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The Mechanisms of Weight Gain in Sleep-Deprived Individuals

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Abstract
The obese population in America has grown during the last century. During these years as well, American's have been sleeping less. Cross sectional studies show that there is a correlation of the two factors, and indeed find a greater number of overweight individuals amongst the sleep-deprived population. Though they are unclear, studies attempt to establish possible mechanisms through which weight gain occurs. Results of studies show that sleep deprivation may influence leptin and ghrelin levels, which can cause hunger, and excessive caloric intake. Sleep-deprived individuals also have an increased opportunity to eat during the wakeful nighttime hours. In the sleep-restricted state, activity levels may decrease, and though the extra hours spent awake also cause a small increase in energy expenditure, there is over-compensation in energy intake, leading to weight gain. Obstructive Sleep Apnea also plays a role in obese individuals' sleep patterns, and may lead to further weight gain. Without excessive caloric intake it may be possible to maintain weight in the sleep-deprived state. This paper will review a number of studies and give an overview of some of the possible methods though which sleep-deprivation causes obesity.

Introduction
Sleep was once thought of as a process during which the both the body and brain are at rest and inactive. But in modern times, starting in approximately 1950, sleep has been studied and discovered to be an active process, necessary to maintain many of the body's functions (National Institute of Neurological Disorders and Stroke, NINDS, 2014).

In 1937, Alfred Loomis, along with E. Newton Harvey, and Garret Hobart, used the EEG to map out what they considered five stages of sleep, A-E. In 1953, Eugene Aserinsky and Nethaniel Kleitman established the REM sleep stage. In 1957 Dement and Kleitman mapped out the sleep stages as they are known today, dividing sleep into NREM (non-rapid eye movement) and REM (rapid eye movement) sleep. NREM sleep consists of four stages, each with varying brain wave activity, and increasing in depth as the stage number increases. These stages are cyclical, repeating throughout the night. Following the four stages, there is a period of REM sleep (Shepard et al., 2005). REM sleep is associated with rapid eye movements, increased heart rate, and dreaming (NINDS, 2014).

With vast research and many studies conducted since, the understanding of the roles that sleep plays in the body has been greatly advanced and its necessity has been studied extensively (Shepard et al., 2005).

Sleep is both a circadian rhythm and a homeostatic cycle. A circadian rhythm is natural rhythm, approximately twenty-four hours long, and controlled primarily by the suprachiasmatic nucleus. The natural timing of the cycle is based on the light-dark cycle (NIGMS, 2012). Though they are naturally occurring, circadian rhythms can be altered and affected by environmental factors. One of the circadian rhythms is the body's drive for alertness and wakefulness, which gets higher throughout the daytime hours and decreases during the night. The circadian sleep-wake cycle, along with the body's homeostatic drive for sleep, which increases based on the amount of prior wakeful hours, keep the body in a proper rhythm, maintaining the appropriate amount of necessary sleep (Dijk and Lockley, 2002).

The necessary amount of sleep in humans varies with age, ranging from 16–18 hours in infancy to 7-8 hours in the average adult (NINDS, 2014). In the last century, America has seen an approximate 20% decrease in the amount of sleep it gets. This has had economic, social, and health consequences. Sleep deficiency has been associated with numerous health risks such as hypertension, stroke, obesity, and diabetes. Sleep is necessary to support many vital systems in the body, and adequate sleep is necessary for optimal cognitive performance, without which many accidents may occur (Institute of Medicine (US) Committee on Sleep Medicine and Research, 2006).

Parallel to the decrease in sleep, the incidence of obesity in America has drastically increased. Studies conducted by the National Health and Nutrition Examination Survey (NHANES) show that more than one third or 34.9% of adults over age 20 are considered obese. Additionally, of American children and adolescents ages 2–19, approximately 16.9% are obese (Ogden et al., 2014). Obesity is a result of excessive weight gain, which occurs primarily when the energy expenditure of the body is less than energy intake, resulting in a net gain. Over time, if the gain continues, the weight accumulates and results in obesity. Other factors that may cause obesity are environmental factors, genetics, illnesses, and medications (CDC 2011).

The correlation between sleep deficiency and weight gain has been examined and many cross sectional and epidemiological studies found a relationship of sleep deprivation with obesity. Studies show an association of a higher body mass index (BMI) in individuals who generally sleep fewer hours than the recommended sleep time. Over all, the studies point at a relationship

of sleep deprivation to obesity primarily in the pediatric and young adult population (Beccuti and Pannain, 2011). Though these results were most evident in the young and middle-aged population, one study found that there is a greater likelihood (3.7 times in men and 2.3 times in women) for obesity in older individuals who sleep less than 5 hours per night (Patel et al., 2008). Though the data is evident of a correlation between the two, there are many factors that may contribute to the obesity and the findings are not entirely conclusive (Patel and Hu, 2008). This paper will investigate the mechanisms involved in sleep deprivation that result in weight gain and obesity?

