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Meta-analysis

Effect of sleep duration on dietary intake, desire to eat, measures of food intake and metabolic hormones: A systematic review of clinical trials

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SUMMARY

Background and aims: Sleep, as well as diet and physical activity, plays a significant role in growth, maturation, health, and regulation of energy homeostasis. Recently, there is increasing evidence indicating a possible causal association between sleep duration and energy balance. We aimed to examine the relationship between sleep duration and food consumption, energy intake, anthropometric characteristics, and appetite-regulating hormones by randomized controlled trials (RCTs).

Methods: Electronic literature searches were conducted on Medline, Web of Science, and Google Scholar until July 2020. The search was conducted with the following words: "Sleep Duration", "Circadian Rhythm", "Sleep Disorders" in combination with "Obesity", "Overweight", "Abdominal Obesity", "Physical Activity", "Energy Intake", "Body Mass Index", "Lipid Metabolism", "Caloric Restriction", Leptin, "Weight Gain", and "Appetite Regulation" using human studies.methods

Results: After screening 708 abstracts, 50 RCTs (7 on children or adolescents and 43 on adults) were identified and met the inclusion criteria. In general, the findings suggested that sleep restriction may leads to a significant increment in energy intake, fat intake, body weight, appetite, hunger, eating occasions, and portion size, while protein and carbohydrate consumption, total energy expenditure, and respiratory quotient remained unaffected as a result of sleep restriction. Serum leptin, ghrelin, and cortisol concentrations were not influenced by sleep duration as well.

Conclusion: Insufficient sleep can be considered as a contributing factor for energy imbalance, weight gain, and metabolic disorders and it is suggested that to tackle disordered eating it may be necessary to pay more attention to sleep duration.

1. Introduction

Sleep disturbances are very common and endemic in modern countries [1]. Approximately 50% of the elderly population suffers from sleeplessness and related sleep problems [2]. Due to dysregulated secretion of melatonin, a hormone with potent antioxidant actions and role in circadian synchronization [3], short sleep durations are associated with several diseases such as diabetes, hypertension, hypercholesterolemia, myocardial infarction, and stroke [4,5].

The amount of energy intake and quality of life can be affected by changes in the circadian cycle. So much so that some recent studies have shown an association between sleep disturbance and overweight/obesity [6,7], with short sleep duration being associated with reduced leptin and raised ghrelin levels, thus increasing appetite and favoring adiposity [8,9]. Such a hormonal shift results

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in augmented appetite while leads to hedonic hunger and hence increasing intake of hyper-palatable foods [10]. More critically, this dysregulation can ensue in obesity due to excess chronic energy intake. Interestingly, a comprehensive program of improved lifestyle can be effective in stimulating weight loss by approximately 10% within 16–26 weeks [11], whose change is associated with a meaningful improvement in sleeping, as well as in the hormonal and cardiovascular systems [12,13].

Not only obesity, but also metabolic syndrome is associated with sleep disturbances [14], seemingly due to a poor lifestyle such as smoking, alcohol intake, unhealthy dietary patterns, low physical activity, and job stress [15]. Obesity, in turn, is a preventable health failure that is described as excessive fat accumulation that results in impaired health, enhanced morbidity, and mortality worldwide [16]. Energy imbalance between calorie expenditure and calorie intake is thought of as the main cause of obesity [17]. Obesity is associated with multiple comorbidities such as cardiovascular diseases, diabetes mellitus, some types of cancer, hypertension, sleep apnea, stroke, among other things [18]. Globally, statistics attest that obesity has become twofold since 1980 so that 39% of the adult population was overweight and 13% obese in 2014 [19]. Annual healthcare expenses that are spent on obesity in the United States are estimated to be $147 billion to $210 billion [20]. In addition, obesity has been considered the fifth leading cause of death [21].

Given the importance of the aforementioned background, our objective was to systematically review the relationship between sleep duration and food consumption, energy intake, anthropometric characteristics, and appetite-regulating hormones through randomized controlled trials (RCTs).

2. Methods

2.1. Search strategy

Electronic literature searches were conducted on Medline, Web of Science, and Google Scholar until July 2020. Our search was performed based on the publisher databases Elsevier, Wiley Online, and Springer by using the medical subject heading (MeSH) and non-MeSH terms ("Sleep disturbance"[tiab] OR "Sleep disorder"[tiab] OR "Sleep Disorders"[Mesh] OR "Sleep Disorders, Circadian Rhythm"[Mesh] OR "Sleep Deprivation"[Mesh] OR "Sleep"[Mesh] OR "REM Sleep Parasomnias"[Mesh] OR "REM Sleep Behavior Disorder"[Mesh] OR "Sleep, REM"[Mesh] OR "Dyssomnias"[Mesh] OR “napping”[tiab] OR “short sleep duration”[TW] OR “long sleep duration”[TW] OR “sleep duration”[TW] OR “sleep hours”[TW] OR "Night Terrors"[Mesh] OR “night-time sleep duration”[TW] OR "Sleep Disorders, Intrinsic"[Mesh] OR "Sleep Apnea, Obstructive"[Mesh] OR "Dyssomnias"[Mesh]) AND ("Energy Intake"[Mesh] OR "Obesity"[Mesh] OR "Overweight"[Mesh] OR "Eating"[Mesh] OR "Caloric Restriction"[Mesh] OR "Appetite"[Mesh] OR "Eating Disorders"[Mesh] OR “food consumption” [TW] OR “dietary intake” [tiab] OR "Appetite Regulation"[Mesh]). Unpublished records and grey literature were not included in this review.

