



Disordered eating behaviors and sleep disturbances



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ARTICLE INFO

Article history:

Received 20 July 2012

Received in revised form 8 January 2013

Accepted 30 January 2013

Available online 21 February 2013

Keywords:

Disordered eating behaviors

Sleep disturbances

Adulthood

ABSTRACT

The aim of the present study was to investigate if disordered eating behaviors predicted the development of sleep disturbances. A total of 870 students participated at baseline, 592 one year later (T1) and 305 two years later (T2). The Eating Attitudes Test-40 was used to assess global disordered eating behaviors, dietary concerns (DC), bulimic behaviors (BB) and social pressure to eat (SPE). Sleep disturbances were assessed by two items related to difficulties initiating sleep (DIS) and maintaining sleep (DMS). A sleep disturbance index (SDI) was calculated by summing DIS and DMS scores. Results revealed that global disordered eating behaviors at baseline predicted DIS, DMS and SDI at T1 and T2. Students with increased BB and SPE scores at baseline were more likely to experience sleep onset and sleep maintenance difficulties in the long term. These results suggest that assessment and correction of eating behaviors might prevent sleep disturbances.

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1. Introduction

Empirical and clinical findings indicate that eating disturbances and sleep difficulties are associated. The continuous desire to lose weight, use of purgatives, dissatisfaction with body shape, feeling overweight and the fear of becoming overweight have all been associated in a sample of college students with difficulties maintaining sleep and with the perception of not having had a restorative sleep (Seigel, Broman, & Hetta, 2004). Abnormal eating habits have also been associated with less sleep in a community study upon 7812 college students (Makino, Hashizume, Yasushi, Tsuboi, & Dennerstein, 2006). A previous cross-sectional study from our group (Soares et al., 2011) also revealed that global eating disorders, particularly bulimic behaviors (BB) and social pressure to eat (SPE), were associated with increased difficulties initiating and/or maintaining sleep among university students.

Studies based on clinical samples show that patients suffering from anorexia nervosa experience sleep alterations (Benca & Schenck, 2005; Lauer & Krieg, 2004). Sleep architecture is altered (decreased slow wave activity and REM sleep), sleep length is reduced and sleep efficiency is decreased in underweight anorexics (Delvenne, Kerkhofs, Appelboom-Fondu, Lucas, & Mendlewicz, 1992; Levy, Dixon, & Schmidt, 1988; Nobili et al., 1999; Walsh, Goetz, Roose, Fingerroth, & Glassman, 1985). It has also been documented that weight loss, starvation and malnutrition can all affect sleep. One of the earliest reports, the Minnesota experiment, showed that effects of semi-starvation included

a pre-occupation with food, binge eating, irritability, weakness, dry skin, hair loss and sleep disturbances (Garner, Vitousek, & Pike, 1997; Keys, 1948). After weight restoration following previous weight loss, sleep architecture and sleep continuity tended to improve (Lacey, Crisp, Kalucy, Hartmann, & Chien, 1975; Pieters, Theys, Vandereycken, Leroy, & Peuskens, 2004).

The association between disturbances of eating behaviors and sleep difficulties is also supported by the existence of the *sleep-related eating disorder* (International Classification of Sleep Disorders-2, ICSD-2; AASM, 2005) and by the proposed *night eating syndrome* (Allison et al., 2010; Howell, Shenck, & Crow, 2009). The former is characterized by recurrent episodes of involuntary eating and drinking during nocturnal arousals (most frequently from slow-wave sleep), which can have adverse health consequences (AASM, 2005). Patients usually do not recall these episodes the next morning and purgative behavior is not present. Episodes of nocturnal bingeing with high caloric foods and bizarre substances are frequent. Medical problems are common, mainly as a consequence of weight gain (e.g. hypertension, diabetes mellitus and obstructive sleep apnea). Dental consequences can also occur (Howell et al., 2009). The latter syndrome is characterized by atypical sleep and eating patterns. Subjects may experience evening hyperphagia (excessive wakeful eating after the last meal and before falling asleep), difficulties initiating and maintaining sleep, nocturnal awakenings associated with food intake and morning anorexia (Howell et al., 2009). Because this syndrome is often associated with binge eating or bulimia nervosa, it is not included as a separate category in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders Diagnostic (DSM-IV; APA, 1994) or the second edition of the International Classification of Sleep Disorders (ICSD-2; AASM, 2005) and its nosological status remains controversial (Allison et al., 2010; Striegel-Moore et al., 2006).

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It has been observed that regularly restricting sleep duration, which occurs more frequently in modern societies, can interfere with the metabolic and nutritional status/balance of the body (Crispim et al., 2007). Eating behavior is regulated by a complex interplay of peripheral endocrine stimuli and central neurotransmitter systems, as well as by circadian rhythms and by environmental cues (Passani & Blandina, 2011). Levels of leptin decrease and of ghrelin increase which, in turn, enhance appetite, food intake and body mass index (Crispim et al., 2007). For example, Bjortvatn et al. (2007) observed that short sleep durations were associated with elevated body mass index (BMI) and increased obesity in a sample of 8860 subjects.

The nature of the relationship and biological mechanisms involved in the association between eating and sleep problems is not completely clear. However, it is likely that the hypothalamus plays a central role in the interface between the sleep–wake cycle, feeding behavior and arousal/stress reactivity. For instance, the lateral hypothalamic area (LHA), which produces the peptides orexins A and B, plays a key role in the regulation of both ingestive behavior and the sleep–wake cycle. Several lines of evidence support the role of orexins as modulators of responses to stress (Winsky-Sommerer et al., 2004). Thus, several hypothalamic systems, such as the orexinergic and the histaminergic system, located in the tuberomammillary nucleus (TMN), are at the heart of different homeostatic functions, illustrating the intricate relationship between wakefulness, stress reactivity and feeding.

