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CLINICAL REVIEW

# Sleep and weight-related factors in youth: A systematic review of recent studies

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SUMMARY

There has been much speculation about mechanisms driving the pediatric sleep/obesity link. However, conjectures have not been matched by a systematic review of pediatric data investigating sleep and behaviors directly influencing weight. This raises the risk of biased, "cherry-picked" interpretations and hampers focused research efforts. This systematic review synthesized recent studies, discussed literature gaps, and provided recommendations for future research on sleep and weight-related factors in youth. Of the 4,302 articles (published between 2012 and 2017) initially screened, 86 were included in this review, which investigated the relationship between sleep and dietary intake, altered eating behavior, physical/sedentary activity, or hormones regulating hunger/satiety. Despite prior systematic reviews indicating associations with body mass, this systematic review of proposed mechanisms revealed highly variable findings and studies showed a high risk of bias. There were some consistent patterns showing no cross-sectional association between sleep duration and caloric intake (despite experimental evidence), and shorter or later sleep associating with greater sedentary or screen time. Considerable variability in methodology, weak sleep and dietary intake measurement, and a paucity of longitudinal or experimental designs likely contributed to variability in results. Findings highlight the need for more rigorous measurement and design methods in order to move the field forward.



## Introduction

Pediatric obesity is the most common chronic health condition of childhood, with 1/3 of American youth meeting criteria for overweight/obesity [1]. Changing established weight-related habits (e.g. diet and exercise) is notoriously difficult, and interventions targeting these behaviors have shown only moderate success [2]. Thus, research is needed to elucidate additional mechanisms of excessive weight gain in youth to further improve obesity prevention and intervention efforts. Sleep has been proposed as a promising target, with meta-analyses indicating a 58–89% increased risk for developing overweight/obesity among youth not meeting recommended sleep guidelines [3].

Researchers have been quick to respond to these promising findings. Hart and colleagues published a hallmark review of pediatric sleep and overweight/obesity in 2011, which indicated that sleep duration, quality, and timing were all related to BMI and body fat percentage in youth [4]. Their review also called for future research on mediators of the pediatric sleep/obesity relationship, having identified a mere three articles investigating these pathways in children. Since then, there has been tremendous growth in the related research literature, including mechanistic studies that have sought to identify how sleep may drive weight gain. Proposed mechanisms have included sleep-related changes in dietary intake and physical activity related to diminished self-control, negative emotions, greater exposure to the obesogenic environment, and changes to hormones regulating hunger and satiety (e.g., leptin, ghrelin) [5–8].

Despite their contributions, reviews thus far have been limited by their restricted focus on narrow aspects of sleep (e.g., duration) or weight-relevant factors (e.g., diet), have combined child and adult literature (despite developmental differences in sleep, obesity definitions and rates, and contextual factors related to diet and physical activity), or have limited their scope to studies using specific types of measures. Importantly, no review to date has systematically assessed the risk of bias in this literature, which

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**Abbreviations**

| | |
|---|---|
| BMI | body mass index |
| MVPA | moderate-to-vigorous physical activity |
| PA | physical activity |
| PSG | polysomnography |
| REM | rapid eye movement |
| RTI | Research Triangle Institute |
| SE | sleep efficiency |
| SES | socio-economic status |
| RoB | Risk of Bias |
| SOL | sleep onset latency |
| SSBs | sugar-sweetened beverages |
| SWS | slow wave sleep |
| TIB | time in bed |
| TST | total sleep time |
| WASO | wake after sleep onset |

limits suggestions for moving the field forward. If the field is to clarify mechanisms underlying the pediatric sleep/obesity relationship, it needs to assess all relevant sources of information in the context of their limitations, identify potential patterns in findings, and suggest specific directions for conceptual and methodological growth.

This review aims to fill that gap by systematically reviewing the recent pediatric literature on relationships between multiple critical markers of sleep (including duration, timing, variability, and quality) and weight-related factors (including dietary intake, altered eating behavior/patterns, physical or sedentary activity, and hormones regulating hunger and satiety) that may drive the sleep/obesity relationship. In doing so, this review will capture, evaluate, and synthesize the current state of the literature. It will also shed light on common methodological or statistical issues that impact the risk of study bias and may influence interpretation. Based upon those findings, it then provides concrete recommendations for how to move the field forward by directing researchers towards promising areas and away from "dead ends," suggesting ways to overcome common methodological or design gaps, and proposing promising areas for targeted meta-analytic work. Finally, this review seeks to identify which relationships, if replicated, could translated from bench to bedside (e.g., by investigating whether intervening on sleep markers, for example, in the context of pediatric weight management intervention, may influence weight-related behaviors or weight status).