2.2. Eligibility criteria

Studies were considered if they met these inclusion criteria: 1) full-text publication was written in English; 2) RCTs (either parallel or cross-over); 3) evaluation of the association between sleep duration and dietary intake, desire to eat, measures of food intake, hormones related to hunger modulation, and anthropometric measures.

2.3. Exclusion criteria

Studies were excluded if they: 1) were non-RCTs; 2) were animal studies, conference papers, reviews, letters, editorial articles; 3) had a lack of sufficient data for the outcomes of interest in short or long sleep and normal sleep duration.

2.4. Data extraction

The following data were obtained from the included studies: the last name of the first author, publication year, range or mean age of participants, study design including crossover and parallel study, number of participants, gender, study duration, and primary outcomes. Summary of each study included can be seen in Table 1.

3. Results and discussion

The initial search identified a total number of 708 articles. After screening relevant abstracts, 58 articles were designed for critical perusal. Seven articles were excluded because the association between sleep duration and dietary factors was not evaluated. Another article was excluded because the data was reported elsewhere. Finally, the current updated systematic review covered 50 papers (Fig. 1) and a total number of 3387 participants, including 1079 children and adolescents and 2308 adults. The highest number of individuals was noted in Arora et al., research ($n = 593$) [22,23] and the smaller number in Hibi et al. (2017) and Schmid et al. (2008) studies ($n = 9$ for both studies) [24,25]. Seven papers encompassed children and adolescents [22,26–31].

3.1. Energy intake

Energy intake was investigated in 30 out of the 50 studies. Restricted sleep condition led to a trend toward more calorie consumption in 3 studies which one of them it occurred during the snack period [26,32,33]. Short sleep conditions indicated higher energy intake in 13 studies [29,31,34–44]. Two papers reported an increase in energy intake only from snacks after sleep curtailment ($P = 0.026$, 0.03) [45,46]. In one of the studies, women had a 15.3% increase in energy intake during short sleep relative to habitual sleep and men had a 9.2% increase, with differences between sexes [34]. Energy intake declined in 3 studies as a result of prolonged sleep duration [27,47,48]. In 7 studies, energy intake remained unaffected by sleep curtailment [49–55]. In contrast to other studies, one study reported a reduction in energy intake by 13% in the ad libitum meal and upon short sleep condition ($P = 0.031$) [28].

One study reported no remarkable discrepancies between short and long periods of sleep for total energy intake with participants consuming over 1000 kcal/d [51]. However, change in energy intake among participants was considerably variable, so that 5 participants consumed fewer calories, 3 participants consumed roughly the same, and 4 participants consumed more calories following the short versus long sleep conditions. In another study [22], after intervention based on a New Nordic Diet in both groups of short and long sleep, no differences in energy intake was detected between two tertiles of sleep condition ($P = 0.49$); energy intake nevertheless trended to increase by inadequate sleep condition in the end. Hogenkamp et al. suggested that overeating following sleep restricted condition may be as a result of two independent mechanisms: a homeostatic drive to compensate nocturnal energy deficit, and an increase in reward pathway susceptibility to calorie-dense food [56]. Furthermore, another study reported that following short sleep condition, participants had more

Table 1
Summary of each study included.

