, there is a major role of some ADs in treating NES as well as there is a partial role of others in inducing NES.

LIMITATION

1. The lack of a control in the present study makes the validation of the results of data further assessments.

2. The study sample was small and consisted only of depressed patients. Consequently, it would be inappropriate to generalize our findings.

3. There were no available studies finding the relation between NES and ADs, and this makes this part in our study lacking comparison with other works.

4. This study was cross-sectional; therefore, the causality relationship cannot be tested and also the possibility that NES may be a part of depression cannot be excluded in this study design. Thus, longitudinal studies are necessary to better understand the relationship between depression and NES.

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CONFLICTS OF INTEREST

There are no conflicts of interest.

REFERENCE

Adami GF, Meneghelli A, Scopinaro N (1999). Night eating and binge eating disorder in obese patients. Int J Eat Disord 25:335–338.

Allison KC, Engel SG, Crosby RD, de ZM, O'Reardon JP, Wonderlich SA, *et al*. (2008a). Evaluation of diagnostic criteria for night eating syndrome using item response theory analysis. Eat Behav 9:398–407.

Allison KC, Lundgren JD, O'Reardon JP, Martino NS, Sarwer DB, Wadden TA, *et al*. (2008b). The night eating questionnaire (NEQ): psychometric properties of a measure of severity of the night eating syndrome. Eating Behav 9:62–72.

Allison KC, Lundgren JD, O'Reardon JP, Geliebter A, Gluck ME, Vinai P, *et al*. (2010). Proposed diagnostic criteria for night eating syndrome. Int J Eat Disord 43:241–247.

American Psychiatric Association (2000).Diagnostic and statistical manual of mental disorder, text revision. 4th ed. Washington, DC: American Psychiatric Association.

Aronoff NJ, Geliebter A, Zammit G (2001). Gender and body mass index are related to the night-eating syndrome in obese outpatients. J Am Diet Assoc 101:102–104.

Beesdo K, Jacobi F, Hoyer J, Low NC, Höfler M, Wittchen HU (2010). Pain associated with specific anxiety and depressive disorders in a nationally representative population sample. Soc Psychiatry Psychiatr Epidemiol 45:89–104.

Belanoff JK, Kalehzan M, Sund B, Fleming Ficek SK, Schatzberg AF (2001). Cortisol activity and cognitive changes in psychotic major depression. Am J Psychiatry 158:1612–1616.

Birketvedt GS, Florholmen J, Sundsfjord J, Osterud B, Dinges D, Bilker W, *et al*. (1999). Behavioral and neuroendocrine characteristics of the night eating syndrome. JAMA 282:657–663.

Blaine B (2008). Does depression cause obesity? A meta-analysis of longitudinal studies of depression and weight control. J Health Psychol 13:1190- –1197.

Bremmer MA, Beekman AT, Deeg DJ, Penninx BW, Dik MG, Hack CE, *et al*. (2008). Inflammatory markers in late-life depression: results from a population- based study. J Affect Disord 106:249–255.

Ceru-Bjork C, Andersson I, Rossner S (2001). Night eating and nocturnal eating-two different or similar syndromes among obese patients. Int J Obes Relat Metab Disord 25: 365–372.

Colles SL, Dixon JB, O'Brien PE (2007). Night eating syndrome and nocturnal snacking: association with obesity, binge eating and psychological distress. Int J Obes (Lond) 31:1722–1730.

De Zwaan M, Roerig DB, Crosby RD, Karaz S, Mitchell JE (2006). Night time eating: a descriptive study. Int J Eat Disord 39:224–232.

Gadalla T, Piran N (2008). Psychiatric comorbidity in women with disordered eating behavior: a national study. Women Health 48:467–484.

Garland EJ, Remick RA, Zis AP (1988). Weight gain with antidepressants and lithium. J Clin Psychopharmacol 8:323–330.

Geliebter A (2002). New developments in binge eating disorder and the night eating syndrome. Appetite 39:175–177.

Gluck ME, Geliebter A, Satov T (2001). Night eating syndrome is associated with depression, low self-esteem, reduced daytime hunger, and less weight loss in obese outpatients. Obes Res 9:264–267.

