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## ***The effect of sleep duration on changes in body composition during calorie deficit for obese individuals: A literature review***

### **Abstract:**

**Aim:** To investigate the effect of sleep duration on body composition for people with a BMI over twenty-five, and who are in a calorie deficit. Also, to evaluate whether sleep restriction affects the levels of leptin and ghrelin, and whether a change in these hormones is associated with an increased risk of overweight and obesity. **Methods:** A literature search was performed in the databases Oria and PubMed. Eight studies were included, and five of them compared sleep restriction and calorie deficit to a non-exposed group. **Results:** A majority of these studies reported a significant difference in what the weight loss consisted of; the exposed groups lost less fat, and more fat-free mass compared to the non-exposed groups. The studies showed a decrease in leptin concentrations after sleep restriction, but no significant difference in ghrelin concentrations. **Conclusion:** Adequate amounts of sleep are a factor that has a major impact on body composition during a weight reduction phase, and the findings provide evidence that sleep duration plays a key role in the appetite-regulating hormone leptin.

**Hensikt:** Å undersøke effekten av søvnvarighet på kroppssammensetning for personer med en BMI over tjuefem, og som er i et kaloriunderskudd. Det blir også evaluert om søvnrestriksjon påvirker nivåene av leptin og ghrelin, og om en endring i disse hormonene er assosiert med økt risiko for overvekt og fedme. **Metode:** Et litteratursøk ble gjennomført på databasene Oria og PubMed. Åtte studier ble inkludert, og fem av dem sammenlignet søvnrestriksjon og kaloriunderskudd med en ikke-eksponert gruppe. **Resultat:** Flertallet av de inkluderte studiene rapporterte en signifikant forskjell i hva vekttapet besto av; de eksponerte gruppene mister mindre fett, og mer fettfri masse sammenlignet med de ikke-eksponerte gruppene. Studiene viste en nedgang i leptin konsentrasjoner etter søvnrestriksjon, men ingen signifikant forskjell i ghrelin konsentrasjoner. **Konklusjon:** Tilstrekkelig mengder søvn er en faktor som har stor innvirkning på kroppssammensetningen i en vektreduksjonsfase, og funnene gir bevis for at søvnlengden spiller en nøkkelrolle i det appetittregulerende hormonet leptin.

**Key words:** Sleep restriction, Calorie deficit, Body composition, Body mass, Leptin, Ghrelin

## 1 Introduction

Sleep is a vital component for both physical and mental health in human beings (1). Despite the fact that there is convincing evidence indicating the importance of sleep, we sleep less today than we did before (2) and sleeping as little as possible is often regarded as an admirable behavior in today's society. The recommended amount of sleep is 7-9 hours for adults, 8-10 hours for teenagers, and 9-11 hours for school aged kids (1).

A systematic review with data from twenty countries shows that from 1905 to 2008 there has been a decline in the sleep duration among children/adolescents by more than 1 hour per night (2). Data from two representative surveys in the adult population in Norway shows that the prevalence of insomnia has increased from 11.9% to 15.5% in the period 2001-2011, and approximately 13.6% report that they are not satisfied with their sleeping habits (3).

The trend of reduced sleep duration has increased in parallel with an increase in the prevalence of obesity that has nearly tripled since 1975 and has become an epidemic worldwide (4). Despite the fact that obesity can be prevented, almost two billion adults were overweight in 2016, and approximately forty million children under the age of five were overweight or obese in 2020 (4). Overweight and obesity are defined as abnormal or excessive fat accumulation that may impair health. Body mass index (BMI) is a simple measure of weight-for-height that is commonly used to classify overweight and obesity in adults. BMI is defined as *a person's weight in kilograms divided by the square of his/her height in meters (kg/m<sup>2</sup>)* (4). The risk for noncommunicable diseases such as cardiovascular diseases, type 2 diabetes, musculoskeletal disorders, and some types of cancers, increases, with increased BMI (4).