First author and year	Number of participants by gender	Age average	Type of intervention	Duration	Stated primary outcome
St-Onge (2011)	15 women 15 men	N/A	crossover	6 days Wings:5 d Washout:1 d	• EI: increased ($P = 0.023$) • Fat intake: increased ($P = 0.01$) • Pro intake: increased ($P = 0.08$) • Number of eating occasion: increased in 20 of the 26 participants and decreased by 1 in 3 participants • CHO: no difference ($P = 0.19$) • TEE: no difference ($P = 0.832$) • RQ: no difference • Fiber: no difference ($P = 0.16$)
Shechter (2013)	10 females	28.0 ± 2.3	crossover	3 days, with a 4-week washout	• 24-h EE: increased ($P = 0.012$) • RQ: no difference ($P = 0.93$)
Hjorth (2016)	530 % 51 boy % 49 girl	9.9 ± 0.6	crossover	3 months	• EI: no difference ($p = 0.49$) • Pro intake: increased ($p = 0.53$) • CHO intake: increased ($p = 0.01$) • Fat, energy: decreased while consuming NND compared with control ($p = 0.01$) • HOMA-IR: increased when consumed NND compared with controls ($p = 0.32$) • Fiber: increased when consuming NND compared with control ($P = 0.36$) • Weight: increased when consumed NND compared with controls ($P = 0.14$)
Beebe (2013)	41 typically developing adolescents (14–16 years old)	15	crossover	3-week Wings:5-night Washout:2 night	• EI: increased ($p = 0.098$) • Fat intake: no difference ($p = 0.175$) • CHO intake: not difference ($p = 0.070$) • Pro intake: no difference ($p = 0.481$)
Abbasi (2012)	23 women, 23 men	65 ± 4.6	N/A	8 weeks	Increased sleep duration: • Cortisol: decreased ($p = P = 0.008$) • TEI: decrease ($P = 0.02$) • CHO intake: decreased ($p = 0.01$) • Fat intake: decreased ($p = 0.04$) • Weight: no difference ($p = 0.07$) • Protein intake: no difference ($p = 0.283$)
Hart, C.N (2015)	12 females	41.7 ± 10.3	parallel	7 days	Comparing short and long sleep: • TEI: no difference ($P = 0.44$) • Leptin: no difference ($P = 0.73$) • Ghrelin: no difference ($P = 0.09$) • Insulin: no difference (p = 0.06) • CHO and fat intake: no difference (CHO, $P = 0.68$ and fat = 0.06) • Pro intake: greater percentage during the long sleep condition ($P = 0.01$) • HOMA-IR: no significant difference ($P = 0.24$)
Speath, A.M (2015)	Sleep restriction subjects, $n = 36$	21–50	parallel	14 or 18 consecutive days	• RMR: reduced ($P = 0.032$) • RQ: no significant difference (sleep restricted subject: $P = 0.57$/ control subject: $P = 0.22$) • Weight: no significant difference ($P = 0.097$) • Caloric and macronutrient intake: no difference ($P > 0.05$)
Hanlon, E.C (2016)	11 men 3 women	23.4 ± 0.8	crossover	1 month	• Leptin: decreased ($P = 0.01$) • The nocturnal peak ghrelin occurred earlier in restricted sleep compared with normal sleep conditions ($P = 0.002$) • Hunger: increased ($P = 0.02$) • Appetite: trend to increase ($P = 0.07$) • EI: first meal ($P = 0.24$), During the snack period (P = 0.085) • CHO intake: first meal ($P = 0.25$), During the snack period ($P = 0.218$) • Pro intake: first meal (P = 0.52), During the snack period ($P = 0.072$) • Fat intake: first meal ($P = 0.31$), During the snack period ($P = 0.062$) • Cortisol: morning achrophase increased ($P < 0.0001$)
Cizza, G (2014)	125	41	parallel	76 days	Increased sleep duration: • Fasting Insulin: decreased ($P < 0.001$)
Brondel (2010)	12 men	22 ± 3	crossover	2 days	• EI: increase during breakfast ($P < 0.01$) and dinner ($P < 0.001$) • Hunger: before the breakfast was greater ($P < 0.001$) and before dinner ($P < 0.05$) • Fat intake: higher at dinner ($P < 0.001$) • CHO intake: no difference
Hart (2013)	37 children (57% were male)	9.6	crossover	2 weeks	Increased sleep duration: • EI: decreased ($P = 0.04$)

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Table 1 (*continued*)

First author and year	Number of participants by gender	Age average	Type of intervention	Duration	Stated primary outcome
					• Macronutrient consumption: no significant difference ($P > 0.05$) • Mean percent kcal from fat, CHO and Pro were not difference: Fat ($P = 0.27$), CHO ($P = 0.53$), Pro($P = 0.49$) • Leptin: decreased ($P = 0.04$) • Ghrelin: no significant difference ($P = 0.21$) • weight: decreased ($P < 0.001$)
Calvin (2013)	11 men and 6 women	24.8	parallel	3 weeks	• EI: increased ($P = 0.014$) • Leptin: no difference ($P = 0.27$) • Ghrelin: no difference ($P = 0.21$) • Activity EE: no difference ($P = 0.62$)
Neadeltcheva (2009)	5 women and 6 men	39 ± 5	crossover	14 days	• EI from snack: increased ($P = 0.026$) • With higher CHO content ($P = 0.04$) and relatively less fat ($P = 0.08$) and Pro ($P = 0.08$) • EE: no difference (136 ± 437) • RMR: no difference (38 ± 162) • Leptin: no difference ($P = 0.76$) • Ghrelin: no difference ($P = 0.45$)
Dzaja (2004)	10 males	28 ± 31	crossover	2 days	• Ghrelin: increased • Cortisol: no significant difference
Hogenkamp (2013)	16 men	23 ± 0.9	crossover	1 day	• Portion size: was larger from snacks ($P < 0.01$) • Hunger: increased ($P = 0.02$) • Ghrelin: increased ($P = 0.04$)
Chapman (2013)	14 males	23 ± 0.8	crossover	2 days	• EI: increased ($P = 0.04$ and p = 0.01) • Ghrelin: increased
Speath (2013)	225 (44.9% women)	31	parallel	5 days	• Weight: no difference (p > 0.04) • EI: increased ($P = 0.003$) • Fat intake: no difference • Pro intake: no difference • CHO intake: no difference
Klingenberg (2012)	21 male adolescents	16.8	crossover	3 days	• EI (kJ): 13% less ($p = 0.031$) • Fat intake: increased ($P = 0.063$) • Ghrelin and Leptin: no difference • EE: increased • RQ: reduced ($P = 0.022$) • Appetite: decreased
Markwald (2013)	Adults (50% women, $N = 16$)	22.4	crossover	5 days	• EI: increased ($P < 0.05$) • CHO intake, g: increased ($p < 0.05$) • Fat intake, g: increased ($p = 0.32$) • Pro intake, g: no difference ($p = 0.82$) • Ghrelin and Leptin: no difference • Hunger: decreased • TEE: increased following LS($P < 0.01$) • Weight: increased ($P < 0.001$)
Schmid (2009)	15 adult men	27.1	crossover	2 days	• TEI (kcal): no difference ($p = 0.70$) • Fat intake (%): increased ($p = 0.06$) • CHO intake (%): consumption of sweet and salty snacks did not differ between the 4 h and 8 h ($p > 0/54$) • Pro intake (%): no difference ($p = 0.61$) • Leptin and Ghrelin: no difference • Appetite ($p = 0.17$), hunger ($p = 0.68$) and satiety (0.62).
Simpson (2010)	145 Adult men and women (49% women)	30.42	Simple RCT	7 days	• Leptin: increased ($p < 0.001$)
Spiegel (2004)	12 adult men	22	crossover	4 days	• CHO intake: increased ($p = 0.03$) • Pro intake: no difference ($p > 0.05$) • Appetite: increased ($p = 0.01$) • Leptin: decreased ($P = 0.04$) • Ghrelin: increased ($p = 0.04$)
Neadeltcheva (2010)	3 women and 7 men	41	crossover	14 days	• Weight: no difference ($p = 0.24$) • Cortisol: no difference • Leptin: declined in parallel with the loss of weight ($P = 0.001$) and without a significant independent effect of sleep loss • Hunger: increased • RMR: decreased • RQ: increased • Ghrelin: increased
Bosy-Westphal, (2008)	14 women 23–38	30.5	parallel	8 nights	• EI: increased ($p < 0.05$) • Leptin: increased ($p < 0.05$) • Appetite & hunger: no difference • TEE: no difference • Cortisol: no difference • RMR: no difference

