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Sleep deprivation in development of obesity, effects on appetite regulation, energy metabolism, and dietary choices

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Abstract

Sleep deprivation, which is a decrease in duration and quality of sleep, is a common problem in today's life. Epidemiological and interventional investigations have suggested a link between sleep deprivation and overweight/obesity. Sleep deprivation affects homeostatic and non-homoeostatic regulation of appetite, with the food reward system playing a dominant role. Factors such as sex and weight status affect this regulation; men and individuals with excess weight seem to be more sensitive to reward-driven and hedonistic regulation of food intake. Sleep deprivation may also affect weight through affecting physical activity and energy expenditure. In addition, sleep deprivation influences food selection and eating behaviours, which are mainly managed by the food reward system. Sleep-deprived individuals mostly crave for palatable energy-dense foods and have low desire for fruit and vegetables. Consumption of meals may not change but energy intake from snacks increases. The individuals have more desire for snacks with high sugar and saturated fat content. The relationship between sleep and the diet is mutual, implying that diet and eating behaviours also affect sleep duration and quality. Consuming healthy diets containing fruit and vegetables and food sources of protein and unsaturated fats and low quantities of saturated fat and sugar may be used as a diet strategy to improve sleep. Since the effects of sleep deficiency differ between animals and humans, only evidence from human subject studies has been included, controversies are discussed and the need for future investigations is highlighted.

Keywords: **appetite: dietary intakes: food reward system: obesity: sleep deprivation: snacks**

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Introduction

Sleep physiology

Sleep is a basic need for humans[1]. Humans spend almost a third of their life in sleep. Sleep is needed for removing toxic metabolites, synaptic plasticity and memory consolidation. It is also required for normal metabolic activity, hormone regulation, tissue repair and optimal function of cardiovascular and immune system[2].

Sleep cycle has two main phases: non-rapid eye movement (NREM) and rapid eye movement (REM)[3,4]. NREM has three stages: N1, N2 and N3. N1 is the lightest stage of sleep, and is the transition state between wakefulness and deep sleep. It is associated with muscle relaxation, and lasts for a few minutes. N2 is deeper and is associated with decreased body temperature and heart rate. It constitutes 50% of total sleep time. N3, which is also called the slow-wave sleep (SWS), is the deepest stage of sleep. At this stage, the body is in complete relaxation and the person hardly wakes up. NREM sleep is essential for physical recovery and repair. A reduction in NREM results in physical and metal fatigue, reduced performance and impaired cognitive function[5,6].

REM sleep is the stage where most dreams occur[4]. It is characterised by quick eye movement behind the eyelids, muscle relaxation, irregular breathing, elevated heart rate and increased brain activity. NREM stage accounts for 75% of the total sleep time, leaving the remaining 25% for REM stage. Stages of sleep are repeated regularly throughout the night. Sleep cycle starts with N1, deepens through N2 and N3, and ends in REM. After the REM sleep, the cycle starts again. The duration of each sleep cycle is approximately 90 min. Each night has four to six sleep cycles. As the night progresses and the morning approaches, the length of NREM sleep decreases and that of REM increases[1,4,7].

It is postulated that sleep is regulated by interaction of two processes: process S and process C[8,9]. Process S or homoeostasis process controls the amount of sleep required for restoration of physical and mental functions. It keeps the balance between the need for sleep and wakefulness, and determines the amount of sleep according to the duration of wakefulness and the amount of sleepiness. During waking hours, adenosine levels gradually increase in the brain, and lead to a feeling of sleepiness and the desire to sleep[7].

Process C or circadian process regulates the timing of sleep according to body's internal clock, also called the circadian clock[7,10]. The circadian clock is located in the suprachiasmatic nucleus (SCN) that are a group of neurons situated above the optic chiasm in the brain[11]. The clock is influenced by external cues such as light and temperature and internal cues such as hormones. In turn, the activation of SCN targets hypothalamus,

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thereby regulating the rhythmic secretion of many hormones and body temperature. In dark, the SCN stimulates pineal gland to produce melatonin, which promotes sleep, whereas in light, SCN stimulates production of cortisol to promote wakefulness[12]. Thus, melatonin and cortisol have opposite alterations during a 24 h period: melatonin levels are lowest during day and are highest at night, while cortisol levels are lowest at midnight, gradually increase during night sleep, peak at approxmately 9 am, and then gradually decline to reach its minimum again at midnight[13]. An overview of the proposed mechanisms for sleep physiology and diurnal fluctuations of serum melatonin and cortisol is demonstrated in Fig. 1.

Circadian rhythm plays an important role in maintaining sufficient sleep, and disruption of this rhythm by any factor, such as shift work, can result in sleep problems. Apart from circadian rhythm, the time of sleep is regulated by chronotype. Chronotype is a natural propensity in individuals to sleep or be active and alert at certain times during a 24 h period[14]. There are two main chronotypes: morning and evening. Individuals with morning chronotype sleep and wake up early and prefer to be active in the morning while those with evening chronotype prefer to sleep and wake up late. Chronotype is influenced by genetics but factors such as age and sex affect it[15]. In general, infants are morning chronotype, young adults are evening chronotype, and in middle and old ages people become morning chronotype again[14,16]. Morning chronotype is more frequently seen among women while men are generally evening chronotype[15].

Sleep deprivation

Sleep deprivation, also known as sleep deficiency or sleep insufficiency, occurs when the amount and/or quality of sleep is reduced[17]. This may occur when a person does not get sufficient sleep that he/she needs, sleeps at wrong time (e.g. sleeps a part of day instead of night), or has sleep disorders. In low sleep efficiency, the duration of deep sleep, i.e. stage 3 of NREM (N3), is usually reduced[18]. Sleep deprivation also occurs when the total time spent in bed does not change but the actual time spent asleep reduces[19]. US National Sleep Foundation has recommended sleep durations for different age groups as follows: 14–17 h for newborns, 12–15 h for infants, 11–14 h for toddlers, 10–13 h for preschoolers, 9–11 h for school-aged children, 8–10 h for teenagers, 7–9 h for young adults and adults and 7–8 h for older adults[20].

Surveys performed in different parts of the world indicate that sleep deprivation is more or less prevalent even among healthy individuals. According to the statistics released on World Sleep Day 2019, adults in 12 countries throughout the world (Australia, Brazil, Canada, China, France, Germany, India, Japan, Netherlands, Singapore, South Korea and the USA) had on average 6·8 h sleep in weeknights and 7·8 h in weekends[21]. Overall, 62% of these adults did not feel they were getting enough sleep. In the USA, 35·3% of adults reported <7 h sleep during a typical 24 h period[22] and 7·1% were diagnosed at least once with a sleep disorder[23]. A meta-analysis of surveys conducted in low- and middle-income countries (Iran, Turkey, China, Russia, Georgia, Pakistan, India, Bangladesh, Haiti, Brazil, Mexico, Venezuela, Peru, Ecuador, South Africa, Argentina and Ghana) demonstrated a sleep duration of 7·6 h and 32·8% prevalence of poor sleep quality in working age adults[24]. Another meta-analysis estimated a prevalence of 36% for poor sleep quality, 37% for sleep duration of <7 h, 22% for insomnia and 10% for sleepiness in industrial workers[25].

Sleep deprivation may result from a variety of medical conditions. These include but not limited to psychiatric diseases (e.g. depression and anxiety)[26], illnesses that bring along physical pain[27], asthma[28], sleep apnea[28], insomnia, parasomnias, Alzheimer's and Parkinson's disease, and restless legs syndrome[29]. Healthy individuals may experience sleep deficiency as a result of interference of work, school and social activities with sleep time, psychological pressures of modern life, environmental factors such as noise, light, temperature, extensive use of electronic devices, and application of recreational substances such as alcohol, caffeine and nicotine[30,31]. Night shift work[32], ageing[33] and food insecurity[34] are also factors that have reduced sleep duration/quality over the last decades[30,31].

Apart from feeling less refreshed during the day, sleep deprivation has negative effects on agility, productivity, efficiency to perform daily activities and health status[17,35]. Sleep deprivation leaves adverse effects on cognitive performance and brain function, cardiovascular health, immune system, endocrine homoeostasis and mood[36]. It predisposes individuals to obesity, and as a consequence to insulin resistance, type 2 diabetes, dyslipidaemia and other metabolic diseases[37,38]. Accordingly, sleep disorder is suggested as a predictor of increased mortality[23,39], and sleep extension has been introduced as a strategy to attenuate obesity risk and cardiometabolic dysfunction[40].

