Chapter 2

The Role of Sleep in the Control of Feeding Behavior

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Chapter Outline

INTRODUCTION

Sleep duration has been shown to be inversely related to obesity risk, and short sleepers are at increased risk of large weight gain over time (Patel & Hu, 2008). However, these epidemiological observations do not infer a causal role of short sleep duration (SSD) on obesity. Nevertheless, several explanations have been proposed to elucidate the role of SSD on obesity risk. One involves a reduction in physical activity due to increased fatigue whereas others involve increases in food intake, either because of increased time spent awake (opportunity to eat) or as a result of hormonal changes that trigger increased appetite/hunger (Penev, 2007). However, all scenarios propose a positive energy balance, either via reduced energy expenditure or increased energy intake, which would explain the association with obesity and large weight gain.

Several intervention studies have been performed in an attempt to determine whether SSD precedes the development of obesity and could be an explanatory factor in the etiology of obesity (Bosy-Westphal et al., 2008; Brondel, Romer, Nougues, Touyarou, & Davenne, 2010; Nedeltcheva et al., 2009; Schmid et al., 2009; St-Onge et al., 2011). Such studies have measured energy expenditure and energy intake during periods of sleep restriction compared with habitual sleep in normal sleepers (defined as sleeping 7–9 h/night). The amount of sleep permitted in those studies ranged from 4 to 6 h/night in sleep restriction studies to no sleep at all in sleep deprivation studies. The effects of sleep duration on components of energy balance (energy expenditure and energy intake) have been reviewed extensively by St-Onge (2013) and Penev (2012). In brief, on the basis of current literature, one would conclude that SSD leads to weight gain/obesity via increased food intake rather than as a result of a reduction in metabolic rate (energy expenditure). In fact, Shechter, Rising, Albu, and St-Onge (2013), Klingenberg et al. (2012), Markwald et al. (2013), have shown that sleep restriction increases 24-h energy expenditure measured in a metabolic chamber as a result of the extended time spent awake and the energetic cost associated with the wake state. Resting metabolic rate is not fundamentally altered by sleep restriction. However, the extent to which 24-h energy expenditure is increased with sleep restriction does not match the observed increase in energy intake associated with a similar degree of sleep restriction in clinical interventions. Additional and more extensive studies are needed to examine the effect of sleep restriction on voluntary physical activity. It is possible that restricting sleep leads to increased fatigue, which would prompt one to choose not to exercise or to do so at a lower intensity and thus energetic cost. However, this has not been verified in a clinical study. If this were the case, the reduction in energy expenditure associated with reduced voluntary physical activity would accentuate the state of positive energy balance that results when one is placed in a condition of sleep restriction relative to habitual sleep.

Although alterations in energy expenditure and energy intake are likely involved in the etiology linking SSD to obesity, the preponderance of evidence surrounds its effects

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on food intake. Thus, the purpose of this chapter is to review the effect of sleep restriction on food choice and the neurological pathways that guide these decisions.

EFFECT OF SLEEP RESTRICTION ON HUNGER AND FOOD INTAKE

It is now generally well accepted that sleep restriction leads to increased food intake. Spiegel, Tasali, Penev, and Van Cauter (2004) were among the first to ask participants about their feelings of hunger and appetite after a 2-day period of sleep restriction (4 h time in bed (TIB)) relative to extended sleep (10 h TIB). Participants provided hourly ratings, on a visual analog scale ranging from 0 to 10 cm, to questions such as "How hungry do you feel right now?" and "How much would you enjoy eating sweets, salty foods, starchy foods, fruits and fruit juices, vegetables, meat/poultry/fish/eggs, and dairy products?" The mean of all ratings for hunger was 24% higher after the two nights of sleep restriction relative to sleep extension, and the mean appetite rating for all categories of foods combined was 23% higher. Furthermore, the increase in appetite ratings for high-carbohydrate, calorie-dense foods, such as sweets and salty and starchy foods, after sleep restriction relative to sleep extension tended to be greater than that for other food categories (33–45% higher vs. 17–21% higher, respectively). However, some limitations of this study are worth noting. First, all participants were males and the sample size was small ($n = 12$). Also, participants were on constant intravenous glucose infusion as their sole source of calories throughout the study. The lack of food consumption may have amplified the effect of sleep restriction on hunger and appetite ratings. Moreover, these measurements were not followed by tests of ad libitum food consumption and, although subjective ratings are correlated with feeding behavior (Drapeau et al., 2005; Griffioen-Roose, Finlayson, Mars, Blundell, & de Graaf, 2010; Parker et al., 2004), it remained unknown whether participants would actually consume more of the foods they reported wanting on a visual analog scale. On the other hand, caloric intakes and energy source were very well controlled and identical under both conditions, removing an element of variability from their study.