Table 1 (*continued*)

First author and year	Number of participants by gender	Age average	Type of intervention	Duration	Stated primary outcome
Benedict (2011)	14 males	22.6 ± 0.8	crossover	4 weeks	• Insulin: no difference • Ghrelin: no difference • Weight: increased ($p < 0.05$) • Leptin: increased ($p < 0.05$) • RMR: reduced ($p < 0.05$) • EE: reduced ($P < 0.0001$) • Cortisol: higher during TSD ($p < 0.003$) • Insulin: no difference ($p > 0.42$) • Ghrelin: increased ($p < 0.02$)
St-onge (2012)	14 men and 13 women	30–45	crossover	3 weeks (23 days)	• Insulin: no significant difference ($P = 0.33$) • Leptin: no significant difference ($P = 0.16$) • Ghrelin: no significant difference ($P = 0.23$)
Schmid (2008)	9 men	24.2 ± 1.0	parallel	2 Weeks	• Hunger: increased in total sleep deprivation sleep than 7 h sleep ($P = 0.020$) or 4.5 h sleep ($P = 0.041$) • Ghrelin: increased ($P = 0.048$) • Leptin: no difference ($P = 0.93$)
Luccassen (2013)	125	41	parallel	76 days	• EI, portion size and number of eating occasions: 1) no difference between chronotypes were observed in TEE (in working day = 0.373 and in non-working day, $P = 0.922$), portion size (in working day, $P = 0.065$ and in nonworking day = 0.751), or number of eating occasions (in working day, $P = 0.175$ and in non-working day, $P = 0.568$). The amount of food intake after 20:00 in evening types was higher than morning types in both working day ($P < 0.001$) and nonworking day ($P = 0.028$) • No difference in total amount of fat ($P = 0.14$), CHO ($P = 0.84$) or Pro ($P = 0.89$)
Nedeltcheva (2009)	11 (5 females/6 males)	39 ± 5	crossover	14 days	• Cortisol: no difference ($P = 0.55$) • Insulin: no difference
Reynolds (2012)	14 males	27.4 ± 3.8	N/A	9 days	• Insulin: increased ($P < 0.001$) • HOMA-IR: no difference • Cortisol: increased ($P = 0.002$) • Leptin: increased ($P = 0.001$)
Shechter (2012)	14 males/13 females	30–45	crossover	2 weeks	• RMR: no difference • RQ: no difference • Appetite: no difference • EI: increased particularly from fat
Thomson (2012)	245 females (6 months) but 198 had weight change assessed through 24 months	45.5 ± 10.4	N/A	24 months	• Weight status: less weight loss
Broussard (2016)	19 men	23.5 ± 0.7	crossover	4 weeks	• Ghrelin: increased after RS ($P < 0.01$). • Leptin: no difference • Weight: average body weight was not significantly different ($P = 0.09$) • Total caloric and macronutrient intake: unaffected, but increased from snacks ($P = 0.03$), Primary from carbohydrate ($P = 0.02$)
Cedernaes (2014)	14 males	22.3 ± 0.5	crossover	4 weeks	• Hunger: increased • Glucose values: no difference ($P = 0.14$)
Mcneil (2017)	12 men/ 6 women	23 ± 4	crossover	2 weeks	• Appetite: increased • Hunger: increased ($P = 0.04$) • EI: no difference
Simon (2015)	31 adolescents	14–17	crossover	3 weeks	• Hunger: no difference ($p > 0.1$) • EI: increased after SR
Reid (2014)	54 (26 male)	30.6	parallel	7 days	• EI: no difference
Mcneil (2016)	12 men/ 6 women	23 ± 4	crossover	2 weeks	• Pro intake: no difference • Fat intake: no difference • TEI: trend to increase ($P = 0.05$) • CHO intake: increased ($P = 0.04$) • TEE: no difference
Dennis (2016)	66 (48.5% women)	21–50	parallel	13 days	• EI: no difference ($p = 0.12$) • Pro intake: increased ($P = 0.01$) • Saturated fat intake: increased ($P = 0.02$)
Speath (2014)	44 Female ($n = 21$)	21–50	parallel	7 nights (5 nights follow up)	• EI: increased ($P < 0.001$) • Fat intake: increased ($P = 0.024$) • Pro intake: decreased ($P < 0.001$) • CHO intake: no difference ($P = 0.97$)
Tan (2016)	49 men	30–65	parallel	6 months	Increased sleep duration: • EI: decreased ($P = 0.006$) • Weight: decreased