Objective

The current review focuses on two main topics: mechanistic pathways for the role of sleep deprivation in development of obesity and the current knowledge on the relationship between sleep deprivation and food and dietary choices. In more details: first of all, an overview on the description, causes, consequence and prevalence of sleep deprivation is presented. Then, scientific evidence for the link between sleep deprivation and obesity is reviewed, and mechanistic pathways for the effect of sleep deprivation in promoting obesity are discussed. Possible effects of sleep deprivation on energy intake, appetite regulation, physical activity and energy expenditure will be described. Also, the role of sex and weight status in the effect of sleep deprivation on appetite control and food selection will be stated. Impact of sleep deprivation on food selection, diet quality, consumption of snacks and desire for consumption of sweets are among issues that will be discussed in more details. At the end, the relationships are reversed and evidence for possible effects of eating habits, diet quality and dietary components on sleep health will be presented. Applying these diet considerations may help in reducing sleep problems in affected individuals.

Role of sleep deprivation in development of obesity

The relationship of sleep deprivation and weight gain has been highlighted in previous investigations[41]. Longitudinal cohort

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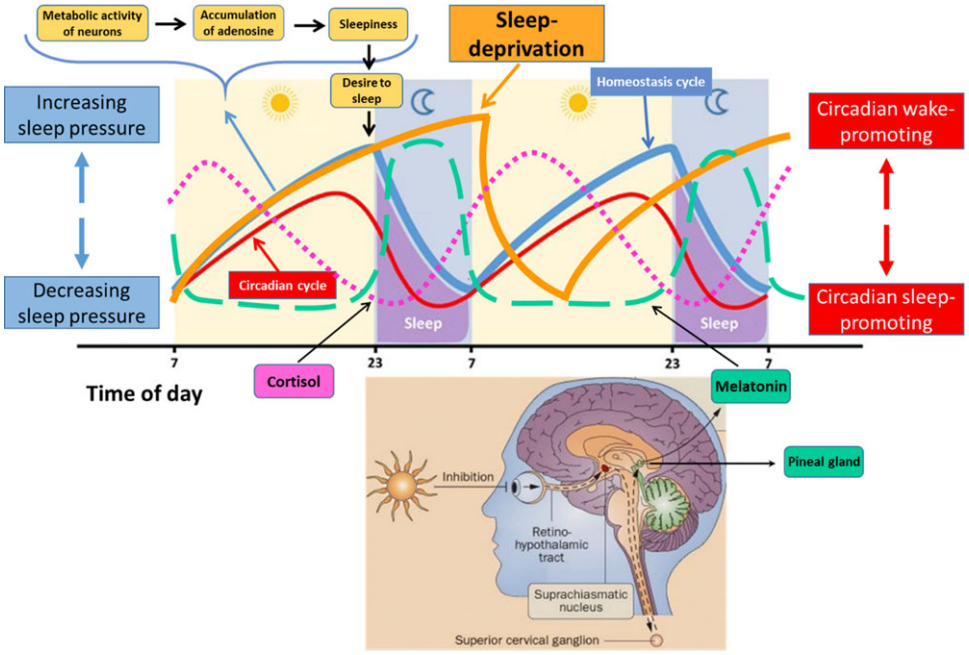

Fig. 1. Proposed schematic of sleep and wake regulation by two homoeostatic and circadian processes as well as a simplistic view of diurnal changes in melatonin and cortisol levels. Homoeostatic process (blue curve) is hypothesised to be regulated by adenosine levels in the brain. In homoeostatic cycle, neuronal activity gradually increases adenosine levels in the brain, reaching its highest level concomitant with sleepiness and desire to sleep at the time of sleep. In circadian process (red curve), sunlight inhibits the circadian clock located in the suprachiasmatic nucleus (SCN), thus inhibiting the SCN signals. In dark, SCN signals stimulate the pineal gland to produce melatonin, a molecule synthesised from tryptophan that has an important role in regulation of sleep–wake cycle. SCN also sends signals to the superior cervical ganglion, which regulates circadian rhythm in other parts of the body such as heart and circulatory system. In sleep-deprivation, the regular sleep–wake cycle is disrupted and this leads to misalignment of homoeostatic (demonstrated with brown colour) and circadian processes, leading to a number of brain and systemic dysregulations. Melatonin levels (dashed green curve), which are positively associated with drowsiness and sleep, are lowest during the day and highest during the night. Conversely, cortisol levels (dotted pink curve) are lowest at midnight, gradually increase during night sleep, peak at approximately 09.00, and then gradually decline to reach a minimum again at midnight.

studies have shown the fastest weight gain in people who get the least amount of sleep[42]. Also, meta-analyses of cohort studies in both children[43] and adults[44] have emphasised short sleep duration as a predisposing factor for obesity. For instance, a meta-analysis of 22 cohort studies on adults showed that <7 h sleep compared with 7–8 h sleep was associated with an odds ratio of 1·26 (95% confidence interval (CI): 1·17, 1·36) for obesity[45]. Also, a meta-analysis of 33 cohort studies on children and adolescents revealed an obesity risk of 1·57 (95% CI: 1·36, 1·81) for short sleep duration[46]. Results of meta-analyses on cohort studies have been confirmed by meta-analysis of randomised controlled trials (RCT), in which strategies that improve duration and/or quality of sleep reduced body weight[47].

Each hour sleep deprivation has been associated with 0·16 and 0·35 kg/m^2 increase in body mass index (BMI) in adolescents[48] and adults[49], respectively. Accumulation of fat occurs especially in central body areas even when total body fat does not change. As an example, in an RCT on healthy non-obese individuals, alterations in total body fat did not differ between sleep restriction (14 d sleep restriction with 4 h sleep opportunity) and control conditions, but total abdominal fat increased during sleep restriction, with significant increases evident in both subcutaneous and visceral abdominal fat masses[50]. Also, in a study on resident physicians, those with poor sleep quality reported greater weight gain and higher frequency of abnormal waist circumference after beginning of residency compared with physicians with better sleep quality[51].

In weight loss programmes, sleep disturbance during weight loss has been associated with an elevated risk of failure to lose weight[52]. Moreover, higher sleep fragmentation, which is determined by measuring the number of wake-ups, led to less weight reduction in those who participated in weight loss programmes[53]. Energy restriction programmes may have more adverse effects on sleep-restricted individuals. Nedeltcheva *et al.* reported that energy-restricted individuals under insufficient sleep had a higher loss of fat-free mass whereas those with adequate sleep showed higher loss of fat mass[54]. Sleep curtailment decreased the proportion of weight lost as fat by 55% and increased loss of fat-free mass by 60%.

The association of short sleep duration and obesity seems to be stronger in younger ages. This has been clearly shown in a meta-analysis of observational data collected from 634,511 participants with ages ranging from 2 to 102 years[49]. The relationship of short sleep duration and obesity had odds ratios of 1·89 (95% CI: 1·46, 2·43) and 1·55 (95% CI: 1·43, 1·68) in children and adults, respectively. Short sleep duration in childhood may predict adulthood obesity. A 32 year prospective birth cohort study showed that shorter childhood sleep times were associated with higher adult BMI values, and the association remained after adjustment for adult sleep time and potential confounders such as early childhood BMI, parental BMI and adult physical activity[55].

It is worthwhile noting that it is not only sleep deprivation that acts as a risk factor for obesity but oversleeping may also

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function as a risk factor, suggesting a U-shaped relationship between sleep duration and BMI. However, there may be a sex-specific effect in this proposed U-shaped relationship[56]. In this regard, a 10 year cohort study in women showed that both habitual short and long sleep durations were risk factors for general obesity in women aged <40 years[57]. Habitual long sleep duration was also a risk factor for central obesity in young women. But, this kind of association was not observed in a meta-analysis which included prospective studies of both sexes[58]. Hence, long sleep duration may be the risk factor of obesity only in women. Since there is still controversy for the existence of such curvilinear association between sleep duration and BMI especially in men[56] more studies examining consequences of long sleep duration are needed. Consistent with what was mentioned for short sleep duration, long sleep duration is also associated with increased mortality risk[39].

In the following sections, we will present some of the mechanisms by which sleep deprivation may contribute to obesity.

