

Non Respiratory Sleep Disorders In
Obese: A Mini-ReviewNevin FW Zaki^{1*}, Abdelbaset Saleh² and Magda A Ahmed²¹Department of Psychiatry, Faculty of Medicine, Mansoura University, Egypt²Department of Chest Medicine, Mansoura University, Egypt

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Abstract

Obesity has become an epidemic worldwide. The health hazards and consequences of obesity are multiple. We will try to briefly go through the interrelationship between obesity and various sleep disorders in this mini review. Poor dietary behaviors resulting in obesity will also affect the sleep quality and might lead to breathing related sleep disorders. Improving dietary habits and prevention of obesity should be included within the management plan of various sleep disorders.

Obesity is not only linked to sleep related breathing disorders but also affects sleep quality, duration, circadian pattern, restless leg syndrome, and sleep-related eating disorder.

Introduction

The World Health Organization (WHO) has defined overweight and obesity as abnormal or excessive fat accumulation that is considered a health risk [1]. The Body Mass Index (BMI) is a simple index that is commonly used to classify underweight, overweight and obesity in adults. It is defined as the weight in kilograms divided by the square of the height in meters (kg/m²). A person with a BMI of 25 or more is considered by WHO to be overweight, while obesity is defined as having a BMI of 30 or more (CDC 2010). Overweight and obesity are strong risk factors for cardiovascular diseases and type-2 diabetes and are major contributors to early deaths. These metabolic disorders are intensely increasing among adults in the Eastern Mediterranean Region. Data for adults aged 15 years and older from 16 countries in the Region show the highest levels of overweight and obesity in Egypt, Bahrain, Jordan, Kuwait, Saudi Arabia and United Arab Emirates. The prevalence of overweight and obesity in these countries ranges from 74% to 86% in women and 69% to 77% in men (WHO 2016).

Obesity is associated with an increased risk for adversative health concerns, including diabetes, cardiovascular disease, arthritis, depression, and cancer [2].

Obesity and Sleep Duration

A meta-analysis of cross-sectional epidemiologic studies comprising over 600000 people has demonstrated a clear association between short sleep duration and elevated BMI in both adult and pediatric populations [3]. A systematic review of both cross-sectional and longitudinal studies also supports a consistent association between short sleep duration and concurrent as well as future obesity [4]. Short sleep duration was associated with increasing obesity in Chinese men, and a per-hour increase in sleep duration was associated with obesity in Chinese women, although the mechanism is unclear. However, the associations were not consistent with gender, suggesting these associations could be non-causal [5]. Furuncuoğlu 2016 found that most of the patients (79.6%) sleeping longer than 8 hours -long sleepers were either obese (49%) or morbid obese (30.6%) and long sleep duration was significantly common compared to control group (79.5% vs. 6%; and $P < 0.001$) [6].

Lakerveld 2016 examined the cross-sectional association between sleep duration and total time spent sitting, time spent sitting at work, during transport, during leisureliness and while watching screens. Shorter sleep duration was associated with more screen time sitting. No significant associations were found with total or other domains of sedentary behavior. The observed (lack of) associations were consistent across the five European countries under study, and they did not find differences by gender, age or weight status [7].

Gardner (2015) examined data from the 2007-2008 NHANES to determine whether the relationship between sleep duration and BMI varies based on age [8]. Sleep duration was assessed both continuously and categorically. In both cases, significant interactions were observed. Post-hoc analyses of these interactions identified a pattern such that younger adults demonstrate a relatively linear negative association between sleep duration and BMI, middle-aged adults show a more U-shaped relationship, and older adults show a more attenuated relationship, with a possible association only for very short sleep duration.

