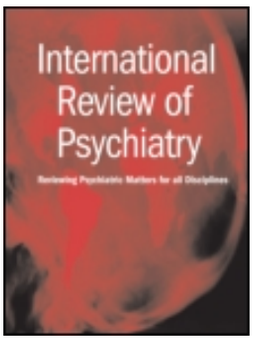

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Sleep, obesity, and weight loss in adults: Is there a rationale for providing sleep interventions in the treatment of obesity?

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Abstract
Rates of obesity and sleep disturbances are substantial in adults. A number of cross-sectional, longitudinal, and experimental studies have found that insufficient sleep and possibly longer sleep are associated with obesity and related eating patterns. Methodological discrepancies and limitations in the literature create ambiguity about the nature and potential mechanisms underlying these relationships. Insomnia and circadian patterns in eating and sleeping have also been examined in relation to weight. Although these studies are not as extensive as those examining sleep duration, the extant literature suggests possible associations between obesity and both insomnia (particularly when combined with short sleep duration) and circadian eating behaviours. However, research has only just begun to examine the benefits of combining sleep interventions with obesity treatment. The goal of the current review is to summarize research examining behavioural sleep patterns and disorders in relation to obesity, to discuss methodological considerations, and to provide an overview of studies examining whether addressing sleep disturbances can augment weight loss treatment effects. We conclude that future studies are needed that take into account sleep duration, sleep disorder co-morbidity, and chronobiology to explore the impact of sleep interventions on weight loss.

Introduction

Obesity is a world-wide public health problem associated with a number of serious health conditions and early mortality (Flegal et al., 2013; Guh et al., 2009). In 2009–2010 more than 35% of men and women in the USA were obese, defined as a body mass index (BMI) ≥ 30 kg/m^2 (Ogden et al., 2012), and recent obesity rates reported by the International Association for the Study of Obesity approximate 25% in countries such as England, Germany, and Spain, 30–35% in Jordan and Venezuela, and 40–45% in Egypt and Kuwait, particularly among women (www.iaso.org).

Insufficient sleep is also recognized as a significant public health problem given its strong association with injury, chronic diseases, and mortality (Buxton & Marcelli, 2010; Grandner et al., 2010). In the 2009 Behavioral Risk Factor Surveillance System survey of 74,571 men and women in the USA, 35% of the adult population reported sleeping less than the recommended 7–9 h of sleep per night (McKnight-Eily et al., 2011). When defined more conservatively (sleeping on average 6 h or fewer per night) and examined internationally, the prevalence of short sleep duration is roughly 10% in the USA, Canada, and the UK and ranges from 1–7% in countries such as the Netherlands and Italy, respectively (Bin et al., 2013). Interestingly, long sleep duration, generally defined in epidemiologic studies as sleeping more than 9 h per night, is also associated with deleterious health conditions and premature mortality and may be a more ubiquitous problem, occurring in approximately 25–40% of adults across the world (Bin et al., 2013; Gallicchio & Kalesan, 2009). The physiological and behavioural effects of too much sleep and the underlying factors contributing to long sleep duration, however, have received minimal attention relative to insufficient sleep.

Numerous cross-sectional and some longitudinal studies have shown an association between sleep duration, particularly short sleep duration, and obesity. Perhaps more convincingly, laboratory studies have also demonstrated that sleep deprivation induces physiological states and behavioural patterns associated with increased BMI and obesity. For example, short-term experimental studies mimicking a natural eating environment (i.e. ad libitum food access not caloric restriction) have found that sleep restriction is associated with increased caloric intake, meals per day, and preference for energy-dense foods (Markwald et al., 2013; Nedeltcheva et al., 2009; Spaeth et al., 2013). Further, sleep restriction induced in the laboratory setting has been shown to decrease spontaneous physical activity under

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free-living conditions (Schmid et al., 2009). Collectively, the scientific evidence is compelling that sleep and weight should be considered in relation to one another in the context of health and behavioural evaluations and interventions. However, the causal relationships between sleep patterns, metabolic functions, behaviours and weight gain are only beginning to be elucidated. Given the relatively modest long-term effects of standard behavioural weight loss programmes, sleep interventions are increasingly being recognized as a potentially promising new target to improve weight loss outcomes. Yet surprisingly, little research to date has focused on combining sleep interventions with weight loss interventions.

The objective of the current review is to outline cross-sectional, prospective and experimental studies examining behavioural sleep disturbances in relation to obesity and to summarize the preliminary research that has examined the impact of combining established sleep interventions with weight loss interventions. We seek to broadly answer the following question: is there a rationale for combining empirically supported sleep treatments with obesity treatment? Practical considerations of this information and areas in need of further study will be discussed.