Energy intake in sleep deprivation

Most obesity cases result from positive energy balance due to extra energy intake[59]. Meta-analyses of RCT indicate that partial sleep deprivation increases total energy intake compared with habitual sleep[60,61]. Cross-sectional studies have also shown the association of sleep deprivation and increased energy intake[62–65]. Most of this extra energy is consumed in the form of snacks especially after dinner[66] as discussed later in this article. This increased energy intake is assumed to be a physiologic adaptation to compensate the augmented energy expenditure due to additional wake hours to prevent weight loss[66]. However, easy access to snacks, increased desire for energy-dense snacks after sleep deprivation, and in some cases increased hunger, leads to overeating and weight gain[67], as will be discussed in the following sections.

Despite the above justifications, sleep deprivation has not increased energy intake in some trials with short but severe restriction, for example, after two nights of 4 h sleep[68] or one night of <5 h sleep[69]. It is likely that psychological stress caused by severe sleep deprivation reduces food intake while in prolonged sleep deprivations a sort of adaption occurs. The lack of the relationship of sleep deprivation with energy intake in some cross-sectional studies can also be justified. In an analysis of NHANES data, short sleepers had lower mean percentage of energy from main meals but higher energy intake from snacks[70]. Although energy intake did not associate with sleep duration, eating main meals and snacks at or after 20.00 in short-duration sleepers was suggestive of eating behaviours that may, in the long-term, lead to positive energy balance. Also, in a cross-sectional study on Japanese women, energy intake was lower in women with low sleep efficiency compared to participants with the middle and high sleep efficiency[71]. Since the participants were young Japanese women, the authors justified this unexpected finding to participants' desire to control their energy intake to prevent obesity, and thus attributed the observed low sleep efficiency a consequence of this eating control.

Appetite regulation in sleep deprivation

Increased food intake is driven by homoeostatic or hormonal and non-homoeostatic mechanisms. In the following sections, we describe these mechanisms in detail.

Homoeostatic regulation of appetite

There is compelling evidence in the literature indicating increased hunger and reduced fullness and satiety following lack or insufficient sleep. A meta-analysis of 15 RCTs showed that both partial and total sleep deprivation increase hunger and daily energy intake[72]. The increased hunger has been observed in all fasted, pre-prandial[73,74] and sated states[75]. In agreement, in a study on adults with overweight and habitual short sleep duration, extending sleep from ~ 5·6 h to ~ 7·1 h led to 14% decrease in overall appetite and 62% decrease in desire for sweet and salty foods[76]. However, not all investigations have reported the change in hunger in sleep restriction[68,77]. Contradictions could be due to differences in the protocol of investigations. For instance, in *ad libitum* eating condition, participants were less likely to feel hunger than in calculated and fixed amounts of food. The time of waking in the morning, the time interval between awaking and hunger assessment and the type of activities that participants performed after sleep deprivation (e.g. watching television compared with playing games) may have also caused such contradictions in the reports of hunger induction by sleep deprivation[78].

The role of gut hormones. Leptin and a number of gut hormones are involved in the regulation of appetite and food intake[79]. Leptin and ghrelin have been speculated to play a role in development of obesity in sleep deprivation conditions[80]. Leptin is primarily produced by adipose tissue in amounts proportional to body fat masses[81]. It is an important anorexigenic hormone which induces satiety and reduces food intake. Ghrelin is mainly secreted by endocrine cells of the stomach and is the chief orexigenic hormone that stimulates hunger. The effect of sleep on leptin and ghrelin levels has not been completely elucidated. Some cross-sectional[51,64,82,83] and interventional studies[75,84–86] have shown alterations in blood ghrelin and/or leptin concentrations in directions to increase hunger or decrease satiety (i.e. augmented ghrelin and reduced leptin). The same direction of alteration in leptin has been reported for cholecystokinin in a cross-sectional study of adults with obesity, in which low sleep efficiency was associated with lower postprandial cholecystokinin levels[87]. As expected, poor sleep quality has the same relationship as short sleep duration with the mentioned appetite-regulating hormones[51,87,88].

Leptin. The decreased leptin concentration after sleep deprivation may play a role in stimulation of obesity in insufficient sleep conditions. In this regard, in a cross-sectional study, after adjustment for plasma leptin levels, adiposity indices were no longer associated with short habitual sleep[83]. The decrease in leptin may be due to increased activity of sympathetic nervous system which occurs following sleep deprivation and has an inhibitory effect on leptin secretion from adipose tissue[89]. The inverse relationship between sleep deprivation and leptin levels

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may be reciprocal, meaning that it is not only sleep deprivation which prevent leptin release, but leptin may in turn contribute to deep sleep[90]. Hirota *et al.* reported that plasma leptin levels were independently associated with sleep quality in patients with obesity and type 2 diabetes, although no association was observed in non-obese patients[90], suggesting that the association between sleep deprivation and leptin concentrations may be stronger in individuals with obesity, as also evidenced in female children in a population-based study[91]. Since individuals with obesity generally have augmented levels of leptin and considering that due to adiposity they are at increased risk of poor sleep quality[92], the higher levels of leptin in individuals with obesity may suggest leptin's potential to improve sleep quality of these patients to compensate, at least partly, for the adverse effects of obesity on their sleep[90].

Negative reports and controversies. There are also interventions in which the decreased leptin was not accompanied with an increase in ghrelin[93] or vice versa[94,95]. Literature also contains reports denying the effect of sleep deprivation on appetite-regulating hormones[66,68,96–101], and a meta-analysis of RCT did not show significant effect of sleep restriction on leptin and ghrelin levels[72]. In contrast, a meta-analysis of both cross-sectional and experimental studies indicated that short sleep duration was associated with increased ghrelin level in cross-sectional studies, but sleep deprivation increased leptin levels in experimental investigations[102]. Reasons of controversies may be incompatibility between studies regarding the age, sex, and weight of participants[103], the energy balance and diet composition during experiments, the extent of sleep deprivation and the type of exercise and activities that participants were allowed during waking hours. For instance, the increased leptin levels in studies where participants had obesity seems reasonable due to the leptin resistance that obesity induces[104]. More details on the effects of sex and diet composition on sleep are given in the following sections.

Non-homeostatic regulation of appetite, the role of food reward system

Brain reward system. The paradox of increased food and energy intake in the lack of change in appetite-regulating hormones, e.g.[97] may be explained by the brain reward system, which in addition to homoeostatic factors regulate the eating. Brain reward system is responsible for stimulating behaviours that increase the likelihood of survival and reproduction[105]. Rewarding stimulus for such a system is any stimulus, object, activity or situation that elicits pleasure and is thus attractive and motivating. Food is one of these stimuli[106]. Non-homoeostatic appetite regulation operates independently of homoeostatic regulation. Thus, the increased activity of the reward system and subjective food valuation could occur in the absence of hunger and alteration in appetite-related hormones (ghrelin, leptin, insulin and cortisol)[99]. Food reward consists of two components: appetitive that refers to "wanting" or motivation to consume a food and consummatory which returns to the hedonic aspects of a food and leads to "liking" a food[106].

Neuroanatomy of the food reward system. Parts of cortical and subcortical areas of brain are known to evaluate food stimuli[107]. At the cortical region, the anterior insula cortex, lateral orbital frontal cortex and anterior cingulate cortex have roles in governing appetitive choices and food preferences. Thus, the activity of cortical areas relates to the "liking" component of food reward. At subcortical regions, the amygdala and ventral striatum are engaged in managing motivation to eat, what is called "wanting"[107]. In fact, the amygdala is involved in modulation of emotional feeding and reward processing[108,109]. Reward information from the motivational and hedonic structures is sent to the nucleus accumbens and then to the lateral hypothalamic area to control feeding behaviours[110–112]. In abnormal conditions, the function of the reward system is impaired and leads to over- or under-eating. For instance, patients with anorexia or frail elderly lose weight because they suffer from loss of pleasure in eating and appetite[106]. In contrast, in some obesity cases, hyper-sensitisation of motivational structures in brain leads to excessive food "wanting"[112]. Such condition also occurs in cases of drug addiction.