Overweight and obesity are a result of the amount of energy supplied in the form of food being greater than the amount of energy the body utilizes. The excess energy is stored as fat and if this is maintained over a period of time, the body composition becomes deteriorated. Obesity is today the most common nutritional problem in the industrialized world; however, - overweight and obesity are an increasing health problem also in developing countries (5, p. 340). With the increasing prevalence of overweight and obesity, the incidence of diabetes and other obesity-related diseases is also increasing (5, p. 358). Most of the world's population live in countries where overweight and obesity today kills more people than underweight (4).

Previous research indicates that sleep deprivation can modify the amount, composition, and distribution of a person's total food intake, and increased proportion of calories from snacks (6, 7, 8). The changes in total energy intake did not increase the daily energy consumption, and a shorter amount of sleep can therefore be a possible risk factor for overweight and obesity.

Sleep is an incredibly complex condition, and it affects all systems of both body and brain (1, 9). Sleep affects, among other things, the production of leptin and ghrelin, which are hormones that regulate the feeling of hunger and satiety. Leptin is a hormone that acts as a feedback mechanism, sending signals to hypothalamus to inhibit food intake and regulate long-term energy balance. Ghrelin is a more fast-acting hormone that affects meal initiation and short-term regulation of energy balance (10). Decreased leptin levels and increased ghrelin levels have been correlated with increased hunger in individuals exposed to sleep restriction (11).

With a positive energy balance over time, the amount of body fat can become so large that one eventually develops obesity (5, p. 85). In order to be able to regulate body weight and the proportion of body fat, individuals have to be in a calorie deficit consistently and over a longer period of time. Calories are a unit of energy and are defined as *the amount of energy required to raise the temperature of one gram of water one degree Celsius*. A calorie deficit is when individuals consume fewer calories than are consumed through basal metabolic rate (BMR), thermal effect of food and energy consumption related to daily activities and movements (5, p. 75).

The effect of sleep duration on changes in body composition during calorie deficit for obese individuals with an BMI over twenty-five is relevant to investigate because more individuals are suffering from overweight and obesity in today's society than before. It is important to examine the relation between sleep, metabolic- and endocrine factors to determine whether reduced sleep duration leads to metabolic- and endocrine changes. The main aim of this literature study is to investigate the effect of sleep duration on body composition for people with a BMI over twenty-five, and who are in a calorie deficit. A second aim is to investigate the effect of sleep restriction on the levels of leptin and ghrelin-, and whether an altered level in these hormones is associated with an increased risk of overweight and obesity.

## **2 Method**

The literature search was performed using the electronic databases PubMed and Oria. The keywords used were «sleep loss», «body mass», «weight», «sleep restriction», «insufficient sleep», «sleep curtailment» and «sleep duration», and the words had to be in abstract or title. The search provided an initial result of 212 articles on PubMed, and 552 articles on Oria. Based on the inclusion- and exclusion criteria described below, 208 articles were excluded from PubMed, and 548 from Oria.

### **2.1 Inclusion- and exclusion criteria**

The search was restricted to articles after the year 2010, and all articles that were included had to be published in English, peer-reviewed, conducted on humans, and the study design had to be an observational study or a randomized controlled trial. The inclusion criteria for this literature review were studies with a sample size larger than  $n=10$ , and the individuals had to be adults (19-64y) with a BMI over twenty-five. In addition to studying sleep duration, the subjects had to be in a calorie deficit where the goal was weight loss. Studies that included subjects with chronic insomnia or other significant health problems, and studies that included subjects that worked night shift were excluded.

After excluding the mentioned factors, there were eighteen studies left in PubMed, and fifty-nine in Oria. Remaining studies were reviewed by title and abstract and resulted in exclusion of a total of sixty-nine studies. In the end, eight articles were chosen based on the criteria: four articles from PubMed, and four from Oria.



