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Optimal sleep: a key element in maintaining a healthy bodyweight

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Abstract

As obesity rates rise globally, addressing modifiable lifestyle factors, such as sleep, presents an opportunity for public health interventions. This review explores the growing evidence linking sleep duration, quality and timing with weight management and dietary behaviours throughout the life course. Observational studies associate short or irregular sleep with increased obesity risk, poor diet quality and metabolic disturbances. Plausible mechanisms include decreased physical activity, heightened hedonic and/or emotional eating, dysregulated appetite signals and circadian misalignment of metabolism, which contribute to a positive energy balance. Unravelling the bidirectional relationship between sleep and weight is challenging; poor sleep exacerbates weight gain, while obesity-related comorbidities such as obstructive sleep apnoea further impair sleep. Despite promising evidence from sleep-restriction studies showing increased energy intake, long-term randomised controlled trials (RCTs) examining interventions designed to improve sleep with weight management as an outcome are lacking. A handful of short-term interventions suggest benefits in reducing energy intake or improving dietary quality, but their effects on weight loss remain inconclusive. This review calls for robust, well-powered RCTs that integrate sleep, diet and physical activity interventions to evaluate the potential of sleep as a core component of obesity prevention and treatment strategies. Currently, there is insufficient evidence to support sleep-focused interventions as a mandatory element in clinical weight-management programmes.

The search for weight-management solutions amidst an expanding global obesity pandemic is an ever-present challenge for humanity. The implications for comorbidity and economic burdens are grave without critical action to prevent this public health failure from getting any worse⁽¹⁾. Significant advances are being made in the pharmaceutical treatment of obesity⁽²⁾, but these are not widely available and not suitable for prevention of weight gain in most people^(3,4). Pharmaceutical treatments offer hope for management of obesity as a disease to prevent comorbidity and disability, but ultimately are not a solution for the societal failure to adequately address the increasing trend for gaining excess weight early in life. Although the root causes of the growing obesity epidemic are pervasive and complex, gaining a full understanding of how socioecological factors and health behaviours interact will provide a firmer theoretical footing for preventative public health interventions. As a small but significant component of this, sleep behaviours are gaining more recognition for their influence on weight management and diet quality⁽⁵⁾, with substantial evidence showing that self-reported estimates of usual sleep duration that fall short of recommended amounts (habitual short sleep duration) is associated with approximately a 40 % increased risk of obesity^(6,7).

Sleep is essential for normal physical, mental and social functioning. Adults should aim to sleep for 7–9 h every night⁽⁸⁾. Inadequate sleep increases the risk of mortality by 13 % and costs the UK economy an estimated £50 billion per year⁽⁹⁾. Sleep is a keystone modifiable factor in optimising a healthy lifespan. Healthy sleep habits support adherence to other fundamental building blocks of health such as a balanced, nutritious diet and regular physical activity, yet sleep behaviours have historically been undervalued as an essential target in lifestyle interventions for weight management. Sufficient good quality sleep is crucial for mental health, repairing damage, immunological responses to infection, cognitive development, physical growth and brain function⁽¹⁰⁾.

Parameters of healthy sleep behaviour and function fall under the domains of sleep duration, sleep quality, sleep timing and sleep regularity. Sleep duration in free-living study populations can be self-reported (for example, through sleep diaries or questionnaires that ask for the time of going to sleep and waking up) or objectively measured (most commonly using wearable actigraphy devices) as time in bed, sleep duration (sleep onset until final wake onset) or actual sleep duration (the same as sleep duration but discounting periods of being awake). Sleep quality can also be evaluated using actigraphy, routinely generating scores for the following: sleep latency, the time between 'lights out' and 'fell asleep'; sleep efficiency, expressed as sleep duration as a percentage of time in bed; sleep fragmentation, the number of awakenings as a proportion of

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sleep time; and wake after sleep onset, the duration of all wake periods between sleep onset and offset. Sleep quality is also often assessed as a global subjective score using the validated Pittsburgh Sleep Quality Index (PSQI) which ranges from 0–21 and is based on seven components including sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications and daytime dysfunction⁽¹¹⁾. Finally, either by subjective or objective methods, sleep timing can be evaluated using the times of sleep onset, offset and midpoint and sleep regularity can be investigated by analysing day-to-day variability in many of the above parameters: sleep duration, awakenings, sleep onset, offset and midpoint⁽¹²⁾.

Multiple parameters of good sleep function have been linked to weight management, including duration and quality of sleep and the regularity of sleep timings and durations from night-to-night or between workdays and non-workdays (13-16). Importantly, the relationship between obesity and sleep function is bidirectional. Obesity may exacerbate inadequate sleep duration and sleep quality mediated through the impact of comorbidities such as obstructive sleep apnoea⁽¹⁷⁾, type 2 diabetes symptoms⁽¹⁸⁾, gastroesophageal reflux disease(19), musculoskeletal pain(20) and mental health problems such as depression and anxiety(21). Accordingly, weight loss or prevention of weight gain may result in reduced daytime sleepiness(22), reduced obstructive sleep apnoea(23) and reduced sleep disturbance and sleep problems (24,25), although the evidence is mixed regarding effects on sleep duration/quality⁽²⁶⁾. However, evidence is accumulating that short, poor quality and/or irregular sleep precedes weight gain and therefore there is an opportunity to exploit the potential to modify sleep habits as an additional strategy in the prevention and treatment of overweight and obesity. This review presents an updated synthesis of established and emerging research on the role of optimising sleep duration, timing and quality in supporting lifestyle interventions for body weight management. The focus is specifically on nocturnal sleep in non-shift workers, with daytime sleep and napping beyond the scope of this appraisal^(27,28).

Mechanisms for the role of sleep in body weight management

The mechanisms mediating the effect of poor sleep on increased risk of weight gain have been extensively reviewed elsewhere (29-31). In summary, evidence suggests that short sleep duration leads to an increase in hedonic eating (leading to greater consumption of highly palatable energy-dense foods)^(32–34), an increase in emotional eating⁽³⁵⁾, decreased physical activity^(36,37) and dysregulation of appetite hormones (38-40). It is also likely that irregular sleep timings or social jetlag may lead to derailment of homeostatic systems that regulate appetite and reward⁽⁴¹⁻⁴³⁾. The discordance between sleep midpoint times across the work/non-work parts of the week known as 'social jetlag' is likely to disrupt normal hunger and satiety signalling due to circadian misalignment between internal biological clocks and the timing of external cues such as eating and sleeping⁽⁴⁴⁾. There is also emerging evidence that social jetlag is associated with unfavourable alterations to the gut microbiome that could be implicated in increased risk of weight gain⁽⁴⁵⁾.