Methods

For this thematic review, PubMed searches were conducted on the following key words: sleep AND obesity OR body mass index OR weight. Secondary searches were also conducted utilizing more specific keywords related to sleep (e.g. sleep duration, sleep timing, insomnia) and obesity (e.g. energy intake, energy expenditure). To represent the most current understanding of the literature, publications from 2008 to the present, particularly systematic reviews and meta-analyses, were given preference when possible, although earlier studies were included if they were identified as being highly influential. Given the broad and extensive nature of the topic, we focused this review on studies of adult humans, although we briefly summarize key cross-sectional and longitudinal paediatric findings when they are highlighted in selected adult/children's reviews. Those interested in studies of sleep and obesity in non-human animals are referred to Mavanji et al.'s review (2012). To further focus this review we elected not to focus on sleep and medical conditions that are often co-morbid with obesity (e.g. type 2 diabetes, coronary heart disease, hypertension, see Grandner et al. in this edition). Similarly, we will not discuss the well-established role of obesity as a causal risk factor for sleep disordered breathing (see Romero-Corral et al., 2010). We will, however, briefly discuss the possibility that treatments for sleep apnoea might impact weight loss as an important direction for future research. Finally, it should be noted that there is considerable individual variation in sleep duration. Some adults sleeping habitually less than 7 h per night or greater than 9 h per night will have neither discernible daytime consequences nor increased medical/psychiatric risk. For the purposes of short hand, in this manuscript, we refer to 'short' and 'long' sleep based on epidemiological surveys of the adult general population with the caveat that some 'short sleepers' and some 'long sleepers' are satiating their normal sleep need and may not be at increased risk for physiological and psychological disorders.

Sleep duration and obesity risk

Cross-sectional studies

Several cross-sectional studies have reported an association between short sleep duration and increased BMI and/or obesity. In 2008 several reviews on the topic were published (Cappuccio et al., 2008; Knutson & Van Cauter, 2008; Marshall et al., 2008; Patel & Hu, 2008; Van Cauter & Knutson, 2008), two focusing exclusively on children (Chen et al., 2008; Hart & Jelalian, 2008). Generally speaking, these reviews concluded that there is an overall pattern of evidence that those who sleep less tend to be heavier, most clearly so in children, less consistently in adults (see Table 1). A more recent critical analysis of the literature identified 16 additional cross-sectional studies (nine in adults, seven in children) that were not included in the 2008 reviews (Nielsen et al., 2011), and these studies showed a pattern of results similar to those reported in the 2008 reviews. However, the authors cautioned against a single scoring of each study in favour of or not in favour of an association between short sleep duration and obesity risk, because this approach misleadingly gives equal weight to each study and overlooks important methodological problems (e.g. measurement problems and failure to consider confounders and/or mediators). These methodological considerations are discussed below.

A number of studies have shown a U-shaped curvilinear relationship between sleep duration and BMI in adults (Table 1), indicating that both long and short sleep duration, but not normal duration, are associated with increased obesity risk. However, in a recent narrative review, Chaput (2013) points out that this U-shaped association has been found mostly in studies using self-reported sleep duration and, consistent with conclusions made by Knutson and Turek (2006), the effect of inadequate sleep on obesity and related health indicators appears more deleterious than prolonged sleep. The majority of studies reporting a positive association between long

Table 1. Summary of selected 2008 reviews evaluating cross-sectional (CS) and longitudinal (L) associations between sleep duration and weight.

Authors (year)	No. of studies reviewed (type of study); age group	Summary of findings	General conclusions
Cappuccio et al., (2008)	(1) 17 adults	(1) A pooled regression analysis in adults further revealed a 0.35 kg/m^2 increase in BMI for each hour of sleep reduction per day.	There is a consistent pattern of increased odds of being a short sleeper if you are obese in both children and adults. There is a 60% to 80% increase in the odds of being a short sleeper among those who are obese.
	(2) 12 children	(2) In children the pooled odds ratio for short duration and obesity was 1.89 (1.46 to 2.43; $p < 0.0001$) and in adults it was 1.55 (1.43 to 1.68; $p < 0.0001$).	
Marshall, Glozier & Grunstein (2008)	(1) 17 (CS) adults	(1) Five studies found a negative linear association[a] (one did not find an association at age 40 and another did not find an association at 50–86 years); six found a U-shaped relationship;[b] six found no association.	Most studies suggest CS and L associations between short sleep duration and obesity in adults, although there are contrasting studies; association between long sleep and obesity is less clear. Research is far more consistent in children, with short sleep duration and obesity associated in both CS and L studies. In adults, studies evaluating sleep and weight over time with repeated valid measures and enough power to evaluate subgroups are needed. RCTs of sleep modification to treat obesity in people with either very long or very short sleep may be warranted.
	(2) 11 (CS) children	(2) Eight found a negative linear association;[a] one found an association with overweight/obesity; one found an n-shape association;[c] one found no association.	
	(3) 5 (L) adults	(3)Three showed a negative linear association[a] (one with a weak u-shaped association);[b] two found no association.	
	(4) 4 (L) children	(4) Four found a negative linear association.[a]	
Patel & Hu (2008)	(1) 19 (CS) adults	(1) 11 found a clear association between short sleep duration and increased BMI (two reported mixed findings by gender); four found a U-shaped association;[b] five found no association.	Overall support for an association between short sleep duration and weight. Results are fairly uniform in paediatric studies. Findings among adult studies are more mixed; relationship may be more complex in adults. Need for large prospective cohort studies with repeated objective measures. RCTs are needed to assess the effect of sleep-promoting interventions on obesity.
	(2) 11 (CS) children	(2) All 11 found an association between short sleep duration and obesity.	
	(3) 3 (L) adults	(3) All longitudinal studies in both adults and children found a relationship between short sleep duration and later overweight/obesity risk.	
	(4) 2 (L) children	(4) All longitudinal studies in both adults and children found a relationship between short sleep duration and later overweight/obesity risk.	
Van Cauter & Knutson (2008)	(1) 25 (CS) adults	(1) 21 found an association between sleep duration and BMI.	Partial chronic sleep restriction may increase risk of weight gain in both children and adults.
	(2) 13 (CS) children	(2) 13 found an association between sleep duration and BMI.	
	(3) 5 (L) adults	(3) Four out of five found a longitudinal association between sleep duration and weight gain.	
	(4) 4 (L) children	(4) Four found a longitudinal association between sleep duration and weight gain.	

