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Time for Bed: Diet, sleep, and obesity in children and adults.

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Abstract

Sufficient sleep is necessary for optimal health, daytime performance and wellbeing and the amount required is age-dependent and decreases across the lifespan. Sleep duration is usually affected by age and several different cultural, social, psychological, behavioural, pathophysiological, and environmental factors. This review considers how much sleep children and adults need, why this is important, what the consequences are of insufficient sleep and how we can improve sleep. A lack of the recommended amount of sleep for a given age group has been shown to be associated with detrimental effects on health including effects on metabolism, endocrine function, immune function, and haemostatic pathways. Obesity has increased worldwide in the last few decades and the World Health Organization has now declared it a global epidemic. A lack of sleep is associated with an increased risk of obesity in children and adults, which may lead to future poor health outcomes. Data from studies in both children and adults suggests that the relationship between sleep and obesity may be mediated by several different mechanisms including alterations in appetite and satiety, sleep timing, circadian rhythm, and energy balance. Moreover, there is evidence to suggest that improvements in sleep, in both children and adults, can be beneficial for weight management and diet and certain foods might be important to promote sleep. In conclusion this review demonstrates that there is a wide body of evidence to suggest that sleep and obesity are causally related and recommends that further research is required to inform policy, and societal change.

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Introduction

Sufficient sleep is necessary for optimal daytime performance and wellbeing. Sleep has many diverse functions including in important role in fighting and recovery from infection, brain development and cognitive performance including alertness and memory as well as in mood, emotion, and behaviour regulation. The amount of sleep we need varies with age, but evidence suggests that there are several different social, environmental, and health-related factors reasons as to why both children and adults may not obtain sufficient sleep[1]. This may have major effects on both mental and physical health. Children and adolescents who have insufficient sleep may have problems paying attention, may misbehave, and they may feel angry or impulsive or have mood swings and their performance may be impaired[2]. And a lack of sleep has major effects on driving performance[3].

Sleep deprivation also has major effects on metabolism, endocrine function, and immune and haemostatic pathways and is associated with increased reporting of fatigue, tiredness, and excessive daytime sleepiness[4]. Too little sleep in adults is also associated with adverse health outcomes, including total mortality[5], stroke and coronary heart disease[6], type 2 diabetes, hypertension[7,8] and obesity[9].

This review specifically considers what happens to our risk of developing obesity when we don't get enough sleep. This is of particular importance as there has been a Worldwide increase in the prevalence of obesity with the World Health Organization recognising it a global epidemic[10]. Recent evidence from prospective longitudinal studies suggests that in children, short sleep may precede the development of overweight or obesity[11]. This is turn may lead to future poor health and increased risk of developing cardiovascular and other diseases including the sleep disorder Obstructive Sleep Apnoea (OSA) which itself can lead to increased risk of obesity[12]. This is of particular concern given the increased risk of poor Covid-19 outcomes associated with obesity[13,14] and with obesity related conditions such as OSA[15-17].

There are many possible underling mechanisms, including alterations in appetite and appetite and satiety hormones[18,19], circadian rhythm, sleep timing and regularity[20,21] and energy balance[22-24], that could be responsible for the association between sleep and obesity. Furthermore, there is some evidence to suggest that sleep extension may have beneficial metabolic effects[25] .This suggests that sleep may be of importance for metabolic and weight balance. It is possible that there may also be a bi-directional relationship between sleep and

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diet and diet and sleep which needs to be considered, along with ways in which we may be able to improve our sleep daily-work life/ school life balance, including improving shift work rotas, evaluating school start times, and eating foods that might help promote sleep. Given that there is a growing body of evidence that suggest that sleep and obesity are causally related this review will consider what has been done to date and what more needs to be done with regards to sleep research, policy and societal change[26-27].

How much sleep do we need?

Whilst it is accepted that sleep need varies considerably with age and that babies and children need more sleep there is a large variability in reported sleeping times within the different age groups. The National Sleep foundation suggests that toddlers might require 11-14 hours, school children aged 6-13years might require 9-11 hours whereas teenagers might require 8-10 hours and that this decreases across the age groups to around 7-9 hours in adults[28].