Since then, studies have been undertaken to determine whether food intake would be altered by sleep restriction relative to habitual sleep. In an inpatient study, Nedeltcheva et al. (2009) assessed energy intakes over two periods of 14 days differing in TIB, either 8.5 or 5.5 h, in a crossover, randomized design. Overweight men and women participated in that study. When subjected to the restricted sleep period, participants ate an average of 300 kcal more than when they spent 8.5 h in bed. Food intake distribution over the day was such that snack energy intakes, but not meal energy intakes, differed between sleep periods. Specifically, energy intakes from snacks increased and the snacks chosen were higher in carbohydrates and lower in fat and protein during the period of sleep restriction relative to habitual sleep. Moreover, the rise in snack energy intakes was mostly observed in the evening/overnight period rather than during the daytime hours. A similar degree of overeating was also observed over a single test day performed after four nights of 9 or 4 h TIB (St-Onge et al., 2011). In that study, participants consumed approximately 300 kcal more during the short sleep period compared with the habitual sleep phase and this tended to be most pronounced in women, who specifically increased their intakes of fat and saturated fat in the SSD period. However, in that study, there was no effect of sleep restriction on late-night eating. Other studies have also reported increased energy intakes after periods of SSD (Bosy-Westphal et al., 2008; Brondel et al., 2010), although one study did not note this effect (Schmid et al., 2009). In that study, food intake was assessed the day after two nights of either 8- or 4-h TIB conditions.

The data by Nedeltcheva et al. (2009) showing increased evening/night intakes with SSD are in line with more recent information by Spaeth, Dinges, and Goel (2013) showing that restricting sleep leads to increased energy intakes at night. In that study, adults were randomized to five nights of 4- or 10-h TIB (sleep extension). Participants who were randomized to the sleep restriction protocol gained weight relative to those who were randomized to the sleep extension protocol. Energy intakes in the former group were higher than in the latter. There was no difference in the distribution of macronutrients in the diet between protocols. Also, in the sleep-restricted participants, meal number increased during the days when bedtimes were delayed to achieve sleep restriction compared with baseline (prerestriction days). In those participants, the distribution of energy intake throughout the day shifted over time. In particular, participants consumed fewer calories in the morning/early afternoon hours (8:00 a.m. to 3:00 p.m.) and more calories in the overnight period (10:00 p.m. to 4:00 a.m.).

NEUROENDOCRINE CONTROL OF FOOD INTAKE AND SLEEP DURATION

There has been much research to determine how sleep restriction affects food intake and the control of energy balance. Most studies have examined if sleep duration alters hormonal signals of hunger and satiety, mostly focusing on leptin and ghrelin. Although early studies have found that restricting sleep duration increases ghrelin (Benedict et al., 2011; Spiegel et al., 2004; Taheri, Lin, Austin, Young, & Mignot, 2004) and decreases leptin relative to habitual sleep (Spiegel et al., 2004; Taheri et al., 2004), this has not been universally observed. In fact, several studies have failed to show that restricting sleep leads to changes in leptin and ghrelin relative to habitual sleep (Nedeltcheva et al., 2009) whereas others have found opposite results—that sleep