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Table 1 (*continued*)

First author and year	Number of participants by gender	Age average	Type of intervention	Duration	Stated primary outcome
					• EE: no difference
Arora (2016)	593	30–80	parallel	7 days	• Weight = increased ($P = 0.031$)
Hibi (2017)	9 men	23 ± 2	crossover	4 days	• EE: no difference • RQ: no difference • Leptin: no difference ($P = 0.172$) • Hunger: increased ($P = 0.004$) • Appetite: increased
Wang (2018)	36 80% women	35–55	parallel	8 weeks	• Leptin = decreased ($P = 0.029$) • RQ = reduced ($P = 0.033$) • Weight = no difference ($P = 0.87$) • Ghrelin = no difference ($P = 0.069$)
Rangan (2017)	368 55% male	2–6	parallel	1.3 years	• EI: increased ($P = 0.006$) • Fat intake: trend to increase ($P = 0.05$) • Saturated fat intake: increased ($P = 0.048$)
Petrov (2017)	51	2–4	parallel	2 weeks	• Fat intake: increased ($P = 0.003$) • CHO intake: decreased ($P = 0.002$)
McNeil (2017)	43 men ($n = 24$) women ($n = 19$)	31 ± 7	crossover	2 weeks	• EI: increased ($P = 0.01$)
Tajiri (2017)	16 female	21–22	crossover	3 nights	• EI: no difference ($P > 0.05$) • Leptin: no difference ($P > 0.05$) • Insulin: increased in SS ($P < 0.05$) • Cortisol: increased in SS ($P < 0.05$)
Yang (2019)	24 female	18–55	crossover	2 weeks	• Hunger: increased ($P = 0.013$) • Pro intake: increased (meal = 0.046/total = 0.035) • Fat intake: increased (meal = 0.008/snack = 0.036) • Total = 0.006 • Portion size: increased ($P = 0.014$)
Alkhatib (2017)	43	18–64	parallel	4 weeks	Increased sleep duration: • CHO intake: trend to reduce ($P = 0.09$)

N/A, not available; TEE, total energy expenditure; EE, energy expenditure; EI, energy intake; RQ, respiratory quotient; HOMA-IR, homeostatic model assessment of insulin resistance; NND, new Nordic diet; TEI, total energy intake; CHO, carbohydrate; Pro, protein; RMR, resting metabolic rate.

consumption of calorie and grams of food, indicating an increased hedonic drive, an increased level of homeostatic hunger or both [38].

3.2. Protein intake

Twenty out of fifty studies calculated the protein intake. Protein consumption tended to be higher during the period of short sleep than the habitual sleep in 5 studies [22,34,37,53,57]. Also, Hart et al. presented a higher protein consumption for the short sleep group compared to the long sleep conditions [51]. Short sleep conditions showed a lower percentage of calories from protein in 2 studies [40,44]. No difference in protein consumption was observed in nine records [9,26,27,32,33,36,39,49,50]. One study displayed that post-dinner protein intake was 42% higher during sleep loss [35]. Nedeltcheva et al., in turn, did not report any effect of sleep restriction on protein intake from meals but less content of protein from snacks was observed [45]. Protein intake did not differ as a consequence of long sleep duration [47]. In brief, it can be concluded that protein intake was unaffected following sleep changes.

3.3. Fat intake

Fat intake was reported in 22 papers. One study showed a trend toward increased fat intake ($P = 0.05$) [31]; significant associations between short sleep and increased fat consumption and a higher percentage of calories from fat were reported in 9 [28,30,34,36,40,42,44,49,57], saturated fat in 3 [31,34,53], whereas 7 other studies found no significant difference across the experimental conditions [26,27,32,33,39,50,51].

Three studies presented a decrease in fat consumption while sleep duration increased ($P < 0.001$, $P < 0.05$, $P = 0.04$) [22,35,47]. Hjorth et al. demonstrated that habitually long sleeping children following a New Nordic Diet consumed less fat (saturated and monounsaturated fatty acids) when compared with controls [22], while another study showed relatively less fat content from snacks as a result of sleep debt ($P = 0.08$) [45]. Observing sex discrepancies, in the investigation of St-Onge et al., women undergoing short sleep tended to intake a larger percentage of calories by total fat (38.5 ± 9.4% for short sleep and 32.8 ± 9.0% for habitual sleep; $P = 0.05$) and saturated fat (12.3 ± 5.0% for short sleep and 10.0 ± 5.0% for habitual sleep; $P = 0.06$) [34]. In summary, it can be deduced that sleep restriction is accompanied by more fat intake.