## Methods

This review was conducted in adherence with the PRISMA guidelines for reporting outcomes in systematic reviews [9].

### *Systematic search strategy*

To identify eligible studies, researchers first identified subtopics within the areas of sleep, physical activity, dietary intake, eating behaviors/patterns, and hormones regulating hunger/satiety. Subtopics and associated subject headings, MeSH terms, and keywords were used to search PubMed, PsychInfo, and Web of Science (detailed in Table S1). The final search date for all databases was November 13, 2017. Reference lists of selected articles (including review articles) were also manually reviewed for articles that may have been missed during the electronic database searches.

*Inclusion criteria.* Researchers retained peer-reviewed, full-text-accessible studies that were published in English between 2012 and 2017. Only studies conducted in youth ages 0–18 y who had not yet entered college were included. Sleep-related independent variables included: duration (total sleep time - TST), time in bed (TIB), timing (bedtime, wake time, sleep midpoint, chronotype), quality (sleep onset latency – SOL), wake after sleep onset (WASO), sleep efficiency (SE), nighttime awakenings, subjective sleep quality, subjective daytime sleepiness, parent-report sleep disorder symptoms, variability (night-to-night variability, social jetlag), and PSG-derived architecture variables (time or percentage of sleep spent in Stage 1, 2, SWS, REM). Outcome variables included: dietary intake (overall dietary quality, total caloric intake, specific macronutrients, sugar-sweetened beverages – SSBs, caffeine, sweets/desserts, fruits and/or vegetables, snacks, fast foods), altered eating behavior or patterns (food appeal, stress or emotion-driven eating, disordered eating, meal-skipping), physical activity (sedentary time, screen time, light physical activity, moderate-to-vigorous physical activity - MVPA, perceptions of physical activity, cardiorespiratory fitness), and hormones regulating hunger or satiety (leptin, ghrelin, adiponectin, orexin). For more information on specific measures used by studies to operationalize any of these constructs, see Supplemental Tables 7–10.

*Exclusion criteria.* Due to the potential for non-sleep influences on weight-related factors, studies conducted with clinical populations (diagnosed developmental delay, medical or psychiatric disorder) were excluded. This included non-insomnia sleep disorders with a known medical foundation (e.g., obstructive sleep apnea, periodic limb movement disorder).

### *Study selection and data charting*

After removal of duplicate papers, the first author (KNK) reviewed all titles/abstracts for inclusion/exclusion criteria. The second author (MLC) independently reviewed approximately 50% of these abstracts, and inclusion decisions were compared until a reliability index of .95 was reached. Disagreements were resolved via discussion. First author (KNK) extracted the following information from the full-text of included studies: study authors, publication year, study location, study design, sample size, age range, sleep measurement, sleep variable, outcome variable measurement, outcome variable, and key findings. The resulting extracted tables were separated into four main outcome variables: dietary intake, physical activity, hormones, and altered eating behaviors/patterns. When studies included information about multiple outcome domains (e.g., total caloric intake and MVPA), they were considered in each domain separately. The second author (MLC) then cross-referenced all extracted information.

### *Risk of bias*

Experimental studies were critically assessed for risk of bias using the Cochrane system [10]. Observational studies were appraised using a modified version of the Research Triangle Institute (RTI) 13-item bank [11], in which one question about overall study quality was dropped to avoid redundancy when computing an overall risk of bias index. Studies with multiple outcomes of interest had risk of bias assessed separately for each outcome. Bias risk appraisals were independently completed by both reviewers (KNK and MLC) and discrepancies were discussed until a 100% consensus was achieved. A risk of bias index was computed by dividing the total number of items rated "no" (indicating low risk of bias) by the total number of items assessed, consistent with methods used in previous systematic reviews [12]. The following cut points were used to deem overall study quality: 0–.40 = high risk of bias, .41–.7 = medium risk of bias, and .71–1.0 = low risk of bias.

*Final study sample*

As shown in Fig. 1, 6,204 citations were identified via the electronic database searches. After removing duplicates, 4,307 citations were screened for inclusion/exclusion criteria, of which 86 unique studies were retained. Broken down, this resulted in 48 studies reviewed within the area of dietary intake, 19 within the area of eating behavior/patterns, 50 within the area of physical activity, and seven within the area of hormones.