restriction increases leptin (Bosy-Westphal et al., 2008; Omisade, Buxton, & Rusak, 2010; Pejovic et al., 2010; Simpson, Banks, & Dinges, 2010) and decreases ghrelin (Dzaja et al., 2004). The reasons for these discrepant results are not clear, and plausible explanations for these discrepancies have been the topic of a review of literature (St-Onge, 2013). Potential sex differences have been proposed, but only a limited number of studies examining the effect of sleep restriction on a food-intake-related mechanism included participants of both sexes. The largest study to date, by St-Onge, O'Keeffe, Roberts, Roy Choudhury, and Laferrere (2012), found that men, but not women, had increased ghrelin after three nights of sleep restriction relative to habitual sleep whereas women, but not men, had increases in glucagon-like peptide-1 concentrations under those same conditions. Leptin concentrations were not affected by sleep duration in men or women. This study is the only study to date to have explored sex differences in the hormonal response to sleep restriction and the first to assess the role of sleep duration on glucagon-like peptide-1 concentrations. These data illustrate potentially completely different mechanisms in which sleep restriction leads to increased food intake in men and women—one implicating increased hunger in men and one implicating reduced satiation in women.

Other potential explanations for the varied leptin/ghrelin results between studies include differences in the degree of sleep restriction and state of energy balance of the participants. It is known that leptin and ghrelin concentrations are affected by the body's internal energy status (i.e., they respond to alterations in energy balance such that corrections in energy intake and energy expenditure can be made to restore balance). If restricting sleep leads to overeating, as described above, then any difference in leptin and ghrelin observed between conditions of habitual and restricted sleep, performed in ad libitum feeding paradigms, could be in part explained by the differences in food intake.

Leptin and ghrelin are considered neuroendocrine hormones. These adipose and gastric-derived hormones produce hypothalamic signals to stop or start eating by initiating crosstalk between several important brain regions. These signals lead to changes in neuronal activity patterns that affect cognition, decision-making, and pleasure. Such processes then guide behavior. Orexins A and B, which are synthesized by lateral hypothalamic neurons, are also considered to provide a link between sleep–wake regulation and the neuroendocrine control of food intake (Hanlon & Van Cauter, 2011). Orexin neurons are active during wake and quiescent during sleep, and they activate neuropeptide Y neurons in the arcuate nucleus, leading to increased appetite. These neurons project to the dopaminergic ventrotegmental area and nucleus accumbens, which are involved in the hedonic control of food intake.

Thus, studies have been undertaken to examine the neuronal pathways involved in the control of food intake under various levels of sleep restriction. When sleep deprivation is enforced, the orexin system is overactive to maintain wakefulness against one's pressure to sleep, suggesting that neurons of the dopaminergic system would be stimulated to promote feeding behaviors (Hanlon & Van Cauter, 2011). Benedict et al. (2012) performed a functional magnetic resonance imaging (fMRI) study with 12 healthy, young, normal weight men in which scanning took place in the morning after a 7-h TIB sleep opportunity or total sleep deprivation (TSD). Participants consumed a standardized dinner (700 kcal) the night before and a light breakfast (125 kcal) approximately 15 min before scanning. During the scan, participants were shown pictures of low- and high-calorie foods, which they later rated as appetizing or not. TSD resulted in greater activation in the right anterior cingulate cortex in response to food images relative to sleep. Furthermore, participants rated more high-calorie food images as appetizing after a night of TSD compared with a night of sleep. The extent of the activation in the anterior cingulate cortex during TSD was significantly correlated with food ratings (appetizing). These data are particularly interesting because dopaminergic input via the mesocorticolimbic pathways is received by the anterior cingulate cortex and projected to the striatum, which is involved in the regulation of hunger motivation.

St-Onge, McReynolds, et al. (2012) also examined neuronal responses to food image stimuli after five nights of either 4- or 9-h TIB in normal weight, healthy men and women. Food intake was strictly controlled over the first 4 days but was ad libitum on the day immediately before the scan. Under conditions of sleep restriction, food stimuli led to increased activation of the putamen (thalamus), pulvinar (lentiform nucleus), orbitofrontal cortex, cingulate gyrus, precuneus, and inferior parietal lobule. When participants spent 9 h TIB, food images significantly activated the inferior parietal lobule, middle frontal gyrus, and hypothalamus. Finally, when sleep states were compared, food stimuli increased activation in the putamen, nucleus accumbens, thalamus, insula, orbitofrontal cortex, precentral gyrus, lentiform nucleus, precuneus, cuneus, and supramarginal gyrus to a greater extent in the sleep restriction phase relative to habitual sleep. Those regions are generally known for their association with emotional responses to stimuli and motivation and reward systems. Moreover, the authors observed that the neuronal responses to food stimuli in the restricted sleep state were similar to those observed in participants after a period of negative energy balance and weight loss (Rosenbaum, Kissileff, Mayer, Hirsch, & Leibel, 2010), and they proposed that restricting sleep may send neuronal signals analogous to energy deprivation, which would then prompt corrective actions to seek and obtain food. This would support the behavioral data obtained in that

study showing that participants eat approximately 300 kcal more in the sleep-restricted state than the sleep-replete state (St-Onge et al., 2011).