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Orexin has been proposed to play a role in obesity, narcolepsy and sleep apnea. It has an integral role, coordinating central control of sleep and physical activity. It has so many intraneuronal connections to the degree that when dysfunction of the orexin system occurs, a great number of regulators and behavioral systems are also affected. Orexin activates the cholinergic system in the basal forebrain leading to modulation of attention and activity [9]. It has been suggested that Orexin system leads to an increase in energy expenditure and that increased orexin provides protection against development of obesity, and this is possible through different mechanisms; like an increase in synthesis or release of orexin peptides or changes in expression of the orexin receptor [10]. Additionally, the effect of Orexinergic peptides on the hypothalamic pituitary thyroid axis remains controversial and might further provide data about the exact role of Orexin in obesity. In fact, although the researches of Orexin and Thyroxine-Releasing Hormone (TRH) neural circuits suggests that TRH neuronal activity is affected by orexin input, the nature of this input and the exact role of ORX (excitatory or inhibitory) remains unclear [11].

The relationship between Orexin and obesity is multifaceted, and in many cases it is difficult to determine whether factors such as physical activity level, sleep quality, or circulating Orexin levels are a consequence of or a causal factor for the development of obesity [12].

Obesity and Sleep Quality

In a study conducted by Rahe 2015, 65.3% of the participants reported good sleep quality (PSQI \leq 5) and 34.7% poor (PSQI $>$ 5) sleep quality [13]. He observed a significant association of poorer sleep quality assessed by the continuous PSQI score with general obesity and high body fat, adjusted for socio-demographic and lifestyle factors. Further adjustment for depressive symptoms and somatic comorbidities attenuated the relationship. The observed association was mainly driven by the PSQI components sleep latency; sleep disturbances, and daytime dysfunction. In another study conducted by (Sun 2016), Poor sleep quality was significantly negatively correlated with overweight/obesity in men but not in women [14]. Additional adjustment for education level, occupation, marriage status, smoking, alcohol consumption, body pain, and health status did not attenuate the correlation among men. The adjusted OR per sleep quality score hour was 1.07 (1.01-1.14) for over-weight/obesity, suggesting that for one score increase in sleep quality, obesity/overweight risk increased by 7 % in men.

Magee (2016) recruited 8,932 Australian employees who participated in the Household, Income and Labor Dynamics in Australia Survey [15]. Structured interviews and self-report questionnaires collected information on sleep quality, obesity, and relevant demographic, health, and work-related variables. Distinct subtypes of sleep quality were identified. General linear modeling examined the associations of sleep quality subtypes with Body Mass Index (BMI) and waist circumference. Five distinct sleep quality subtypes were identified: Poor Sleepers (20.0%), Frequent Sleep Disturbances (19.2%), Minor Sleep Disturbances (24.5%), Long Sleepers (9.6%), and Good Sleepers (26.7%). BMI, waist circumference, and physical activity differed among the sleep quality subtypes, with similar results observed in males and females. Participants who slept less than 6 hours a night had significantly higher BMI, waist circumference, and body fat relative to those who slept 7-8 hours. Those who slept less than 5 hours had a 16% higher

prevalence of general obesity (BMI \geq 30 vs. $<$ 25 kg/m²) (95% [CI]: 0.08-0.24) and a 9% higher prevalence of abdominal obesity (waist circumference: women \geq 88 centimeters, men \geq 102 centimeters; 95% CI: 0.03-0.16) compared to those who slept 7-8 hours [16].

On the contrary Crönlein 2015 in a study conducted on Two-hundred and thirty-three patients with a confirmed diagnosis of severe and chronic insomnia without co-morbidity showing objectively impaired sleep quality were compared with respect to their body mass index with control data derived from a representative population survey matched in gender and age. As a result, patients with insomnia showed a lower body mass index (23.8 kg m⁻² versus 27.1 kg m⁻²; $P <$ 0.0005). Suggesting that patients with chronic insomnia do not exhibit overweight [17].

Restless Leg Syndrome and Obesity

Restless Legs Syndrome (RLS) (Willis-Ekbom disease) is characterized by "an urge to move while at rest relieved by movement and worse in the evening and at night". Iron deficiency in the basal ganglia and low dopamine may play a role in pathogenesis [18]. The International Restless Legs Syndrome Study Group has revised the diagnostic criteria of the syndrome to make it easier for clinicians to diagnose the disorder and hence treat it [19].