3.4. Carbohydrate intake

Nineteen out of 50 studies investigated the effect of sleep duration on the consumption of carbohydrates. Ten articles reported that carbohydrate intake was unaffected by sleep duration or there was no significant association between them [26,27,32,34,36,39,44,49–51]. Four articles demonstrated that short sleep duration resulted in greater consumption of carbohydrates [9,33,35,45], whereas one study indicated greater energy intake particularly from carbohydrates across snacks [46]. Markwald et al. indicated that altered eating patterns were observed during sleep loss as a result of changes in circadian phase and the circadian timing of awaking and also resulted in greater total daily food intake especially carbohydrate to meet the increased needs of energy that contributes to weight gain [35]. After several nights of sleep loss, adolescent's diet, as well as more calorie and carbohydrate consumption, were specified by higher glycemic index and

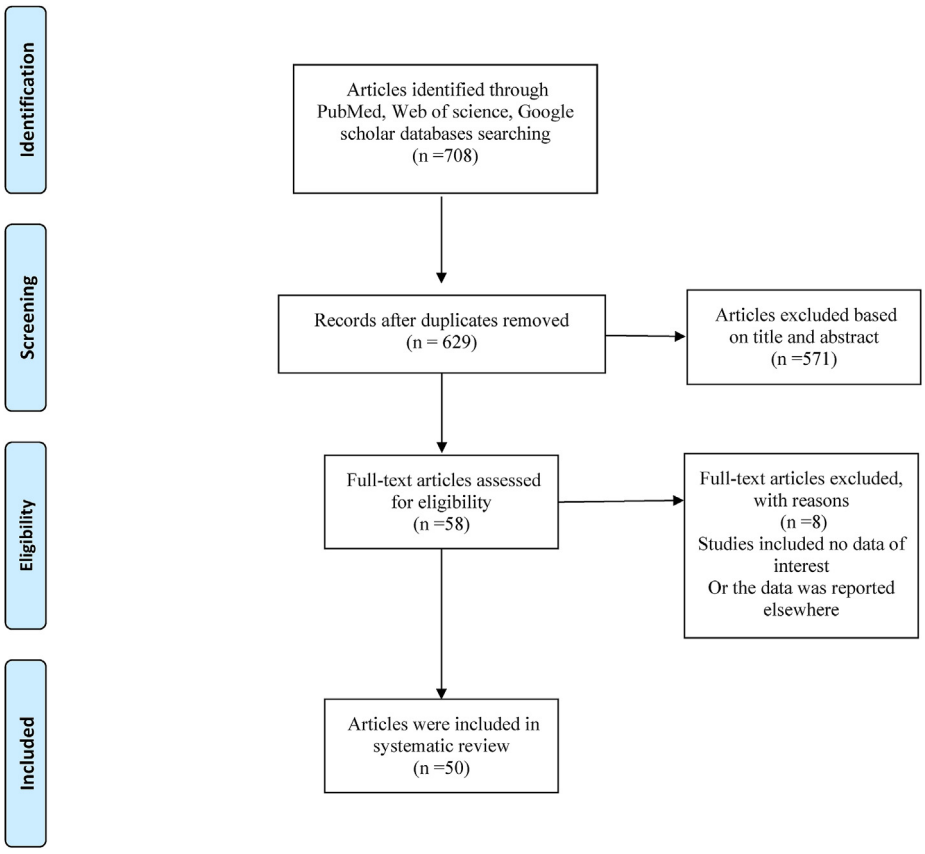

Fig. 1. Flow chart for study examined and included into the systematic review.

glycemic load, sleep restricted teens may reinforce reward pathway, comprising consumption of sweet foods [26]. Simple sugars cause dopamine release in reward centers [58]. Several mechanisms were suggested by Van Cauter et al. which may result in this relation: reduced glucose tolerance, decreased insulin sensitivity, increased sympathovagal balance, increased cortisol levels, increased ghrelin levels, reduced leptin levels, up-regulation of orexin neurons activity and changes in appetite regulating hormones [59]. Also, 2 records showed that longer sleep duration led to a decrease in the percentage of energy consumed from carbohydrates [47,60]. In contrast, one of the studies suggested that more sleep duration resulted in an increment in carbohydrate intake [22], and lower consumption of carbohydrates following shorter sleep was reported in one record [30]. Therefore, differences in sleep duration were not influenced by carbohydrate consumption when taken into consideration in most studies.

3.5. Fiber intake

In one study, fiber intake, as well as sugar and sodium, was not affected by sleep duration ($P = 0.16$) [34]. On the other hand, habitually short sleep children who had consumed the same diet (New Nordic Diet) of the long sleep group had a higher dietary fiber intake [22]. Such a finding is in line with one study in which subjects consumed 42% more calories as after-dinner snacks during sleep loss with more fiber content [35].

