In: Hormones and Behavior
Editor: Davis Simonsen

ISBN: 978-1-62417-767-5

Chapter V

Altered Energy Balance in Response to Sleep Restriction

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Abstract

Many epidemiological studies suggest that short sleep duration leads to weight and fat mass gains over time. Sleep restriction (5.5 hours of sleep/night) has also been shown to alter weight loss success, by promoting a greater loss of fat-free mass coupled with a decrease in fat mass loss when compared to individuals who received 8.5 hours of sleep per night. Current evidence suggests that sleep restriction may lead to increased food intake but does not appear to result in decreased energy expenditure. Sleep restriction coupled with clamped energy intake has been suggested to alter the neuroendocrine regulation of appetite, through increases in the levels of ghrelin, combined with decreases

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in leptin. However, these variations may be subdued by an adequate access to food. Current evidence also suggests that sleep restriction leads to decreases in insulin sensitivity and acute insulin responses to glucose. Conversely, shortsleepers have glucose responses that are similar to averagesleepers, but at the cost of an increase in insulin release, which may be the result of decreased insulin sensitivity over time. Recent studies also provide evidence that sleep restriction enhances susceptibility to food stimuli, especially for energy-dense, high-carbohydrate foods. Furthermore, reduced sleep quality, defined as decreases in slow-wave sleep, has been suggested to alter proper neuroendocrine functions. However, these alterations only seem to occur following total sleep deprivation since slow-wave sleep is usually preserved during partial sleep restriction. In summary, current evidence suggests that the severity (number of hours per night) and duration (number of days) of sleep restriction are likely important factors in determining the extent of metabolic and non-homeostatic changes that may alter energy balance.

Keywords: Sleep restriction, energy intake, energy expenditure, non-homeostatic drive to eat, neuroendocrine/metabolic hormones

Introduction

In a society which is operational 24 hours a day, 7 days a week, many choose to voluntarily decrease their sleep time, which may in turn lead to metabolic and health problems over time [1; 2]. Even if sleep is considered to be the most sedentary of all human activities, it may in fact help facilitate appetite control and promote the maintenance of a healthy body weight [3]. Furthermore, short sleep duration and disinhibited eating behaviour trait have been suggested to play an important role in promoting obesity [4], highlighting the fact that body weight control is multi-factorial and involves "non-caloric" factors.

Many epidemiological studies have noted that adults [5] and children [6] who are short sleepers (< 6 hours of sleep/night in adults and < 10 hours of sleep/night in children) tend to have greater body weight, body mass index (BMI), body fat percentage and abdominal circumference when compared to average sleepers (7-8 hours of sleep/night in adults and 10-11.9 hours of sleep/night in children). Short sleep duration was further associated to an increased risk of weight and fat mass gains over time in adults [5], making it an important risk factor for the development of obesity with odds ratios exceeding other well-known contributors (*e.g.* high dietary lipid intake and the non-participation in high-intensity physical activity, with odds ratios of 1.64 and 2.03 respectively vs. 3.81 for short sleep duration) [4]. Similar results were observed in children aged between 5 and 10 years, where short sleep duration was determined as being the most important risk factor for excess weight gain when compared to other well-known contributors (*e.g.* parental obesity, $\geq$ 3 hours of television viewing and physical inactivity, with odds ratios of 2.39, 2.08 and 1.45 respectively vs. 3.45 for short sleep duration in boys and girls combined) [7].

This chapter reviews the literature on the effects of sleep restriction (SR), total sleep deprivation (TSD) and short sleep duration on energy metabolism, appetite control and weight gain. Furthermore, the potential effects of altered sleep duration and sleep quality on the neuroendocrine regulation of appetite and the non-homeostatic drive to eat will be discussed.