Sleep and Obesity

An elevated body mass index (BMI) is a major risk factor for heart disease, stroke, type 2 diabetes, and other chronic diseases. Overweight individuals are defined as having a BMI of 25–30 kg/m^2, and obese individuals having a BMI >30 kg/m^2[29]. Increasing evidence supports a link between short sleep and the development of obesity. Our initial meta-analyses of cross-sectional studies demonstrated that sleep was associated with obesity in both children and adults[9] and our more recent meta-analyses of prospective studies, indicates that "short sleep" which was defined by age proceeds subsequent weight gain and obesity in infants, children and adolescents[11], which suggests a causal relationship. Furthermore, similar results were reported in the study of Poorolajal et al in children and adolescents aged 5-19 years[30], in Chinese children and adolescents[31], and in preschool children[32].

Sleep disorders and Obesity

Obstructive Sleep Apnoea (OSA)

Sleep-related breathing disorders, such as OSA, are contributing factors for the development of cardiovascular disease (CVD)[33]. In OSA, during sleep there is a repetitive interruption of

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ventilation caused by collapse or partial occlusion of the airway. Obesity is a risk factor for the development of OSA as fat deposits around the neck can increase the occlusion. OSA is associated with sleep loss, and it is proposed that this may in turn lead to further metabolic and hormonal changes leading to increased appetite and further weight gain thus potentiating a vicious bi-directional pathway[12]. The onset of CVD in the context of obesity and OSA begins early in childhood and better recognition of obesity and OSA in children is of paramount importance to clinicians[34]. Whilst Marin et al., showed that men with severe OSA benefited from continuous positive airway pressure (CPAP) treatment and had a reduction in cardiovascular events[35]. In a more recent randomised control trial CPAP therapy didn't result in a statistically significant reduction in the incidence cardiovascular events in patients with OSA[36].

Mechanisms

The growing body of evidence suggests that sleep and obesity are causally related and there are several lines of evidence to suggest plausible mechanisms which will be briefly considered.

Appetite control Leptin and ghrelin

Short sleep appears to influence various hormonal responses which may lead to dysregulation of appetite control, affecting both huger, satiety and appetite control[19,37] which would increase appetite. In a randomised cross over trial conducted in healthy young men, Spiegel et al demonstrated that acute sleep deprivation was associated with a decrease in the satiety hormone leptin and an increase in the hunger hormone ghrelin[37]. Furthermore, although caloric intake was maintained by a glucose infusion it was observed that the change in the ghrelin-to-leptin ratio was associated with an increase in hunger. Likewise, in a study of 1,024 volunteers aged 30–60 y from the Wisconsin Sleep Cohort Study, it was found that in individuals who slept less than 8 h (74.4% of the sample), the observed increased in BMI was proportional to the reduction in usual sleep levels. Short sleep was associated with also associated with lower leptin and higher ghrelin levels[19]. On a long-term basis these changes in hormone levels and associated increase in appetite could be associated with increased caloric intake and obesity and disruption in energy balance.

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Sleep Timing, Eating Patterns, and Obesity

Studies have shown that eating patterns are affected by sleep. In one study of 27,983 women aged 35 to 74 years of age. It was shown that eating patterns among women varied with sleep duration, they also found that the tendency for eating during conventional eating hours (between breakfast and dinner) and that eating snacks was dominant in short sleepers with an increased intake of fat and sweets and a decreased intake of fruit and vegetables[38]. In the U.S., a study of 8,550 preschool-aged children also found that those who were exposed to three household routines of regularly eating the evening meal as a family, obtaining adequate night-time sleep, and having limited screen-viewing time had a lower prevalence of obesity (~40%) than those children who were not exposed to such routines[39]. In a study of 308 community-recruited children (age 4–10 years), sleep duration in obese children was shorter and showed more variability on weekends compared with schooldays. Sleep variability on schooldays was also positively associated with triglyceride levels in the obese children[40]. In a more recent study by Simon et al it has been shown that an earlier bedtime was associated with lower BMIz scores, lower intake of added sugars and lower sweet/ dessert food servings in pre-school children[41].