Greeno CG, Wing RR, Marcus MD (1995). Nocturnal eating in binge eating disorder and matched-weight controls. Int J Eat Disord 18:343–349.

Heo M, Pietrobelli A, Fontaine KR, Sirey JA, Faith MS (2006). Depressive mood and obesity in US adults: comparison and moderation by sex, age, and race. Int J Obes 30:513–519.

Jelliffe DP, Jelliffe EFP, Zerfas A, Neumann GG (1989). Community nutritional assessment. Oxford: Oxford University Press.

Lundgren JD, Allison KC, O'Reardon JP, Stunkard AJ (2008). A descriptive study of non-obese persons with night eating syndrome and a weightmatched comparison group. Eat Behav 9:343–351.

Milaneschi Y, Corsi AM, Penninx BW, Bandinelli S, Guralnik JM, Ferrucci L (2009). Interleukin-1 receptor antagonist and incident depressive symptoms over 6 years in older persons: the InCHIANTI Study. Biol Psychiatry 65:973–978.

Orhan FO, Ozer UG, Ozer A, Altunoren O, Celik M, Kareaslan MF (2011). Night eating syndrome among patients with depression. Isr J Psychiatry Relat Sci 48:212–217.

O'Reardon JP, Allison KC, Martino NS, Lundgren JD, Heo M, Stunkard AJ (2006). A randomized, placebo-controlled trial of sertraline in the treatment of night eating syndrome. Am J Psychiatry 163:893–898.

Pasquali R, Vicennati V (2000). Activity of the hypothalamic-pituitary-adrenal axis in different obesity phenotypes. Int J Obes Relat Metab Disord 24(Suppl 2):S47–S49.

Pattern SB, Williams JVA, Lavorato DH, Brown L, McLaren L, Eliasziw M, *et al*. (2009). Major depression, antidepressant medication and the risk of obesity. Psychother Psychosom 78:182–186.

Rand CS, Macgregor AM, Stunkard AJ (l997). The night eating syndrome in the general population and among postoperative obesity surgery patients. Int J Eat Disord 22:65–69.

Root TL, Thornton LM, Lindroos AK, Stunkard AJ, Lichtenstein P, Pedersen NL, *et al*. (2010). Shared and unique genetic and environmental influences on binge eating and night eating: a Swedish twin study. Eat Behav l l:92–98.

Rosmond R (2004). Obesity and depression: same disease, different names. Med Hypotheses 62:976–979.

Sinha MK, Ohannesian JP, Heiman ML, Kriauciunas A, Stephens TW, Magosin S, *et al*. (1996). Nocturnal rise of leptin in lean, obese, and non-insulindependent diabetes mellitus subjects. J Clin Invest 97:1344–1347.

Striegel-Moore RI, Franko DL, Thompson D, Affenito S, Kraemer HC (2006). Night eating: prevalence and demographic correlates. Obesity (Silver Spring) 14:139–147.

Stunkard AJ, Grace WJ, Wolff HG (1955). The night-eating syndrome; a pattern of food intake among certain obese patients. Am J Med 19:78–86.

Stunkard A, Berkowitz R, Wadden T, Tanrikut C, Reiss E, Young L (1996). Binge eating disorder and the night eating syndrome. Int J Obes Relat Metab Disord 20:1–6.

Stunkard AJ, Allison KC, Lundgren JD, Martino NS, Heo M, Etemad B, *et al*. (2006). A paradigm for facilitating pharmacotherapy at a distance: sertraline treatment of the night eating syndrome. J Clin Psychiatry 67:1568–1572.

Vander Wal JS (2012). Night eating syndrome: a critical review of the literature. Clin Psychol Rev 32:49–59.

Walker BR (2001). Activation of the hypothalamic-pituitary-adrenal axis in obesity: cause or consequence? Growth Horm IGF Res 11(Suppl A):S91–S95.

Yokogoshi H, Wurtman RJ (1986). Meal composition and plasma amino acid ratios: effects of various proteins or carbohydrates, and of various protein concentrations. Metabolism 35: 637–642.

Zimmerman U, Kraus T, Himmerick H, Schuld A, Pollmacher T (2003). Epidemiology, implications and mechanisms underlying drug-induced weight gain in psychiatric patients. J Psych Res 37: 193–220.