Association between Short Sleep Duration and Weight Gain

As briefly discussed, many epidemiological studies have noted a positive association between short sleep duration and weight gain in adults and children [8]. Based on a pooled regression analysis, Cappuccio *et al.* [2] determined that a decrease in 1 hour of sleep per night was associated with a BMI that is 0.35 kg/m^2 greater, thus suggesting that body weight is relative to the amount of hours spent sleeping per night. Chaput *et al.* [9] also recently observed that short sleep duration ($\leq$ 6 hours of sleep/night) is associated with increases in abdominal adiposity in adults, when compared to those who sleep on average $\geq$ 7 hours/night, over a 6-year follow-up period. In a similar matter, the association between sleep duration and waist circumference in children remained significant even after adjusting for BMI, thus suggesting that short sleep duration (< 10 hours of sleep/night) is a stronger predictor of central fat deposition compared to overall adiposity [10]. These findings are of particular concern because abdominal adiposity is correlated with a number of metabolic anomalies. In addition to sleep duration, sleep timing seems to also be an important predictor of weight gain. Gaina *et al.* [11] observed that morning-type children (*i.e.* children that wake earlier) tend to have a lower BMI when compared to evening-type children (*i.e.* children that go to bed later). Furthermore, Olds *et al.* [12] indicated that morning-type children were less likely to be obese when compared to evening-type children, despite similar sleep durations. This study also mentions that evening-type children were almost twice as likely to have low levels of moderate-to-vigorous physical activity participationand were 2.9 times more likely to watch television and play video games for more hours than those recommended in guidelines. Lastly, a recent study in adults [13] noted that those who went to bed later consumed more kilocalories after 8pm when compared to those who went to bed earlier, which was a positive predictor of BMI after controlling for sleep duration and sleep timing. Studies assessing the effects of sleep extension on weight change in short sleepers are needed to evaluate the potential benefits of prolonging sleep duration in these individuals. Preliminary results from a randomized controlled trial that is currently under way in obese short sleepers (< 6.5 hours of sleep/night) noted that participants who increased their sleep duration reported a better mood and ability to focus, a decrease in sleepiness during the day, more willingness to exercise, as well as a decrease in caffeine intake and less cravings for sweet and salty foods during the evening [14].

Furthermore, a longitudinal study by Chaput *et al.* [15] noted that short sleepers ($\leq$ 6 hours of sleep/night) who increased their sleep time to 7-8 hours per night had adiposity gains, which were similar to average sleepers (7-8 hours of sleep/night) when measured over a 6-year follow-up period. In summary, these results suggest that shifting sleep duration (*i.e.* from short to average duration) may have beneficial effects on overall mental and physical health, and may lead to a lower adiposity gain over time. Future sleep intervention studies should also concentrate on sleep timing and promoting earlier sleep times which may aid in preventing weight gain over time.

Lastly, it is important to target the root causes of habitual short sleep duration (*e.g.* insomnia, anxiety, longer work days, shift work) since the reasons can be very different between individuals, and not all short sleepers ($\leq$ 6 hours of sleep/night) require additional sleep time [16].