3.6. Hunger

Seventeen studies checked out hunger ratings. Restricted sleep conditions produced a notable increase in ratings of hunger in 11 studies [9,13,24,25,32,36,54,56,57,61,62]. Several articles discussed that changes in daily rhythm of 2-arachidonoylglycerol (2-AG), caused by sleep loss may be conducive to an increase in hunger, appetite and food intake related to sleep restricted situation [9,34–36,45]. One of the suggested mechanisms for previous positive findings was alteration of neuronal pathways that regulate reward behaviors following sleep debt [34] and also greater brain's calorie demand during prolonged wakefulness is a contributing factor to higher subjective feeling of hunger as reported in Schmid et al. study [24]. Despite no observed changes in hunger, when viewed in general, less feeling of fullness was reported in men after short sleep in one study [34]. In one article, hunger ratings showed neither effect of sleep condition nor sleep condition by time interaction, but without controlling for order, retest, practice, age, and BMI [51]. In 3 records, feeling of hunger did not differ during short sleep period [29,41,49]; an appealing feeling was nevertheless related to pictures of sweets/desserts as observed by Simon et al. [29] while Markwald et al. found a declined hunger as a consequence of sleep curtailment [35]. In other words, hunger ratings may increase as a result of inadequate sleep when condensing the results.

3.7. Appetite

In 3 articles, global appetite did not differ between normal sleep and restricted sleep conditions [41,42,49]. Conversely, a trend toward increase was showed by Hanlon et al. ($P = 0.07$) [32], and a significant increase in 3 studies ($P < 0.01$) [9,25,54] and decrease in another one [28]. Taken together, these results based on 8 articles provide underpinning for concluding that appetite is augmented under insufficient sleep.

3.8. Eating occasion

The individuals of St-Onge et al., investigation had 4.96 ± 1.2 eating occasions during habitual sleep and 6.08 ± 1.4 during short sleep, with the number of eating occasions increasing in 20 of the 26 participants while did not change in 3 participants and decreased by 1 out of 3 participants [34]. In another study, no change was detected between chronotypes, although morning types tended to eat more frequently than evening types during working days [50].

3.9. Portion size

Portion size became larger after total sleep deprivation in 2 studies ($P < 0.01$) [56,57]. According to Jung et al. the fact of increase in portion size as a result of sleep loss suggest that the overeating may represent a homeostatic compensatory response. This homeostatic response is probably produced to compensate energy deficit that is caused by sleep restriction [63]. In contrast, no difference between the 2 chronotypes was observed in one article, albeit larger portions from morningness at eveningness were observed [50].

3.10. Body weight

Collectively, 14 out of 50 records investigated the effect of sleep duration on weight status. Six studies reported that weight status was not under the effect of sleep duration or there was no significant association between them [28,39,40,46,47,64]. Four out of the articles showed that the reduction in sleep duration led to weight gain [22,23,35,41]. Several mechanisms were suggested in multiple studies: alteration in measure of some hormones (cholecystokinin, glucagon like peptide-1) may increase consumption of food during sleep deprivation [35]. Furthermore, insufficient sleep makes difference in eating behaviors. An evidence from epidemiological studies showed that overeating at night may lead to weight gain [65]. In addition, a group of orexin agents including neurons in hypothalamus regulate feeding behavior and appetite [66]. These orexin neurons react to sleep debt and can regulate the control of reward such as food and motivation [67]. One study indicated the possibility of achieving successful weight loss in women without a good sleep quality and with short sleep duration (<7 h/night) [68]. A paper showed that overall weight loss did not significantly differ between restricted sleep and normal sleep, but subjects with shorter sleep lost less percentage of fat in comparison with normal sleep [13] Also, 2 papers demonstrated weight loss following more sleep duration [27,48]. Thus, it can be inferred that poor sleep may hinder weight loss.

3.11. Ghrelin

Nineteen out of 50 studies investigated the effect of sleep duration on ghrelin levels. Eleven studies reported that sleep duration not significantly influenced ghrelin concentrations [27,28,32,35,37,41,45,49,51,64,69]. In contrast, 8 articles showed that a decrease in sleep duration resulted in increased ghrelin concentrations [13,24,38,46,56,61,70,71]. These findings can be as a result of different mechanisms, such as: The endocannabinoid (eCB) system gets involved in the control of appetite, energy homeostasis and sleep. The eCB system consists of cannabinoid CB1 and CB2 receptors. The endogenous agonists of these receptors are 2-AG and N-arachidonoylethanolamin [32]. It seems that the concentration 2-AG may increase in a state of sleep debt. Thus, it leads to a greater desire to eat more amount of food [9,34–36,45]. Therefore, the change in the levels of ghrelin and 2-AG after inadequate sleep may stimulate hedonic response that makes individuals consume foods which are highly palatable [32,56]. So, according to these findings, the orexigenic hormone ghrelin was unaffected by sleep duration when viewed collectively.