Table 1: Characteristics of the included studies

Study	Study design	Selection	Intervention
Nedeltcheva, A.V. et al (2010) (12)	RCT	n=10(f=3, m=7) BMI=25-32 Age=35-49	14 days of moderate calorie deficit with 8.5 or 5.5 hours of nighttime sleep.
Wang, X. et al. (2018) (13)	RCT	n=36 (f=29, m=7) BMI=25-40 Age=35-55	8-week with calorie deficit with or without sleep restriction (calorie deficit: n=15; calorie deficit + sleep restriction: n=21).
Kline, C.E. et al (2021) (14)	POS	n=125(f=114, m=11) BMI=34.1 ± 4.6 Age=50.3 ± 10.6	12-mo behavioral weight loss intervention, with assessments of sleep, bodyweight and -composition.
Sparks, J.S. et al (2020) (15)	RCT	n=28(f=21, m=7) BMI=25-40 Age=35-55	8-week with calorie deficit with or without sleep restriction (calorie deficit: n=12; calorie deficit + sleep restriction: n=16)
Hart, C.N. et al (2014) (16)	RCT	n=12(f=12) BMI=25.8-38.4 Age=41.7 ± 10.3	two nights of short (5 hours) and two nights of long (9 hours) sleep.
Nedeltcheva, A.V. et al	RCT	n=11(f=5, m=6)	two 14-d stays in a sleep laboratory with ad libitum access to palatable food

(2008) (17)		BMI=26.5 ± 1.5 Age=39 ± 5	and 5.5-h or 8.5-hour sleep.
Chaput, J.P. et al (2012) (18)	POS	n=123 (f=53, m=70) BMI=33,2 ± 3,6 Age=41,1 ± 6,0	15-24 weeks with a calorie deficit. BF, sleep quality, and sleep duration was measured.
Verhoef, S.P.M. et al (2013) (19)	POS	n=98 (f=73, m=25) BMI=28-35 Age=20-50	2-month with a very low energy-diet followed by a 10-mo period of weight maintenance. Bodyweight, -composition, energy intake, PA and sleep were measured before and after the weight loss, and 3- and 10-mo follow-up.

RCT= randomized crossover study, POS= Prospective observational study, BF= body fat mass, PA=physical activity

### **3 Results**

Eight studies studying the effect of sleep restriction and caloric restriction among adults with a BMI equivalent to overweight and obesity (>25) were included in this literature study.

Table 1 shows the background characteristics of the included studies. Among these studies there were two types of study design: randomized crossover study and prospective observational study. Five of these studies compared the effect of sleep restriction with a control group that had an adequate duration of sleep (<8h), and the remaining three studies included a behavioral weight loss intervention with sleep restriction and calorie deficit, see table 2.

Seven of the eight included studies reported measurements on body weight pre- and post-intervention (12, 13, 14, 15, 17, 18, 19). Six studies measured changes in body composition, where all of them reported changes in body fat (12, 13, 14, 17, 18, 19) and four of these included measurements on fat-free mass (12, 13, 14, 17). Five of the eight studies reported levels of leptin (12, 13, 16, 17, 19), and included measurements on leptin concentrations, and four of these also had measurements of ghrelin concentrations (12, 13, 16, 17).

Table 2: Reported changes in body weight, body composition as fat-free mass and total body fat, leptin, and ghrelin at baseline and after the intervention.