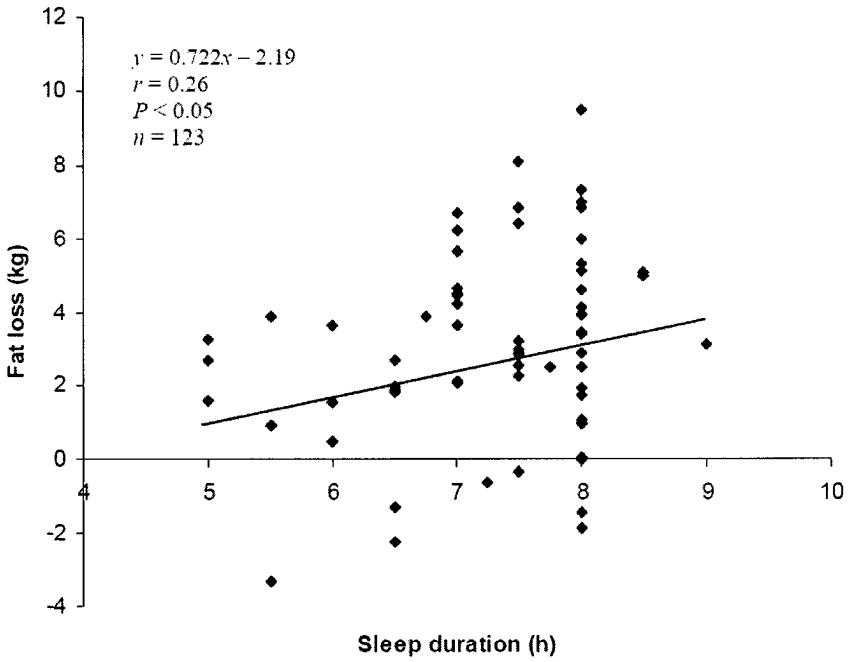

Figure 1. Sleep duration in relation to absolute body fat mass loss following a supervised weight loss intervention (*i.e.* 600-700 kilocalorie/day decrease in energy intake) in adults after adjusting for age, sex, baseline body mass index and the change in total energy intake. Note: h, hours; kg, kilogram. Figure from Chaput and Tremblay [18], reproduced with permission of S. Krager AG, Basel, Obesity Facts, Vol. 5, p. 564, © 2012.

Association between Short Sleep Duration and Weight Loss Success

SR and/or habitual short sleep duration has been shown to undermine weight loss success [17]. Chaput and Tremblay [18] recently observed that total sleep time at baseline predicted fat mass loss in overweight and obese adults subjected to moderate caloric restriction, where an increase in 1 hour of sleep per night was associated with a decrease of 0.7 kg in fat mass after adjusting for covariates (Figure 1). Following a 14-day energy restriction diet, Nedeltcheva *et al.* [19] noted that participants who slept 5.5 hours per night saw a greater loss in fat-free mass coupled with a decrease in fat mass loss when compared to participants who slept 8.5 hours per night, despite similar body weight lost. The sleep restricted group also reported greater perceived hunger ratings and saw a greater than expected decrease in their resting metabolic rate based on fat mass and fat-free mass lost. Conversely, a study which evaluated the potential changes in resting energy expenditure following a 17-week caloric restriction (-300 kilocalories/day on average) in habitual short (< 6 hours of sleep/night) and average (7-9 hours of sleep/night) sleepers noted that sleep duration was not associated with a greater than predicted decrease in resting energy expenditure [20]. The discrepancy in these results may be in part explained by the study designs used (short-term in-laboratory intervention vs. long-term intervention under free-living conditions), the populations tested (average sleepers who are sleep restricted vs. habitual short sleepers) and the proportion of

body weight lost as fat (25% in the sleep restricted group vs. 73% in habitual short sleepers). It may be hypothesized that caloric restriction interventions may have a greater than expected effect on the resting energy expenditure of average sleepers who are subjected to SR when compared to habitual short sleepers, who may either be accustomed to shorter sleep durations and/or may not require more than 6 hours of sleep per night [16]. However, future studies on this subject would be needed before drawing any further conclusions.