CS, Cross-sectional; L, Longitudinal; RCTs, Randomized controlled trials.
[a]Short sleep is associated with increased weight.
[b]Both short and long sleep are associated with increased weight.
[c]Short and long sleep are associated with less obesity.

sleep and weight have rarely sought to disentangle co-morbid medical or psychiatric factors (e.g. depression) that may play a prominent role in this relationship. Thus, it is not clear whether sleeping more than 9 h is inherently unhealthy or whether long sleep is secondary to other mental or medical disorders that are associated with overweight.

Observational studies

While several cross-sectional studies have found a significant relation between sleep duration, particularly short sleep duration, and obesity risk, these studies provide no information related to temporal sequence and, hence, do not allow causal inferences to be made. It is therefore important to turn to prospective studies to better understand the association between sleep duration and subsequent weight gain over time.

Studies that have examined sleep duration and later weight gain in observational prospective studies have yielded consistent results in children and somewhat more mixed results in adults. All of the eight longitudinal paediatric studies have found a significant inverse association between short sleep duration and later weight gain and/or obesity risk (Magee & Hale, 2012; Nielsen et al., 2011). Magee and Hale (2012) identified 13 adult observational prospective studies. Approximately 62% (8 out of 13) of these studies found evidence indicating that short sleep predicted weight gain. Four found an association between both short and long sleep duration and weight gain, supporting a U-shaped association (Chaput et al., 2008; Hairston et al., 2010; Lopez-Garcia et al., 2008; Watanabe et al., 2010); four found an association between short sleep duration (but not long sleep) and later weight gain (Gunderson et al., 2008; Hasler et al., 2004; Nishiura & Hashimoto, 2010; Patel et al., 2006); five found no significant associations between sleep duration and subsequent weight gain (Gangwisch et al., 2005; Lauderdale et al., 2009; Littman et al., 2007; Marshall et al., 2010; Stranges et al., 2008).

Another review similarly concluded that most, but not all, longitudinal studies find short sleepers to experience greater increases in weight over time; however, average reported weight gains tend to be modest, and there is substantial variability among studies (Lucassen et al., 2012). For example, in the largest longitudinal study to date examining sleep duration in relation to later weight gain (N = 68,183), the Nurses' Health Study, women free of co-morbid disease who slept 6 h and those sleeping ≤ 5 h were 12% and 32% more likely to have a 15 kg weight gain over 16 years compared to those sleeping 7 h, respectively (Patel et al., 2006). In the Quebec Family Study, short-duration sleepers (5–6 h) gained 2 kg more and long-duration sleepers (9–10 h) gained 1.6 kg more than average-duration sleepers (7–8 h) over 6 years, with a 27% and 21% elevated risk of developing obesity for short and long duration sleepers, respectively (Chaput et al., 2008). Among adults ≥ 60 years of age in Spain, women, but not men, who slept ≤ 5 h/night and those who slept > 8 h/night were more likely to gain ≥ 5 kg in 2 years (Lopez-Garcia et al., 2008). In another study, risk of developing obesity was increased in those with a sleep duration of < 5 h and in those sleeping 5–6 h (odds ratios of 1.91 and 1.50, respectively) relative to those sleeping 7–8 h in a sample of Japanese adults (Watanabe et al., 2010). However, these findings were significant in men, but not women, suggesting inconsistent findings related to gender. A more recent study not included in the above reviews (Xiao et al., 2013) found that among US men and women 51–72 years of age (N = 83,377), those who were not obese at baseline and reported less than 5 h of sleep per night had a 40% greater chance of developing obesity over 7.5 years than those sleeping 7–8 h (odd ratio of 1.45 for men and 1.37 for women).