Methods

Information was obtained online with access to many online publications through the Touro College library. Key words included sleep-deprivation, obesity, weight gain, leptin, ghrelin, energy expenditure, OSA. Additionally, references made by literature reviews were helpful in conducting research.

Discussion

Many studies have been performed in an effort to establish the relationship of decreasing sleep with increasing likelihood of obesity. The studies examine whether the shortened sleep times have an effect on weight, and via which mechanisms this may occur. Three commonly discussed factors that may contribute to the weight gain in sleep-deprived individuals are energy intake (consumption of food), energy expenditure, and hormonal leptin and ghrelin levels.

Leptin and ghrelin

Leptin and ghrelin are two essential hormones in the maintenance of the body's energy balance. These two hormones have opposite effects on the body. Leptin induces a satiety and fullness, and ghrelin, hunger and appetite. Leptin is primarily released by adipose tissue into the bloodstream and send the brain signals regarding the energy state of the body. Leptin signals receptors at the hypothalamus, which affect hypothalamic neurons and various neuropeptides. Leptin is known to have anorexic (appetite suppressing) effects. Ghrelin, is released primarily by the stomach and signals the hypothalamus, which thereafter has orexic (appetite inducing) effects (Klok et al., 2007). Because of the correlation found between sleep and weight gain, it is presumed that perhaps leptin and ghrelin, which are closely related to food intake and regulation, are affected by sleep restriction. Studies examine this detail, and show inconsistent results.

One study that examined the sleep-weight gain relationship was performed on 12 average men, 22 +/- 2 years of age, and with average BMIs 23.6+/- 2 kg/m2. The participants did not smoke or take any medication. They all regularly slept 7 to 9 hours, and they had not crossed time zones in the last four weeks before the study. The study consisted of two randomized periods, six weeks apart. Weight did not change during the time between the two studies. For the first night of study, participants were given a restricted diet, while during the day, they ate their regular diets. During the second day of study, they received a glucose infusion of 5 g/kg/ 24 hours. After both periods of the study- two nights of 10 hours in bed, and two nights of 4 hours in bed, blood samples were taken at 20-minute intervals, from 8 am to 9 pm. Results showed that in the sleep deprived state, leptin levels were 18% lower and ghrelin levels 28% higher than in the normal sleep state (Spiegel et al., 2004). These results accounted for the elevated hunger levels in the sleep-deprived state. Consequently, increased hunger usually causes increased food intake. However, these studies did not measure exact food intake of the participants, so there is no clear evidence of the increased energy intake.

Another study performed on 1,024 volunteers in the Wisconsin Sleep Cohort Study found a decrease in leptin levels with sleep deprivation. They underwent polysomnography and answered questionnaires to measure sleep. In the sleep-deprived state, (5 hours versus 8) a morning blood test showed a 15.5% decrease in leptin and 14.9% higher ghrelin levels (Taheri et al., 2004). Consistent with the findings by Spiegel et al. these results can likely cause increase in hunger and food intake, leading to weight gain.

Though these results show a clear effect of sleep on leptin and ghrelin, not all studies yielded such results. Numerous reasons may be responsible for the discrepancy in the results. One reason might be that in the study by Spiegel et al., the blood samples were taken after participants underwent constant glucose infusion, which could perhaps yield results that differ from those of a regular food diet.

Another study was in search of a relationship of partial sleep deprivation to weight gain and insulin sensitivity in women. It was performed on 14 healthy women, age 23-38, who were weight stable, with no recent illness, non- smoking, non-lactating, and not on any medication besides oral contraceptives. They were all in the follicular phase of the menstrual cycle, slept 7.5 to 9 hours on an average night, did not cross time zones in last month, and had no sleep complaints. Participants were studied after 2 nights of 8 hours of sleep, after 4 nights of consistent decrease in sleep (minus one additional hour nightly) and then 2 nights of recovery sleep. Food was not restricted, though alcohol was. Their diet was carefully recorded using computer software. After performing a glucose tolerance test, blood was collected in 15-minute intervals for 90 minutes. Leptin levels showed a 24% increase after 2 nights of approximately 5.5 hours in bed versus 9 hours in bed. Ghrelin levels were stable. This may be due to the extra caloric intake during those days of study, as will be discussed below. Insulin

sensitivity did not appear to be affected by sleep restriction (Bosy-Westphal et al., 2008).