How Sleep Restriction May Alter Energy and macronutrient Intake

There are many causes that underlie voluntary SR, whether it be in part due to longer work days, increased night and shift work, increased social and/or family demands, as well as possible mental distress (*e.g.* depression and anxiety) which may cause insomnia [1; 21]. As previously mentioned, it is also possible that certain individuals do not require more than 6 hours of sleep per night [16]. In certain instances, SR may promote a positive energy balance by increasing the amount of time available to eat, as well as amplifying fatigue, an effect that could lead to a decrease in the motivation to exercise [5]. However, there is currently not enough substantial evidence in support of any meaningful effects of sleep restriction on the different components of energy expenditure. These factors, as well as others which may potentially influence energy balance in response to SR, are presented in Figure 2. In regard to EI, short sleepers ($\leq$ 6 hours of sleep/night) are more likely to have irregular eating habits, to snack between meals, to use an excessive amount of food seasoning, and to consume less vegetables when compared to average sleepers [22; 23]. These results are in part supported by a study in adolescent short sleepers (< 8 hours of sleep/night) who reported consuming relatively higher EI from fat and were twice as likely to consume $\geq$ 475 kilocalories per day from snacks [24]. A small number of controlled crossover studies have previously evaluated the effects of SR on *ad libitum* energy and macronutrient intake, but conflicting results have ensued. Certain studies observed no significant effect of SR (5.5 hours of sleep/night for 14 nights; 4 hours of sleep/night for 2 nights) [25; 26] and TSD (1 night) [27] on energy and macronutrient intake when measured inside the laboratory setting. However, Nedeltcheva *et al.* [25] reported an increase in snack intake between 7pm-7am, which was mainly characterized by an increase in carbohydrate intake, despite no significant increase in total energy and macronutrient intake during 14 days of SR (5.5 hours of sleep/night). Additionally, a study that measured energy and macronutrient intake inside the laboratory (breakfast and lunch) and under free-living conditions (afternoon snacks and dinner) showed greater total energy and fat intake following 2 days of SR (4 hours of sleep/night), when compared to the control condition (2 days of 8 hours of sleep/night) [28]. This increase was mainly characterized by greater total EI during breakfast and dinner, as well as greater fat intake during dinner only. A controlled intervention study also noted significantly greater EI using dietary records over 4 days of increasing SR (1 night of 7 hours, 2 nights of 6 hours and 1 night of 4 hours of sleep/night) when compared to baseline (2 nights of more than 8 hours of sleep/night) [29]. There is indeed some evidence suggesting that SR, especially when measured under free-living conditions, may lead to greater energy, fat and snack intakes. However, the SR interventions imposed in these studies are short-term and are often imposed

in average sleepers, limiting our ability to generalize these results to habitual short sleepers. Furthermore, the multi-factorial causes of habitual short sleep duration makes it more difficult to understand the potential reasons behind the short sleep-obesity link [5].