In the study by St-Onge, McReynolds, et al. (2012), foods were categorized as healthy or unhealthy. In an exploratory analysis of the data separated by food category, they further noted that unhealthy food stimuli specifically activated areas of the middle and superior frontal gyrus, right inferior frontal gyrus, left inferior parietal lobule, postcentral gyrus, and insula after sleep restriction whereas the inferior parietal lobule and medial temporal gyrus were activated after a period of habitual sleep (St-Onge, Wolfe, Sy, Shechter, & Hirsch, 2014). Moreover, relative to restricted sleep, habitual sleep selectively activated regions of the right thalamus, left precuneus, and middle cingulate gyrus in response to unhealthy relative to healthy foods. It was concluded that the activation of cognitive control mechanisms, when faced with appealing food stimuli, is not as well recruited after a period of restricted sleep, which may explain the apparent lack of restraint, or control, leading to increased intakes of snacks and higher fat foods in these conditions.

It is of interest that the inferior parietal lobule is activated by food stimuli in general and by unhealthy foods to a greater extent than healthy foods under periods of restricted and habitual sleep (St-Onge et al., 2014). De Havas, Parimal, Soon, and Chee (2012) have found that TSD reduces connectivity in the inferior parietal lobule of the default mode network, a network of brain regions that deactivates in response to externally driven tasks and activates in response to internally driven cognition tasks. They report the inferior parietal lobule as being involved in cognitive operations related to bodily awareness. This reduction in default-mode network inferior parietal lobule connectivity is observed after TSD. Our studies (St-Onge, McReynolds, et al., 2012; St-Onge et al., 2014) and the study of Benedict et al. (2012) were performed after acute severe sleep restriction. Perhaps either longer sustained periods of sleep restriction beyond five nights or TSD are needed to fully compromise this cognitive network.

The acute sleep restriction studies of Benedict et al. (2012), St-Onge, McReynolds, et al. (2012), and St-Onge et al. (2014) illustrate different neuronal networks that are involved in the response to food stimuli that implicate greater reward valuation after sleep restriction compared with habitual sleep as an explanation for increased food intake in this condition. Killgore et al. (2013) further examined whether self-perceived sleepiness, despite adequate, or normal, self-reported sleep duration, may be related to neuronal responses to food stimuli. Participants underwent fMRI scanning while viewing pictures of high- and low-calorie foods and rated the image on the basis of desire to eat ("How much would you like to eat this right now?"). Sleepiness scores on the Epworth Sleepiness Scale were positively correlated with appetite ratings, and this association tended to be stronger in women than in men. Furthermore, a single cluster in the ventral medial prefrontal cortex, an area important for evaluating the reward value of objects, regulating emotional responses, and controlling behavior, was also negatively correlated with sleepiness. Finally, in women, but not men, there was a negative correlation between the activation of the ventral medial prefrontal cortex and self-reported overeating ("Do you feel you eat more than you intend to?"). The authors concluded that daytime sleepiness was associated with a reduced activation of the ventral medial prefrontal cortex in response to high-calorie foods and that this was predictive of difficulty curtailing food intake, particularly in women. These results further support the notion that sex differences exist in food intake control responses to sleep restriction.

SLEEP RESTRICTION AND FOOD CHOICE

It is of interest to note that poor sleep quality is also associated with decision-making complacency and lower decision-making self-esteem in adolescents. Telzer, Fuligni, Lieberman, and Galvan (2013) found that hyperactivity of the insula occurred when processing positive stimuli in teens and that this was correlated with greater risk-taking likelihood, more decision-making complacency, and decision-making panic. They also found that reduced functional coupling of the insula and dorsolateral prefrontal cortex was associated with decision-making complacency and low decision-making self-esteem and vigilance, and that reduced functional coupling of the ventral striatum and dorsolateral prefrontal cortex was associated with increased likelihood of engaging in risk-taking behaviors and decision-making complacency. Therefore, if similar effects of poor sleep on decision-making and risk-taking behaviors are observed with SSD, one might expect that individuals with poor sleep quality or SSD would also make poor decisions with respect to their food choices.