Numerous other studies also yielded results that were inconsistent with each other. Certain studies show increase in leptin, others show a decrease, while others do not show any significant change.

One study was performed on eleven people, men and women, with an average BMI of 26.5, for two two-week periods in random order. In the restricted sleep they slept 5.5 hours, compared to 8.5 hours in the habitual sleep. They had an unrestricted diet throughout the study. The results showed that in the sleep deprived state, leptin and ghrelin levels were not affected significantly. These results show that perhaps in response to sleep loss, the body may have an altered response to restricted food, and the hormonal levels remain more stable than in the body's usual state. This would account for the stable leptin and ghrelin levels across the two periods of study (Nedeltcheva et al., 2009).

One other study observed that in nine average weight men after one night of normal sleep, one of partial sleep restriction and one total sleep deprivation, leptin levels did not show a significant change, while ghrelin levels were 22% higher in total sleep deprivation than in normal sleep (Schmid et al., 2008).

Clearly, many studies yield conflicting results on the effect of sleep on leptin and ghrelin. Two studies we discussed show entirely opposite results (Spiegel et al. 2004 and Bosy-Westphal et al., 2008). Why are the observations to the various studies inconsistent?

Perhaps it is the fact that certain factors were sometimes unaccounted for in these particular studies, such as genetics, individual's environment, race, and height (Spiegel et al., 2004). Additionally not all of the studies measured and accounted for precise weight of the participants, which is perhaps necessary to determine the effect of one factor on another. Additionally, as mentioned above, the diets of the participants in the studies were not consistent with each other, which may have had an effect on the body's endocrine response.

The inconsistent results of the many studies suggest that the leptin and ghrelin levels may or may not change and contribute to weight gain in response to sleep restriction. However, even studies that do not show a direct change in leptin and ghrelin, do show greater caloric intake, likely due to hunger and increased opportunity to eat.

The effect that sleep has on energy expenditure must also be taken into account. Intuitively, in the sleep-deprived state, because of fatigue, energy expenditure should decrease. However, studies show that a sleep deprived individual experiences extra wakeful hours during which he/she is burning more calories than if he/she had been sleeping. However, because during those wakeful hours the individual is likely fatigued, activity levels are lower than regular wakeful hours. Therefore, the overall energy expenditure in the sleep-deprived state may increase only slightly.

Energy intake versus expenditure

To maintain weight one must be in energy balance, when energy consumed is equal to energy expended. Weight gain occurs when the body is in positive energy balance, when the person takes in more calories than they burn. Perhaps even without an observed hormonal change, the body reacts to sleep deprivation with increased hunger, and thus increased caloric intake. Sleep may also have an effect on metabolism and energy expenditure? Many of the studies mentioned above, searched for the effect of sleep deficiency on hunger levels, energy intake, and energy expenditure, in addition to its role in leptin and ghrelin levels.

According to Spiegel et al. participants recorded their hunger levels after both periods of the study- restricted and normal sleep times- by answering how hungry they felt at every hour from 9 am to 9 pm, after the second night of sleep. They were also asked to describe their appetite, answering how much they would like to eat 7 different foods (sweet, salty, starchy, fruits, vegetables, meat, dairy etc.) Results showed that consistent with the decreased leptin and elevated ghrelin levels found in this study, 24% higher hunger levels were found, as well as 23% higher appetite levels, in the sleep deprived state. The appetite increase was highest for calorie and carbohydrate dense foods. Increased appetite for protein rich foods was not significantly increased in sleep-deprived state (Spiegel et al., 2004). The study by Schmid et al. also showed an increase in average hunger ratings of 3.9 after total sleep deprivation as opposed to 1.7 after a 7-hour time in bed, consistent with increased ghrelin (Schmid et al., 2008).

Presumably, the increase in hunger accounts for greater caloric intake in sleep-deprived individuals, causing weight gain. However, in the studies by Spiegel et al. (2004) and Schmid et al. (2008), exact caloric intake during the study periods was not measured, and thus we cannot necessarily assume that there was extra consumption of calories or how many. Additionally, during the study periods, activity levels were kept low (reclining, sitting etc.), and they did not examine whether or not there is a change in energy expenditure or energy balance. Without this information we cannot determine if there would be a theoretical net weight gain. To examine these

details, further study on energy expenditure is needed.