Obese persons have decreased dopamine D2 receptor availability in brain [20], and this might elevate their risk of developing RLS. However, studies examining the associations between obesity and RLS are limited. In a large cohort study conducted in 2009 by Gao and his colleagues whom found that the prevalence of the diagnostic criteria recommended by the International RLS Study Group increased progressively with increasing BMI and waist circumference in the study patients. The finding was independent of age, smoking status, anxiety levels, use of medications, and comorbidity with a number of chronic illnesses. A similar association was found between obesity in early adulthood (age 18 or 21) and RLS prevalence in mid-life or later (age 40 years and higher), the findings imply that obesity might be a risk factor for the development of RLS [21].

Additionally, in a more recent cohort study it was found that obesity and hypercholesterolemia were highly associated with an increased risk of developing RLS. This association was independent of age, ethnicity, smoking status, physical activity, alcohol intake, antidepressant use, phobic anxiety score, history of MI, history of stroke and menopause. On the other hand, there was no similar associations with hypertension in the same study group [22].

It is essential to determine the direction of the relationship between both disorders in order to understand more about their pathophysiology and thus prevention and treatment regimes.

Sleep-Related Eating

Recurrent nocturnal eating episodes represent the main presentation in patients affected by Night Eating Syndrome (NES) as well as in those affected by Sleep-Related Eating Disorder (SRED). Although NES is considered to be an eating disorder, SRED is classified among parasomnias (AASM 2014). Nocturnal Sleep-Related Eating Disorder (NSRED) denotes a type of sleep disorder characterized by recurrent episodes of unconscious eating during the night with partial or complete amnesia of the incident. It has been known to result in weight gain from eating high caloric foods. There

have been reports of patients consuming uncooked food, placing inappropriate amounts of condiments on food, consuming hot food/beverages which resulted in burns, cutting food which resulted in ripping their fingers, and consuming other unusual items such as cooking oil or cleaning solutions [23].

In a recent study by Vinai et al 2014 trying to delineate the differences between night eating syndromes they compared polysomnographic and personality characteristics of patient whom are nocturnal eaters, they concluded that nocturnal eaters share several psychological behaviors with patients suffering from eating disorders, and have distinctive features allowing one to distinguish them from Binge eaters. They do not have definite sleep disorders, further supporting the hypothesis that their eating episodes are due to an eating disorder more than to a parasomnia.

Careful assessment and diagnosis of this sleep disorder in obese subjects is necessary to plan the appropriate therapy and prevent other obesity consequences in this group of patients [24].

Obesity and Sleep Apnea

Obstructive sleep apnea is characterized by temporary, but intermittent episodes of upper airway obstruction resulting in cessation of breathing. Apnea termination requires arousal and the resulting frequent awakenings lead to daytime symptoms such as daytime sleepiness, and impaired concentration [25].

Obesity is associated with anatomical changes that predispose to upper airway obstruction during sleep. These changes include excessive adiposity around the pharynx and chest increase neck circumference, decrease in lung volume, deformities in nose and thus compromise a load upon the respiratory tract and may lead to breathing related sleep disorders [26]. Furthermore, Lakerveld (2016) stated that the incidence of EDS was 8.2%, while its persistence and remission were 38% and 62%, respectively. Obesity and weight gain were associated with the incidence and persistence of EDS, while weight loss was associated with its remission. Significant interactions between depression and PSG parameters on incident EDS showed that, in depressed individuals, incident EDS was associated with sleep disturbances, while in non-depressed individuals; incident EDS was associated with increased physiologic sleep propensity. Diabetes, allergy/asthma, anemia, and sleep complaints also predicted the natural history of EDS [27].

In A population-based sample of men (n=1896, aged 40-79 years) and women (n=5116, age≥20 years) whom responded to questionnaires at baseline and follow-up after 10-13 years. Sleep problems were assessed through questions about Difficulties Initiating Sleep (DIS), Difficulties Maintaining Sleep (DMS), Excessive Daytime Sleepiness (EDS), and insomnia. Although overweight and obese subjects reported more sleep problems at baseline, there was no independent association between BMI level at baseline and development of new sleep problems [28].

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