Responsiveness to food (e.g. preference for highly palatable foods and greater responsiveness to external cues in eating) mediated the relationship between sleep disturbances and obesity in a cross-sectional analysis of data from 588 school-aged children in Spain⁽⁴⁶⁾. Similarly, higher disinhibition (a predisposition

towards consuming excessive palatable food in response to external food cues, particularly following triggers such as emotional stress) mediated the relationship between poorer sleep quality and higher BMI in a US adult population (n 602) with a mean age of 39 years⁽⁴⁷⁾. However, a cross-sectional study in 5900 European adults (mean age 52 years) found that workplace sedentary behaviour, but not dietary behaviours such as sugarsweetened beverages or fast-food consumption, mediated the relationship between sleep duration and obesity⁽⁴⁸⁾. Mediation analyses of this nature in cross-sectional studies, where lifestyle factors are putative mediators, are limited by several issues, including lack of temporality, heterogeneity in (mostly selfreported) sleep parameters and tools used for assessment, inaccuracies in assessment of lifestyle factors (e.g. dietary assessment, physical activity, sleep habits) and unaccounted for confounders. Only randomised, controlled trials can show a causal link between suboptimal sleep parameters and dysregulation of eating behaviours or physiological markers of appetite regulation, and these are limited in number.

Relationships between sleep, diet and maintaining a healthy weight in adults

Although assessments of sleep duration in population studies tend to rely on subjective tools, estimates of short sleep prevalence are consistent across high-income industrialised countries at around 34–36 %^(49,50). Observational evidence clearly points to striking associations between optimum sleep duration and maintaining a healthy body weight in both adults and children^(51–59) but the complexities of this bidirectional relationship are not yet fully elucidated, and it is likely that there are unmeasured confounding factors limiting the acuity of theoretical frameworks.

Dietary intake and physical activity fall on the causal pathway between short sleep and weight gain. There is an abundance of observational evidence suggesting that higher energy and sugar intakes, low fibre and a lack of fruit and vegetables, as well as suboptimal micronutrient intakes, are linked to short sleep (50,60-64) and/or irregular sleep timings (50,65-67). Systematic reviews that have evaluated evidence for relationships between sleep (duration, quality or timing/regularity) and dietary behaviours are summarised in Table 1. Those that addressed relationships between shorter sleep duration and diet have reported mixed findings for low adherence to the Mediterranean diet (70,72) and higher dietary inflammatory index (69), but stronger evidence for associations with higher total sugar and confectionary intake, and higher soda and soft drink intake (74).

A clearer picture emerges from systematic reviews on the Mediterranean diet and sleep quality, with greater dietary adherence being related to shorter sleep latency and higher global self-reported scores of overall sleep quality⁽⁷⁰⁻⁷²⁾. A minority of studies found relationships between poorer sleep quality and higher dietary inflammatory index⁽⁶⁹⁾, and there was some evidence for negative associations between sleep quality and soft/sugar-sweetened drink consumption and positive associations between sleep quality and intakes of fruits and/or vegetables⁽⁷¹⁾. In relation to sleep timing, a systematic review of 17, mostly crosssectional, studies investigating relationships between social jetlag and diet reported moderate evidence linking social jetlag to less healthy dietary patterns, lower fruits and vegetables intakes and higher sugar-sweetened beverages intakes⁽⁶⁸⁾, which presents diet as a key explanatory factor for a link between social jetlag and obesity⁽⁸⁴⁾, alongside reduced physical activity⁽⁸⁵⁾.

Table 1. Systematic reviews or meta reviews of observational studies that have investigated relationships between nocturnal sleep parameters, dietary intakes or eating behaviours published since 2019, organised by population type

| Authors | Population | Study designs included* | Sleep parameters | Diet/eating behaviour parameters | Adiposity parameters | Meta- analysis | Outcome |
|---|---|-------------------------------|---------------------------------|--|-------------------------|-------------------|---|
| General popul | lation | | | | | | |
| Arab <i>et al.</i> , 2023 ⁽⁶⁸⁾ | General population, excluding pregnant or lactating women. Means for age 3–56 years | Cross-sectional, longitudinal | SJL (exposure) | Dietary intakes: total energy, micro-/macro nutrients, food groups, and/or dietary patterns (outcomes) | Not included | N | 17 studies (1 longitudinal). 13/17 low risk of bias (Newcastle Ottawa Quality Assessment scale 'good quality'). Total energy (7 studies): 3 positive association (higher intake associated with social jetlag); 1 negative; 3 ns. Nutrients Protein (5 studies): 2 positive association; 3 ns. Carbohydrate (8 studies): 2 positive association; 2 ns. Fat (4 studies): 3 positive association; 1 ns. SFA (4 studies): 3 positive association; 1 ns. Food groups F&V (12 studies): 7 negative association; 5 ns. Dairy (7 studies): 6 ns; 1 negative. SSB (6 studies): 4 positive association; 2 ns. Sweet/sugar intake (7 studies): 4 positive association; 3 ns. Eggs/meat (5 studies): 4 ns; 1 positive association. Grains (8 studies): 3 negative association; 5 ns. Dietary patterns (7 studies) 5 negative association with healthy dietary patterns; 2 ns. |
| Coxon <i>et al.</i> , 2024 ⁽⁶⁹⁾ | General population. Age ranges spanned 17–≥ 70 years | Cross-sectional studies | Sleep duration or sleep quality | Dietary Inflammatory Index (DII) | Not included | N | 14 cross-sectional studies in total. 11 studies as high quality (Agency for Healthcare Research and Quality score). Higher DII was associated with shorte sleep duration in 2/7 studies and with poorer sleep quality in 4/12 studies. |