Sleep deprivation and food reward system. A large body of evidence indicates that sleep deprivation alters reward-driven eating behaviour[113]. Animal and human subject studies have shown that sleep deprivation increases motivation for reward[114]. There are two hypotheses by which the reward system reacts to inadequate sleep. In the first hypothesis, the sensitivity of the reward system to food cues increases, thus individuals have augmented responsiveness to food rewards[115,116]. In this regard, Demos *et al.* showed that compared to sufficient sleep (9 h/night), short sleep (6 h/night) caused greater activity to food images in brain regions of reward processing (e.g nucleus accumbens and putamen) and sensory/motor signalling (i.e. right paracentral lobule) effects that are mostly seen in individuals with obesity[115]. Also, St-Onge and colleagues reported an increased neuronal activity in response to food stimuli after restricted sleep (4 h/night) compared with habitual sleep (9 h/night)[116]. In the second hypothesis, insufficient or low-quality sleep causes weaker neuronal reactivity in reward-related regions in response to food intake, thus more inspiring food rewards (i.e. delicious high-energy foods) may need to induce neurons in the reward system or alternatively more foods need to be consumed to compensate for the reduced reward response and induce good pleasure[113]. Mechanisms involved in both of these hypotheses result in overeating.

Areas of the brain that are affected by sleep deprivation and the manner of changes are subject to controversy in neuroimaging investigations. For instance, Greer *et al.* reported that one night sleep deprivation suppressed activity in the cortical regions involved in hedonic evaluation and augmented that in the amygdala, leading to increased desire for consumption of high-energy foods[107]. In contrast, Katsunuma *et al.* found that one night sleep deprivation (39 h wakefulness) activated the right anterior insula in response to food images but the activity of the amygdala remained unchanged although in optimal sleep condition both amygdala and anterior insula had low activity[117]. Likewise, six nights of sleep restriction increased neural activity in response to food stimuli in brain regions associated with

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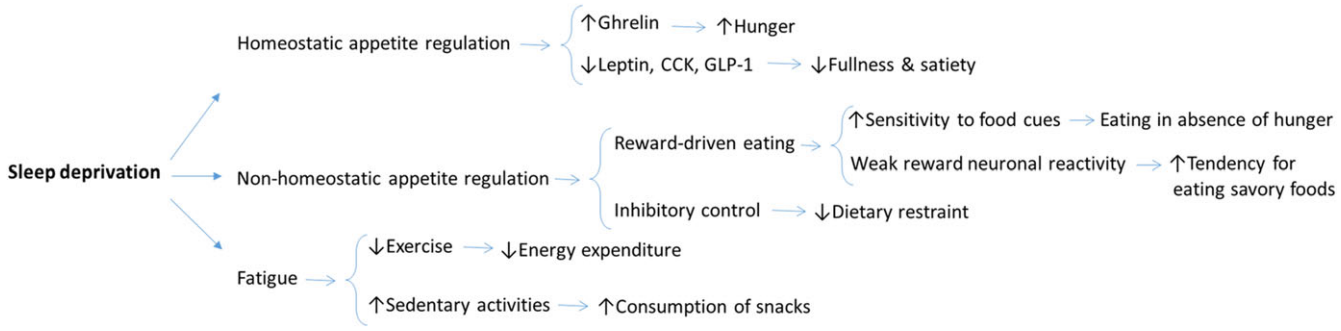

Fig. 2. Possible mechanisms by which sleep deprivation lead to overweight and obesity. CCK, cholecystokinin; GLP-1, glucagon-like peptide-1.

reward[116]. Also, Benedict *et al.* reported that one night sleep deprivation increased neural activation in the right anterior cingulate cortex in response to food images[118]. Although these studies seem contradictory at the first glance, an overview of their results suggests that sleep loss may modulate neural activity in at least one of cortical or subcortical regions. In the majority of the above examples, it was the cortical region (involved in liking) that increased its activity following sleep loss[116–118], but suppression of cortical neurons when accompanied with heightened subcortical neuron activity also associated with augmented food desirability[107].

Inhibitory control. In addition to homoeostatic and reward-driven regulation, as a third mechanism, sleep deprivation impairs inhibitory control over eating. Inhibitory control or dietary restraint is defined as the ability to resist a potent stimulus, such as a palatable food[119]. The interaction between food reward system and inhibitory control predicts food intake[120]. Hence, a strong food reward perception along with low levels of inhibitory control may lead to overeating. Acute sleep deprivation reduces the ability to exert cognitive inhibition toward food stimuli[121]. This low inhibitory control along with hormonal signals for hunger from the gut and/or increased motivation towards food stimuli from the reward system intensifies the risk of obesity. An overview of the possible mechanisms of sleep deprivation in the development of obesity has been given in Fig. 2.

Interaction of different systems. The hormonal, cognitive, emotional and behavioural systems that control feeding behaviour influence and interact with one another[122,123]. For instance, specific areas of the brain reward system, such as the hypothalamic arcuate nucleus, have receptors for ghrelin and leptin. Moreover, ghrelin levels have been positively correlated with increased sensitivity to dairy odour and higher intensity ratings to food odors including dairy and vanilla[124]. In addition, the homoeostatic regulation has a strong control overeating under normal circumstances, but in abnormal conditions like adiposity, depression or sleep deprivation, particularly when the person lives in an obesogenic environment with easy access to highly energetic and palatable foods, the homoeostatic control may be weakened[125]. Thus, the outcome of the interaction of these systems will finally determine the eating behaviour.

Physical activity in sleep deprivation

Weight gain in sleep deprivation seems to be partly due to decreased exercise and physical activity as a result of fatigue and tiredness[68]. In contrast, good sleep quality may predict increased physical activity through its positive effect on emotion regulation[126]. On the opposite side of exercise, sedentary activities may be increased during sleep deprivation[69,127–129]. Sedentary activities not only have low energy requirements but they may also promote consumption of high-energy snacks, further predisposing individuals to overweight. A hypothetical scheme for the possible effect of sleep deprivation on physical activity has been depicted in Fig. 3.

Nonetheless, controlled trials for the effect of sleep deprivation on physical activity are inconsistent and all cases of increased[73,130,131], decreased[68,127,132] or of no effect[69,133,134] in the amount and/or intensity of physical activity have been reported. These controversies may have resulted from diversity in study protocols. For instance, the observation of increased physical activity has been mostly in trials with one night of sleep deprivation[73,131] or two nights of fragmented sleep[130] where the severity of sleep deprivation was mild. Decreased physical activity has been reported during prolonged (14 d) sleep deprivation[127] or in studies where participants were not allowed to exercise and perform brisk physical activities[68]. Under normal living conditions (not laboratory) and *ad libitum* eating and free exercise, a moderated sleep curtailment may not largely affect physical activity[133,134]. The time of sleep restriction also seems to matter. For instance, in a trial on normal-weight men sleep restriction during the second half of night reduced physical activity more than sleep restriction during the first half[135], indicating that sleep during the second half of the night has a more important effect on feeling freshness during the next day. In agreement, McNeil *et al.* reported that participants with delayed sleep had more moderate-intensity physical activity than those with advanced wakeup time, although vigorous-intensity physical activity was greater following advanced wakeup[131].

Energy expenditure

Like physical activity, the effect of sleep deprivation on daily energy expenditure has not been completely elucidated and all modes of increased[66,136–138], decreased[139] and no effect[50,97,140–142] have been reported. There are many reasons

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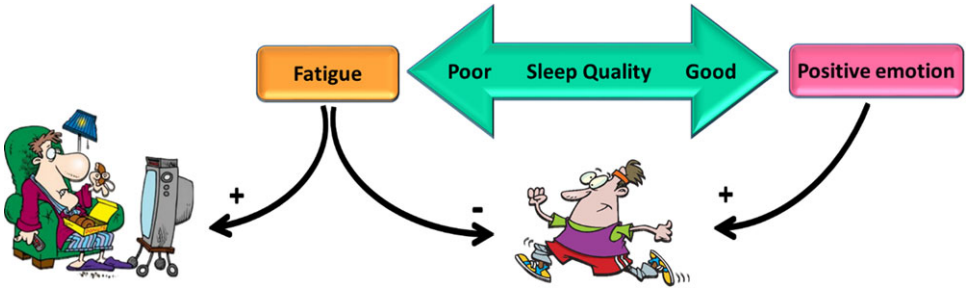

Fig. 3. Hypothetical scheme for the possible effect of poor sleep quality on physical activity.

for the controversies such as the difference in sleep deprivation protocol, very low sample size of some trials, the type of activities that were allowed during waking hours, and age, sex and BMI of participants. Studies largely differ in sleep deprivation protocol, such as total *v.* partial, moderate *v.* severe, and acute *v.* chronic sleep deprivation as well as the time of sleep deprivation. For the last item, depriving from deep or slow-wave sleep may have a different impact on energy expenditure than depriving from REM[143].