A functional magnetic resonance imaging (fMRI) study has shown that acute sleep deprivation enhances the brain's response to hedonic food stimuli[42]. Total sleep deprivation with sleep was associated with an increased activation in the right anterior cingulate cortex in response to food images, which was independent of calorie content and pre-scan hunger ratings. These results suggest that sleep loss enhances hedonic stimulus processing in the brain underlying the drive to consume food.

Circadian clock shift work and energy balance

Many of the biological processes run on an approximate 24-hour cycle that is controlled by the master clock located in the suprachiasmatic nuclei in the brain. It in turn synchronises clocks located in individual tissues in the body. It is directly influenced by environmental clues, the most important one being light, and allows the circadian rhythm to be aligned with the night and day light cycle. The sleep-wake cycle is one such circadian rhythm. Although according to the two-process model of sleep regulation which was first proposed in 1982[43] this (known as Process C) is only part of the sleep regulatory process. This model suggests that one's drive to sleep is governed not only by the regular 24-hour circadian pacemaker but

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also by the homeostatic pressure (Process S) which predicts the propensity to sleep. This increases the longer one has been awake and is determined by the amount of slow-wave activity (stage N3) during sleep. These processes are particularly important in shift workers who may be required to sleep at a time when their homeostatic pressure is low and hence have difficulty in initiating or maintaining sleep or need to stay awake when the homeostatic pressure is high. The circadian pacemaker also governs the timing of many behavioural, physiological, and metabolic processes for example the 24-hour variation in leptin, glucose, and insulin levels[44].

Animal studies have suggested that sleep loss leads to changes in the circadian clock which alters metabolism, and affects energy stores, but these effects could also be the result of the result of a stress response rather than the effect of sleep loss per se. In shift workers circadian desynchronization occurs as the individuals consume food and sleep out of phase with the normal clocks. This has adverse metabolic effects and has effects on glucose control and energy balance which are thought in part to be responsible for the observed increase in diabetes, obesity, and cardiovascular events in shift workers[45-46]. Adverse alterations in the oscillating clock genes, within human adipocytes, have also been associated with obesity[47]. A study in Japanese male workers has also shown that in shift workers, those individuals who reported sleeping ≤5 hours had an increased relative risk of obesity as compared to those who slept 5–7 hours. But, by contrast, there was no significant increase in short sleeping men who were not shift workers[48]. A more recent study suggests that sleep regularity is an even stronger predictor of all-cause mortality than sleep duration[21].

Chronotype

There is growing evidence to suggest that evening chronotype individuals have a higher risk of obesity and a worse metabolic profile[49]. Furthermore, it has been suggested that this may in part be a result of food preference. In a more recent study of teenagers, it was found that those individuals reporting later sleep timing were more likely to consume sugary/caffeinated beverages and high-energy-dense, nutrient-poor foods and to consume more food into the throughout the day and into the nighttime hours compared with their early sleep timing peers[50]. Circadian rhythms change around puberty, probably due to hormonal changes. Adolescents display a delayed circadian sleep phase (evening type) with a preference for going to bed late and getting uplater than adults. The sleep phase delay can be further

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exacerbated by late-night socializing and entertainment, or regularly studying late and the resultant reduction in required sleep is likely to cause difficulty waking up in the morning for school, college, or work.

Genes and genetic studies

There are several genes that have been identified that influence our circadian rhythm and the timing of sleep including the 'clock' genes *Per*, *tim*, and *Cry* that influence our circadian rhythms and the timing of sleep. The expression of genes may also change depending on whether someone is in the awake or sleep state.

Animal studies using clock gene mutant mice have shown that homozygous C57BL/6J clock mice are obese and hyperphagic, have a greatly attenuated diurnal feeding rhythm, and develop metabolic complications, including hyperglycemia, hyperlipidemia, and hyperleptinemia[51]. Whilst these results are far from conclusive, they provide sufficient evidence to warrant further investigations into the relationship between the disruption of the circadian sleep rhythm and the risk of metabolic diseases.