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|---|--|--|---|---|----------------------|-------------------|---|
| Authors | Population | Study designs included* | Sleep parameters | Diet/eating behaviour parameters | Adiposity parameters | Meta- analysis | Outcome |
| Fallah <i>et al.</i> , 2024 ⁽⁷⁰⁾ | Not specified. Age ranges spanned 11–84 years | Cross-sectional, longitudinal, case-control | Sleep duration, sleep quality | Adherence to the Mediterranean diet | Not included | N | 20 studies in total (2 cohort, 18 cross-sectional). 15/20 low risk of bias (Newcastle Ottawa Quality Assessment scale 'high quality'). 6/10 Mediterranean diet adherence associated with better sleep duration outcomes. 3/3 Mediterranean diet adherence associated with shorter sleep latency. 1/3 Mediterranean diet adherence associated with lower occurrence of low sleep efficacy. 9/9 Mediterranean diet adherence associated with better overall sleep quality. |
| Godos <i>et al.</i> , 2021 ⁽⁷¹⁾ | General population excluding < 3 years old. Age ranges spanned 8–84 years | Cross-sectional, longitudinal | Sleep quality | Dietary patterns, diet quality indices, food groups, carbohydrate quality | Not included | N | 25 observational studies in total. NIH quality score: good 1; fair 12, poor 12. Dietary patterns A posteriori-derived: 1/1 prudent dietary pattern inversely associated with sleep onset latency. A priori derived: 6/6 Mediterranean dietary pattern positively associated with sleep quality. Diet quality indices 2/3 studies found positive associations between healthier diet quality scores and sleep quality. Food groups 3 studies; sleep quality positively associated with fruit &/or vegetables (2/3), wholegrains (1/3), milk (1/3), fish (1/3*) and negatively associated with soft/sugar-sweetened drinks (2/3). *1 also found negative association. |
| Godos <i>et al.</i> , 2024 ⁽⁷²⁾ | General adult population, excluding pregnancy and patients with end-stage degenerative diseases. Means for age 30–73 years | Cross-sectional, cohort, case-control | Sleep features (sleep quality, duration) | Mediterranean dietary pattern, Mediterranean diet score | Not included | N | 23 studies (4 longitudinal but cross-sectionally analysed). No risk of bias assessment. In those without health conditions, 7/8 studies in Mediterranean countries and 5/6 studies in non-Mediterranean countries found positive associations between Mediterranean diet adherence and better sleep quality/fewer insomnia symptoms. In those without health conditions, 2/3 studies in Mediterranean countries and 2/2 studies in non-Mediterranean countries found positive associations between Mediterranean diet adherence and longer sleep duration. |

Table 1. (Continued)

| Komada <i>et al.</i> , 2020 ⁽⁷³⁾ | General population [see below section on children], excluding | All observational | General sleep outcomes (outcome) | Consumption of milk, yogurt, cheese, or other dairy products (exposure) | Not included | N | 4 studies included (1 longitudinal, 3 cross-sectional). 1/4 low risk of bias (Newcastle Ottawa Quality Assessment |
|--|---|----------------------------------|-------------------------------------|---|--------------------------|---|---|
| 2020 | breast-fed babies Age ranges spanned 12 weeks–81 years | | | (ехрозите) | | | scale 'high quality'). Association between late sleep midpoint and lower dairy intake in university students (1 study) and between higher milk consumption and longer sleep in girls (1 study). |
| Shahdadian et al., 2022 ⁽⁷⁴⁾ | General population [see below section on children] Age ranges spanned 4–56 years | All observational | Sleep duration (exposure) | Intake of sugar or sugary drinks (outcome) | Not included | Υ | 34 studies included in systematic review (all cross-sectional) and 13 studies included in meta-analysis. 7/13 low risk of bias (Newcastle Ottawa Quality Assessment scale 'high quality'). 26/34 found associations between short sleep duration (generally < 7–8 h in adults) and increased total sugar and confectionary intake. Short sleep associated with greater odds of higher soda and soft drink intake (adults OR: 1·20 [1·12, 1·28], 2 studies). |
| Zerón- Rugerio et al., 2023 ⁽³⁵⁾ | Adults without diagnosed chronic diseases, eating disorders, sleep conditions, or emotional disorders. Age ranges spanned 18–65 years | Cross-sectional and longitudinal | Not specified | Eating behaviours. | Obesity and weight loss. | N | 15 studies included (12 cross-sectional). 11/15 low risk of bias (Academy of Nutrition and Dietetics Quality Criteria Checklist 'positive'). 5/5 found associations between poor sleep quality and emotional eating. Other studies found associations between poor sleep quality and external eating (1), low eating competence (2), greater hunger (1), disinhibited eating behaviours (3), and higher cognitive restraint (2). 2/2 found associations between short sleep duration and emotional eating. 3 studies found emotional eating mediated relationship between inadequate sleep and obesity. 2 studies found disinhibited eating behaviour mediated relationship between inadequate sleep and obesity. |

| Table 1. (Contin | nued) | | | | | | |
|--|---|----------------------------------|--|---|--|-------------------|---|
| Authors | Population | Study designs included* | Sleep parameters | Diet/eating behaviour parameters | Adiposity parameters | Meta- analysis | Outcome |
| Children and | adolescents | | | | | | |
| Alibabaei et al., 2021 ⁽⁷⁵⁾ | Healthy children and adolescents ≤ 19 years old | Cohort, cross-sectional | Any measure of duration or quality of sleep | Any dietary pattern or dietary index derived by <i>a priori</i> or <i>a posteriori</i> approach (intervention/ exposure) | Not included | N | 14 studies (12 cross-sectional, 1 cohort, 1 RCT). Risk of bias not assessed. 9/12 studies found higher <i>a priori</i> diet quality indices or healthier <i>a posterior</i> dietary patterns was associated with longer sleep duration. 5/5 studies found healthier diets to be associated with earlier sleep midpoint or sleep timing. 1/1 study found healthier diets to be associated with lower social jetlag and 1/1 study found healthier eating habits (not skipping breakfast) was associated with better sleep quality. |
| Dutil <i>et al.</i> , 2022 ⁽⁷⁶⁾ | Children and adolescents aged 5–18 years, excluding those with diagnosed medical diseases/conditions other than obesity | Longitudinal and cross-sectional | Sleep timing (exposure), including sleep onset/ sleep offset, bedtime/ wake- up time, or midpoint of sleep | Missing breakfast, restrained eating score, emotional eating score, external eating score, healthy diet pattern, diet quality score, energy intake, food groups consumption | BMI, waist-to-height ratio, obesity, fat mass %, waist circumference | N | 20 studies on adiposity (3 longitudinal, serious risk of bias; 17 cross-sectional, no serious risk of bias). Quality of evidence assessed by GRADE framework 'very low'. 13 studies on eating behaviour/diet with serious risk of bias (0 longitudinal); quality of evidence assessed by GRADE framework 'very low'. Adiposity 1/1 longitudinal study found an association between later bedtime in adolescence and increase in BMI into adulthood. 1/1 longitudinal studies reported a longitudinal association between reoccurring late bedtime over multiple timepoints and higher BMI and waist circumference in children. 1/1 longitudinal study found no association between sleep onset/sleep midpoint and BMI. 7/17 cross-sectional studies in children and adolescents found an association between sleep timing and adiposity. Eating behaviour/diet patterns/food group intakes 9/13 cross-sectional studies in children and adolescents found an association between more favourable sleep timing and more favourable eating behaviour (3), healthier dietary patterns (1), diet quality score (1), healthier food group intakes (3), and lower fat intake (1). |