A positive effect of sleep deprivation on energy expenditure was mostly seen in investigations where subjects had to spend at least 3 d inside calorimetric units. Staying for several days inside these units may cause a sort of psychological stress in participants because of limitations that such units impose, for instance, in eating, exercise, daily routine activities and communication with people as desired[144]. Such kind of stress may have also occurred by prolonged (40 h) sleep deprivation[138]. Stressful conditions may increase cortisol and/or norepinephrine levels, augment heart rate[139] and subsequently increase metabolic rate[145]. However, investigations that give participants some sort of freedom for eating, physical activity, and doing daily activities did not find a significant increase in energy expenditure[50,97,141,142]. The extent of the increase is estimated to be ~ 4–5% equal to ~ 377–460 kJ/day[146]. This amount is not large enough to compensate the increased energy intake during waking hours. For this increased energy expenditure, carbohydrates have shown to be oxidised more than fats, thereby promoting fat accumulation and weight gain[130,147].

Basal metabolic rate

Few studies have examined the association of sleep deprivation with metabolic rate, and again findings are with controversy. In a cross-sectional study on obese individuals with short sleep duration, poor sleep quality was associated with increased resting energy expenditure[147]. The authors reasoned that secretion of cortisol and epinephrine during short sleep stress may be the cause of this increase. In contrast, in a cross-sectional study on children and adolescents, suboptimal sleep was associated with lower basal metabolic rate and physical activity and higher sedentary behaviours[129]. There are also cross-sectional studies that did not find a relationship between sleep deprivation and basal metabolic rate[148,149]. On the other hand, two RCT reported decreased metabolic rate following sleep deprivation. In one study, Nedeltcheva *et al.* reported that energy restriction with 5·5 h sleep/night for 14 d decreased resting metabolic rate and 24 h plasma epinephrine concentrations compared with energetic restriction with 8·5 h sleep/night[54]. In agreement, Spaeth *et al.* reported that 4 h sleep/night for five nights reduced resting metabolic rate and 12 h recovery sleep returned it to baseline levels[150].

According to what was stated in this and the previous two sections, there is still much work to be done to complete our understanding of the effect of sleep deprivation on physical activity, total and resting energy expenditure and thermic effect of food; for the latter, evidence is scarce.

Sex-specific effects

There is a large difference in the sleep and metabolic consequences of sleep deprivation between men and women. A global sleep dataset of 11·14 million nights from 69 650 non-shift workers aged 19–67 years from forty-seven countries showed that men tended to sleep less than women across the lifespan, but nighttime awakening was more prevalent in women, which was coincided with early to middle adulthood, i.e. a life stage associated with child-rearing[151]. The prevalence of evening chronotype is higher in men than women, meaning that men generally wake up later in the morning and go to sleep later at night than women[152]. This could be because the timing of the circadian rhythm of core body temperature and pineal melatonin secretion occurs earlier in women than men[153]. A study of intrinsic circadian rhythm in a group of men and women aged 18–74 years revealed that the intrinsic circadian period is shorter in women (24 h 5 min in women *v.* 24 h 11 min in men) and that a greater proportion of women have intrinsic circadian periods shorter than 24·0 h (35% *v.* 14%)[154].

Hormonal regulation of appetite in sleep restriction may also differ between sexes. Nymo *et al.* reported that while in men shorter habitual sleep duration and worse sleep quality was associated with increased basal and postprandial ghrelin, in women lower sleep quality was associated with decreased postprandial ghrelin[87]. Also, in a study by St-Onge *et al.*, sleep deprivation led to an increase in ghrelin in men and a decrease in glucagon-like peptide-1 in women without significant change in leptin levels[155]. In the same line, a cohort study on 655 children showed an association between chronic curtailed sleep and lower leptin levels at age 7 years in girls, but not boys[91]. In contrast, in another cohort of 502 adolescents shorter sleep was

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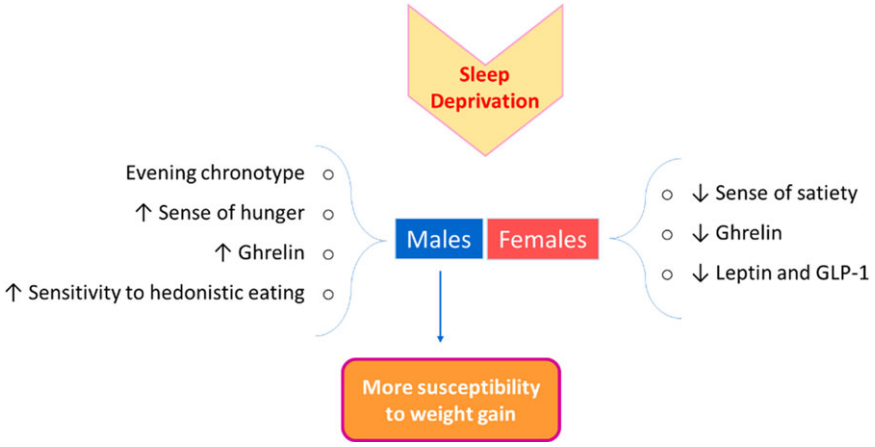

Fig. 4. The effect of sleep deprivation on males and females. GLP-1, glucagon like peptide-1.

associated with lower leptin in males[91]. Although somehow contradictory, the current findings mostly support the notion that the increased food consumption after sleep restriction may be caused by increased feeling of hunger in men and weakened satiety in women. Circadian misalignment has proved to reduce fullness in women and increase cravings for energy-dense and savory foods in men, suggesting that men may be more sensitive to hedonic regulation of food intake[156]. This diverse and sex-specific hormonal response to sleep restriction may partly justify the lack of sleep deprivation effect on appetite hormones in studies that have not separated their results by sex.

Men and women also show a different weight response to sleep deprivation; i.e. men appear more susceptibility to weight gain than women. For instance, in RCT with sleep restriction for five nights men gained more weight than females[66,157]. Also, a cross-sectional study in elderly aged 67–96 years showed that the risk of increased BMI in sleep durations <5 h was greater in men than women[158]. Cross-sectional investigations on children and adolescents have also shown an inverse correlation between sleep duration and BMI in boys but not girls[159,160]. The reason of this sex-specific effect of sleep deprivation on weight is not clear but hormonal fluctuations in women[161] and the dominant hedonic aspects of food selection in men[156] may partially explain it. For the latter, a large cross-sectional study on school-aged children in China showed that short sleep duration was associated with increased risk of more sugar-sweetened beverage intake among younger children and boys and less vegetable and fruit intake among older children and girls[162]. Also, in a trial after chronic sleep restriction, men showed a trend towards decreased implicit positive attitude for low-energy foods whereas women did not show a significant change[163]. A summary of the sex-specific effect of sleep deprivation is depicted in Fig. 4.

Influence of weight status

Neural response to sleep deprivation depends on weight status. Jensen *et al.* found that in sleep restriction, both normal-weight and overweight/obese adolescents showed higher activity in reward regions irrespective of their weight status[164]. However, while adolescents with normal-weight demonstrated concomitant increases in neural activity in brain regions associated with food reward and inhibitory control, adolescents who were overweight/obese showed only increases in brain areas related to food reward without a change in inhibition-related brain processing[164]. This suggests that in sleep restriction, individuals who are overweight/obese may be at a higher risk of food overconsumption compared with their normal-weight counterparts. Interestingly, in sufficient sleep conditions, adolescents with overweight/obesity had higher level of activity in inhibition-related regions than their normal-weight counterparts but in sleep restriction they showed a considerable decrease in neural activity of such regions[164]. Contrary to the results observed by Jensen *et al.*[164], Duraccio and colleagues[165] found that adolescents with normal weight demonstrated increased perception of food appeal following sleep restriction but adolescents with overweight/obesity consistently indicated high scores of food reward, which did not differ between habitual and restricted sleep conditions[165]. However, both groups showed poor scores of food-related inhibitory control task following sleep restriction[165]. These results suggest that sleep restriction increases the risk of weight gain in both normal-weight and overweight/obese adolescents but adolescents who were overweight/obese are at greater risk. A summary of the possible influence of weight status in homoeostatic regulation of appetite has been depicted in Fig. 5.