Hogenkamp et al. (2013) explored the effects of sleep deprivation on a computer-based task to self-select portion sizes for a meal relative to a night of 8-h TIB. Young, normal-weight, healthy men underwent one night of 8-h TIB followed by 1 day of a fixed meal and food intake diet before being randomly allocated to the TSD or 8-h TIB night. The portion size task was performed at the same time the next morning and was followed by a controlled, 650-kcal breakfast and a second portion-size task. During the task, participants were shown pictures of seven different meal foods and six different snack foods, each presented in 51 different portion sizes ranging from 83 to 750 kcal. Self-reported hunger was greater after the night of TSD relative to the 8-h TIB night. Overall, portion sizes chosen on the computer task were larger after TSD than sleep and in the fasted versus the fed state. In the fasted state, food type did not affect portion size choice between TSD and sleep. However, after breakfast,

larger portions of snack foods, but not meal items, were chosen after TSD relative to sleep. The authors concluded that two independent mechanisms may be involved in the effect of sleep on feeding behavior: homeostatic and hedonic. However, food intake was not measured in this study, and it is unknown whether participants would have actually consumed what they reported they would in the portion task. Nevertheless, based on data from studies of food intake, one would expect that the results obtained by Hogenkamp et al. (2013) would be reflective of actual consumption patterns. One study has been conducted to investigate the association between the results of this task and actual and concluded that screen-based measures of portion-size selections were a valid method to assess energy intake in humans (Wilkinson et al., 2012).

Another study by the same group examined economic decision-making specific to food purchases after one night of TSD or one night of 8-h TIB (Chapman et al., 2013). Young, normal-weight healthy men underwent one night of 8-h sleep followed by a day of controlled food intake before undergoing either TSD or 8-h TIB in a crossover design. The next morning, participants ate a fixed 650-kcal breakfast at 8:00 a.m. and performed a mock supermarket task immediately after. For this task, participants were given approximately $50 (USD) to purchase from an array of 20 high-calorie and 20 low-calorie foods. Participants were aware of each food's price, energy density, and weight. In two subsequent trials, prices were manipulated such that high-calorie foods were either 25% cheaper or 25% more expensive than in the first trial. Participants were asked to spend as much of the money as possible and told that they were not permitted to make money savings. After the night of TSD, participants bought 9% more calories and 18% more grams of food than after the night of 8-h TIB. Making changes to the price of high-calorie foods did not alter the effect of TSD on purchasing behaviors. These data suggest that food purchasing may represent another mechanism through which a lack of sleep could promote food intake and put individuals at increased risk of large weight gain over time.

CONCLUSIONS

Studies to date have established that restricting sleep duration leads to alterations in food choices resulting in increased intakes of snacks and high-fat foods. Evidence suggests that SSD leads to changes in the hormonal regulation of appetite (although the exact mechanism remains to determined) and in the neuronal control of feeding behavior. It seems that, at least in individuals who do not regularly have SSD, restricting bedtimes leads to increased appetite and poor food choices. Leptin, ghrelin, and glucagon-like peptide-1 have been proposed to act at the hormonal level whereas the brain reward centers could be involved at the neuronal level. These imply that homeostatic and hedonistic controls of food intake could be affected by SSD. Future studies are needed to examine the contributions of each pathway to increased obesity and the effect of sex on this relationship.

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REFERENCES

Benedict, C., Brooks, S. J., O'Daly, O. G., Almen, M. S., Morell, A., Aberg, K., et al. (2012). Acute sleep deprivation enhances the Brain's response to hedonic food stimuli: an fMRI study. *Journal of Clinical Endocrinology and Metabolism*, *97*(3), E443–E447.