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Table 1. (Continued)

| Grimaldi et al., 2023 ⁽⁵⁹⁾ | Adolescents from general population aged 10–19 years | Longitudinal (≥ 2 assessments) | Any aspect of sleep (duration, timing, quality) | Dietary intake of foods or nutrients, amount or timing of food consumption, energy expenditure | BMI, % fat, risk of obesity | Y | 28 studies in total. 21 studies included in meta-analysis. Not stated how many overall at low risk of bias (adapted version of the Newcastle–Ottawa Scale). Adiposity 18 studies included on sleep and adiposity. Studies with sleep at baseline (T1) and adiposity at later time point (T2) including in MA: Longer sleep/higher sleep quality/lower sleep disturbances (T1) associated with anthropometric indices (T2) (n 15; $r = -0.06$ [-0.09 , -0.03] $P < 0.001$) and shorter sleep/lower sleep quality (T1) associated with higher odds of Ob (T2) (n 6; OR: 1.30 [1.08 , 1.56] $P < 0.01$) Studies with adiposity at baseline (T1) and sleep at later time point (T2) including in MA: Higher anthropometric indices (T1) not associated with poorer sleep (T2) (n 7; $r = -0.01$ [-0.01 , -0.00] $P > 0.05$) Diet 4 studies included on sleep and diet/eating behaviour. No MA possible. No consistency between studies |
|---|---|--------------------------------|---|---|-----------------------------|---|--|
| Komada et al., 2020 ⁽⁷³⁾ | General population (including children), excluding breast-fed babies | All observational | General sleep outcomes (outcome) | Consumption of milk, yogurt, cheese, or other dairy products (exposure) | Not included | N | [See section on general population] Association between higher milk consumption and longer sleep in girls (1 study). |

| Table 1. (Contin | | | | | | | |
|---|--|----------------------------------|---|---|----------------------|-------------------|--|
| Authors | Population | Study designs included* | Sleep parameters | Diet/eating behaviour parameters | Adiposity parameters | Meta- analysis | Outcome |
| Krietsch <i>et al.</i> , 2019 ⁽⁷⁷⁾ | Children and adolescents aged 0–18 years, excluding clinical populations | Longitudinal and cross-sectional | Sleep duration, time in bed, sleep timing, sleep quality, sleep variability | Dietary intake of nutrients/food groups, eating behaviour | Not included | N | intake or eating behaviour in total. 44/57 observational studies at high risk of bias, 2/57 at low risk of bias (Cochrane risk of bias tool). Not possible to determine number of studies finding associations for each category from report. Diet quality About three-quarters of studies find longer sleep duration associated with higher diet quality; results more variable for sleep variability and timing. Energy intake Higher energy intake associated with shorter, later, poorer, and more variable sleep in some studies - inconclusive. Sugar-sweetened beverage intake Some, not all, find associations with shorter, more variable sleep - inconclusive. Fruits and vegetables Longer sleep duration or earlier sleep timing associated with more F&V consumption in half of studies but inconclusive. Sweets and desserts Longer sleep duration associated with less sweets/desserts consumption in half of studies but inconclusive. Breakfast skipping associated with shorter sleep duration and later sleep timing. Inadequate literature highlighted for: eating in response to external cues, stress, or emotions; disordered eating; hunger and satiety; |
| Shahdadian <i>et al.</i> , 2022 ⁽⁷⁴⁾ | General population [including children] | All observational | Sleep duration (exposure) | Intake of sugar or sugary drinks (outcome) | Not included | Y | [See section on general population] Short sleep associated with greater odds of higher sugar and confectionary intake (children OR: 1·1 [1·10, 1·21], 5 investigations). Short sleep associated with greater odds of higher soda and soft drink intake (children OR: 1·18 [1·05, 1·32], 5 studies). |

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Table 1. (Continued)

| Ward <i>et al.</i> , 2020 ⁽⁷⁸⁾ | Children aged 0–5 years, excluding studies including only participants with preexisting chronic conditions or only overweight/obese | Longitudinal and cross-sectional | Sleep duration, variability or quality; night wakings; fragmented sleep; number of sleep episodes; improved sleep pattern | Quantitative measures of dietary intake | Not included | N | 33 studies in total (16 longitudinal, 17 cross-sectional). Average risk of bias score 5·1/8 for cohort studies and 4·6/8 for cross-sectional studies (Newcastle Ottawa Scale). Energy intake 2/2 showed reduced sleep duration associated with higher energy intake Dietary patterns/nutrient intakes Substituting unsaturated fat for saturated fat at 13 months associated with longer sleep duration at 3 years (1 cohort study). Healthier dietary patterns associated with longer sleep duration (3 cohort studies). Breastfeeding Mixed results on sleep outcomes (1 favourable, 1 null, 4 unfavourable) Introduction of solids Early introduction of solids or complementary foods associated with increased sleep duration (2/3 cohorts) |
|---|---|--------------------------------------|---|--|--------------|---|---|
| Ward <i>et al.</i> , 2021 ⁽⁷⁹⁾ | Children aged 6–12 years, excluding studies including only participants with pre- existing chronic conditions | Longitudinal and cross- sectional | Sleep quality or quantity | Nutrients, food, food groups, dietary patterns, or eating behaviours | Not included | N | 42 studies in total (5 longitudinal, 37 cross-sectional). Average risk of bias score 5·2/8 for cohort studies and average score not given for cross-sectional studies (Newcastle Ottawa Scale). Cohort studies: short sleep duration was associated with higher energy from fat intake (n 1), lower diet quality (n 1), and greater consumption of SSBs (n 2). Cross-sectional studies: 10/13 found short sleep duration was associated with lower measures of diet quality, 1/1 found lower sleep quality associated with unhealthy dietary pattern, and 2/2 found later bedtimes associated with increased energy intake after the evening meal or unhealthy dietary pattern. 2/2 studies found that high consumption SSB was associated with unfavourable sleep outcomes. |
| Zhang <i>et al.</i> , 2020 ⁽⁸⁰⁾ | Children aged < 5 years | Longitudinal and cross- sectional | Sleep duration | Eating behaviours | Not included | N | Eating behaviour exposures at study level not presented |