## Results

*Overview of study designs, measurement methods, and risk of bias*

See Fig. 2 for breakdown of study design and measurement methods within each domain. Experimental studies generally had a medium risk of bias (see Tables S2-6 in the Supplemental Materials for item-by-item ratings of each study), with lack of participant blinding, allocation concealment, and reporting on missing/incomplete data being most common. Risk of bias was much higher among observational studies. Primary areas of biases included unvalidated/unreliable measurement, lack of participant/rater blinding, and common method variance (e.g., using the same rater for both sleep and dietary intake or same device to measure sleep and physical activity). Most observational studies reported secondary analyses on data initially collected for other purposes, making it difficult to evaluate selective outcome reporting. Because of the intrinsically-objective nature of assays, hormone-related studies generally had a lower (medium) risk of bias, but other measurement issues remained common, including the risk for possible circadian effects on hormone levels.

*Synthesis of results*

*Dietary intake.* See Table 1 for key findings within each subsection of dietary intake, Table S2 for review of risk-of-bias by study, and Table S7 for comprehensive review of study characteristics and main findings. Overall, findings were mixed. Experimental data fairly consistently showed that restricting time in bed (TIB) increased overall caloric intake, despite cross-sectional studies generally finding no such relationship (although most cross-sectional studies had serious measurement limitations that could have attenuated associations). Sleep was not related to fast food or macronutrient (fat, carbohydrate, or protein) intake in most cross-sectional (and all experimental) studies. Evidence linking total sleep time (TST) and sugar-sweetened beverages (SSBs) or fruits/vegetables was inconclusive, with both areas lacking high-quality experimental studies and showing inconsistencies across the higher-quality observational studies. Longer TST tended to correlate with higher scores on measures of dietary quality, which differed across studies but encompassed more intake of fruits, vegetables, and lean proteins and less processed foods. However, that area also lacked higher-quality longitudinal or experimental designs. Conversely, there was strong experimental and higher-quality cross-sectional evidence linking short sleep with greater intake of sweets/desserts. Although fewer in number, most relevant studies found that later and more variable sleep was associated with snacking, greater caloric intake, and consumption of unhealthy foods over fruits and vegetables. Most studies found no relationship between sleep quality or PSG-derived variables and dietary intake.

Taken together, results indicate some promising and less-promising areas for growth. Patterns of findings do not support