Measurement issues

Some inconsistencies in the literature on sleep duration and obesity are likely explained by methodological differences, including variability in definitions of normal and short sleep, inconsistencies in distinguishing normal sleepers from long sleepers, and frequent use of self-reported sleep measures. Definitions of normal sleep duration vary substantially, ranging from at least 6 h to 9 h across studies (Patel & Hu, 2008). Similarly, in the meta-analysis of 18 studies by Cappuccio et al. (2008), short sleep was variably defined as ≤ 5 h (11 studies), ≤ 6 h (five studies), ≤ 6.5 h (one study), and < 8 h/day (one study). Moreover, studies that have not found a link between sleep duration and obesity may be due to long sleepers being combined with 'normal' sleepers and use of statistical methods that have forced a linear relationship in modelling the sleep–weight relationship (Patel & Hu, 2008). Reliance on self-reported versus objective measures of both weight and sleep has also varied across studies, with many studies utilizing only a single-item, self-reported measure of sleep duration. It is possible some of the inconsistencies in the literature are due to measurement error.

Newer studies using objective measurement. There have been more recent advances in measuring sleep duration objectively using actigraphy. Actigraphy measures of sleep are derived from wrist-worn triaxial accelerometer data with application-validated algorithms to quantify total sleep time and other sleep

continuity parameters (Lichstein et al., 2006). Significant cross-sectional associations between both actigraphy and diary measures of short sleep duration and elevated BMI were recently found independent of objectively measured sleep-disordered breathing in a sample of 310 midlife women participating in the Study of Women's Health Across the Nation (SWAN) sleep study (Appelhans et al., 2013). These findings are consistent with those of the Coronary Artery Risk Development in Young Adults (CARDIA) Sleep Study, which also found a cross-sectional association between wrist actigraphy-measured shorter sleep duration and higher BMI in a community-based study of adults in early middle age (Lauderdale et al., 2009). Two additional cross-sectional studies using actigraphy (Patel et al., 2008; van den Berg et al., 2008) have also found a significant association between reduced sleep duration and elevated BMI in older adults, a population in whom both obesity rates and sleep disturbances (e.g. decreased REM sleep and sleep efficiency, increased sleep latency) are more prevalent (Ogden et al., 2012; Ohayon et al., 2004) and in whom the sleep duration–obesity association has not been established via brief self-report measures (Gangwisch et al., 2005). The few studies that have utilized objective measures of sleep duration to examine the longitudinal association between sleep duration and subsequent weight gain have not found a significant relationship (Appelhans et al., 2013; Lauderdale et al., 2009; Vgontzas et al., 2013b). More research using objective sleep measures are clearly needed to better elucidate the extent to which sleep duration predicts weight over time.

Additional methodological considerations

In addition to measurement concerns, longitudinal studies have not consistently adjusted for potential confounds of the sleep duration–weight gain relationship (e.g. age, socio-economic status, chronic diseases, medications). This limits the ability to make comparisons between studies and to make causal inferences. Importantly, only five of the 13 studies (38%) in Magee and Hale's review adjusted for other sleep-related problems (e.g. waking in the night/insomnia, snoring status) that are associated with obesity and also influence sleep duration. None of the five studies adjusting for sleep problems included (1) objective measures of sleep apnoea, (2) a diagnosis of insomnia ascertained by clinical interview, or (3) an evaluation of restless leg syndrome or a periodic limb movement disorder. Although these types of measures are certainly more challenging to collect in large prospective studies, future research that incorporates objective measures of sleep, sleep disorder diagnoses and/or clinical indices into statistical models are needed to move the state of the science forward.

It is important to note that the distinction between confounders and mediators is often confused (see Babyak, 2009 for a thorough discussion), and it is not always clear when to consider a variable a confounder or a potential mediator of the association between sleep duration and obesity. Simply adjusting for variables that are associated with sleep duration and obesity (e.g. emotional factors, physical activity and appetite/dietary patterns) in statistical analyses (i.e. treating them as confounders) may limit the ability to examine the mechanisms through which short sleep duration impacts weight change (Nielsen et al., 2011). Research examining mediators of short sleep duration and obesity is very limited. A notable exception is the Penn State Cohort (Vgontzas et al., 2013b), which found that the longitudinal relationship between self-reported short sleep duration and incident obesity was mediated by poor sleep complaints (e.g. difficulties falling and staying asleep, excessive daytime sleepiness) and emotional stress, suggesting that self-reported short sleep duration may be a marker of other sleep complaints and chronic psychological distress. These factors may ultimately be more important to target in weight loss programmes than sleep duration itself.