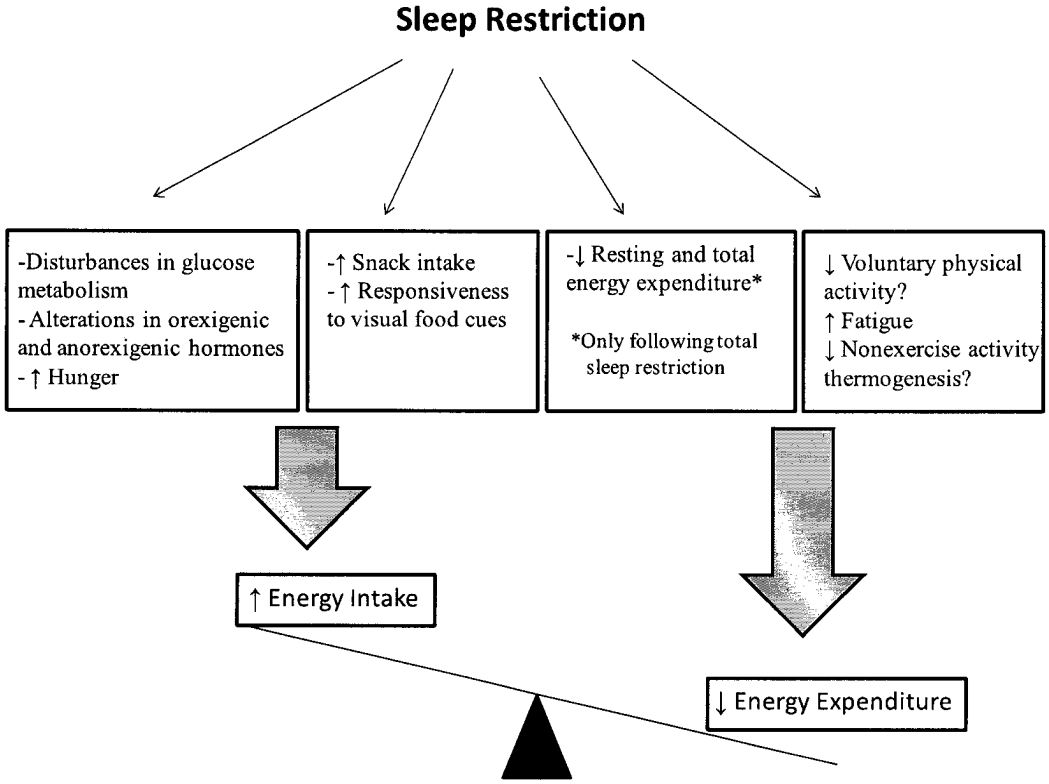

Figure 2. Potential mechanisms related to sleep restriction which may lead to alterations in energy balance. This figure illustrates certain potential mechanisms by which sleep restriction may influence energy balance, by increasing energy intake and/or decreasing energy expenditure. Figure adapted from Chaput *et al.* [3].

How Sleep Restriction May Alter Energy Expenditure

Current evidence suggests that SR does not alter total energy expenditure (TEE), the thermic effect of food (TEF) and resting energy expenditure (REE) [25; 29]. A 6-year prospective cohort study noted no significant difference in REE between short- (< 6 hours of sleep/night), average- (7-8 hours of sleep/night) and long-duration (> 9 hours of sleep/night) sleepers [5]. However, a significant decrease in REE (≈5%) and postprandial energy expenditure (≈20%), measured before and after a standardized breakfast respectively, were observed following 1 night of TSD [27]. Taken together, these results suggest that acute decreases in energy expenditure (EE) may be dose-dependent, where TSD conditions are needed in order to induce any changes in TEE, REE and/or TEF. In addition to sleep duration, slow-wave sleep (SWS) has been shown to have an essential energy preserving function by decreasing core body temperature and oxygen consumption during sleep [30; 31]. Many

studies have shown that SWS is maintained during SR [25; 26; 32-35]. Conversely, its absence during TSD has been suggested to have a potential effect on daytime EE [27], which may in part explain the discrepancy in REE and postprandial EE values noted between studies that have subjected participants to SR *vs.* TSD [36]. However, the effect of different sleep stages, such as SWS, on EE remains to be thoroughly evaluated since it is unknown whether SWS suppression may indeed lead to alterations in daytime EE. As for physical activity energy expenditure (PAEE), 2 studies saw no significant difference in this variable when assessed inside the laboratory [25] and under free-living conditions [29] following SR. Conversely, a study noted greater PAEE under free-living conditions following SR [28], while another saw a decrease in PAEE and moderate-to-vigorous physical activity participation after 4pm following SR [26]. Since both these studies followed a similar design and evaluated PAEE with accelerometry under free-living conditions in participants following similar sleep durations and timing (4 hours of sleep/night for 2 nights from ≈ 2h30-6h30am during the SR condition), it may be hypothesized that inter-individual variations, rather than physiological alterations, may in part explain the effect of SR on PAEE.

Possible Factors for Altered Energy Balance: Neuroendocrine and Metabolic Hormones

Following a sufficient amount of sleep, cortisol levels usually peak 30-45 minutes after awakening and decrease throughout the day, attaining its nadir approximately 12 hours later [37]. However, SR has been shown to alter the release of cortisol levels, where its levels decline more slowly throughout the day, thus leading to greater cortisol levels in the afternoon and evening [38-44]. Additionally, Omisade *et al.* [39] noted that morning cortisol levels were significantly reduced following 3 hours of sleep for 1 night when compared to 10 hours of sleep for 1 night, while afternoon and evening cortisol levels were higher post-SR. These results thus show that the slope of decline in cortisol levels throughout the day was lower following SR. Increased cortisol levels in the evening have been suggested to result in decreased insulin sensitivity the following morning [37; 45]. Furthermore, increases in sympathetic nervous system activation in response to stress has been shown to inhibit the secretion of leptin and insulin [46]. It has been previously suggested that the down-regulation of the hypothalamic-pituitary-adrenal (HPA) axis may fail to occur following SR, which results in greater evening cortisol levels [47]. And so, greater sympathetic nervous system activity and evening cortisol levels induced by SR may cause disturbances in glucose metabolism and/or the release of certain metabolic hormones (*i.e.* leptin and insulin). These neuroendocrine and metabolic hormone variations due to SR are presented in Figure 3.