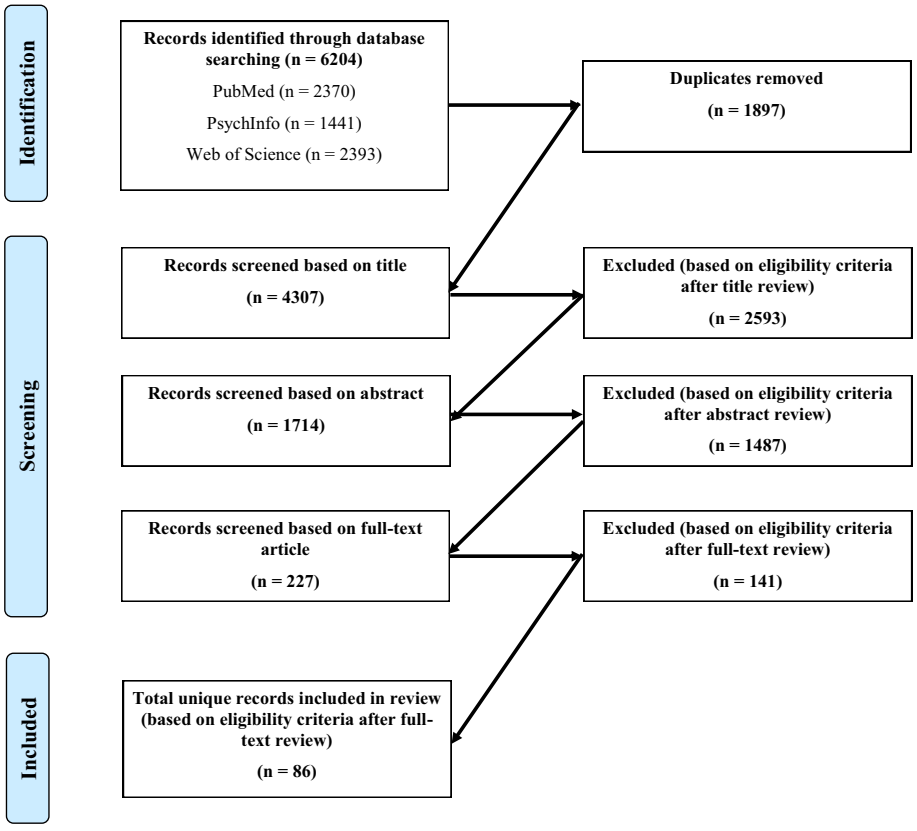


**Fig. 1.** Flow chart of data charting.

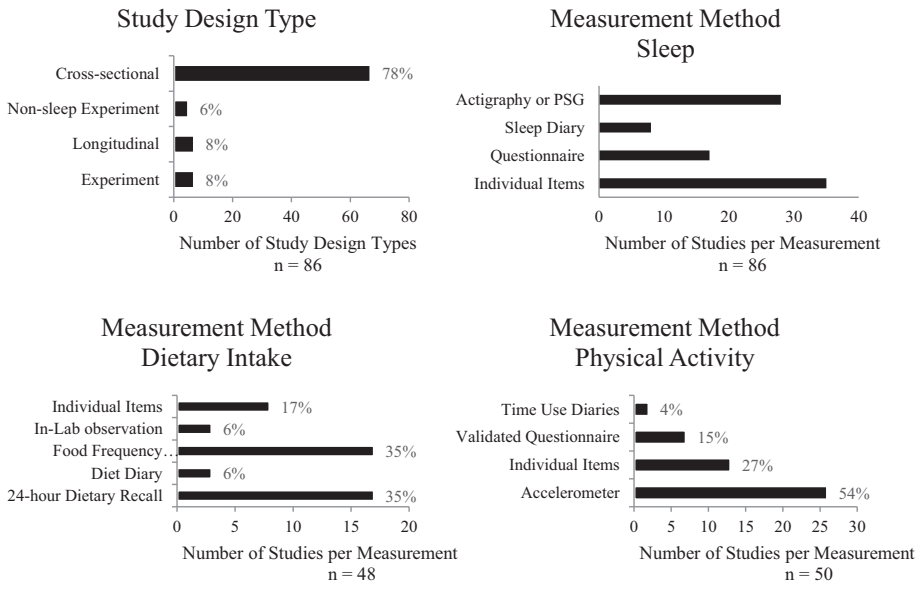


**Fig. 2.** Breakdown of study design and measurement of sleep, dietary intake, and physical activity. Note: PSG = polysomnography.

**Table 1**
Summary of findings linking sleep with various domains of dietary intake.

| Factor | Key Findings |
|---|---|
| Caloric intake | Findings differ by methodology. All experimental studies find restricting TIB increases caloric intake; cross-sectional evidence shows no association. No studies find the opposite. Some evidence that later, poorer, and more variable sleep associated with greater caloric intake, perhaps most in the evening. |
| Macronutrients | Growing evidence for null or negligible effect. Most (about 2/3) cross-sectional and all experimental studies find no association between TST and fat, carbohydrates, or protein intake; remaining studies yield positive and negative results. Some indication that more variable sleep relates to higher carbohydrate and fat intake. |
| SSBs or Caffeine | Inconclusive evidence. Half of studies find longer TST associated with less SSB consumption; other half find no relationship. Mixed literature on caffeine. Small but more consistent literature finds that more variable sleep is related with greater SSB consumption. |
| Sweets and Desserts | Inconclusive but suggestive evidence. Half of cross-sectional studies find longer TST associated with less sweets/dessert intake; other half find no relationship. None report the opposite relationship. Stronger experimental evidence linking short TST with increased sweets/dessert consumption. No strong findings with other sleep measures. |
| Fruits and vegetables | Inconclusive evidence. Half of studies (including one longitudinal) find longer TST associated with greater fruit/vegetable consumption; other half (including one longitudinal) find no relationship. No studies report the opposite relationship. Small but more consistent literature show that later sleep timing or chronotype relates to less fruit/vegetable intake. |
| Snacking | Growing evidence for null or negligible effect. Most (about 2/3) studies find no association with TST; remaining studies (including one longitudinal) find longer TST associated with less snacking. Small but consistent literature finds later sleep timing or greater sleep variability related to more snacking. |
| Fast Food | Growing evidence for null or negligible effect. Most (about 2/3) cross-sectional studies find no association with TST; remaining studies find longer TST related to less fast food. |
| Dietary Quality | Growing evidence for positive relationship. Most (about 3/4) cross-sectional studies find that longer TST is associated with various measures of higher dietary quality; no studies showed the opposite. Variable findings relating sleep timing/variability with dietary quality. |