A more recent study has used two-sample Mendelian randomization to assess the association of genetically predicted sleep traits with adiposity and vice versa to evaluate the direction of any potential causal effect[52]. This study constructed genetic instruments for all adult adiposity traits from data from the Genetic Investigation of ANthropometric Traits (GIANT) consortia, a meta-analysis of ~59 studies from across the UK and Europe[53] and combined those for child-BMI generated from the Early Growth Genetics (EGG) consortia, a meta-analysis of ~20 studies from across the UK and Europe[54]. It provides robust evidence to suggest that there is a robust casual evidence for both unidirectional and bidirectional relationships between sleep and adiposity. They suggest that poor sleep and weight gain may contribute to a feedback loop that is detrimental to the overall health of the individual. For example, they were able to demonstrate that insomnia symptoms increased mean waist circumference (WC), BMI, and waist hip ration (WHR). They also demonstrated that higher hip circumference, WC, and adult BMI increased odds of daytime sleepiness. It was noted that genetic epidemiological studies of this kind include a disproportionate representation of individuals of European ancestry and further studies are required that include samples from individuals with a variety of ancestries[52].

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Other factors

Whereas homeostatic appetite circuits located in the hypothalamus can affect motivating behaviours like food seeking and selection through hormones such as leptin and ghrelin' non-homeostatic hedonic factors, hallmarked by pleasure and reward signalling, may also regulate appetite and favour the consumption of energy-dense food[55].

There are several neurotransmitters that are involved in the sleep wake cycle including those which 'switch off' or decrease the activity of cells important for arousal or relaxation. One important transmitter GABA is associated with sleep, muscle relaxation, and sedation. Whereas norepinephrine and orexin (also called hypocretin) and adenosine are important in keeping some parts of the brain active when we are awake. The extracellular level of adenosine, which acts on the forebrain increase during periods of wakefulness and dissipate during sleep. These increased levels appear to be associated with an increased level in the pressure to sleep.

Two important sleep hormones are cortisol which is sometimes known as the 'stress hormone and melatonin the 'sleep hormone. Cortisol release raises sugar levels and blood pressure. The levels increase in early hours to prepare for wakefulness and then decline during the day. Whereas melatonin which has been shown to synchronize the circadian rhythms is made in the pineal gland and promotes relaxation and drowsiness ready for sleep. Its production is light sensitive. Darkness prompts the pineal gland to start producing melatonin while light causes that production to stop. Melatonin helps improve the onset, duration, and quality of sleep. Generally, as melatonin levels increase, cortisol levels decrease and vice-versa. Hence high cortisol will be associated with low melatonin and less propensity to sleep.

Melatonin is produced in the body from its amino acid precursor tryptophan this is initially hydroxylated to 5-hydroxytryptophan (5HT) and then decarboxylated to form serotonin before being converted into melatonin (see Figure 1). Several cofactors are needed to convert tryptophan to melatonin, but these can be obtained from a healthy diet. For example, B vitamins are found in tuna, lentils and avocados, magnesium in whole grains, salmon and green leafy vegetables, calcium from broccoli and dairy products and vitamin C from bell peppers and oranges.

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Other important neurotransmitters include acetylcholine, histamine, adrenaline and the endocannabinoids, which are endogenous lipid-based neurotransmitters. The latter regulate a variety of central nervous system processes including appetite and are affected by sleep restriction[56].

Sleep and Weight Loss

A study conducted in 10 healthy adults has suggested that insufficient sleep may influence what kind of tissue is lost on a calorie-controlled diet and may undermine a person's ability to lose weight as fat[57]. In this randomized two-period two-condition crossover study, individuals who were either overweight or obese and who reported sleeping 6.5–8.5 hrs per night were randomly assigned to first sleep either 5.5 or 8.5 h each night in conjunction with moderate caloric restriction. The study was then repeated for the other sleep time. The individuals were monitored in a closed clinical research environment. The results showed that when the lost around 3kg in weight for both time periods. But when they slept for 8.5 h they lost most of their weight as fat mass but when sleeping 5.5 h they lost mainly fat-free mass. The individuals also reported an increase in appetite when they slept only 5.5hrs. The study suggests that sleep restriction may affect an individual's ability to lose metabolically active fat mass.