As for glucose metabolism, glucose utilization is greatest during wake and lowest during SWS, with intermediate levels occurring during rapid eye movement (REM) sleep and light non-REM sleep [49]. This may simply be explained by variations in oxygen consumption rate during sleep, where its values are greatest during REM sleep and lowest during SWS [31]. And so, when participants are subjected to TSD, nocturnal glucose levels were found to be lower [27], which may be due to the decrease (or lack of) SWS coupled with greater EE associated with a prolonged waking state [50; 51]. Furthermore, when participants were subjected to SR or TSD, certain studies have shown that glucose levels are significantly

increased following a standardized breakfast without an adequate, subsequent rise in insulin, thus leading to a decrease in the effectiveness of insulin-mediated glucose uptake [27; 38; 52]. However, the response of insulin to the rise in glucose levels following midday and evening meals were the same between the SR/TSD and control conditions in these studies. Further evidence suggests that 5 days of SR (4 hours of sleep/night) lead to an increase in the fasting (*i.e.* measurement prior to breakfast) insulin to glucose ratio [53], thus suggesting that a decrease in insulin sensitivity may have transpired. Additionally, non-obese short sleepers (< 6.5 hours of sleep/night for 6 months) had glucose responses to an intravenous glucose tolerance test that were similar to non-obese average sleepers (7.5-8.5 hours of sleep/night), but at the cost of a significantly greater increase in insulin release [54], which may be the product of decreased insulin sensitivity over time. The participants in each group were carefully matched for gender and ethnic differences, as well as exercise habits. This study further confirmed that insulin sensitivity in short sleepers was almost 40% lower when compared to average sleepers. Lastly, decreases in SWS for 3 days also lead to decreases in insulin sensitivity [55], which suggests that an adequate amount of SWS, in addition to total sleep duration, is needed to maintain proper glucose metabolism. Based on these results, it may be hypothesized that SR, habitual short sleep duration and/or decreased sleep quality may lead to the development of insulin resistance, independently of changes in BMI and/or body weight.