Laboratory studies and potential mechanisms

Sleep deprivation and feeding related hormones. Compelling experimental work has been undertaken that elucidates potential mechanisms through which sleep loss may induce weight gain. Highly controlled laboratory studies have found that sleep deprivation impacts the signalling systems that underlie hunger and satiety (Benedict et al., 2011; Spiegel et al., 2004). In particular, reduced sleep duration has been associated with decreased leptin, a hormone produced in adipose tissue that influences metabolism and suppresses appetite, and with increased ghrelin, a gastrointestinal peptide that stimulates hunger. Perhaps the most frequently cited laboratory study demonstrating alterations in appetite regulation with sleep curtailment is one conducted by Spiegel et al. (2004). In this study of healthy men, sleep restriction (2 days of 4 h in bed) with controlled energy intake using intravenous glucose infusion was associated with overall reductions in leptin by 18%, elevations in ghrelin by 28%, and increased ratings of hunger, global appetite, and cravings for high carbohydrate foods (Spiegel et al., 2004). More recent work using a controlled energy intake design (standardized meals during the experimental sleep sessions) has shown that as little as one night of sleep loss (24 h of continuous wakefulness) in healthy male adults is associated with increased ghrelin and hunger ratings

(Benedict et al., 2011). However, in some laboratory studies using ad libitum food access, which more closely resembles the food environment in developed countries and produces a positive energy balance versus a restricted energy balance, ghrelin and leptin levels remained unaffected by sleep restriction in both a sample of healthy men (N = 15) who spent 14 days of 5.5 h/night in bed (Schmid et al., 2009) and in a sample of healthy men and women (N = 11, 45% women) who spent 2 days of 4.25 h/night in bed (Nedeltcheva et al., 2009). Interestingly, despite there being no alterations in leptin and ghrelin levels, sleep-restriction was nevertheless associated with increased snacking, particularly on snacks with high carbohydrate content between 7:00 p.m. to 7:00 a.m. (Nedeltcheva et al., 2009). In other sleep-restriction studies with ad libitum food access, one which restricted healthy women to one day of only 3 h of sleep (Omisade et al., 2010) and another which restricted healthy men and women to five consecutive nights of 4 h in bed (Simpson et al., 2010), sleep restriction was associated with elevated leptin levels. In the latter study, greater increases were seen in women compared to men.

Sleep restriction combined with caloric restriction. Nedeltcheva and colleagues (2010) tested the hypotheses that sleep restriction can attenuate the effect of a reduced calorie diet on adiposity, hunger ratings, and leptin and ghrelin concentrations in 10 overweight middle-aged adults. Participants were placed on 14 days of moderate caloric restriction (caloric content restricted to 90% of resting metabolic rate) combined with either 8.5 h or 5.5 h of sleep per night (in random order with at least 3 months apart). Compared to the normal sleep condition, sleep curtailment decreased the proportion of weight lost as fat by 55% and increased the loss of fat-free body weight by 60%, providing powerful evidence that sleep plays a role in the maintenance of fat-free body mass when combined with reduced caloric intake. This study also found that sleep curtailment increased ratings of hunger and ghrelin, which is consistent with the results found in Spiegel et al.'s (2004) study in which increases in ghrelin were associated with sleep deprivation in combination with a calorically restricted diet. These studies have important implications for the potentially critical role of normalizing sleep in weight loss management programmes. Specifically, they suggest that insufficient sleep may compromise the efficacy of and adherence to common weight loss strategies (i.e. dietary energy-restriction, food cravings, failure to loss adipose tissue).

Sleep restriction, energy intake and the role of timing of sleep and eating. Several experimental studies have shown that sleep deprivation is associated with increased caloric consumption, number of meals eaten per day, and tendency to choose energy-dense foods (for a comprehensive review see Chaput, 2013). In the largest and perhaps most well controlled study of its kind, participants who were sleep-restricted (5 nights of 4 h in bed per night) showed substantial behavioural dietary effects (Spaeth et al., 2013). Specifically, they consumed 130% of daily calorie requirements, consumed more meals per day, and consumed 550 additional calories between 10 p.m. and 4 a.m., a greater percentage of which were derived from fat. These findings suggest the interesting possibility that in addition to chronic sleep restriction, the circadian timing of caloric intake, specifically night eating, might increase susceptibility to weight gain.