Citations: [13–40, 41–60], detailed in Tables S2 and S7.
Note: TST = total sleep time.

further inquiry into TST and macronutrient intake; simple counts of fats, carbohydrates and protein seem to have weak or negligible links to pediatric sleep. Similarly, further cross-sectional studies on TST and total caloric intake seem unlikely to move the field past a correlational literature saturated with null effects and prone to confounding factors. However, high-quality experimental studies of caloric intake that not only manipulate sleep duration, but also sleep timing, variability, or quality could further clarify a cause-and-effect relationship. Given the growing trend in cross-sectional findings linking TST with dietary quality, it would be reasonable to invest in further examining these relationships through high-quality observational studies or experimental manipulations. Similarly, because the most rigorous study designs with lowest risk of bias consistently linked TST with sweets/dessert consumption, further research explicating this link appears promising. Finally, given the small literature linking measures other than sleep duration to dietary intake, future studies should consider investigating the role of sleep timing, variability, and quality.

*Altered eating behaviors/patterns.* See Table 2 for key findings within each sub-section of altered eating behaviors/patterns, Table S3 for review of risk-of-bias by study, and Table S8 for comprehensive review of study characteristics and main findings. There were very few studies examining sleep and altered eating behaviors or patterns, most with a high risk of bias. Most cross-sectional studies did not indicate relationships between sleep and snacking, disordered eating, or stress-eating. Despite the potential to draw interesting parallels to hormonal studies, sleep was also not clearly linked to self-reported hunger or satiety. However, findings did indicate some trends worth further examination. Experimental studies tended to indicate that shorter and later sleep was associated with greater food appeal and desire to eat sweet or high-glycemic-index foods, and a growing number of longitudinal

**Table 2**
Summary of findings linking sleep with various domains of altered eating behavior and patterns.

| Factor | Key Findings |
|---|---|
| Hunger and Satiety | Inconclusive evidence. Some experimental and cross-sectional studies find shorter TST or less SWS is associated with higher hunger ratings and the rest find no association; no studies showed the opposite. No indication for connection between TST and satiety ratings. |
| Food Appeal | Growing evidence of positive effect. Small but growing experimental evidence that restricting TIB increases appeal of sweet, salty, fatty, starchy, or high glycemic foods. |
| Eating in Response to External Cues, Stress, or Emotions | Inconclusive evidence. Literature is very small and cannot be compared across various outcome measures. Individual studies suggest that shorter, more disrupted, or more variable sleep associated with greater external or emotion/stress-driven eating. |
| Disordered Eating | Inconclusive evidence. Literature is very small and cannot be compared across various outcome measures; however, several individual studies indicate that later, more disturbed, or shorter sleep associated with disordered eating. |
| Breakfast-skipping | Growing evidence of positive effect. Fairly consistent cross-sectional and longitudinal findings that shorter and later sleep related to more breakfast-skipping. |

Citations: [15,16,27,28,34,39,40,42,45,49,50,54,63–70], detailed in Tables S3 and S8.
Note: TST = total sleep time.

and cross-sectional studies found links to greater breakfast-skipping. A handful of studies found that sleep quality, variability, and architecture were related to various measures of altered eating behaviors.

Individual studies indicated interesting results, but were hard to compare due to differing outcome measures. It will be important for researchers to establish and more consistently use the best measures of constructs such as hunger and satiety, food appeal, and emotional eating. These would likely include both questionnaires (e.g., Power Food Scale) [61] and behavioral tasks (e.g. Behavioral Choice Task) [62] that are validated in youth and shown to discriminate between healthy weight and overweight samples. Future studies that assess frequency and timing of meals across multiple days (e.g., via ecological momentary assessment or dietary recalls) would be particularly useful in clarifying the relationship between TST or sleep timing with meal-skipping. Given the strong cross-sectional findings, experimental research in this area is also warranted.