Impact of sleep deprivation on food selection

As stated above, a part of eating behaviours following sleep restriction is driven by reward-driven food intake, which results in "eating in the absence of hunger". Available evidence indicates that contribution of this reward-driven eating in the increased food intake is larger than hormonal factors even in adequate sleep but more importantly in sleep deprivation[99,118], reviewed in Alonso-Alonso *et al.*[166]. In this regard, a systematic review showed that insufficient sleep leads to increments in energy and fat intake, body weight, appetite, hunger, eating occasions and portion size without affecting leptin, ghrelin and cortisol concentrations, suggesting the dominance of

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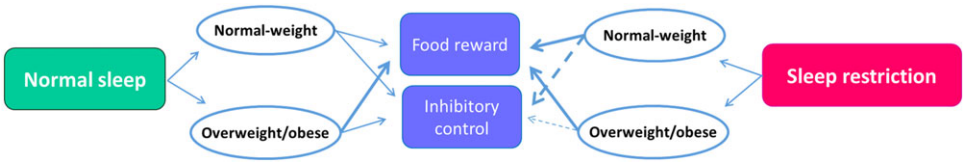

Fig. 5. Differential response to sleep restriction according to body weight. Both normal-weight individuals and individuals who are overweight/obese are assumed to stimulate the food reward system more strongly during sleep deprivation, but individuals who are overweight/obese indicate a weaker inhibitory control over eating compared with normal-weight individuals.

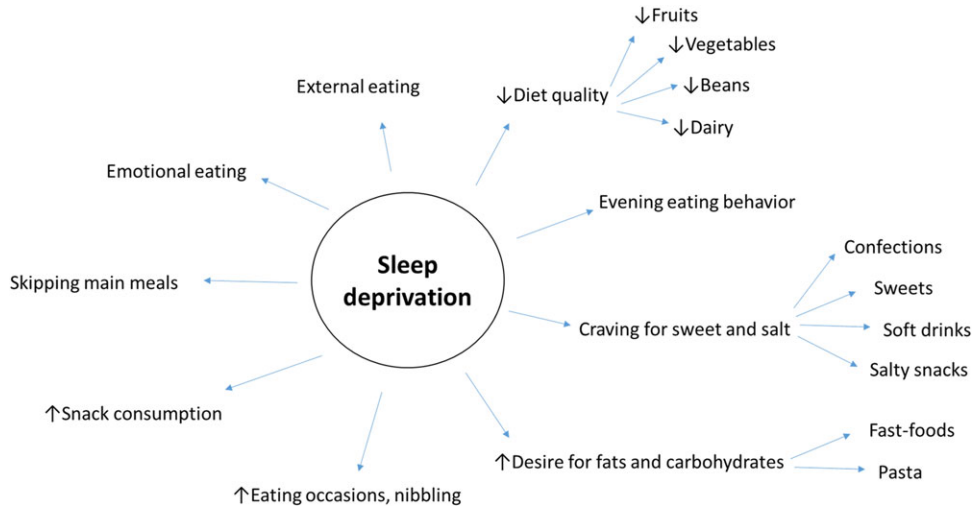

Fig. 6. Effect of sleep deprivation on eating behaviours and food selection.

non-homoeostatic mechanisms over the hormonal or homeostatic factors[61]. Also, in a cross-sectional study on normal-weight young women, scores of emotional eating (i.e. eating in response to aroused emotional states) and external eating (i.e. eating in the presence of food irrespective of hunger) were higher in women who had poor sleep quality (but not short sleep duration)[167]. Upon administration of snacks, participants who scored high on emotional eating and reported short sleep consumed higher amounts of snacks. Similarly, an RCT on normal-weight young men showed that in sated state (i.e. following breakfast), men who experienced one night total sleep deprivation chose larger snack portions, further indicating the predominance of reward-driven eating over homoeostatic signals[168]. Also, five nights of sleep restriction (6·5 h sleep/night) in adolescents did not affect their self-reported hunger or desire for non-sweet foods but pictures of sweet/dessert foods were more appealing for them[169].

Sleep deprived individuals usually look for and consume palatable foods, foods that are mostly unhealthy, i.e. energy-dense with high fat and/or high sugar content[70,170–173]. Craving for unhealthy foods may be due to alteration in taste function including sweet, salt and even umami or sour tastes[174]. Extended use of such unhealthy foods may disrupt appetite regulation by inducing addiction-like deficits in brain regions involved in the reward system, including the lateral hypothalamus, nucleus accumbens, ventral tegmental area, prefrontal cortex and amygdala[166,175]. In an RCT in normal-weight individuals, after five nights sleep restriction with 4 h sleep/night, greater activation in parts of the brain reward system was seen upon viewing unhealthy foods (pepperoni pizza, doughnuts, chocolate bars and candy) compared with healthy foods (carrots, yogurt, oatmeal and grapes) whereas such increased activity did not occur after normal sleep[176]. In another RCT, the increased brain connectivity from the dorsal anterior cingulate cortex to the bilateral anterior insula after one night total sleep deprivation correlated with increased fat and decreased carbohydrate intake in healthy adults[177]. A summary of the effect of sleep deprivation on food selection and eating behaviours is presented in Fig. 6.

Effect of sleep deprivation on diet quality

Compelling evidence from cross-sectional investigations indicates an inverse relationship between sleep deprivation and diet quality. This inverse association has been reported, for instance, in adolescents aged 10–16 years[178], postmenopausal women[64] and women with diverse age, race, and ethnicity[62] although postmenopausal women with ≥8 h sleep have also shown poor diet quality[64]. Furthermore, social jetlag, which is the difference in the average sleep duration between weekdays and weekends, has been associated with negative food choices even in cases of adequate weekly sleep duration[179]. Interestingly, weekend catch-up sleep did not prevent the adverse effect of short weekday sleep on unhealthy dietary choices[179,180].

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Sleep deprivation usually induces cravings for consumption of carbohydrates/starches, fast foods and sweets[52]. A cross-sectional study on resident physicians indicated that additional work was associated with higher intake of cereals, bread and pasta, and lower intake of fruits and beans, along with lower score of adapted healthy eating index[51]. Consistently, in motor freight workers, adequate sleep was associated with more healthful food choices[181]. In agreement, a systematic review of interventions that used sleep extension as a strategy to improve cardiometabolic health suggested that sleep extension may be associated with reductions in overall appetite, desire for sweets and salty foods, and increased percentage of daily energy intake from protein[182]. Although there are RCT denying the selection of high *v.* low energy foods under sleep deprivation states[86,183], most of trials agree with the effect of insufficient sleep on activation of the reward system and selection of high energy foods [52,157,167,169,176,177,184]. Unhealthy habits such as skipping breakfast[171], skipping main meals[70], and higher ingestion of snacks[70] have been reported in individuals with insufficient sleep.

Sleep insufficiency has also been associated with low diet quality in children and adolescents. Lower rate of fruit and vegetable consumption has been reported in sleep-deprived school-aged children in China[162], children and adolescents of Australia[185], Spanish children[186] and US adolescents[172]. In the same line, an analysis of data from 10 453 children from five European countries revealed that one additional hour of sleep was associated with increased consumption of fruits and vegetables[187]. Also, in a group of Danish 2–6-year-old children, those with more variable sleep duration consumed less fruit and vegetables and a higher amounts of added sugars and sugary beverages[63]. Also, in a cross-sectional study on 118 462 Korean adolescents, short sleep duration of <6 h was associated with higher intake of soft drinks and confectionaries, and poor sleep quality with 7–8 h sleep was associated with lower intake of fruits, vegetables and milk, and higher intake of soda, soft drinks, fast-food, instant noodle and confectionaries[188]. Given the importance of childhood habits in the formation of adulthood eating habits, results of these investigations remind the importance of childhood sleep in adulthood health status. Sleeping at earlier bedtimes may result in healthier eating behaviours such as eating low glycemic index foods, fruits and dairy in adolescents[189].