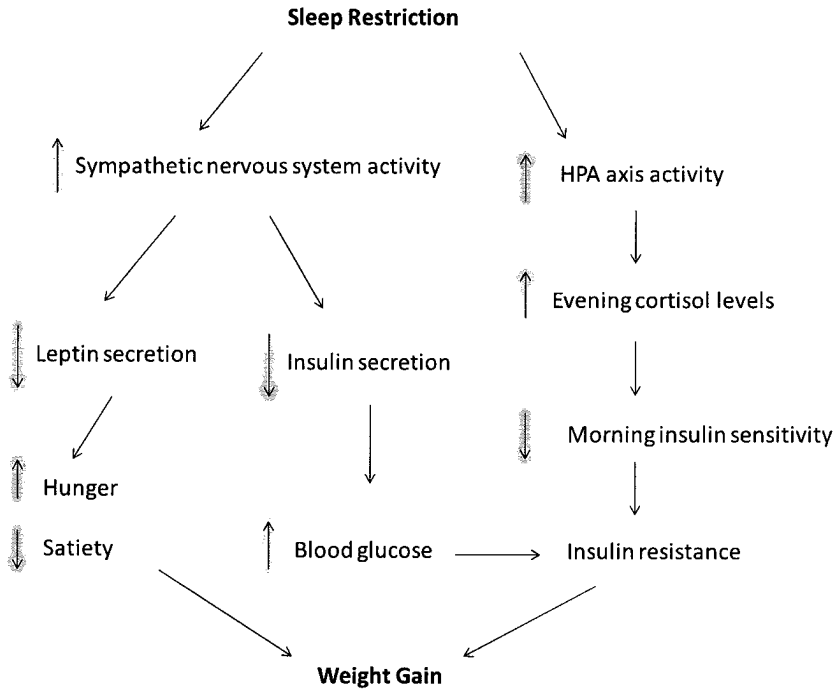

Figure 3. The neuroendocrine and metabolic hormone variations induced by sleep restriction, which may lead to weight gain. This figure illustrates the effects of sleep restriction on the neuroendocrine system, thus leading to alterations in cortisol and certain metabolic hormones in a way which may induce weight gain. Note: HPA, hypothalamic-pituitary-adrenal. Figure adapted from Zimberg *et al.* [48].

Sleep *per se* has been suggested to have an inhibitory effect on EI, with leptin levels attaining its zenith at night and ghrelin levels increasing during the 1st half of the night before decreasing during the 2nd half of the night despite no increase in food consumption at this time [43]. Chaput *et al.* [56] noted that short sleepers (< 6 hours of sleep/night) had mean leptin levels that were lower than that predicted by their fat mass, thus suggesting that habitual short sleep duration may decrease leptin levels. Furthermore, certain crossover studies, which imposed SR interventions with clamped caloric intakes saw a decrease in leptin levels following the SR condition (4 hours of sleep/night for 2 nights; 4 hours of sleep/night for 6 nights) [42; 52]. Conversely, other crossover studies that imposed different SR interventions (5.5 hours of sleep/night for 14 nights; 4 hours/night for 2 nights; 1 night of TSD; 1 night of TSD and 1 night of 4.5 hours of sleep/night) with *ad libitum* access to food noted no changes in leptin levels between sleep duration conditions [25-27; 57]. And so, it may be hypothesized that free access to food will not lead to alterations in leptin in response to SR. Similar results were found for ghrelin, where increases in this hormone were only noted following SR (4 hours of sleep/night for 2 nights) coupled with standardized EI [42]. These results thus suggest that leptin and ghrelin variations ensued by SR may be subdued by an increase in EI and/or adequate access to food. Furthermore, nocturnal increases in ghrelin during the 2nd half of the night only seem to occur following TSD when given adequate access to food [27]. These results are further supported by a study from Schmid *et al.* [57], who noted a significant increase in ghrelin following 1 night of TSD when compared to 1 night of 7 hours of sleep. Conversely, no significant differences in ghrelin levels were noted in the same participants following 1 night of 4.5 hours of sleep when compared to 1 night of 7 hours of sleep. Based on these results, it may be hypothesized that an increase in EI following SR may be needed in order for leptin and ghrelin values to be similar as when measured following adequate sleep durations. However, as previously mentioned, certain studies observed no differences in EI between sleep duration conditions (5.5 hours of sleep/night for 14 nights; 4 hours of sleep/night for 2 nights; 1 night of TSD) when participants had *ad libitum* access to food inside the laboratory setting [25-27], while others noted an increase in EI following the SR conditions (4 hours of sleep/night for 2 nights; 1 night of 7 hours, 2 nights of 6 hours and 1 night of 4 hours of sleep/night) when measured under free-living conditions [28; 29]. Consequently, the reasons behind the effects of SR on food intake still seem to be obscure at best. Many reasons may underlie the discrepancy in results noted by different studies, such as the severity (number of hours per night) and duration (number of days) of SR interventions used, the timing of SR (earlier vs. later in the night, which may alter circadian rhythm), the study designs employed (controlled in-lab vs. free-living conditions), as well as the populations tested (*e.g.* men vs. women, habitual average- and/or shortsleepers, the exclusion criterions followed).

In summary, greater cortisol levels during the evening due to SR may alter insulin sensitivity, which may then negatively affect glucose uptake and potentially lead to the development of insulin resistance over time, independently of changes in BMI and/or body weight. Furthermore, increases in abdominal adiposity, which has been previously associated with habitual short sleep duration [9], may be a possible consequence of greater evening cortisol levels and altered insulin sensitivity. It is also hypothesized that leptin and ghrelin variations ensued by SR may be subdued by an increase in EI and/or adequate access to food. Hence, future studies would be needed to evaluate the effects of different SR severities

(number of hours per night), as well as *ad libitum* vs. controlled caloric intake on leptin and ghrelin levels within a same study design.