Energy expenditure can be measured using indirect calorimetry, metabolic chambers, heart rate and other methods. Indirect calorimetry and metabolic chambers measure the level of carbon dioxide production to assess energy expenditure.

In the study by Bosy-Westphal et al. careful measurement of caloric intake was measured during the entire study via computer. Resting energy expenditure was measured using indirect calorimetry, and total energy expenditure was measured using 24-hour heart rate monitoring. Physical activity was also measured with pedometers. Energy intake significantly increased from baseline to sleep deprived state, while energy expenditure did not show a significant change. The average increase in calories was 415 +/- 471 kcal/day, and caused a net weight gain (Bosy-Westphal et al., 2008). Because there was no change in energy expenditure seen, we can assume that in prolonged sleep deprivation the individuals would experience significant weight gain. This provides further evidence that sleep deprivation causes an increase in caloric intake and body weight, despite the fact that there was no significant endocrine response to sleep deprivation.

However, the study did observe a change in thyroid hormone levels, so further research is required to examine more specific results of the effect sleep restriction may have on energy expenditure, such as measuring total energy expenditure using a caloric chamber (Bosy-Westphal et al., 2008).

This leads us to another study that did indeed measure specific total energy expenditure, using a caloric chamber. The study shows a significant increase in energy expenditure in the sleep-deprived state due to the extra hours of time spent awake. Ten women, with average BMIs were studied under shortened sleep and habitual sleep, 4 and 8 hours, 3 days of each, while kept on a strictly controlled diet. The results showed significantly greater energy expenditure of approximately 92 kcal in the shortened sleep period. This study provides evidence that there may be an increase in energy expenditure in the sleep deprived state, however, there is an overcompensation of energy intake, which leads to weight gain (Shechter et al., 2013).

Another study that measured energy intake versus expenditure found that with unlimited food allowance, the participants did not consume a significant amount of extra calories during meals, however, they did increase their intake of snack calories between the hours of 7pm-7am, with more carbohydrates and less protein/fat snacks (Nedeltcheva et al., 2009). The fact that the increase was only significant between 7pm-7am, not 7am-7pm, highlights the fact that during the nighttime hours when sleep is restricted participants found time to consume significantly more snacks, leading to an increase in daily calories.

When one is sleep deprived and awake during nighttime hours, not only is there elevated hunger during the day, but there is also increased opportunity to eat during the extra hours spent awake. More so, the nature of nighttime eating is snacking, which can cause unintentional excessive caloric intake.

The total energy intake was 297 kcal more in sleep-deprived state. Additionally, during both sleep deprived and habitual sleep state, there was a surplus of calories, due to the participants' eating habits. Some individuals had a greater 'propensity' to eat, while others not .The study also found a slight change in energy expenditure between the two sleep states. The difference, which they considered insignificant, was 136 +/- 437 kcal more per day in the restricted sleep period (Nedeltcheva et al., 2009). Even if the change in total energy expenditure was considered significant, there is a surplus of an average 160 kcal per day in the sleep-deprived state. If we were to apply this information to a general population one can assume that the overcompensation in calories would cause weight gain in individuals who are consistently sleep deprived.

Perhaps if participants were more active, the studies would find a significant increase in energy expenditure, and there would no longer be the overcompensation of calories. However, because of fatigue and sleepiness, activity levels should presumably be lower when one is sleep-deprived. Studies show that the activity levels of sleep-deprived individual are indeed lower.

Another study involved 30 men and women, ages 30-49, and with BMIs between 22-26. No shift workers, or individuals that had travelled across time zones within 4 weeks of the study were allowed. Smokers, diabetics, those with excessive caffeine intake, or neurologic issues were also disqualified. The study was divided into two stages of 6 days each. In the shortened and normal sleep stages, the participants were given 4 hours in bed and 9 hours in bed, respectively, with no naps during the day. They measured sleep by polysomnography. For the first 4 days of each period, their food intake was allotted and strictly measured. The last two days were also carefully measured, but participants were allowed to make their own food choices. On day 4 they were asked how they would rate their hunger, how full they felt, how energetic, how much they could eat, and how much they would like to eat

foods that were sweet, salty, savory etc. After measuring the energy intake and expenditure, results showed that women consumed 15.3% more in the restricted sleep period, with a 39% increase in fats. Men consumed 9.2% more. Additionally they ate more often in the sleep-restricted stage. While they consumed more, no significant change in energy expenditure was found, though the point of peak activity observed was higher in the normal sleep period. In the normal sleep period, participants spent a greater percentage in sedentary activity, a lesser percentage in light activity, and a significantly greater percentage of time doing heavy activity, than in the restricted sleep period. Overall the study showed greater feelings of sluggishness and less energy during the shortened sleep period and there was no significant change in total energy expenditure over the course of the study (St.-Onge et al., 2011).