Benedict, C., Hallschmid, M., Lassen, A., Mahnke, C., Schultes, B., Birgir Schioth, H., et al. (2011). Acute sleep deprivation reduces energy expenditure in healthy men. *American Journal of Clinical Nutrition*, *93*, 1229–1236.

Bosy-Westphal, A., Hinrichs, S., Jauch-Chara, K., Hitze, B., Later, W., Wilms, B., et al. (2008). Influence of partial sleep deprivation on energy balance and insulin sensitivity in healthy women. *Obesity Facts*, *1*(5), 266–273.

Brondel, L., Romer, M. A., Nougues, P. M., Touyarou, P., & Davenne, D. (2010). Acute partial sleep deprivation increases food intake in healthy men. *American Journal of Clinical Nutrition*, *91*(6), 1550–1559.

Chapman, C. D., Nilsson, E. K., Nilsson, V. C., Cedernaes, J., Rangtell, F. H., Vogel, H., et al. (2013). Acute sleep deprivation increases food purchasing in men. *Obesity (Silver Spring)*, *21*(12), E555–E560.

De Havas, J. A., Parimal, S., Soon, C. S., & Chee, M. W. (2012). Sleep deprivation reduces default mode network connectivity and anti-correlation during rest and task performance. *Neuroimage*, *59*(2), 1745–1751.

Drapeau, V., Blundell, J., Therrien, F., Lawton, C., Richard, D., & Tremblay, A. (2005). Appetite sensations as a marker of overall intake. *British Journal of Nutrition*, *93*(2), 273–280.

Dzaja, A., Dalal, M. A., Himmerich, H., Uhr, M., Pollmacher, T., & Schuld, A. (2004). Sleep enhances nocturnal plasma ghrelin levels in healthy subjects. *American Journal of Physiology Endocrinology and Metabolism*, *286*(6), E963–E967.

Griffioen-Roose, S., Finlayson, G., Mars, M., Blundell, J. E., & de Graaf, C. (2010). Measuring food reward and the transfer effect of sensory specific satiety. *Appetite*, *55*(3), 648–655.

Hanlon, E. C., & Van Cauter, E. (2011). Quantification of sleep behavior and of its impact on the cross-talk between the brain and peripheral metabolism. *Proceedings of the National Academy of Sciences of the United States of America*, *108*(Suppl 3), 15609–15616.

Hogenkamp, P. S., Nilsson, E., Nilsson, V. C., Chapman, C. D., Vogel, H., Lundberg, L. S., et al. (2013). Acute sleep deprivation increases portion size and affects food choice in young men. *Psychoneuroendocrinology*, *38*(9), 1668–1674.

Killgore, W. D., Schwab, Z. J., Weber, M., Kipman, M., Deldonno, S. R., Weiner, M. R., et al. (2013). Daytime sleepiness affects prefrontal regulation of food intake. *Neuroimage*, *71*, 216–223.

Klingenberg, L., Chaput, J. P., Holmback, U., Jennum, P., Astrup, A., & Sjodin, A. (2012). Sleep restriction is not associated with a positive energy balance in adolescent boys. *American Journal of Clinical Nutrition*, *96*(2), 240–248.

Markwald, R. R., Melanson, E. L., Smith, M. R., Higgins, J., Perreault, L., Eckel, R. H., et al. (2013). Impact of insufficient sleep on total daily energy expenditure, food intake, and weight gain. *Proceedings of the National Academy of Sciences of the United States of America*, *110*(14), 5695–5700.

Nedeltcheva, A. V., Kilkus, J. M., Imperial, J., Kasza, K., Schoeller, D. A., & Penev, P. D. (2009). Sleep curtailment is accompanied by increased intake of calories from snacks. *American Journal of Clinical Nutrition*, *89*(1), 126–133.

Omisade, A., Buxton, O. M., & Rusak, B. (2010). Impact of acute sleep restriction on cortisol and leptin levels in young women. *Physiology and Behavior*, *99*(5), 651–656.

Parker, B. A., Sturm, K., MacIntosh, C. G., Feinle, C., Horowitz, M., & Chapman, I. M. (2004). Relation between food intake and visual analogue scale ratings of appetite and other sensations in healthy older and young subjects. *European Journal of Clinical Nutrition*, *58*(2), 212–218.