*Physical activity.* See Table 3 for comprehensive review of study characteristics and main findings from physical activity (PA) articles, Table S4 for risk-of-bias by study, and Table S9 for detailed review of study characteristics and findings. Studies consistently associated shorter and later sleep with greater sedentary and screen time. This held across all study designs and all levels of risk of bias. In contrast, results relating TST to any other accelerometer-based PA markers (e.g., moderate-to-vigorous PA - MVPA, energy expenditure, activity count/epoch) were mixed. Even cross-sectional and experimental "exemplar studies" directly contradicted each other, with positive, negative, and null findings. When assessed subjectively, PA tended to be either unrelated or positively related to TST. Like other areas, a greater proportion of the (albeit smaller) literature on sleep timing (but not sleep efficiency or sleep architecture) suggested this is a promising area for exploration.

Next-steps in the area of physical activity should include experimental studies that examine the causal influence of changing duration, timing, or stability of sleep on sedentary time or screen

**Table 3**
Summary of findings linking sleep with physical activity.

| Factor | Key Findings |
|---|---|
| Sedentary Time | Growing evidence for effect. Most cross-sectional studies find that shorter and later sleep is associated with more sedentary time. |
| Screen Time | Growing evidence for effect. Most studies (about 2/3, including one longitudinal and one experimental) find that shorter TST is associated with more screen time use. Later, more variable, or poorer quality sleep is associated with more screen time use in greater proportion (albeit smaller) of studies. |
| Objective Light PA | Inconclusive evidence. Mixed results - studies find null, positive, and negative relationships with TST. |
| Objective MPA, VPA | Growing evidence for null or negligible effect. In this small literature, TST is consistently unrelated to MPA or VPA. Some indication that later sleep timing or higher sleep efficiency is associated with less MPA (but not VPA). |
| Objective MVPA | Inconclusive evidence. About half of studies find no relationship with TST; the remaining half find both positive and negative associations. Later sleep timing associated with less MVPA in half of studies, with the other half finding no relationship. Most studies find no relationship with SE. |
| Energy Expenditure or Activity Count per Epoch | Inconclusive evidence. Severe experimental TIB restriction increases spontaneous PA and energy expenditure, whereas moderate TIB restriction decreases average activity count/epoch. |
| Questionnaire- or Time Use Diary-based PA | Inconclusive evidence. About half of studies find that longer TST associated with greater questionnaire-based PA, whereas the other half (including one longitudinal and one experimental) find no association. Although literature is small, findings consistently show greater daytime sleepiness (but not later sleep timing) related to lower questionnaire-based PA. |
| Self-Reported PA | Inconclusive evidence. About half of studies find no relationship and the other half find that longer TST is related to greater moderate self-reported PA or greater odds of meeting recommendations for daily PA. |
| Cardiorespiratory Fitness and Strength | Inconclusive evidence. Very small literature. Some evidence for a positive relationship between TST and muscle strength (but not cardiorespiratory fitness). |

Citations: [16,21,25,32,34,36,37,39–41,43,45–47,51,60,71–97,105], detailed in Tables S3 and S9.
Note: PA = physical activity; MPA = moderate physical activity; MVPA = moderate-to-vigorous physical activity; TST = total sleep time; TIB = time in bed.

time use, perhaps particularly in the evening. Despite mixed findings, meta-analysis on sleep and accelerometer-based PA could illuminate possibly under-powered effects. Although not the focus of the current review, there were a growing number of studies that found interesting within-person, temporal relationships between sleep and various measures of PA that should be further explored.

*Hormones regulating hunger and satiety.* See Table 4 for comprehensive review of study characteristics and main findings from articles reviewing hormones, Table S5 for risk-of-bias by study, and Table S10 for detailed review of study characteristics and findings. Overall, studies connections sleep and hormones regulating hunger and satiety were highly mixed. Over half of the studies, spanning design type and risk of bias, found no associations between any sleep marker and leptin or ghrelin. The remaining studies found both positive and negative relationships, further obscuring the picture. No studies indicated a relationship between TST and adiponectin or orexin.

Biomarkers such as leptin and ghrelin are very sensitive to circadian effects and even minor uncontrolled influences (e.g., health status, body fat). As such, future studies in this area must take great care in their study design and implementation, as well as controlling for various confounding covariates. At a minimum, studies must control for time-of-day of blood draws; ideally, circadian phase of study participants would also be considered. Readers are referred to Hagen and colleagues' 2015 review for a more thorough discussion of future directions.