3.12. Leptin

Twenty out of 50 studies examined the effects of sleep duration on leptin levels. Thirteen studies reported that leptin concentration was not affected by sleep duration [13,24,25,28,35,37,45,46,49,51,55,61,69]. Hart et al. found that increased sleep duration led to decreased leptin concentrations [27]. In addition, short sleep duration resulted in enhanced leptin levels in 3 records [41,72,73] that may alter hedonic reaction to food in the central nervous system [74,75] and influence eating behavior. Conversely, 3 articles demonstrated that a reduced sleep period lowered leptin concentrations [9,32,45]. In this way, the anorexigenic hormone leptin was unaffected by sleep duration taking into account most studies.

3.13. Cortisol

Nine out of 52 articles described the connection between sleep duration and cortisol levels. Four of the articles reported that changes in sleep duration did not affect cortisol concentrations [13,41,70,76]. Three studies suggested that sleep restriction can lead to higher cortisol levels [55,61,73]while one study showed reduced cortisol concentrations under prolonged sleep duration [47]. Hanlon et al. reported that mean 24-h cortisol concentrations did not differ between normal sleep and restricted sleep conditions, but morning achrophase of cortisol increased in restricted sleep in comparison with normal sleep conditions [32]. Therefore, while 5 studies indicated no significant association between sleep hours and cortisol levels, a couple of studies showed a harmful relationship between sleep restriction and higher cortisol status.

3.14. Insulin

Eight out of 50 studies investigated the effect of sleep duration on insulin levels. Five articles showed that insulin concentrations were not affected by sleep duration [41,51,61,69,76]. Cizza et al., in turn, reported lower fasting insulin levels as a result of increased sleep hours [77] while 2 papers demonstrated that insulin levels increased following sleep loss [55,73]. Along these lines, sleep duration did not influence insulin levels in general.

3.15. Homeostasis model assessment of insulin resistance (HOMA-IR)

No noticeable effects regarding differences in sleep duration were found for HOMA-IR in 2 studies [51,73]. Habitually short sleeping children who consumed a New Nordic Diet had a larger increase in HOMA-IR values when compared with controls [22].

3.16. Energy expenditure

Altogether, 12 out of 50 papers analyzed the effects of sleep duration on energy expenditure. Seven trials did not report any significant effect of sleep duration on energy expenditure [25,33,34,37,41,45,48]However, 3 studies suggested that reduced sleep time led to enhanced energy expenditure [28,35,78]. Also, Brondel et al. found that sleep-restricted subjects had a positive energy balance which means that energy expenditure was lower than energy intake in this group [36]. In addition, resting energy expenditure and postprandial energy expenditure decreased as a result of short sleep situation in a record [61]. Furthermore, energy

expenditure can liaise to racial groups, Spaeth et al. reported that African American get extra weight than Caucasians during sleep restriction, but they did not have more caloric intake [40].

3.17. Respiratory quotient

Collectively, 8 out of 50 studies had checked the effect of sleep duration on respiratory quotient but 5 of them did not mention the significant effect of sleep duration [25,34,40,42,78]. Conversely, short sleep duration led to an elevation in the respiratory quotient in one study [13], while it reduced following declined sleep period in 2 records [28,64].

3.18. Resting metabolic rate

Resting metabolic rate was discussed in 8 papers which in 4 of them it did not vary significantly between sleep conditions [28,42,45,69]; in one of them, although Resting metabolic rate did not change significantly, individual increases may be observed after glucose load following restricted sleep condition [41]. Another 3 papers suggested that resting metabolic rate decreased after sleep curtailment ($P = 0.032$, $P = 0.01$, $P < 0.05$, respectively) [13,40,61]. Benedict et al. also detected a 20% lower postprandial metabolic rate during short sleep [61]. Bearing this in mind, a handful of studies corroborate declined resting metabolic rate after sleep deprivation but it should be emphasized that 4 papers suggested no changes.

4. Novelty and limitations

Although the investigated area is already well explored in the literature, this work is a novelty by providing an updated systematic research covering 50 papers in which 40 of them were not assessed in a previous systematic review whose authors worked on a similar setting [79]. Nevertheless, while our study provides a general conclusion, it is not exempt from bias, since residual factors exist due to the difficulty of minutely controlling the variables studied even in the circles of science. For example, there is no gold standardized method for controlling food intake and both self-report measures are a typical problem in nutrition and sleep research because of systematical error [80,81].

5. Conclusions

Overall, in this systematic review, we found that most studies corroborated that sleep restriction leads to a significant increment in energy intake, fat intake, body weight, appetite, hunger, eating occasions, and portion size, while protein and carbohydrate consumption, total energy expenditure, and respiratory quotient remained unaffected. In addition, serum leptin, ghrelin, and cortisol concentrations were not influenced by sleep duration in general. According to a recognized consensus statement, adults must sleep $\geq$7 h a day regularly to promote optimal health, while short sleepers (<6 h of sleep) are more susceptible to deleterious consequences [82,83]. Thus, as a general rule, sleeping $\geq$7 h a day is undeniably the ideal recommendation, but as the combination of full-time work with daily activities and time spent in traffic is unavoidable today for millions of people, personalized strategies ought to be discussed to optimize sleep.

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Author contributions

The author's responsibilities were as follows, BA contributed to the study design, SS, ShS, MA and BA contributed to manuscript draft; HOS contributed to editing of the manuscript; BA supervised the study. All authors read and approved the final manuscript.

Declaration of competing interest

All authors declared no conflicts of interest.

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