Possible Factors for Altered Energy Balance: The Non-Homeostatic Drive to Eat and Appetite

Generally speaking, the non-homeostatic drive to eat is influenced by goal oriented choices made by the higher cortical association areas of the brain, and is based on past experiences and positive associations made with, for instance, specific types of foods [58; 59]. Eating behaviors are often influenced by different cognitive, hedonic, social and environmental factors, which may greatly influence choices in food consumption [59]. To date, few studies have looked at the possible relationship between reward-driven eating behaviour and SR.

Functional MRI results from Holm *et al.* [60] showed a decreased reactivity in the ventral striatum, one of the primary reward centers of the brain, in adolescents with short sleep durations, decreased sleep quality and later sleep onset times when anticipating and receiving a monetary reward. St-Onge *et al.* [61] also noted greater neuronal activation in the orbitofrontal cortex, an area of the brain related to motivation, in response to food cues when compared to non-food cues following SR (4 hours of sleep/night for 6 nights). Similar results were noted following 1 night of TSD, where a greater activation of the right anterior cingulate cortex, which plays a key role in perception, was observed when participants were presented with images of foods with different caloric (high vs. low) content [62].

These participants also rated high calorie foods as being 24% more appetizing following TSD. Taken together, these results suggest that individuals experiencing SR or TSD may have an enhanced susceptibility to food cues and their rewarding properties.

Changes in hunger and appetite have also been shown to occur following SR and TSD. For instance, SR (4 hours of sleep/night) for 2 nights led to a 24% and 23% increase in hunger and appetite ratings respectively for all food categories. Furthermore, this increase in appetite ratings tended to be greatest for energy dense foods that were high in carbohydrates, while appetite for fruits and vegetables increased to a lesser extent [42]. It may also be suggested that hunger ratings may be dependent on the severity of SR, since increases in hunger ratings were greatest following 1 night of TSD, while more subtle increases occurred following 1 night of SR (4.5 hours of sleep/night) [57].

Even though increases in hunger and appetite ratings for energy dense foods seem to occur following SR and TSD, its effects on actual food intake seem to be contradictory. Certain studies, which subjected participants to SR (4 hours of sleep/night for 2 nights), observed that hunger and appetite ratings coincided with food consumption [26; 28]. However, in a study by Benedict *et al.* [62] where participants were subjected to 1 night of TSD, greater hunger ratings were observed the following morning and did not translate into greater EI at that time. It may thus be suggested that increases in appetite and hunger ratings may vary with the severity (number of hours per night) of SR, and that these increases seem to only predict food consumption following partial SR.

Conclusion

In summary, SR may increase the response to certain food cues, which may then predispose certain individuals to increased EI. As for EE, it would seem that TSD only may lead to alterations in TEE, while studies that evaluated the effects of SR on PAEE have ensued conflicting results [25, 26, 28, 29]. It is thus important to bear in mind that the severity (number of hours per night), duration (number of days) and timing (*e.g.* SR during the 1st half of the night vs. 2nd half of the night) of SR are likely important factors in determining the onset and severity of metabolic, non-homeostatic and sleep architectural changes that may alter energy balance.

Future studies are needed to measure the potential alterations in the non-homeostatic drive to eat in habitual short sleepers ($\leq$ 6 hours of sleep/night), when compared to average sleepers (7-8 hours of sleep/night), as well as the relationship between reward center activation in the brain to actual food intake. Additionally, the potential effects of SR on non-exercise activity thermogenesis (NEAT), which may play a substantial role in EE, remains to be elucidated. Taken together, there is some indication that SR may contribute to alterations in EI, the neuroendocrine regulation of appetite, and responses to certain food cues under distinct circumstances.

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