Lifestyle intervention programmes

Sleeping less potentially gives adults and children more time to eat and to engage in other sedentary activities, as exemplified by children and adolescents who like to stay up late to play on their computer or watch TV or to interact with social networks whilst snacking[58]. More opportunities to eat energy dense foods and concomitant tiredness may lead us to less engagement in physical activity. Activation of inflammatory pathways by short sleep may be implicated in the development of obesity[59] and it can up- and down-regulate the expression of genes involved in oxidative stress and metabolism[60]. Finally, insufficient sleep is associated with alterations in mood, attention, impulse control, motivation, and judgment, all of these factors could potentially influence eating behaviours, energy intake, and ultimately BMI in children[61]. In adults, short-term, acute, laboratory, and cross-sectional observational studies indicate that adverse changes in sleep are associated with adverse changes in insulin and glucose response but can be reversed when sleep quantity and quality are restored[62].

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Sleep Extension as a Treatment for Obesity

The sleep–obesity hypothesis is complicated by the possible bidirectional causality pathway and to date few studies have explored the possibility of sleep extension on weight loss. To address this Cizza et al outlined a protocol for a 'proof of concept' study to investigate whether sleep extension could be used as a treatment for obesity[63]. Participants in both the Intervention and Comparison Groups were encouraged to follow the current standard of care for both exercise (60–90 min of activity daily) and diet throughout the study. As of January 2010, it was reported that 109 adult participants had been randomized, 64 to the Intervention group and 45 to the Comparison group. The major limitations of this trial were the relatively small number of participants and the fact that it is not possible to blind the study. The interim analysis of the trial reported that global Pittsburgh sleep scores substantially improved in both groups but that the improvement was significantly more in the intervention Group ($p = 0.02$). This recent literature review however failed to find evidence of any follow-up data.

In a separate study Khatib et al assessed the feasibility of a personalized sleep extension protocol in adults aged 18–64 y who are habitually short sleepers (5 to < 7h)[64]. They found that the sleep extension group (n = 21) who received a behavioural consultation session targeting sleep hygiene significantly increased time in bed and sleep duration as compared to the control group (n = 21) who maintained habitual short sleep. Dietary intake was also determined, and it was found that the sleep extension group reduced intakes of fat (percentage), carbohydrates (grams), and free sugars (grams) in comparison to the control group[64]. There were no significant differences between the groups in markers of energy balance or cardiometabolic health.

An intervention program known as 'The Control, Evaluation, and Modification of Lifestyles in Obese Youth (CEMHaVi)' program aimed to address the observed rise in obesity in young individuals with a 1-year health-wellness program of physical activity and health education for obese youths. The intervention was designed to be enjoyable to improve adherence and consisted of a variety of game based 2-hour activity sessions offered once a week over 12 months, A health education program was offered. Measurements included height, weight, body mass index (BMI), academic performance, sleep habits, and health knowledge. Findings, from the study suggest significant improvements in quality and quantity of sleep ($P < .05$), and obesity ($P < .05$) in the treatment participants were compared with a group who

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chose not to participate[65].

In 2020 we conducted a systematic review and meta-analysis to determine whether interventions beneficial for sleep reduced weight gain in preschool children. We identified five intervention studies in pre-school that had measures of improved sleep duration and quality and prospective weight-related outcomes. Four of these reported improved outcomes for BMI and BMI z-score and demonstrated that improved sleep may be beneficially associated with a reduced weight gain in these children. There were however major limitations to the analysis in that the number of studies was small, the studies had different study designs and none had been specifically designed to look at the effect of sleep extension per se[32].

In a recent study Simon et al demonstrated that shorter sleep duration was significantly associated with diet and in particular sugar consumption in pre-school children[41]. The comprehensive review of 199 studies by Poorolajal et al[30], which included 1,636,049 eligible participants aged between 5 to 19 years, identified factors that protected against increased weight gain in children. These included eating breakfast and physical activity. Whereas, being breastfed for <4 months, watching too much TV, drinking sugar-sweetened beverages, and smoking had a significant detrimental effects on weight management[30]. Similarly in a recent cross-sectional study of children aged 5 to 12 years it has been shown that late sleep patterns, short sleep duration and greater sleep disturbances are significantly related to what and how children eat[66].