Table 1. (Continued)

| Authors | Population | Study designs included* | Sleep parameters | Diet/eating behaviour parameters | Adiposity parameters | Meta- analysis | Outcome |
|--|------------------------------|---|--|--|----------------------|-------------------|---|
| Zhong et al., 2024 ⁽⁸¹⁾ | Adolescents aged 12–18 years | Longitudinal and cross- sectional, excluding studies on restless leg syndrome, sleep apnoea, chronic medical or neurodevelopmental conditions | Sleep features | Dietary factors | Not included | Y | 33 studies in total (6 longitudinal, 27 cross-sectional); 12 fair quality and 21 poor quality (NIH quality assessment). 6 studies included in meta-analysis. Dietary patterns 1/1 found adherence to DASH diet associated with lower odds of insomnia. 1/1 found adherence to plant-based/lean protein pattern had earlier sleep timing. 1/1 found unhealthy diet pattern associated with short sleep duration and 1/1 found Mediterranean diet pattern associated with optimal sleep duration. Food groups No association between vegetable, fruit or milk intake and sleep duration (1/1). No association between consumption of processed foods and sleep problems (OR = 1-32, 95 % CI 0-98, 1-79) |
| von Ash et al., 2023 ⁽⁸²⁾ | Pregnant women | Longitudinal and cross-sectional | Sleep duration, quality, disturbance, and other measures | Dietary patterns, energy and nutrient intakes, and eating behaviours | Not included | N | 23 studies in total (8 longitudinal, 15 cross-sectional); Risk of bias not assessed. Dietary patterns 4/5 found healthier dietary patterns were linked to improved sleep quality. 1/5 found healthier dietary pattern associated with adequate sleep duration. Energy or nutrient intakes 1/3 found that higher energy intakes were associated with sleep quality and 1/5 with sleep duration. 1/1 found that higher fat intake was associated with more sleep disturbance but not duration or quality, and 1/1 that lower fat intake was associated with lower sleep quality but not duration and greater carbohydrate intake was associated with lower sleep quality. Eating behaviours Suboptimal eating behaviours (e.g. night-time eating, late eating, cravings, emotional eating etc) or disordered eating associated with lower sleep quality (3/4) and shorter sleep duration (2/5). |

| Table 1. (Continued) | | | | | | | |
|---|----------------|----------------------------------|---|--|----------------------------|---------------------------------------|---|
| Pauley Pregnar et al., 2022 ⁽⁸³⁾ | Pregnant women | Longitudinal and cross-sectional | Sleep duration, quality, disturbance and other measures | Dietary patterns, diet quality, energy and nutrient intakes, and eating behaviours | Gestational weight gain | Weighted mean effect sizes calculated | 10 studies in total (2 longitudinal/cross-sectional only). High quality n 5, moderate quality n 5 (Downs & Black checklist). Gestational weight gain Associated with lower sleep quality (effect size 0.51, n 2) but effect sizes small for sleep duration (0.04–0.26, n 2). Dietary patterns/diet quality/energy intake Low effect size for sleep quality and duration (0.032–0.158, n 3). Eating behaviours Poor sleep quality associated with shorter interval sight-time fasting interval (effect size 0.658, n 1) |

BMI, body mass index; DASH, Dietary Approaches to Stop Hypertension; DII, dietary inflammatory index; F&V, fruits and vegetables; GRADE, Grading of Recommendations, Assessment, Development, and Evaluations; MA, meta-analysis; N, no; NIH, National Institutes of Health, ns, non-significant; OR, odds ratio; OW/Ob, overweight/obesity; RCT, randomised controlled trial; SFA, saturated fatty acids; SJL, social jetlag; SSB, sugar-sweetened beverages; T1, baseline timepoint; T2, second timepoint; T2D, type 2 Systematic reviews may have also included intervention studies, but outcomes not included here.

The multi-directional links between sleep, eating and physical activity behaviours, alongside psychological and socio-cultural factors, create a complex network of cause-and-effect relationships. Randomised controlled intervention studies can provide an estimate of the potential independent effect size of changes in sleep duration, timing or quality on adiposity and intermediary factors, such as dietary intake and energy expenditure. Indeed, a robust body of evidence from sleep deprivation intervention trials has demonstrated that reductions in sleep duration over short periods of time (1–14 d) increase energy intake in adults by a pooled mean of 385 kcal/d (95 % CI 252, 517, based on meta-analysis of 7 RCTs and 3 non-randomised trials, n 185 sleep-restricted and n 161 control), with no observed compensatory increase in overall energy expenditure⁽⁸⁶⁾. This positive energy balance, if it were to be maintained long-term, could eventually predispose chronically sleep-restricted individuals to weight gain. However, currently, there is a lack of evidence to suggest a long-term impact of chronic sleep-restriction on weight management due to an absence of longer-term RCTs for obvious ethical reasons. However, one RCT of chronic sleep restriction with a longer duration (5 nights/week for 8 weeks) plus energy restriction (95 % of RMR; RMR) was conducted in adults with overweight or obesity (parallel arms, n 36), reporting similar weight loss to an energy restriction-only control⁽⁸⁷⁾, although the study was limited by small sample size and an imbalance between groups in body weight at baseline.

Sleep extension trials in habitual short-sleepers are important because extrapolation of sleep restriction intervention trial results (in those who normally sleep for an adequate duration) to the potential impact of sleep extension (in short-sleepers) may be an over-simplification of the physiological and psychological mechanisms involved. However, there have been fewer studies of the effects of sleep extension on weight management, diet or energy expenditure. A representative summary of diet, adiposity and cardiometabolic health outcomes from published studies in adults where an intervention was designed to extend sleep duration, or to improve sleep quality or timing/regularity (restricted here to a minimum of 2 weeks intervention), is presented in Table 2.

Designing a behavioural sleep extension intervention is problematic due to multiple challenges relating to designing an appropriate control treatment, blinding, potential behaviour change in the individuals allocated to the control group (who may be aware of the aim to extend sleep), burden of data collection/ lifestyle change and compliance to protocol, and adherence to behaviour change counselling. Therefore, many published sleep extension intervention trials are pilot and/or feasibility studies. Sleep extension intervention trials have been conducted in both healthy and clinical populations, with outcomes including dietary intakes, adiposity, energy expenditure, cardiometabolic health markers and mental health (Table 2). Some of these have successfully extended average sleep duration (89,93-95,100,102). A pilot feasibility RCT in young, healthy-weight adults, extended sleep duration for 4 weeks, but at a cost of decreased sleep efficiency⁽¹⁰²⁾. In contrast, no negative effects on sleep quality parameters were reported by several other sleep intervention studies that have successfully extended sleep duration (89,94,95,100). Data from another pilot trial suggested that the effectiveness of a weight loss programme may be enhanced by sleep extension (96), but substantial participant attrition and lack of reported data on sleep duration limited the conclusions that could be drawn; very few larger, fully powered trials addressing the same question have materialised. Nonetheless, two intervention trials with mixed results are worth noting. Firstly, a RCT compared 6-month