## Discussion

### *Summary of results*

This systematic review synthesized studies published after Hart and colleagues' 2011 [4] call for research investigating pathways linking pediatric sleep and obesity. Given the broad scope of this review, the 86 studies published since that 2011 review were included. The aim was to provide a timely and concise update on the current state of the science regarding the mechanisms linking pediatric sleep to obesity. As often occurs as science moves beyond initial work to more thorough examination, findings become more nuanced. All three early mechanistic studies included in Hart and colleagues' review suggested an effect of short sleep on eating behaviors, but the current review's findings were not as conclusive. Systematic review revealed considerable variability in findings linking pediatric sleep with eating behaviors/patterns as well as with dietary intake, physical activity, and hormones. However, findings were consistent enough to suggest that some directions are more promising than others. Although there is no resolution regarding the TST-caloric intake relationship (with distinct discrepancies in experimental vs. correlational findings), links between longer TST and both healthier eating patterns and less sedentary/screen time seem more robust. While less-often studied, greater sleep variability and later sleep timing also show signs of being related to caloric intake, breakfast-skipping, poorer dietary quality and patterns, more screen time, and less moderate-to-vigorous physical activity.

If future mechanistic studies replicate these findings, there may be direct implications for interventional research. Researchers may consider creative ways to expedite this process, such as designing experimental trials that extend or stabilize sleep to identify mechanisms, while also establishing feasibility of future interventions. Researchers may also consider measuring or intervening upon sleep to augment established behavioral interventions (e.g., weight loss trials, pain treatment programs). Although adult studies are often cited to support a relationship between sleep and hormones related to hunger and satiety, this review agrees with Hagen and colleagues' 2015 appraisal that the data in children and adolescents is unconvincing [7]. This apparent developmental discrepancy highlights the importance of studying children directly rather than simply extrapolating from adult findings.

### *Literature gaps, risk of bias assessment, and future directions*

Most observational studies of dietary intake, altered eating behavior, and physical activity showed a high risk of bias using established metrics. This underscores the importance of considering individual and group-based findings within the scope of that literature's limitations. For example, domains in which study findings were highly inconsistent and had a high risk of bias may have had issues with detecting potential effects. Conversely, domains in which higher-quality studies show significant effects despite more mixed findings from studies with high risk of bias suggest that further high-quality work is warranted.

Overall, the literature continues to lack experimental and longitudinal research, particularly pertaining to physical activity. The recent increase in experimental studies since the last pediatric sleep/obesity review (from three in 2011 to thirteen in 2011–2017) [4] highlights the feasibility of these study designs. Given limitations of observational studies, experimental studies (although resource-intensive) seem worthwhile. Cohort studies that investigate developmental trajectories, duration, and chronicity of sleep behaviors would also be valuable considering that sleep, physical activity, and dietary intake, and feeding behaviors all present differently across development. Within the cross-sectional literature, there was a notable reliance on group-level study designs. This offers a limited vantage point, given that childhood health behaviors (e.g., sleep [100], physical activity [101], dietary intake [102]) tend to be individually variable day-to-day. Traditional statistical methods that treat mean-level trajectory deviations as error may not have the resolution to detect non-linear effects or individual differences. Future studies should consider investigating non-linear, temporal, bi-directional, or within-subjects relationships.

Across all domains and study designs, uncontrolled confounding was one of the greatest areas of concern. Many studies did not consider, assess for, or report on what are considered to be standard

**Table 4**
Summary of findings linking sleep with hormones regulating hunger and satiety.

| Hormone | Key Findings |
|---|---|
| Leptin | Inconclusive evidence. Experimental, longitudinal, and cross-sectional studies find null, positive, and negative relationships between TST or other sleep markers and leptin. |
| Ghrelin | Inconclusive evidence. Experimental and cross-sectional studies find null, positive, and negative relationships between TST or other sleep markers and ghrelin. |
| Adiponectin | Growing evidence for null or negligible effect. No evidence for relationship with TST. |
| Orexin | Growing evidence for null or negligible effect. No evidence for relationship with TST. |

Citations: [13,16,24,43,56,98,99], detailed in Tables S5 and S10.
Note: TST = total sleep time.