School Start Times

Adolescents often report spending longer in bed and later wake-up times on weekends compared with school days, which may reflect a both a sleep preference due to their circadian biology but also a need for 'recovery sleep' to compensate for the sleep debt accumulated on school days resulting from early wake up times. To address this, it has been proposed that schools should delay their start times and initial reports suggest that a delayed school start time is associated with the same bedtime but a delayed awakening time, resulting in more sleep[67]. However, a recent review of this subject has indicated that further research on this topic is recommended[68].

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Diet

As previously described, nutrition interventions that act on the neurotransmitters involved in sleep wake cycle could have different impacts sleep. For example, caffeine promotes wakefulness by blocking the receptors to adenosine[69], whereas a well-balanced diet that includes milk and dairy products is effective in improving sleep quality[70]. It is believed that this is because milk contains a high proportion of tryptophan from melatonin is synthesized[71] (see Figure 1). Tart cherries contain high concentrations of melatonin and have a positive effect on insomnia symptoms in the elderly[72].

Conclusion and policy intervention

The potential causal relationship between sleep and obesity is of importance although further studies, are still required in which robust measurements of sleep and potential confounding factors are determined and it remains to be seen if the mechanistic studies conducted in adults are applicable to children. Nevertheless, the effect of sleep deprivation needs to be considered from a societal and health prevention view. Societal pressure and changes in the physical environment may lead to a chronic curtailment of sleep leading to an increase in caloric intake and a reduction in energy expenditure and hence obesity. In turn obesity may lead to an increased risk of OSA related sleep disturbances and further increased risk of weight gain and poor health outcomes[73-75].

The findings from this review suggests that sleep may be an important and potentially modifiable risk factor (or marker) of future obesity and ensuing type 2 diabetes in early life. The studies outlined in this review are important in that they suggest ways in which future behavioural interventions for weight management might be enhanced with the inclusion of sleep education and management. There is evidence to suggest that maintaining good dietary habits and sleep may improve both sleep and health outcomes. Further randomised controlled clinical trials of the effects of sleep extension on the reduction of body weight in obese people are needed.

There is a need for a greater awareness of the importance of adequate sleep in children both for parents and for medical practitioners. Educational programs could be used to empower parents and children to improve sleep quality and maximize quantity.

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The importance of sleep for health, wellbeing, productivity, and safety has been outlined in the recent American Academy of Sleep Medicine position statement[76]. The statement also addresses the need for need for greater emphasis on sleep in education in school, colleges, medical schools, primary care, specialist care, health professionals and workplaces as well as the importance of sleep for in-patient and long-term care and for public health promotion. The need for further research is also addressed[76].

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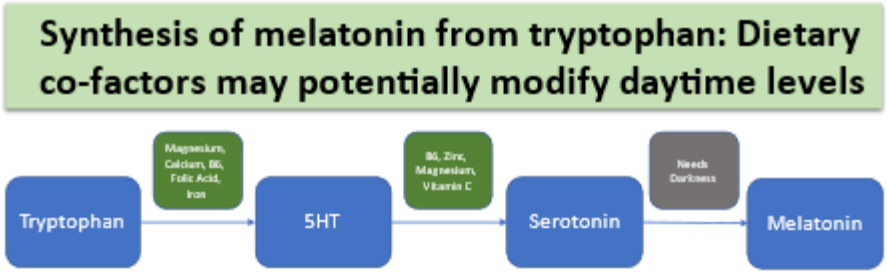

Nutrient	Examples of potential dietary sources
Magnesium	Whole grains, salmon, green leafy vegetables
Calcium	Broccoli, beans, dairy
B Vitamins	Tuna, lentils broccoli, avocados
Folic Acid	Dark green leafy vegetables, avocado, lentils, nuts
Iron	Dark chocolate, lentils, beef, chickpeas
Zinc	Spinach, shellfish, nuts, seeds
Vitamin C	Bell peppers, kiwifruit, oranges, broccoli, tomatoes

Figure 1

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