Study	Bodyweight, kg: mean (SD)	FFM, kg: mean (SD)	BF, kg: mean (SD)	Leptin, ng/ml: mean (SD)	Ghrelin, pg/ml: mean (SD)
Nedeltcheva, A.V. et al (2010) (12)	<i>Exposed</i> Pre: 80.5 (10.3) Post: $\Delta$ -3.0 (1.0)	<i>Exposed</i> Pre: 55.5 (11.2) Post: $\Delta$ -2.4 (1.4)**	<i>Exposed</i> Pre: 25.0 (6.3) Post: $\Delta$ -0.6 (0.6)*	<i>Exposed</i> Pre: 12.4 (9.5) Post: 9.1 (9.2)	<i>Exposed</i> Pre: 73 (38) Post: 84 (47)**
	<i>Non-exposed</i> Pre: 82.0 (11.2) Post: $\Delta$ -2.9 (1.4)	<i>Non-exposed</i> Pre: 55.6 (11.5) Post: $\Delta$ -1.5 (1.3)	<i>Non-exposed</i> Pre: 26.4 (6.4) Post: $\Delta$ -1.4 (0.9)	<i>Non-exposed</i> Pre: 13.1 (10.2) Post: 9.7 (7.2)	<i>Non-exposed</i> Pre: 13.1 (10.2) Post: 9.7 (7.2)
Wang, X. et al. (2018) (13)	<i>Exposed</i> Pre: 99.0 (10.9) Post: $\Delta$ -3.2 (2.5)	<i>Exposed</i> Pre: 53.3 (7.3) Post: $\Delta$ -0.72 (1.2)	<i>Exposed</i> Pre: 42.7 (9.1) Post: $\Delta$ -1.8 (1.7)	<i>Exposed</i> Pre: 46.3 (34.7) Post: $\Delta$ -12.6(16.4)*	<i>Exposed</i> Pre: 406 (183) Post: $\Delta$ 74 (120)
	<i>Non-exposed</i> Pre: 88.1 (8.8) Post: $\Delta$ -3.3 (3.2)	<i>Non-exposed</i> Pre: 50.7 (7.2) Post: $\Delta$ -0.71(0.93)	<i>Non-exposed</i> Pre: 34.3 (7.9) Post: $\Delta$ -1.9 (2.1)*	<i>Non-exposed</i> Pre: 26.8 (19.2) Post: $\Delta$ -5.6 (17.1)	<i>Non-exposed</i> Pre: 427 (297) Post: $\Delta$ 59 (193)

Kline, C.E. et al (2021) (14)	Pre: 92.0 (15.4) 6-mo: -8.5 (5.8) * 12-mo: 0.4 (3.9)	Pre: 51.2 ( 7.9) 6-mo:Δ-1.7 (1.9) 12-mo: Δ0.4 (2.2)	Pre: 40.7 (10.3) 6-mo:Δ-6.7 (5.1) 12-mo: Δ-0.1 (3.1)	-	-
Sparks, J.S. et al (2020) (15)	<i>Exposed</i> Pre:100.8 (9.3) Post: 98.0 (9.9)  <i>Non-exposed</i> Pre: 89.2 (9.2) Post: 86.4 (8.8)	-	-	-	-
Hart, C.N. et al (2014) (16)	-	-	-	<i>Exposed</i> 36.2 (13.1)  <i>Non-exposed</i> 35.9 (12.3)	<i>Exposed</i> 379.4 (103.0)  <i>Non-exposed</i> 404.7 (140.6)
Nedeltcheva, A.V. et al (2008) (17)	<i>Exposed</i> Pre: 77.3 (10.2) Post:Δ-1.9 (1.6)	<i>Exposed</i> Pre: 48.8 (12.2) Post:Δ0.3 (1.3)	<i>Exposed</i> Pre: 25.6 (5.9) Post:Δ1.7 (0.8)	<i>Exposed</i> Pre: 13.3 (10.3) Post: Δ-2.6 (3.7)	<i>Exposed</i> Pre: 1295 (339) Post: 1242 (457)

	<i>Non-exposed</i> Pre: 75.0 (9.0) Post: $\Delta$ -2.1 (2.1)	<i>Non-exposed</i> Pre: 47.8 (11.7) Post: $\Delta$ 0.6 (1.5)	<i>Non-exposed</i> Pre: 24.3 (6.9) Post: $\Delta$ 1.5 (1.0)	<i>Non-exposed</i> Pre: 13.0 (11.8) Post: $\Delta$ -3.1 (4.5)	<i>Non-exposed</i> Pre: 1262 (409) Post: 1311 (572)
Chaput, J.P. et al (2012) (18)	Pre: 94.7 (14.5) Post: $\Delta$ -4.5 (3.9)	-	Pre: 35.7 (8.3) Post: $\Delta$ -3.42	-	-
Verhoef, S.P.M. et al (2013) (19)	Pre: 92.5 (12.7) 3-mo: 84,4 (12.4)*	-	Pre: 38.7 (7.6) 3-mo: 30,9 (8,3)	Pre: 27.5 (16,0) 3-mo: 21,4 (18.5)*	-

BF=body fat, FFM=fat-free body mass,  $\Delta$ =changes in variables after interventions; the difference of the post- and pre-measurement, \*= p-value <0.05, \*\*= p-value <0.01

### **3.1 Body weight**

Seven of eight studies included body weight measurements pre- and post-intervention, and of these, all studies showed weight loss (12, 13, 14, 15, 17, 18, 19).