Patel, S. R., & Hu, F. B. (2008). Short sleep duration and weight gain: a systematic review. *Obesity (Silver Spring)*, *16*(3), 643–653.

Pejovic, S., Vgontzas, A. N., Basta, M., Tsaoussoglou, M., Zoumakis, E., Vgontzas, A., et al. (2010). Leptin and hunger levels in young healthy adults after one night of sleep loss. *Journal of Sleep Research*, *19*(4), 552–558.

Penev, P. D. (2007). Sleep deprivation and energy metabolism: to sleep, perchance to eat? *Current Opinion in Endocrinology, Diabetes and Obesity*, *14*(5), 374–381.

Penev, P. D. (2012). Update on energy homeostasis and insufficient sleep. *Journal of Clinical Endocrinology and Metabolism*, *97*(6), 1792–1801.

Rosenbaum, M., Kissileff, H. R., Mayer, L. E., Hirsch, J., & Leibel, R. L. (2010). Energy intake in weight-reduced humans. *Brain Research*, *1350*, 95–102.

Schmid, S. M., Hallschmid, M., Jauch-Chara, K., Wilms, B., Benedict, C., Lehnert, H., et al. (2009). Short-term sleep loss decreases physical activity under free-living conditions but does not increase food intake under time-deprived laboratory conditions in healthy men. *American Journal of Clinical Nutrition*, *90*(6), 1476–1482.

Shechter, A., Rising, R., Albu, J. B., & St-Onge, M. P. (2013). Experimental sleep curtailment causes wake-dependent increases in 24-h energy expenditure as measured by whole-room indirect calorimetry. *American Journal of Clinical Nutrition*, *98*(6), 1433–1439.

Simpson, N. S., Banks, S., & Dinges, D. F. (2010). Sleep restriction is associated with increased morning plasma leptin concentrations, especially in women. *Biological Research for Nursing*, *12*(1), 47–53.

Spaeth, A. M., Dinges, D. F., & Goel, N. (2013). Effects of experimental sleep restriction on weight gain, caloric intake and meal timing in healthy adults. *Sleep*, *36*(7), 981–990.

Spiegel, K., Tasali, E., Penev, P., & Van Cauter, E. (2004). Brief communication: sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. *Annals of Internal Medicine*, *141*(11), 846–850.

St-Onge, M. P. (2013). The role of sleep duration in the regulation of energy balance: effects on energy intakes and expenditure. *Journal of Clinical Sleep Medicine*, *9*(1), 73–80.

St-Onge, M. P., McReynolds, A., Trivedi, Z. B., Roberts, A. L., Sy, M., & Hirsch, J. (2012). Sleep restriction leads to increased activation of brain regions sensitive to food stimuli. *American Journal of Clinical Nutrition*, *95*(4), 818–824.

St-Onge, M. P., O'Keeffe, M., Roberts, A. L., RoyChoudhury, A., & Laferrere, B. (2012). Short sleep duration, glucose dysregulation and hormonal regulation of appetite in men and women. *Sleep*, *35*(11), 1503–1510.

St-Onge, M. P., Roberts, A. L., Chen, J., Kelleman, M., O'Keeffe, M., RoyChoudhury, A., et al. (2011). Short sleep duration increases energy intakes but does not change energy expenditure in normal-weight individuals. *American Journal of Clinical Nutrition*, *94*(2), 410–416.

St-Onge, M. P., Wolfe, S., Sy, M., Shechter, A., & Hirsch, J. (2014). Sleep restriction increases the neuronal response to unhealthy food in normal-weight individuals. *International Journal of Obesity (London)*, *38*(3), 411–416.

Taheri, S., Lin, L., Austin, D., Young, T., & Mignot, E. (2004). Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Medicine*, *1*(3), e62.

Telzer, E. H., Fuligni, A. J., Lieberman, M. D., & Galvan, A. (2013). The effects of poor quality sleep on brain function and risk taking in adolescence. *Neuroimage*, *71*, 275–283.

Wilkinson, L. L., Hinton, E. C., Fay, S. H., Ferriday, D., Rogers, P. J., & Brunstrom, J. M. (2012). Computer-based assessments of expected satiety predict behavioural measures of portion-size selection and food intake. *Appetite*, *59*(3), 933–938.