Desire for consumption of snacks

Both epidemiological and interventional investigations have shown increased consumption of snacks in sleep deprivation conditions. A large cross-sectional study showed that adults with habitual short sleep duration reported a lower mean percentage of energy intake from main meals but higher energy from snacks[70]. Likewise, a survey on high school students revealed an association between shorter sleep and more frequent cracker consumption[132]. In another cross-sectional study, university students with habitual short sleep ate more often, i.e. more than three meals per day and more frequent snacking, than those with long and sufficient sleep[190]. RCT have indicated the same line of evidence. For instance, two groups of investigators reported that during sleep curtailment in healthy volunteers, meal intake did not change but the amount or energy from snacks increased[97,168]. These findings have been confirmed by other investigators[97,173,191].

Craving for sweetness

Increased consumption of sugar-sweetened beverages and snacks after sleep deprivation may result from the preference for sweet taste under such conditions. Cross-sectional investigations have shown inverse associations between sugar intake and sleep duration[70,192], and an inverse association between sleep efficiency and sweet cravings[178]. RCT have also reported sweet craving in normal-weight young adults after three nights of sleep restriction (5 h sleep/night)[193] or non-obese adults after one night of sleep curtailment with 33% reduction in habitual sleep duration[194]. But, such alteration in sweet taste pleasantness or intensity was not observed after one night total sleep deprivation although feeling of hunger and ghrelin concentration increased[75]. It is likely that stress caused by total sleep deprivation decreases sensitivity to sweet taste[75] as animals with mild stress have shown attenuated sensitivity to food rewards[195].

Sweet liking leads to increased consumption of energy from sugar-sweetened snacks and beverages by sleep-deprived individuals[95,196], augments glycaemic load of the consumed diets[197,198], and enhances risk of obesity[199]. Moreover, sweetness sensitivity is associated with higher preference for sweets and other carbohydrate-dominated foods but lower preference for foods with high content of protein[200]. The low preference for protein is particularly important because proteins alter appetite regulating hormones in a direction that leads to increase satiety and decreased hunger[201]. Sweet sensitivity may also bring along a higher interest in high-fat food odours such as chocolate and peanuts[200], consumption of which may further intensify risk of weight gain.

Appealing macronutrients

The extra energy consumed during sleep deprivation is provided from carbohydrate and fat rather than protein[76]. Numerous cross-sectional and interventional investigations have shown increased fat[141,177,191,202–204] and carbohydrate[97,205] intake in sleep-deprived individuals. Consistently, increased sleep duration and quality following an exercise regimen in adolescent girls with obesity led to significant decreases in food energy density, fat, and sugar consumption[206]. Likewise, transitioning from an insufficient to adequate/recovery sleep schedule decreased energy intake, especially that from fats and carbohydrates[66]. However, there are also documents indicating the increase in fat intake and a decrease in carbohydrate consumption[177,191], suggesting that sleep deprivation may cause a greater desire for consumption of fats compared with non-sweet carbohydrates. In this regard, a cross-sectional study on adults with obesity showed associations between sleep apnea, as a sleep disorder, and a shift from carbohydrate to fat intake[207]. Accordingly, a

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systematic review of RCT showed increased consumption of fat without a change in protein and carbohydrate intake in sleep deprived conditions[61]. It seems that the type of fat is also important. A meta-analysis of nine cohort studies with 14 906 participants of European descent showed associations between sleep duration and lower saturated fatty acid intake in adults aged 20–64 years old and higher total fat and polyunsaturated fatty acids (PUFA), as well as lower carbohydrate intake in women aged 65–80 years[208]. Similarly, a cross-sectional study on Mexican American children showed associations between longer sleep duration and higher percentage of energy obtained from fats, especially from PUFA[209]. On the contrary, in an RCT on adults, sleep deprivation (4 h sleep for five nights) caused increases in the consumption of fats, notably saturated fats[141]. Based on these reports, in sleep deprivation, individuals crave foods high in fat, particularly saturated fats, while in sufficient sleep consumption of food sources of saturated fats seems less appealing.

Time of eating

Although, short-duration sleepers are reported to eat from the earliest time in the morning to the latest time of night[70], extra energy is mostly consumed during evening hours[31,35,97,157,198,210]. For instance, adolescents who experienced five nights with 6·5 h sleep indicated increases in the intake of energy, fat and carbohydrates after 9 pm but not earlier in the afternoon or morning[198]. Also, participants who experienced 4 h sleep/night for 5 d consumed additional energy between 10 pm and 4 am and consumed less energy the next morning[157]. Similarly, participants who had 5 h sleep/night for 5 d consumed a smaller breakfast but 42% more energy after dinner as snacks[31]. Interestingly, the energy consumed after dinner have been more than that consumed at individual meals[31,210]. The cause of tendency to late-night food intake is not known, but it is likely that changes in circadian sleep and awakening time affect eating patterns. For instance, the reduced desire to consume breakfast may be due to short sleep and waking up at a time when the internal circadian clock is still promoting sleep. Also, staying awake during the night may stimulate food consumption because melatonin levels are high during the night and promote sleep, and the pleasure of eating helps to better tolerate a stressful insomnia condition[31]. The additional energy consumed during late-night waking hours compensates for the extra energy needed to stay awake for the extra hours[210]. However, this additional energy may exceed energy needs and thus contribute to weight gain and obesity. In this regard, night eating syndrome is more frequently seen in individuals with obesity[211,212]. Animal studies have shown that when mice eat food at times when they normally sleep, they gain weight even though they have their regular energy intake[213].

The effect of diet on sleep

In previous sections the effect of sleep on dietary intakes was discussed but the literature also contains documents claiming the influence of diet and eating behaviours on sleep health.

Eating habits

A number of investigations have claimed adverse effect of eating habits on sleep. A cross-sectional study on healthy adults indicated that food intake near the sleeping time was associated with poor sleep quality[214]. Also, Gwin and Leidy reported that when compared with skipping breakfast, consuming breakfast resulted in shorter total sleep time but tended to improve perceived sleep quality and sleep onset[215]. Likewise, a survey on grade 5 children in Canada, unhealthy eating habits (e.g. eating fried foods, eating fast food, eating supper alone, eating supper in front of the TV and eating ready-made dinners) were inversely associated with sleep duration and quality[216]. Snacking between meals and after supper was also negatively associated with sleep quality.

Diet quality

Epidemiological investigations have shown that consumption of unhealthy foods may predispose individuals to poor sleep quality. A population-based survey on Brazilian adults during the coronavirus disease 2019 pandemic showed that higher consumption of ultra-processed foods and lower consumption of fresh and minimally processed foods was associated with a higher chance of poor sleep quality[217]. Another survey on adolescents of the same country revealed an association between consumption of ultra-processed foods and anxiety-induced sleep disturbance[218,219]. Also diets with low fibre and high saturated fats and sugar intake was associated with lighter and less restorative sleep with more arousals in normal-weight adults with habitual sleep[220]. In contrast, in a sample of college students in the USA, higher frequency of healthy protein and healthy dairy food consumption reduced the odds of poor sleep quality, while higher consumption of empty calories predicted poor sleep quality[221]. Similarly, in a sample of Japanese college students, the likelihood of poor sleep quality for individuals in the middle and highest tertile of the Food Guide (i.e. well-balanced dietary intakes of meats, fish, eggs, soy products, dairy and fruits) was half of that in the lowest tertile[222]. Fish, eggs, nuts, seeds, and to a lesser extent cereals, legumes, seeds and a number of fruits such as kiwi fruit and tart cherries are food sources of melatonin and on consumption can increase serum melatonin levels[223,224].

Consistent with the positive effect of diet quality on sleep, a large body of evidence indicates adverse effect of food insecurity on sleep outcomes, e.g.[225–229]. For instance, a population-based sample of 10 901 US adults, marginally food secure, low food secure and very low food secure participants, were more likely to report sleep complaints than their fully food secure counterparts[230]. Also, a meta-analysis of cross-sectional studies showed associations between food insecurity and increased risk of sleep disorders[231]. Food insecurity has also been associated with trouble falling and staying asleep[232]. Sleep quality and quantity may mediate the relationship between food insecurity and obesity[233].