The randomized crossover studies of Nedeltcheva et al. (2010), Wang et al., Sparks et al. and Nedeltcheva et al. (2013) reported that the body weight decreased significantly in both the exposed and non-exposed group without any significant difference being found in the amount of absolute or percent weight loss between the groups (12, 13, 15, 17). However, there was a significant difference whether the weight loss came from bodyfat or fat-free mass.

Kline et al. registered that better sleep health (six dimensions: regularity, satisfaction, daytime alertness, timing, efficiency, and duration) were significantly positively correlated with greater weight loss over the 6-mo. interval (14), and Verhoef et al. concluded that sleep duration has benefits of weight loss or vice versa, but no results were significantly positively correlated (19).

### **3.2 Body composition**

When it comes to body composition, four of the eight studies included measurement on fat-free body mass (12, 13, 14, 17), and six studies included bodyfat measurement (12, 13, 14, 17, 18, 19).

The randomized crossover study of Nedeltcheva et al. (2010) reported that both treatments with 8.5 and 5.5 hours of nighttime sleep opportunity were accompanied by comparable weight loss with approximately 3 kg, but there were big differences in what the weight loss consisted of (12). During the 8.5-hours sleep opportunity more than half of the weight loss was fat (2.4 kg), and during the restriction with 5.5-hours nighttime sleep only a quarter of the weight loss was fat; 55% reduction in fat loss (0.6 kg). The sleep restriction group resulted instead in considerably increased with 60% loss of fat-free body mass (2.4 kg) compared to the 8.5hour-group (1.5 kg).

Wang et al. reported that both groups lost a similar amount of body weight, but the proportion of total fat mass lost was significantly greater in the group with an adequate duration of sleep (13). The amount of lost fat mass as a proportion of total mass lost was greater in the control-group (85% vs. 58%), and the amount of fat-free mass was less in the non-exposed group

(17% vs. 39%). In the non-exposed group, the amount of fat lost as a percentage was greater than baseline percent body fat, but in the exposed group the percentage was similar to baseline body fat.

Kline et al. reports that better sleep health (six dimensions) was associated with greater loss of bodyfat, but not fat-free mass (14). Better overall sleep health was associated with greater fat loss, and the dimensions «regularity», «timing», and «efficiency» were associated with fat loss. Overall sleep health was not associated with a change in fat-free mass, but the dimension «high alertness» was associated with greater loss of fat-free mass.

Chaput et al. showed a significant positive correlation between sleep duration and loss of body fat (18). The mean weight loss was 4.5kg, which 76% came from fat stores. They also observed that better sleep quality at baseline was associated with greater fat-loss and percent body fat loss.

### **3.3 Leptin**

Five of eight studies reported levels of leptin pre- and post-intervention, and of these, all studies showed a decrease in leptin levels after sleep restriction (12, 13, 16, 17, 19).

The randomized crossover study of Nedeltcheva et al. (2010) reported that leptin concentrations decreased in parallel with weight loss without a significant effect of sleep restriction (12).

Wang et al. and Verhoef et al. showed that the leptin concentrations were significantly reduced in the group with sleep restriction (13, 19). Verhoef et al. also had a significant positive correlation between the change in leptin and the change in fat mass after 10-mo follow-up.

The studies by Hart. et al. and Nedeltcheva et al. (2008) showed a non-significant decrease in leptin levels after both the short and the long sleep intervention (16, 17). The study by Hart et al. also showed that leptin levels decreased parallel to the participants' loss in fat.