Baron et al. (2011) examined timing of sleep as measured by wrist actigraphy and 7-day sleep logs in relation to dietary patterns and BMI in adults (N = 52; ~50% females) and found that late sleep timing (midpoint of sleep 5:30 a.m. or later) was associated with higher BMI, later meal times, higher caloric intake at dinner and after 8:00 p.m., more fast food, more full-calorie sodas, and less fruit/vegetable consumption in comparison to normal sleep timing (midpoint of sleep < 5:30 a.m.). Later sleepers consumed on average 248 calories more per day than normal sleepers, the majority of which occurred at dinner and after 8:00 p.m. This equates to approximately 2 lb. per month if this sleep pattern persists over time and is not balanced by increased energy expenditure. Of note, dietary intake after 8:00 p.m. was most strongly associated with BMI, independent of sleep duration, suggesting that evening caloric consumption may be a critical behaviour to consider in weight loss interventions. Two recent reviews on delayed timing of eating have been published, with both similarly concluding that, although further research is needed, experimental studies provide good evidence that later/delayed timing of eating independently contributes to weight gain (Allison et al., 2014; Garaulet & Gomez-Abellan, 2014).

Sleep restriction and energy expenditure. Studies examining the effects of sleep restriction on energy expenditure have been mixed. A number of reviews on the topic have concluded that energy expenditure does not seem to be substantially affected by short sleep duration (Chaput, 2013; Klingenberg et al., 2012; St-Onge, 2013). Although it is intuitive that insufficient sleep may be linked to obesity through a decrease in energy expenditure, Markwald and colleagues (2013) found that restricted sleep (5 days of 5 h of sleep/night) was associated with an average increase of approximately 5% in daily energy expenditure compared with a control condition of 9 h of sleep per night. However, individuals who are sleep

deprived in the laboratory setting experience a decrease in spontaneous physical activity during the day under free-living conditions (Schmid et al., 2009). Taken together, it appears that although energy expenditure may increase slightly with sleep deprivation, the sleepiness and fatigue that accompany sleep deprivation could result in less physical activity during the day. More research is needed to understand the possible implications that insufficient sleep may have on physical activity and adherence to weight maintenance strategies, particularly since increased physical activity is a prominent mediator of successful weight loss outcomes (Coughlin et al., 2013).

Sleep deprivation and weight gain. Perhaps due to the challenges of executing extended sleep deprivation protocols and the presumption that long time-frames might be required before sleep curtailment translates into appreciable changes in weight, few experimental studies have manipulated sleep duration to determine direct effects on weight. Two recent laboratory sleep deprivation studies, however, have examined the experimental effects of sleep restriction on weight among those permitted to eat ad libitum from a range of foods (Markwald et al., 2013; Spaeth et al., 2013). In the previously mentioned study of 225 healthy subjects (45% female) between 22 to 50 years of age, Spaeth (2013) showed that five consecutive days of sleep-restriction to 4 h/night in bed yielded a 0.97 kg increase in weight compared to control participants, who had a 0.11 kg increase over the same time period ($p < 0.007$). African-Americans and men were more susceptible to weight gain. In a smaller study ($N = 16$), Markwald et al. (2013) found that 5 days of sleep restricted to 5 h/night in bed yielded a 0.82 kg weight gain in a sample of healthy adults (50% female; mean age = 22.4). Interestingly, this study also found that transitioning from sleep restriction to 'recovery' sleep was associated with weight loss. While both of these studies benefit from the experimental rigour of a controlled environmental setting, it is not clear the extent to which these studies might generalize to the real-world environment with differential availability of food choices, for example. One recent study randomly assigned healthy men ($N = 19$) to sleep restriction (habitual bedtime minus 1.5 h) or a control condition (habitual bedtime) for three weeks and found that sleep restriction, measured by both actigraphy and diaries, was associated with an initial drop in weight during weeks 1 and 2 followed by a significant increase in weight from weeks 2 to 3 (Robertson et al., 2013). These findings suggest that the effects of sleep restriction on weight gain may be time dependent (i.e. there may be a period of time during which individuals may be at greatest risk for physiological and behavioural changes that occur with exposure to sleep reduction), highlighting the methodological concern that single visit studies, both epidemiological and laboratory-based, may be problematic and that longer-term studies, though challenging to execute, are needed.

Summary of experimental evidence and future considerations

In summary, research conducted in the laboratory setting, which demonstrates causality in a way that cross-sectional and longitudinal studies cannot, has demonstrated that sleep restriction promotes weight gain. Research has been more consistent in finding an association between sleep restriction and energy intake than energy expenditure. Both differences in sleep restriction models (e.g. number of days and hours of restriction) and the energy balance model utilized (controlled energy intake versus ad libitum) likely explain inconsistencies in the literature. Recent research has also suggested that later timing of sleep and later timing of food intake may play a role in obesity. These timing factors should also be considered in future sleep restriction/duration studies.