Table 2. Intervention studies that have extended sleep duration, modified sleep timing, and/or aimed to improve sleep quality as an isolated intervention, with intervention duration ≥ 2 weeks in adult and paediatric populations

| Authors | Population | Study design | Sleep inter- vention type | Intervention duration | Outcome domain | Parameters measured (primary outcome denoted by*) | Key findings |
|---|---|--|------------------------------|-----------------------|-------------------|--|---|
| 7.00.1010 | · opulation | Adults | vendon type | | | (p.i.i.a.) cateomic achieved 2) | They intuings |
| Al Khatib et al., 2018 ⁽⁸⁸⁾ | UK, men and women, aged 25 years (95 % CI 22, 30), BMI 22 (95 % CI 21, 24) | Parallel arm pilot feasibility RCT, <i>n</i> 42 | Sleep extension | 4 weeks | Sleep | Actigraphy (sleep duration*, sleep quality) and subjective global sleep quality (PSQI). Sleep hygiene index for compliance monitoring. | ↑ time in bed, sleep period, sleep duration. ↑ sleep fragmentation. ↓ sleep efficiency. NSD in sleep latency. |
| | | | | | Diet | 7 d estimated weight food diaries (energy, macronutrients, caffeine, adherence to UK dietary guidelines). | ↓ intake of carbohydrate (g), free sugars (g), fat (%en) ↑ intake protein (%en). ↑ adherence to dietary guidelines. NSD in energy, total sugar, fibre, saturated fat, alcohol or caffeine intake. |
| | | | | | Adiposity | BMI, BW, FFM, BF%, WC | NSD |
| Baron <i>et al.</i> , 2019 ⁽⁸⁹⁾ | USA, men and women, aged 46 years (95 % CI 37, 56), BMI 27 (95 % CI 25, 30), pre- | Parallel arm feasibility RCT, n 16 | Sleep extension | 6 weeks | Sleep | [Primary outcome not stated.] Actigraphy (sleep duration, sleep quality, sleep onset and offset time). | † total sleep time. NSD in other outcomes. |
| | hypertension/ stage 1 hypertension | | | | Diet | NR | _ |
| | | | | | Adiposity | ВМІ | NSD |
| Baron <i>et al.</i> , 2023 ⁽⁹⁰⁾ | USA, men and women, aged 45 years (sp 11), BMI ≥ 25 | Parallel arm feasibility RCT, n 38 | Sleep extension | 6 weeks | Sleep | [Primary outcome not stated.] Actigraphy (sleep duration, sleep quality, sleep onset and offset time). Epworth Sleepiness Scale for subjective daytime sleepiness. | NSD |
| | | | | | Diet | NR | - |
| | | | | | Adiposity | ВМІ | NSD |
| | | | | | | | |

Table 2. (Continued)

| Duncan <i>et al.</i> , 2020 ⁽⁹¹⁾ & Fenton <i>et al.</i> , 2021 ⁽⁹²⁾ | Australia, men and women, aged 45 (sp 10) years, BMI 32 (sp 4) | Parallel arm RCT, n 116 (3 arms: weight loss (diet and physical activity) + sleep intervention; weight loss intervention only; waitlist control group. | Sleep timing and extension | 6 mo (primary endpoint). | Sleep | Accelerometry (sleep quality) subjective global sleep quality/duration (PSQI). | NSD at 6 mo. ↓ bedtime variability (12 mo). NSD in other outcomes at 12 mo. |
|---|--|--|--|--------------------------------|---|---|---|
| | | | | | Diet | Food frequency questionnaire (120-items) for energy, food groups, macronutrients, micronutrients, caffeine and diet quality) | NSD between weight loss + sleep v. weight loss only groups. Some differences in food groups at 12 mo follow up. |
| | | | Adiposity BW*, BMI, WC | | BW*, BMI, WC | NSD between weight loss + sleep v. weight loss only groups | |
| Haack <i>et al.</i> , 2013 ⁽⁹³⁾ | USA, men and women, aged 48 (sp 12) years, BMI 26 (sp 1), hypertensive or pre-hypertensive | Parallel arm RCT, n 22 | Sleep extension | 6 weeks | Sleep | [BP*] Actigraphy and sleep diary (sleep duration, sleep quality, time of sleep and wake). | ↑ in sleep duration |
| | | | | | Food records (1 d) for energy, macronutrients and sodium. | NSD | |
| | | | Adiposity BW, BMI, % body fat. a RCT, n 18 Sleep 6 weeks Sleep Actigraphy and sleep diary (sleep duration*, sle | | NSD | | |
| Hartescu et al., 2022 ⁽⁹⁴⁾ | UK, men, aged 41 (sp 9) years, BMI 30 (sp 3), increased risk of T2D | Parallel arm RCT, n 18 | Sleep extension | - · · · | Sleep | Actigraphy and sleep diary (sleep duration*, sleep quality). | † in sleep duration, time in bed. NSD in sleep quality outcomes. |
| | | | Diet NR | | NR | - | |
| | | | | Adiposity | BW | NSD | |
| Leproult et al., 2015 ⁽⁹⁵⁾ | Belgium, men and women, aged median 25 (Q1 23, Q3 28) years, BMI median 21 (Q1 19, Q3 24) | Single-arm intervention trial, <i>n</i> 16 | Sleep extension | 6 weeks | Sleep | [Glucose and insulin*] Actigraphy, daily sleep logs, and polysomnography (sleep duration, sleep quality, sleep and wake onset). | ↑ in sleep duration and sleep quality (pre- to post- intervention) |
| | | | | | Diet | NR | _ |
| | | | | | Adiposity | BW | NC pre- to post- intervention |
| Logue <i>et al.</i> , 2012 ⁽⁹⁶⁾ | USA, men and women, aged 18–83 years, BMI 25 and < 40 | Parallel arm pilot RCT, <i>n</i> 46 (<i>n</i> 25 completed) | Sleep extension | 12 weeks | Sleep | Subjective global PSQI (sleep quality) and Sleep Timing Questionnaire. | NSD in sleep efficiency. No other sleep parameter reported. |
| | | | | | Diet | Food frequency questionnaire (food groups) and diet interview (nutrients) | Effects on dietary intakes NR |
| | | | | | Adiposity | BW [⋆] | ↓ in BW greater after sleep + weight loss intervention compared to weight loss only |