Although disordered eating habits have been associated with sleep disturbances, to our knowledge there is no study investigating whether these abnormal habits and body mass index (BMI) could predict subsequent sleep disturbance. Therefore, the aim of the present longitudinal study was to investigate this issue in a non-clinical sample of university students. Results will enable a better understanding of the effects of abnormal eating behavior upon sleep to be obtained, and might contribute to the rationale that acting upon these dysfunctional behaviors would enable sleep disturbances, which are known to affect an individual's daytime mood, work activities and well-being, to be prevented.

2. Methods

This research was reviewed and approved by the Medical Ethics Review Committee of the University Hospital of Coimbra (Macedo et al., 2007).

2.1. Procedure

Students were approached and experimental measures were completed as described previously (Azevedo et al., 2010; Soares et al., 2011). Data were collected at three time-points separated by intervals of one academic year (2000/2001, 2001/2002, 2002/2003), when examinations were not taking place. Baseline was considered the first stage, Time 1 (T1) the 1st follow-up year and Time 2 (T2) the 2nd follow-up year. All participation was voluntary and confidentiality was ensured. Most students returned the questionnaires but individuals were free not to return a questionnaire (when their participation was deemed to have ended) without fear of any form of reprisal.

2.2. Participants

A total of 870 undergraduate students (Mean age, $M = 19.59$ years; $SD = 1.61$; Range = 17–25) participated in the study; 544 were females ($M = 19.5$ years, $SD = 1.56$) and 326 males ($M = 19.8$ years, $SD = 1.68$). The vast majority was single ($N = 861$; 99.0%) and 81.1% ($n = 706$) were Medicine students. Of the students assessed at baseline, 65% ($n = 592$) and 48.5% ($n = 305$) completed the same measures one year (T1) and two years later (T2), respectively. Gender distribution, course allocation and marital status were similar between the three assessments.

2.3. Measures

2.3.1. Eating disordered attitudes/behaviors

The Portuguese version of the Eating Attitudes Test-40 (EAT, Garner & Garfinkel, 1979; Soares, Macedo, Gomes, & Azevedo, 2004) was used to measure the severity of behavioral eating disorders, with particular interest in three specific dimensions: (1) Diet Concerns (DC), (2) Bulimic Behavior (BB) and (3) Social Pressure to Eat (SPE).

The Portuguese EAT-40 was derived from factor analysis based on a sample of 596 female university students. A principal component analysis with varimax rotation (orthogonal method of factor rotation) was applied. A three factor solution was obtained similar to the original EAT-40 factor structure by Garner, Olmsted, Bohr, and Garfinkel (1982). The first dimension, DC, included 17 items, the second dimension, BB, integrated 10 items and the third dimension, SPE, incorporated 6 items. Items with factor loadings lower or equal to .3 in all dimensions (7 items) were excluded. As a result, the Portuguese EAT-40 version includes a total of 33 items. Each item is scored on a 6-point Likert-type scale ranging from “never” (0) to “always” (5).

Based on total EAT scores and DC, BB and SPE scores, different sub-groups were formed. Subjects with total EAT scores 1 standard deviation above the mean formed the group with high total EAT score and subjects with scores 1 standard deviation below the mean formed the group with low total EAT score. Similar methods were used for forming sub-groups for each of the EAT dimensions (DC, BB, SPE).

2.3.2. Body mass index (BMI)

BMI (kg/m^2) was calculated using the formula: body weight in kilograms divided by height in meters squared. Height and weight were self-reported by students at baseline, T1 and T2.

2.3.3. Sleep disturbances

Two items were used to assess sleep. These items were: (1) “I have difficulty in falling asleep”, used to assess difficulties initiating sleep (DIS); (2) “I wake up many times during the night”, used to assess difficulties maintaining sleep (DMS). Each item was scored on a 6-point scale ranging from 0 (never) to 5 (always). An overall sleep disturbance score index (SDI) was calculated by summing the scores from the two individual items (possible range, 0–10) where a higher score indicated greater subjective sleep disturbance. Based on this score, the following groups were formed: (i) good sleepers: students reporting that they “never” or “rarely” had DIS/DMS at baseline and at follow-up ($n = 74$; 24.3%); (ii) persistent sleep difficulties group: students reporting that they “often”, “very often” or “always” had DIS/DMS at baseline and at follow-up ($n = 23$; 7.5%); (iii) onset sleep difficulties group: students who did not report sleep complaints at baseline (never/rarely/sometimes) but suffered from sleep complaints (DIS/DMS) either at T1 (1-year) or T2 (2-years) of the follow-up ($n = 32$; 10.5%); (iv) remission sleep difficulties group: students who reported sleep complaints at baseline but which decreased (never/rarely/sometimes responses) either at T1 or T2 ($n = 22$; 7.2%).

2.4. Data analyses and statistics

The Statistical Package for Social Sciences (SPSS) was applied (versions 12.0–14.0). Kruskal–Wallis tests, Student's t tests and Mann Whitney U tests were used to explore differences between groups with respect to disordered eating behavior and sleep disturbances. Pearson correlation analyses were conducted and Cohen criterion was adopted for size-effect interpretation of coefficient correlations: 0.1 = small; 0.3 = medium; and 0.5 = large (Cohen, 1992).

Hierarchical and logistic regression analyses were applied to investigate which factors predicted sleep difficulties over time. The analyses were carried out for the whole sample and not separately for males and females, as a previous cross-sectional study (Soares et al., 2011)

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