This is intuitive, as a sleep-deprived individual is tired and consequently has less energy for intense activity. Low intensity activity leads to less energy expenditure, and opportunity for more weight gain.

Surprisingly, there was no net change in energy balance observed, after controlling for phase order and gender. However, like Nedeltcheva et al., this study showed an increase of approximately 300 kcal per day in the shortened sleep period. Due to the fact that they are primarily due to the increase in fat and saturated fat calories, the people would also be at risk for cardiovascular disease (St-Onge et al., 2011).

Even though, unlike previous studies, this study did not show a significant effect on participants hunger ratings, there was an observed increase in energy intake. This suggests that the relationship of sleep deprivation and obesity is one primarily based on excessive food intake. The continuous surplus of calories in the sleep-deprived state may eventually lead to obesity, if not coupled with an increase in energy expenditure (St-Onge et al., 2011).

Obstructive Sleep Apnea

There is reason to believe that perhaps the relationship of poor sleep and obesity is a reverse process. The cross sectional data showing a relationship between obesity and sleep deprivation may be due to the fact that as a result of being overweight, obese individuals experience shortened sleep, with poorer quality. This is indeed true in the case of Obstructive Sleep Apnea (OSA). Obese people are twice as likely to experience Obstructive Sleep Apnea, a significantly higher risk, than average weight individuals. OSA occurs in approximately 45% of obese individuals. Additionally, their risk for OSA is increased six times, if they gain even 10% of their normal (baseline) weight. On the other hand, the OSA can show an improvement with weight loss of the same amount. One reason that obesity increases the risk for OSA is that fat situated in the upper airways can cause an increase in the collapsibility of the airway, and increase the risk for apnea. A patient with sleep apnea has interrupted sleep and decreased sleep quality. This leads to sleep deprivation, and the sleep deprivation leads to additional weight gain. The process is cyclical. An obese individual is at risk for OSA. OSA causes sleep deprivation and an increased risk for weight gain. The weight gain causes further worsening of OSA symptoms. Studies propose that weight loss can effectively help the obesity, as well as help improve symptoms of OSA (Romero-Corral et al., 2010).

In light of the above, we can say that the relationship between sleep deprivation and obesity is indeed cyclical. However, as our discussion shows, even amongst the average weight population, individuals who are sleep deprived tend to increase energy intake and gain weight.

Further Research

Though studies have established correlation of the above factors, further research would be helpful to provide more conclusive evidence of the mechanisms involved in sleep deprivation that cause obesity. Discrepancies amongst the studies may be due to environmental factors, genetic predisposition, and metabolic differences that vary with each individual participant. Additionally as mentioned above, participants each have varying propensity to eat more, and gain or lose weight, independent of sleeping habits. Furthermore, the studies discussed were performed on relatively small samples of people. Additional studies with a greater number of participants, and in a broader population of ages could perhaps provide more conclusive results. Moreover, perhaps there is a way to study populations in a free-living environment, rather than in a lab setting, and assess under those circumstances, which mechanisms resulting from sleep deprivation lead to the development of obesity.

Conclusion

Obesity is an epidemic that has many economic, social, and health costs. The effects it has on health and healthcare costs are tremendous. Obesity not only requires treatment itself, but it also leads to many health problems such as cardiovascular disease, hypertension, type two diabetes, stroke, osteoarthritis, gallstones, certain types of cancer, fertility problems in women, as well as other health issues (Kopelman, 2007).

Studies show that if the growing obesity epidemic continues, 86.3% of American adults will be overweight or obese, with 51.1% obesity by year 2030. This could lead to health care costs projected at 860.7-956.9 billion dollars (Wang et al., 2008). Obesity also has many social costs, amongst adults and children alike. Obese people may experience limitations on

physical activity, and there are stigmas associated with being heavily overweight that may cause obese people to be treated differently in the workplace and social forums (Seidell, 1998). In light of the above, if sleep deprivation has a role in the growing obese population, not only is intervention necessary to treat obesity and its side effects, but there must also be intervention to counsel people regarding positive sleeping habits. Beginning with school aged children, they must be educated regarding the benefits of getting enough sleep. Parents should try to assist children in creating healthy sleeping habits. Adults should be careful to try to generally maintain a proper sleep schedule, when social and work responsibilities do not demand late bed times. Overall, a greater awareness must be made to the public that sleep deprivation has serious negative side effects.

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