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Table 2. (Continued)

| Authors | Population | Study design | Sleep inter- vention type | Intervention duration | Outcome domain | Parameters measured (primary outcome denoted by*) | Key findings |
|---|--|--|---|-----------------------|-------------------|--|---|
| Martyn- Nemeth <i>et al.</i> , 2023 ⁽⁹⁷⁾ | USA, men and women, aged 30 years (sd 7·3), BMI 27 (sd 7), type 1 diabetes | Parallel arm pilot feasibility RCT, <i>n</i> 14 | Sleep extension and regularity | 8 weeks | Sleep | [Primary outcome not stated.] Actigraphy (sleep duration, sleep quality (efficiency only), sleep and wake onset, sleep regularity (using midpoint of sleep time)). Epworth Sleepiness Scale. | Improved sleep regularity but NSD in other parameters. NSD in subgroup with short sleep at baseline n 8. |
| | | | | | Diet | NR | _ |
| | | | | | Adiposity | NR | - |
| Reutrakul et al., 2022 ⁽⁹⁸⁾ | USA, women, aged 42 years (sp 3) in sleep intervention group and 39 years (sp 6) in control group, | Parallel arm pilot feasibility RCT, <i>n</i> 14 | Sleep extension | 6 weeks | Sleep | Actigraphy (sleep duration*, sleep quality (efficiency only)) and subjective global sleep quality (PSQI). | NSD |
| | BMI 33 (SD 5), history of gestational diabetes | | | | Diet | NR | - |
| | 8 | | | | Adiposity | BW | NSD |
| So-Ngern et al. 2019 ⁽⁹⁹⁾ | Thailand, men and women, aged 33 years (sp 6), BMI 23 (sp 3) | Crossover arm RCT, n 21 | Sleep extension | 2 weeks | Sleep | [Glucose and insulin*] Subjective sleep assessed by global PSQI. | NSD |
| | | | | | Diet | 2×3 d food diaries (only energy intake reported) | NSD |
| | | | | | Adiposity | BW | NSD |
| Tasali <i>et al.</i> 2022 ⁽¹⁰⁰⁾ | USA, men and women, aged 30 years (sp 5), BMI 28 (sp 1) | Parallel RCT, n 80 | Sleep extension | 2 weeks | Sleep | Actigraphy (sleep duration, sleep quality). | ↑ in sleep duration, sleep onset latency and wake after sleep onset. NSD in sleep efficiency. ↓ nap duration. |
| | | | | | Diet | Energy intake*, measured as sum of total energy expenditure (doubly labelled water) and change in body energy stores (daily home weights and body composition changes using dual-energy x-ray absorptiometry). | ↓ energy intake |
| | | | | | Adiposity | BW, FFM, FM | ↓ BW, FFM and FM |
| Children | | | | | | | |
| Moreno-Frias et al., 2020 ⁽¹⁰¹⁾ | Mexico, male and female adolescents, aged 14–18 years, BMI ≥ 30 kg/m² | Parallel RCT, <i>n</i> 52 | Sleep extension | 4 weeks | Sleep | Subjective sleep assessment by sleep diaries and daily telephone calls (sleep duration, sleep quality). | Groups were not compared statistically. |
| | | | | | Diet | Weekly 2 \times 24 h diet recalls (only energy intake reported) | NSD (‡ energy intakes in both groups) |
| | | | | | Adiposity | BW*, WC | ↓ BW and WC |

Sleep quality is an umbrella term for the following parameters: sleep efficiency, sleep onset latency, sleep fragmentation, wake after sleep onset; it can also be measured as a subjective global score by the Pittsburgh Sleep Quality Index questionnaire.

Abbreviations: AEE, activity energy expenditure; BF, body fat; BW, body weight; CGM, continuous glucose monitor; %en, % energy; FFM, fat-free mass; FM, fat mass; MVPA, moderate to vigorous physical activity; NC, no change (pre- to post-intervention); ND, no significant difference; NR, not reported; PAL, physical activity level; PSQI, Pittsburgh Sleep Quality Index; SJL, social jetlag; T2D, type 2 diabetes; TEE, total energy expenditure; WC, waist circumference.

interventions comprising a weight loss programme (personalised dietary advice and physical activity goals, delivered using behaviour change techniques via in-person counselling sessions, a smartphone app, email and SMS messaging, n 41) with the same weight loss programme plus optimisation of sleep timing and sleep extension in adults with overweight or obesity $(n \ 39)^{(91)}$. There was substantial drop-out by the 6-month primary endpoint: 31 % in the enhanced sleep group and 22 % in the diet and exercise-only group. The enhanced sleep group, who received the sleep intervention via an app and a handbook targeting sleep hygiene behaviour change, reduced sleep time variability and stress management and relaxation techniques, reported reduced bedtime variability⁽⁹¹⁾. However, there were no effects on the Pittsburgh Sleep Quality Index (PSQI; validated questionnaire) global score. Sleep duration forms part of the PSQI global score and was not reported separately. Furthermore, there were no associated effects on energy intake, weight loss, or HbA1C compared to a weight loss only (diet and physical activity) intervention⁽⁹¹⁾. Over a much shorter intervention period (2 weeks), and without a weight loss component, a sleep extension intervention in adults with overweight (parallel arm RCT, n 80) led to reduced energy intakes (270 kcal/d, measured objectively) and body weight compared to habitual sleep(100). The impact on diet quality and eating behaviours was not captured in the latter study, but collectively the evidence suggests that although short-term improvements in sleep habits may lead to reduced energy intake, the impact on body weight may be negligible over longer periods when healthy eating and physical activity advice is already being provided.

An important consideration in the design of multiple target lifestyle interventions for weight management is the risk of overwhelming participants with behaviour change goals, resulting in a failure to incorporate change into daily life and ultimately an adverse effect on weight loss. This was suggested by Marshall et al. who conducted a study evaluating the feasibility of a remotely delivered lifestyle intervention in short-sleeping (< 7 h per night on working days) university employees who had expressed an interest in losing weight (assessed for eligibility by an online questionnaire), involving sleep hygiene counselling in combination with diet and physical activity advice⁽¹⁰³⁾. There were fewer participants with > 1 kg weight loss after 12 weeks of intervention in the sleep + diet + physical activity group compared with the diet + physical activity only group (103). These findings suggest that more intervention development work is needed to optimise an acceptable, achievable programme that incorporates sleep behaviour change into standard weight-management strategies.

Relationships between sleep, diet and maintaining a healthy weight in children and adolescents

Recommended sleep durations per night for infants and children range from 14–17 h for newborns down to 8–10 h for 14–17-year-olds⁽⁸⁾. In 2023, 38 % of 8–16 years old (total sample n 1140 recruited from the NHS Patient Register) participating in the 'Mental Health of Children and Young People in England - Wave 4 Follow up to the 2017 Survey' reported 3 or more nights of problems getting to sleep, waking in the night, or waking early in the previous seven days (25 % in a subsample of 810 excluding those likely to have a mental disorder) (104). Similarly, 34 % of 0–17-year-olds in a US representative 2019–2020 sample (total sample n 67 598 from a population-based nationally representative survey) had parent-reported short sleep duration (105). Extensive evidence exists for an association between sleep duration and

adiposity in childhood. A meta-review of 24 systematic reviews of mainly observational studies investigating nocturnal sleep and adiposity parameters in children up to 18 years found that 68 % of the 443 studies reported an association between adequate sleep duration and reduced adiposity⁽¹⁰⁶⁾, with 58 % of the 26 studies on sleep quality also finding a favourable relationship. However, evidence is less abundant and more mixed for associations between sleep variability/timing and overweight or obesity. To yield insights into directionality, Grimaldi *et al.* (2023) meta-analysed longitudinal studies in children and adolescents aged 10–19 years and reported that shorter sleep or lower sleep quality at baseline was associated with a 30 % increased likelihood of obesity at a later timepoint⁽⁵⁹⁾.