Important moderators may explain why reduced sleep leads to weigh gain in some individuals and not others. For example, Chaput and colleagues (2011) have shown that having a high disinhibition eating behaviour trait (as measured by the Three-Factor Eating Questionnaire) increases the propensity to overeat in those with short sleep duration. Although this has yet to be demonstrated in an experimental study (the 6-year Quebec Family Study was a longitudinal cohort study), it suggests that short sleep duration alone may not explain obesity risk and that hedonic aspects of overeating (i.e. increased pleasure/reward associated with overeating) help explain the association between short sleep duration and overeating and/or weight gain (Chaput, 2013). Indeed, interesting work is being done in the area of neuroimaging, with some studies showing that sleep restriction is associated with increased activation of brain regions sensitive to food stimuli (Benedict et al., 2012; Greer et al., 2013; St-Onge et al., 2012, 2013). (Chaput, 2013). Future research should consider the reward-driven characteristics of eating behaviour to further characterize those with short sleep duration.

Other behavioural sleep disorders and obesity: insomnia

The majority of studies investigating sleep and weight gain have focused largely on self-reported sleep duration in the general population or on the experimental manipulation of sleep duration in healthy

subjects. While data suggest that many individuals without intrinsic sleep disorders voluntarily curtail their sleep due to leisure activities or work-related demand, sleep disorders are highly prevalent and are a common reason why individuals obtain either deficient or longer sleep. As noted above, a major limitation of many of the sleep duration weight findings is that these studies failed to screen or account for the effects of sleep disorders. Many sleep disorders such as insomnia and sleep apnoea are caused by pathophysiological processes, such as autonomic and central nervous system hyperarousal in the case of insomnia disorder and intermittent hypoxia in the case of sleep apnoea, which may have effects on weight gain independent from actual sleep duration.

Insomnia disorder is defined as trouble initiating or maintaining sleep, despite adequate opportunity for sleep, associated with daytime impairment. Recent diagnostic nosologies require symptom duration of at least 3 months (APA, 2013). Many, but not all, patients with insomnia demonstrate curtailed sleep time. To date we are not aware of any studies that have used full insomnia disorder diagnostic criteria to study possible linkages with weight gain. Recent prospective research has shown that higher BMI and obesity are associated with persistent frequent insomnia symptoms among adults independent of important covariates including social background, physical and mental health (Lallukka et al., 2012). Additional longitudinal research has reported that obesity, but not sleep apnoea (apnoea or hypoapnoea index ≥ 5), is a risk factor for new-onset chronic insomnia (Singareddy et al., 2012). In this study, however, insomnia was defined atypically (a positive response to the question: do you feel you have insomnia with a duration of at least one year?). In the analyses the authors atypically differentiated insomnia from poor sleepers who were defined as subjects who did not endorse the insomnia item but who reported moderate to severe difficulty with falling asleep, staying asleep, early morning awakenings and non-restorative sleep. A more recent study by the same group revealed that self-reported poor sleep, but not self-identified 'insomnia' (both defined in the same manner as the above study) and emotional stress mediated the relationship between subjective sleep duration and later obesity (Vgontzas et al., 2013a). Interestingly, studies have shown that insomnia with objective short sleep duration is associated with significant deficits in neuropsychological performance, morbidity, and mortality (Fernandez-Mendoza et al., 2010, 2012; Vgontzas et al., 2010). In fact, Vgontzas et al. (2013a) have recently summarized research suggesting that insomnia with objective short sleep duration is the most biologically severe phenotype of insomnia, given its unremitting course and association with cognitive-emotional and cortical arousal, activation of both limbs of the stress system (the HPA axis and the sympathetic system), and medical complications often co-morbid with obesity (e.g. diabetes and hypertension). In general, the literature linking sleep duration and weight has largely omitted the possible role of clinical insomnia, and future work is needed to determine whether insomnia, one of the most common causes of reduced sleep time, might be an important intervention target to combine with weight loss programmes.

Clinical trials: an examination of sleep and weight loss

Sleep as a predictor of weight loss outcomes

Recent research has begun to describe the relationship between sleep and behavioural weight loss outcomes in clinical trials. In a randomized clinical trial of a commercial weight loss programme, Thomson and colleagues (2012) examined eight components of 245 overweight or obese women's sleep as assessed by the Pittsburgh Sleep Quality Index (Buysse et al., 1989) at baseline and 6 months in relation to weight outcomes at 6, 18 and 24 months. Results indicated that higher sleep quality scores and longer sleep duration were associated with greater success in the weight loss programme (defined as losing $\geq 10\%$ of initial body weight). More specifically, sleeping ≥ 7 h per night and better subjective sleep quality at baseline increased the likelihood of weight loss success at 6 months by approximately 30%. Worse global sleep scores (≥ 5) and sleeping < 7 h per night at 6 months were both associated with a lower likelihood of successful weight loss maintenance at 12 and 18 months, but not at 24 months.

When timing of food intake was examined in relation to weight loss effectiveness in a sample of 420 adults participating in a behavioural weight loss treatment based on a Mediterranean diet, late eaters (those who typically had a lunchtime after 3:00 p.m.) lost significantly less weight than early eaters (lunchtime before 3:00 p.m.) during 20 weeks of treatment (Garaulet et al., 2013). Interestingly, late eaters were more likely to be of the late to bed/late to rise wake/sleep pattern (i.e. evening type), suggesting that shifting timing of food intake and perhaps adjusting sleep timing during weight loss interventions could promote greater weight loss success.

