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EDITORIAL

Sleep and obesity: an introduction

Caterina Lombardo[1]

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Introduction

The paper focuses on the relationship between poor sleep quality/quantity and obesity and aims to document and support the progresses in this field, bringing new contributions to several questions that are still open in the extant literature. The work also introduces to the Topical Collection "Sleep and Obesity" which will cover the contribution of poor sleep in the etiology of obesity in both humans and animals, the epidemiological and diagnostic features that relate poor sleep to obesity as well as the associated features and the factors that could moderate or mediate this relationship. The topical collection is aimed to provide a wide evidence base for clinicians, and researchers and all readers interested in this area.

Prevalence of obesity has grown to epidemic proportions: It has more than doubled, since 1980; in 2014 the world prevalence of overweight among adults aged more than 18 years was 39 % and that of obesity was 13 %. Mortality rate among overweight and obese people is higher than among underweight people [1]. Looking for modifiable factors associated with obesity that may be targeted in preventive interventions, the scientific research has addressed sleep duration, since parallel to the growth of obesity in Western societies there has been reported a systematic reduction of at least 1 h of the mean sleep duration in children and adolescents [2]. A modern epidemic of insufficient sleep has been, however, questioned in adults by several systematic reviews evidencing no significant decrements in self-reported and objective sleep duration over the last half-century [3, 4]. For instance, Yougsteadt and colleagues [4], reviewing studies assessing objective-recorded sleep durations in healthy sleepers over the last half-century, evidenced a relative stability across years and age groups in both men and women, for both PSG and actigraphic data.

Although secular trends in sleep duration still need to be clarified, the negative impact of sleep curtailment on health seems consensually acknowledged. A panel of experts reviewed 5314 published articles dealing with consequences of short sleep and published a consensus report with recommendations for the optimal sleep duration in healthy adults aged 18–60 years [5]. The panel agreed on a recommendation of an average of 7 h or more per night on a regular basis for optimal health.

The main causes of a reduction in the quantity and quality of sleep observed, especially in youngers, may be lifestyles, leaving less and less time to sleep or that include habits that are dysfunctional for sleep such as abuse of coffee and caffeinated beverages or use of technology (mobile phones, tablets, etc.) until late at night [6, 7]. Since sleep and habits related to sleep are factors that can be modified, the study of sleep in relationship to obesity grew up very quickly during the last 15 years. On September, the 1st I conducted a rough literature search on Ebsco data banks (Medline, PsychInfo, PsychLit) and simply took notes of how many records were found using the following keywords: "poor sleep & obesity", "short sleep & obesity", "sleep duration & obesity" and the more general "sleep & obesity". The number of records found across three different years ranges (2000–2005; 2006–2010; 2011–2015) is summarized in Table 1. Obviously, this is

This article is part of the topical collection on Sleep and Obesity.

✉ Caterina Lombardo
caterina.lombardo@uniroma1.it

[1] Department of Psychology, Sapienza University of Rome, Via dei Marsi 78, 00185 Rome, Italy

Table 1 Record founds on Ebsco data banks Medline, PsychInfo, PsychLit (duplicates removed)

Key words	Years		
	2000–2005	2006–2010	2011–2015
Poor sleep & obesity	17	55	168
Short sleep & obesity	27	172	378
Sleep duration & obesity	37	273	718
Sleep & obesity	1099	2390	3798

not a systematic search of the literature (as it goes beyond the scope of the present paper) and not all records found address the target relationship. However, this rough search may give us a very general idea about how the interest toward the relationship between sleep and nutritional status has increased during the last 15 years.

Epidemiological evidence

Most cross-sectional studies and systematic reviews show that sleep characterized by poor quality or short duration is consistently associated with obesity in children [8, 9]. Also in adults a cross-sectional inverse relationship has been reported between sleep duration and obesity [10, 11]. A meta-analysis [12] showed that children with poor or disturbed sleep are about twice as likely to have obesity and adults with poor sleep have a probability of 1.5 more than good sleepers to be obese. In children, the cross-sectional results are also confirmed by longitudinal studies, systematic reviews and meta-analyses; on the contrary from adolescence to adulthood, results are more controversial [13, 14]. A recent systematic review [9] examined 20 longitudinal studies published between October 2004 and October 2010, highlighting again consistency of the results in children and inconsistency in adults. In particular, four of the studies examining adults found a positive association between short sleep and weight gain; four studies found that weight gain was associated with both sleep of short and excessively long duration; five studies, including one using objective measures of sleep quality and duration, did not report any association between quality and/or duration of sleep and weight gain or obesity.

Explaining inconsistency of epidemiological studies conducted in adults

The inconsistency outlined in the previous paragraph may depend on different factors. First of all, the relationship between sleep duration and obesity in adults may be non-linear and U-shaped. In other words, there may be an optimal duration of sleep (around 7 h) that is associated with health and well-being while sleep of longer or shorter duration may increase the risk of obesity [14, 15]. This explanation is consistent with the results of a longitudinal study by Theorell-Haglow and colleagues [16] who showed that in a wide sample of women aged 46 ± 17.5, the subsample of participants aged <40 showed higher obesity if they slept <6 h or more than 9 h.