### **3.4 Ghrelin**

Four of the eight included studies reported levels of ghrelin pre- and post-intervention (12, 13, 16, 17), and the majority of these studies showed no positive correlation between sleep restriction and ghrelin concentrations.

Wang et al., Hart et al., Nedeltcheva et al. (2008) and Verhoef et al. reported no significant difference in ghrelin concentrations between pre- and post-intervention with sleep restriction (13, 16, 17, 19).

The randomized crossover study of Nedeltcheva et al. (2010) was the only study that reported a significant difference in ghrelin concentration after sleep restriction (12). The serum concentration of ghrelin was higher at the end of the period with 5.5-hours sleep.

## **4 Discussion**

The main aim of this literature review was to investigate the effect of sleep duration on body composition for people with a BMI over twenty-five, and who are in a calorie deficit. A second aim was to investigate the effect of sleep restriction on the levels of leptin and ghrelin-, and whether an altered level in these hormones is associated with an increased risk of overweight and obesity.

The results of this literature review show that having a sleep restriction while being in a calorie deficit does not affect the change in body weight. Despite no significant difference in weight loss, the majority of the included studies show that the exposed groups lost more fat-free mass and less bodyfat - they ended up with an inferior body composition than the non-exposed groups (12, 13, 14, 18). The randomized crossover trial of Nedeltcheva et al. (12) reported a significant difference in ghrelin concentration, but the majority of the included studies found no significant changes in ghrelin levels after sleep restriction (13, 16, 17, 19). However, all included studies that measured leptin reported a decrease in leptin concentration after sleep restriction (12, 13, 16, 17, 19). Randomized controlled trials are the most reliable research method when looking at the effect of a measure and comparing two comparable groups, therefore more emphasis was placed on these results versus the prospective observational studies (12, 13, 15, 16, 17).

## **4.1 Body weight**

Seven of eight studies included body weight measurements pre- and post-intervention, and of these, all studies showed weight loss as a result of the calorie deficit (12, 13, 14, 15, 17, 18, 19). The majority of the studies reported that the body weight decreased significantly in both the exposed and non-exposed groups.

Several previous studies show that sleep restriction is associated with an increase in caloric consumption with no increase in activity energy expenditure (5, 7). Benedict et al. also shows that one night without sleep, reduces resting metabolism by 5%, and this combination can naturally lead to weight gain over time (20). Increased caloric intake without any accompanying increase in energy expenditure may therefore contribute to obesity in people who are exposed to sleep restriction over a longer period of time. This is also supported by Markwald et al. that reported weight gain during sleep restriction (6). If sleep restriction is persistent or permanent, it may increase the risk of weight gain and obesity.

## **4.2 Body composition**

Of the included studies, four included measurements on fat-free body mass (12, 13, 14, 17), and six studies included body fat-measurement (12, 13, 14, 17, 18, 19). Four of these studies (12, 13, 14, 18) reported a significant difference in what the weight loss consisted of. The exposed groups lost less proportion of body fat, and more fat-free mass compared to the non-exposed groups.

None of the studies included resistance training in the interventions, and since this form of exercise is a powerful tool to both maintain and build muscle mass, it could have been possible that the loss of fat-free mass could be reduced if the subject had engaged in resistance training during the intervention with both sleep restriction and calorie deficit. A randomized controlled trial from 2020 supports this with results that shows a combination of regular resistance training, and a sufficient amount of sleep provides significant added benefits to body composition (21).

Saner et al. reported findings that may help explain decreased fat-free mass in those with sleep restriction (22). The results of this study show that the frequency of myofibrillar protein synthesis was significantly lower in those who had sleep restriction compared to the group

that also implemented high-intensity interval training. They concluded that high-intensity interval training can be used to counteract loss of muscle mass during sleep restriction (22).

In this literature view, the activity levels are not considered, and it can therefore be assumed that one of the reasons for this is that inadequate sleep results in a lack of energy and surplus in daily life. Thus, they will have a reduced activity level during the day. This can constitute a substantial proportion of the energy consumption, when non-exercise activity thermogenesis (NEAT) and physical activity level (PAL) can be sharply reduced in a calorie deficit.