As is the case for adults, there is a large body of observational evidence, mainly cross-sectional studies, but also including some cohort studies, linking short sleep with diet or eating behaviours in children and adolescents, with several systematic reviews published on this topic (Table 1). Many studies evaluated in these systematic reviews have reported that better diet quality, healthier dietary patterns, higher energy intake and/or lower intake of sugar/ sugar-sweetened beverages are associated with longer sleep duration, earlier sleep timings, lower social jetlag and better sleep quality in children and adolescents^(74–79,81).

Analyses of pooled intervention trials to increase sleep duration in children and adolescents, conducted in school and home settings, have reported mixed results for adiposity and diet(107,108). Yoong and colleagues (2016) included 8 studies that had manipulated sleep duration (restriction or extension) or improved sleep quality, including multicomponent interventions, in children up to 18 years of age⁽¹⁰⁸⁾. Only 2 of these studies aimed to extend sleep duration in children aged ≥ 1 year, one with a 1-week intervention compared to a sleep restriction comparator arm⁽¹⁰⁹⁾ and the other intervening over 6 months with the control arm involving provision of leaflets on child development(110); both studies reported an increased sleep duration, with the former reporting a reduction in energy intake of 134 kcal/d⁽¹⁰⁹⁾, and both reporting a decrease in BMI. A small number of sleep intervention trials (6 months to 5 years follow-ups) in under 5-year-olds were meta-analysed by Miller et al. (2021) demonstrating an overall significant reduction in BMI compared to control⁽⁵⁷⁾. A subsequent systematic review and meta-analysis of sleep intervention trials in children aged 5-17 years of age with overweight or obesity (107) included 8 studies that measured adiposity (7 of which were multicomponent interventions). However, findings were inconclusive: heterogeneity was high across studies, only 5 reported sleep outcomes (only 2 out of 5 reported an improvement), and metaanalysis of data from 5 studies found no significant overall effect on $BMI^{(107)}$.

The evidence base comprising behavioural sleep or sleep plus lifestyle intervention studies with adiposity-related outcomes is small in paediatric populations, but the number that has measured the impact of improvement in sleep on dietary outcomes is even smaller. Although a handful of intervention studies including sleep within a multicomponent intervention exist, it is not possible to isolate the effect of sleep modification from these studies, as reviewed by Liu et al⁽¹⁰⁷⁾. Moreno-Frias and colleagues (2020) conducted a 4-week parallel RCT in Mexican male and female adolescents with obesity aged 14–18 years to extend sleep in addition to a weight loss programme (n 25) compared to weight loss only control group (n 27); they reported a greater decrease in BMI and waist circumference in the sleep intervention group but no differences in energy intake⁽¹⁰¹⁾. However, differences in

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changes between groups were not presented and there were no other details on dietary outcomes reported, so firm conclusions could not be made.

In summary, although the observational evidence suggests that inadequate sleep is a key factor associated with obesity and poor diet, there is a lack of interventional evidence demonstrating that improving sleep duration or other sleep parameters in children will lead to a reduced risk of weight gain and healthier diets. Chronotype, an individual's natural tendency to go to bed and get up in the morning either earlier or later, plays a particularly important role in how inadequate sleep impacts the risk of weight gain and obesity in adolescents, who tend to increasingly develop a later chronotype, peaking in the early twenties(111). Evening chronotypes may be particularly vulnerable due to misalignment between their biological rhythms and societal demands, leading to reduced sleep duration and consequently greater risk of unhealthy eating behaviours and weight gain. Concentrating research in this area could yield new public health interventions to improve the effectiveness of current strategies to prevent child obesity.

Relationships between sleep, diet and maintaining a healthy weight during pregnancy

There are no specific guidelines for sleep duration during pregnancy. Many pregnant women experience increased sleep disruption across all trimesters, with a pooled prevalence estimate of 46 % (95 % CI 37 %, 55 %) during pregnancy, and 70 % (95 % CI 54 %, 82 %) specifically during the third trimester, as assessed by a PSQI score $\geq 5^{(112)}$. A systematic review that had investigated sleep parameters during pregnancy, gestational weight gain and eating behaviours found a low number of relevant articles (10 crosssectional studies and one RCT, although the latter was not a sleep intervention) and reported high heterogeneity across studies, limiting conclusions that could be drawn⁽⁸³⁾. Subsequently, a second systematic review of mainly observational studies, published up to 2021, reported mixed results and concurred that high levels of heterogeneity precluded any clear messages from the evidence base⁽⁸²⁾. There is a shortage of evidence for the impact of sleep hygiene counselling on gestational weight gain or diet.

Conclusions on the evidence supporting inclusion of sleep behavioural interventions as a key element of successful weight management

This review has provided an updated overview of available literature on the impact of inadequate sleep duration, timing/ regularity and quality in adults, children and during pregnancy on weight management and dietary factors. Although the evidence base is growing, and the links between suboptimal sleep and risk of weight gain are consistent across study types and age groups, there remains a lack of quantitative data on the likely effect size of sleep interventions on weight management. Inclusion of sleep hygiene counselling or other behavioural interventions in weight-management programmes and/or clinical guidelines for the management of obesity uses considerable resources and therefore convincing evidence is needed that this would significantly enhance the impact or durability of weight-management approaches. The use of research-grade, validated, wearable devices (e.g. MotionWatch, ActiGraph) that use accelerometry, ideally integrated with other technologies to measure changes in light or variability in heart rate for even greater accuracy, enhances the scalability and validity of research into the impact of sleep

interventions on dietary and physical activity behaviours. These actigraphy devices are now integral tools for accurate assessment of changes in sleep patterns in both clinical and research settings. To date, the totality of the evidence from the few intervention studies that have investigated effects of sleep interventions on weight management is hampered by limitations such as short duration, small sample size, or low generalisability of the study sample. Full integration of sleep behaviour change into clinically-endorsed weight-management programmes would require testing an integrated diet-activity-sleep intervention in a long-term, well-powered RCT to establish superiority over standard weight-management programmes. Currently, there are insufficient data to support the inclusion of intensive sleep behaviour change counselling as a mandatory element of weight-management plans.

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