Behavioural sleep interventions and weight loss

Although the evidence to date largely supports the need for randomized clinical trials to determine whether sleep interventions, particularly those designed to extend sleep in those with insufficient sleep, can impact body weight, to our knowledge only one study

in adults has been published. In the Better Weight-Better Sleep (BWBS) pilot study, 49 overweight or obese (BMI 25–39.9 kg/m^2) adults from a family medicine centre were randomized to either a cognitive behavioural weight management intervention (BW) or to BW combined with a sleep intervention that included cognitive behavioural strategies for the treatment of insomnia (BWBS). Participants in the BWBS arm lost significantly more weight at 12 weeks (Logue et al., 2012). Sleep-related exclusions included diagnosed uncontrolled sleep apnoea, modified Berlin apnoea score >2 (removing obesity and hypertension from the standard score), secondary sleep disorders (restless leg syndrome, narcolepsy/cataplexy, enuresis, sleep walking, sleep-related eating, sleep violence) and hypnotic drug dependency. The sleep management intervention started at week 4. Participants in the BWBS arm lost 5% of initial body weight, a percentage of weight loss that is considered clinically relevant in obesity trials, compared to 2% in the BW group.

Although no weight results are currently published, Cizza et al. (2010) are conducting a randomized, prospective, proof-of-concept study assessing the impact of sleep extension over 12 months on weight. This study is randomly assigning 150 obese participants who sleep less than 6.5 h per night to either a non-pharmacological, behavioural sleep extension intervention conducted in the real world setting or a control condition. Randomization is stratified by age and by presence or absence of metabolic syndrome, and there is no focus on weight management strategies during the 12-month intervention. This study will offer important insights regarding whether behavioural sleep treatment alone impacts weight outcomes.

Positive airway pressure therapy for sleep disordered breathing and weight loss

Although substantial research demonstrates that weight loss significantly improves sleep apnoea (Fujii et al., 2010; Johansson et al., 2011), surprisingly very few studies have evaluated whether positive airway pressure therapy (PAP), which reverses sleep-disordered breathing and improves sleep quality and daytime sleepiness, impacts weight. One recent randomized sham-controlled crossover study trial found that compared to sham therapy, 8 weeks of PAP failed to change BMI and adipose tissue distribution (Sivam et al., 2012). A retrospective study found that regular PAP therapy use was actually associated with increased weight gain relative to patients who did not adhere to PAP therapy (Redenius et al., 2008). A 2-year prospective study, however, found that sleep apnoea patients with hypertension who were treated via PAP therapy lost significant weight relative to patients without hypertension (Saito et al., 2010). A small study by Loube et al. (1997) also found that PAP therapy facilitated weight loss in 36 obese patients with sleep apnoea. It is unclear whether treating sleep fragmentation using PAP therapy has the same potential impact on weight as treating sleep duration. Future work will be necessary to determine the possible benefits of PAP therapy, if any, when combined with weight loss programmes.

Conclusion

Based on the current review, it appears that clinicians and investigators should consider adding sleep to the multifactorial and interactive list of factors that contribute to obesity. There is good experimental and substantive longitudinal data linking short sleep to increased weight gain and obesity as well as to behaviours implicated in weight gain (e.g. snacking). Evidence for the effects of long sleep duration on BMI is less clear. Emerging research also suggests the possibility of chronobiological effects on weight such that evening/nocturnal caloric intake might play an independent role in weight gain. A collective consideration of past reviews combined with more recent research not only illuminates the complexity of understanding the relationship between sleep characteristics and obesity, it highlights the need for further study using both subjective and objective measures of sleep that include clinical indices of common sleep disorders. Because there are many aspects of sleep that appear to impact weight, and because the interactions between these sleep variables are often complex and bidirectional, there is a need to better understand multi-measured sleep components both in combination with and independent of sleep duration. In order to truly disentangle the sleep–obesity relationship, large randomized prospective controlled trials with adequate power to examine important modifying effects of sleep disorder subgroups, particularly patients with insomnia, sleep-disordered breathing and insufficient sleep are needed. Given that the data linking poor sleep to obesity and weight gain are relatively compelling and standard long-term weight loss outcomes relatively modest, clinical weight loss trials should consider studying the potential benefits of treating disordered sleep in overweight/obese patients. For example, behaviour therapy for insomnia, behavioural sleep extension approaches for short sleepers, and light-therapy/melatonin supplementation for circadian rhythm disorders should all be considered as potential treatment approaches. Methods for integrating sleep therapies into weight loss and obesity prevention interventions are only beginning to be developed.

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