The second explanation that may be advanced is the eventual different composition of the samples. The relationship between poor sleep and obesity appears stronger in adult males than in females [17, 18]. Consistently, the results of the study by Theorell-Haglow and colleagues [16] evidence no association between changes in sleep duration and any of the measures of obesity at the follow-up in women aged >40 years at baseline.

Another factor that may explain why the association between short sleep and obesity in adults is inconsistently reported across studies is that the need for sleep decreases with age and, thus, the risks associated with sleep deprivation may vary accordingly: children need more hours of sleep and then sleep deprivation may produce more serious effects on metabolism; on the contrary, as adults need less sleep, their metabolism may be influenced by this variable to a lesser extent than by other factors. In other words, there may be other factors (e.g., stress, mood, etc.) that may mediate the relationship between sleep duration and obesity in adults, thus explaining why some studies find and some studies do not find the relationship.

Which is the most suitable explanation and which are the mediating factors still needs further investigation and available evidence does not allow any exhaustive conclusion. Moreover, the epidemiological literature has a limited generalizability for many methodological reasons, dealing with the definition and assessment of both sleep and nutritional status.

1. *Sleep* The definition and assessment of sleep vary widely: several studies evaluate the effects of short sleep duration while others assess the consequences of poor sleep quality; several studies measure sleep quality/quantity through self-report instruments, others use portable devices (e.g., actigraphs), while others measure objective sleep duration and quality through polysomnography.
2. *Nutritional status* Several studies evaluate the effect of poor sleep on BMI based on self-reported height and weight; other studies use objective measures of height and weight for computing BMI; other studies use other anthropometric measures or assess weight gain in a given period of time (time periods also vary widely).

Experimental evidence

Many experimental studies have also been conducted producing consistent results. In particular, they evidence that acute (1, 2, or 6 consecutive nights) partial sleep deprivation (4 h of sleep allowed) increases hunger, alters metabolism of carbohydrates, reduces leptin levels, and increases ghrelin levels [19–21]. Hypotheses regarding the processes underlying this relationship have also been advanced, suggesting both metabolic and behavioral paths of action. Knutson [22], for instance, suggests that sleep deprivation causes increased activation of the sympathetic portion of the autonomic nervous system, increased production of GH and cortisol, reduced glucose utilization by the brain and thus alters glucose metabolism. According to the author, sleep deprivation produces changes also in eating behavior: since leptin is reduced and ghrelin elevated and since the time available for eating is increased, food intake (especially intake of highly palatable foods) increases and energy expenditure decreases. Chapman and colleagues [23] conducted a meta-analysis for evaluating the effects of three factors potentially associated with obesity: alcohol intake, sleep deprivation and time spent watching TV. The studies included adopted an experimental design, manipulating lifestyle variables and observing the effects of this manipulation on an ad libitum test meal. The results showed that all target variables increased energy intake. In particular, the consumption of alcohol was the factor that produced a wider effect (Cohen's $d = 1.3$ great effect size), followed by sleep deprivation (Cohen's $d = 0.49$ medium effect size) and time spent watching TV (Cohen's $d = 0.2$ small effect size).

Implications and conclusions

If the mechanisms identified by the experimental findings are generalized to the results of the epidemiological literature, it is possible to explain why people sleeping less show higher overweight and obesity rates. However, the possibility to generalize the results of the experimental literature to real life is limited for several reasons. First of all, experimental sleep deprivation is not exactly equivalent to the chronic sleep deprivation consequent to lifestyle or sleep problems, since in the second case, adaptation processes may reduce the consequences of sleep curtailment [24]. Moreover, optimal sleep is very difficult to define, as it may depend not only on duration but also on quality [24]. After Borbely's theory [25], it is known that the quality of sleep (measured as sleep continuity or slow wave sleep percentage or density) increases with longer wakefulness. Due to this homeostatic process, normal sleepers may adapt to temporary sleep curtailment or simply cope with the consequences of getting less sleep sometimes and sleep better when they have the opportunity to sleep after acute sleep deprivation. Optimal sleep quality refers to concepts like high sleep efficiency (minimal waking after sleep onset notwithstanding the duration), few and brief arousals that may cause next-day sleepiness, a sleep-wake cycle very close to the individual circadian type [24]. Similarly, an acute increase of food intake or an acute change in the metabolic hormones is not equivalent to a stable weight gain or to obesity status.

These are some of the main reasons why it is difficult to simply extend the results of the experimental literature to the real life and why the mechanisms that explain the consequences of sleep loss on health still need to be investigated.

Compliance with ethical standards

Conflict of interest The author declares that he has no conflict of interest.

Ethical approval This article does not contain any studies with human participants or animals performed by any of the authors.

Informed consent For this type of study formal consent is not required.

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