Dietary carbohydrates

Investigations claiming the effect of carbohydrates on diet are contradictory and some are with cross-sectional design and thus

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cannot conclusively indicate the cause and effect relationship. A cross-sectional study on 9239 Chinese adults showed associations between higher intake of carbohydrate and longer sleep duration[234]. In agreement, in a study on 4435 Japanese adults, low carbohydrate intake (<50% *v.* ≥50% of total energy) was associated with difficulty in maintaining sleep[235]. Also, consumption of a high-glycaemic index meals shortened sleep onset latency in healthy sleepers compared with a low-glycaemic index meal although it was less effective when consumed 1 h before sleep[236].

Nonetheless, there are investigations from both RCT and cross-sectional type that have reported benefits of a low carbohydrate diet on sleep. For instance, two cross-sectional investigations demonstrated that low carbohydrate diet was associated with better sleep quality in Iranian women[237,238]. Also, a systematic review of RCTs showed that lower carbohydrate intakes lengthen the proportion and duration of N3 stage sleep (referred to as slow wave sleep or deep sleep), while higher carbohydrate ingestions prolong REM stage sleep[239]. Surveys also point to an adverse effect of simple carbohydrates on sleep quality. A survey on Korean adolescents showed a direct relationship of energy drink intake with sleep dissatisfaction and feeling of fatigue[240]. Likewise, a survey on 100 648 Brazilian adolescents suggested that ingestion of sweets and other confectionaries and soda drinks increases the risk of anxiety-induced sleep disturbance[218].

The controversies may be resolved by considering the type of carbohydrates; benefits of carbohydrates are seen with complex and low-glycaemic index carbohydrates while adverse effects are more related to simple and high-glyacemic index carbohydrates. In this context, cross-sectional and longitudinal analyses of a prospective cohort study with 77 860 post-menopausal women showed that higher dietary glycaemic index (dietary added sugars, starch and refined grains) was associated with increasing odds of prevalent and incident insomnia. By contrast, higher intakes of dietary fiber, whole grains, non-juice fruit and vegetables were associated with lower odds of prevalent insomnia[241]. High dietary glycaemic load triggers hyperinsulinaemia and subsequently hypoglycaemia, resulting in secretion of counter-regulatory hormones such as adrenaline and cortisol which cause symptoms like heart palpitations, tremors and irritability that lead to awakening from sleep[241].

Dietary fats

In agreement with studies that have shown benefits of low carbohydrate diet on sleep quality, Bennett *et al.* reported that higher intake of mono-unsaturated fats if substituted for carbohydrate may protect against poor sleep quality in pregnant women[242]. However, Cao *et al.* in a cohort study with five follow-ups on Chinese adults reported that association between fat intake and sleep depends on the time of fat intake; high-fat breakfast was associated with less daytime sleep, which may mean feeling more refreshed during the day, but a high-fat dinner was associated with persistent short sleep[243].

Dietary protein

There is evidence of a protective effect of dietary protein on sleep. A cross-sectional study in Japanese adults indicated that regular exercise combined with high protein intake contributes to good sleep quality[244]. Likewise, in an RCT on adults who are overweight and obese, a greater proportion of protein in an energy-restricted diet improved sleep quality[245]. In agreement, a cross-sectional analysis of 4435 non-shift workers in Japan showed that low-protein intake (<16% *v.* ≥16% of total energy) was associated with difficulty in initiating sleep and poor sleep quality, although high-protein intake (≥19% *v.* <19% of total energy) was associated with difficulty in maintaining sleep[235].

Other effective components

Numerous components in healthy diets may render sleep-promoting properties. An RCT on adults with obesity demonstrated that adults who are obese and poor sleepers may become good sleepers when they consume either the recommended or a higher protein energy-restricted healthy eating pattern[246]. Animal and plant protein in healthy diets provides tryptophan which is an essential amino acid and the precursor of serotonin and melatonin, both are known to play roles in sleep-wake cycle[247]. Evening consumption of tryptophan caused a 130% increase in plasma tryptophan to large neutral amino acids ratio and reduced sleepiness and improved brain-sustained attention processes the following morning[248]. In agreement, a recent meta-analysis of 18 RCT showed that tryptophan supplementation, especially at ≥1 g, can help improve sleep quality[249].

Food sources of protein are good sources of zinc. Numerous studies in both cross-sectional and interventional designs have indicated the effect of zinc on improving the duration and quality of sleep and possible mechanisms of action have been suggested[250]. Fruit and vegetables provide great quantities of magnesium. Observational studies have suggested an association between higher magnesium intake and sleep quality and RCT have shown a positive effect of magnesium supplementation on sleep quality, sleep efficiency and sleep time[251]. Fibre also reduces stage 1 (the first 1–5 min of sleep) and increases slow wave sleep (deep sleep) in individuals with habitually normal sleep[220]. Trials have also documented benefits of pro-, pre- and post-biotics on sleep length and quality[252]. Animal studies have shown that short-chain fatty acids, particularly butyrate, promote sleep[253].

In national surveys in the USA, short (<6 h) and long (>9 h) sleepers indicated lower variety in their diet compared to normal sleepers[254]. Also, short sleepers consumed less vitamin C, lutein, zeaxanthin and selenium[254]. Whether low consumption of antioxidants impairs sleep is not known but sleep deprivation has been associated with increased oxidative stress and reduced serum antioxidant capacity[255,256] and sleep is supposed to restore antioxidant potential of the body[256,257]. Cross-sectional investigations have also shown inverse associations between serum folate, vitamin D and sleep disturbance and a positive association between total carotenoids concentrations and short sleep duration, although an inverse association was also observed between serum vitamin B_{12} levels and sleep duration[258]. Regarding vitamin B_{12}, a cross-sectional study on Chinese patients with type 2 diabetes showed associations between elevated serum vitamin B_{12} level and incidence of insomnia[259]. However, evidence also suggests improvement

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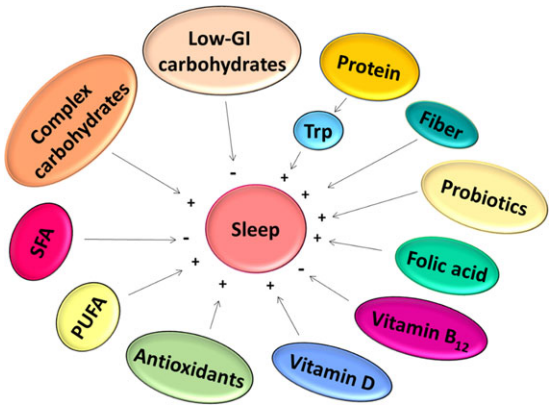

Fig. 7. Nutrients with possible influence on sleep health. GI, glycemic index; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids; Trp, tryptophan.

in daytime alertness, concentration and in shortening and improving sleep following vitamin B_{12} supplementation, likely through its effect on melatonin[260]. The literature for the effects of nutrients on sleep is largely incomplete and somehow contradictory, and many more investigations aimed at exploring the potential of food components on sleep duration and quality should be conducted in the future. A brief overview of the current findings is given in Fig. 7.

Conclusions

Sleep deprivation, which is caused by reduction in the quantity or quality of sleep, disrupts hormonal and neural regulation of appetite, leading to increased sensation of hunger, decreased satiety and intensification of the desire to eat food even in the absence of hunger. Feeling less refreshed during the day may end in lowering physical activity and a willingness to perform sedentary activities, the latter is generally accompanied with consuming high-energy snacks, further increasing obesity risk. Living in an obesogenic environment with plenty of palatable energy-dense foods available, having less time and motivation for doing regular exercise, and being exposed to mental pressures such as stress and depression all act in a consortium with sleep deprivation to push individuals towards obesity. Therefore, sufficient and good quality sleep is necessary for obesity prevention and should be a part of any weight-loss programme. Little data on weekend catch-up sleep have not yielded promising results, but more research should focus on this topic. Whether daytime sleep can compensate nocturnal short sleep also remains to be determined in future. As discussed during the previous sections, much still needs to be done to complete our knowledge of the consequences of different intensities and duration of sleep deprivation on appetite and desire to eat, energy intake and expenditure and physical and sedentary activities, and about the involved mechanisms. The current literature shows an interaction between sleep deprivation and diet quality, which is also largely unknown and is the subject of ongoing research. Moreover, current data have mainly been obtained from cross-sectional investigations and cannot suggest causal relationships. Well-designed RCT with longer periods of sleep deprivation while considering age, sex and weight status effects are needed.

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Authorship

The idea of the work was from M.A., but A.A. contributed with his valuable search to find relevant citations. Both the authors collaborated in the preparation of the manuscript.

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