### **4.3 Leptin**

All five included studies that measured leptin showed a decrease in leptin concentrations after a period with sleep restriction (12, 13, 16, 17, 19). Previous research has shown that participants with sleep deprivation have reduced leptin, and these differences are likely to increase appetite. This is an explanation of the increased BMI observed in people with short sleep duration (23). When the concentration of leptin decreases, the feeling of satiety gets reduced - this can lead to an increase in body fat and then also an increase in body weight over time (11). Previous studies have reported that most obese individuals have increased levels of leptin. But studies show that increased levels of leptin over a longer period of time may result in leptin resistance. Leptin resistance leads to less sensitivity and therefore insufficient response to leptin (24).

### **4.4 Ghrelin**

Five of the eight included studies reported levels of ghrelin (12, 13, 16,17, 19.). Ghrelin is an appetite stimulating hormone, and when the concentration of ghrelin increases, the feeling of hunger heightens. The study by Nedeltcheva et al. from 2010 was the only study that reported a significant difference in ghrelin concentration after sleep restriction. The concentration of ghrelin was higher at the end of the 5.5-hours sleep period than the 8.5-hours sleep period (12). The increase of ghrelin, together with reduced leptin levels gives a strong increase in appetite. This can lead to an increased amount of body fat and body weight over time (25).

### **4.5 Limitations**

The sample sizes in some of the included studies may be a weakness in this literature study, especially considering that the smallest sample sizes only contain 10-12 individuals (12, 13, 15, 16, 17). Several of the studies focused on women in the sample (13, 14, 15, 16).

Considering that the menstrual cycle was not taken into account, this is a weakness in these studies because menstruation can affect the hormone concentrations in the female body. A larger study population with a more even distribution of gender could lead to a more generalizable result. However, although several of the included studies had few participants, there is no evidence to suggest that a larger sample may have changed the conclusion. Another weakness of this literature study is that several of the included studies have a short length of intervention; from two days interventions to two weeks (12, 16). It is therefore unclear whether the findings can be applied to situations where sleep restriction is repeated over a longer period of time, or if the sleep restriction is chronic. Longer exposure to different sleep durations may also be necessary to demonstrate differences in measured hormones and eating behaviors.

Several of the included studies conducted the sleep and analyzes in a laboratory, which can be evasive, and not comparable to the reality in terms of other environmental factors that can affect the subjects nighttime sleep (12, 14, 16, 17). Future studies should therefore have the intervention in the participants' natural environment as it may be able to give a more generalizable result.

Although the results show that weight loss consists of the most fat-free mass due to sleep restriction and calorie deficit, there are several factors that can determine whether the weight loss mainly consists of fat mass. Sleep restriction gives more waking time, and thus more hours where you can consume food. Sleep restriction can also lead to reduced motivation for exercise, and less energy surplus to maintain everyday activity throughout the day. None of the included studies implemented resistance training. Given that resistance training is a strong signal for maintaining muscle mass, it is conceivable that the loss of fat-free mass during sleep restriction could be reduced if the interventions implemented resistance training.

#### **4.6 Future research**

For future research, it is an advantage to include a larger sample size, where the participants live and sleep in their natural environment. This will give the studies a more generalizable result. It would be interesting to see if implementing resistance training in an intervention with sleep restriction and calorie deficit would change the results with loss of fat-free mass. And also, if the sleep restriction has a significant effect on changes on eating habits and dietary intake.

## **5 Conclusion**

After completing this literature study, it can be concluded that shortened sleep duration can affect the incidence of overweight and obesity. Adequate amounts of sleep are a factor that has a major impact on body composition during a weight reduction phase, and the findings provide evidence that sleep duration plays a key role in energy metabolism. If sleep restriction is persistent or permanent, it may increase the risk of weight gain and obesity. Sleep restriction in a calorie deficit had a small impact on the appetite-regulating hormone ghrelin, but a significantly decreasing concentration of leptin levels. The altered level in leptin is associated with an increased risk of overweight and obesity.

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