covariates in other literature (e.g., age, sex, race/ethnicity, SES), and future studies should include results with/without covariates to allow for future meta-analyses. Participants and assessors generally were not (and often could not) be blinded to the predictor (sleep) or outcome variables. As most studies were conducted as secondary data analyses and/or did not report on their *a priori* outcomes of interest, risk of selective outcome reporting was often unclear, and may be underestimated in this review. Within longitudinal and experimental studies, details were often lacking related to randomization, attrition, and detection bias (i.e., how the impact of drop-outs or missing data was assessed). Researchers are encouraged to clearly report on methods for handling randomization, allocation concealment, and missing data in future studies. Common method variance was rampant, with most observational studies using the same reporter or device to measure both predictor and outcome variables. None of the reviewed physical activity studies used separate devices to measure sleep and activity. Beyond statistical issues with shared method variance, this introduces validity concerns, as there are currently no validated devices explicitly developed for both sleep and physical activity monitoring in youth. Readers are directed to Lang and colleagues (2016) [103] for a review of the current challenges in the area of sleep and activity monitoring in adolescence. Future studies should prioritize having independent raters for sleep and outcome variables or using separate (and validated) devices for measuring sleep and physical activity.

Use of unreliable or unvalidated measurement methods was the rule rather than the exception, with 60% of studies measuring via 1–2 items (e.g., "How many hours does your child typically sleep?"). Despite the convenience of this measurement method, concerns about reporting bias, low reliability, and limited scope likely outweigh the benefits. Instead, researchers should consider the combined use of objective and subjective methods with good ecological validity (i.e., actigraphy, sleep diaries) and at least seven d of data (to ultimately capture at least three weeknights and two weekend nights) for accurate sleep pattern representation [104]. Given the small literature, it is hard to know how well positive findings identified in this review will stand up to replication, but early findings encourage researchers to look beyond average sleep duration to more "novel" markers of sleep. This could include measures of sleep quality (e.g., sleep efficiency, sleep onset latency, wake after sleep onset, self-reported sleep quality), variability (e.g., intra-individual standard deviation, weekend oversleep, social jetlag), timing (e.g., bed- and wake-times, chronotype, circadian phase), and architecture (e.g., Stage 1, 2, slow wave sleep, rapid eye movement sleep).

Poor measurement was also particularly problematic in dietary intake studies. Although measuring dietary intake is inherently challenging (e.g., controlling for effects of monitoring or social desirability bias on intake, missing data due to youth unawareness of eating occasions), some methods are more supported than others. In this review, 70% of studies used food frequency questionnaires, which inflates risk of memory bias and limits the scope of information provided. Future studies that need to minimize cost might consider using 24-hour dietary recalls obtained over several days. This would provide valuable information beyond just caloric intake (i.e., day-to-day variability in intake, patterns of intake timing/frequency). Additional studies with direct behavioral observation of meals would allow for more internally valid estimates of dietary intake, feeding behavior, and mealtime interactions. Within the domain of physical activity, almost half of reviewed studies used accelerometers with algorithm-determined measures of sedentary, light, moderate, vigorous, and moderate-to-vigorous physical activity as their main outcomes. This is an encouraging trend, as such devices allow for measurement in the free-living environment and makes comparison across studies easier. Future studies seeking to investigate more novel physical activity targets may examine how sleep impacts differences in physical activity timing (e.g., pattern of activity across the day), frequency, and intensity (e.g., peak activity within an epoch).

Finally, future work should expand on this review to include special populations, specifically those in which sleep and/or weight problems are most common. For example, obesity is highly prevalent in genetic conditions (e.g., Prader–Willi Syndrome, Down's syndrome), as well as obstructive sleep apnea (OSA). Yet, little is known about how sleep and/or treatment status in pediatric OSA impacts weight-related behaviors, or whether these relationships differ in youth who are typically developing vs. medically- or developmentally complex. As discussed by several of the papers included in this review [25,52,74,83,95], youth from low-SES or minority backgrounds are at heightened risk for poor sleep and obesity. Given the vulnerability of this population, focusing research efforts on sleep as a health equity target will also be an important future frontier. Although basic knowledge about interactions between sleep and obesogenic behaviors is still needed, researchers may want to thoughtfully consider the unique societal, environmental, contextual, and family/cultural factors that influence or mediate those relationships. Finally, relatively little mechanistic sleep/obesity work has been done with very young children, despite the fact that sleeping and feeding are the primary activities of infancy. A greater understanding of how these two processes interact early in development and over time could offer insights into early obesity prevention efforts that target sleep and feeding together.

### *Limitations*

Findings of this review should be considered in light of several limitations. Only peer-reviewed published articles were included, which could have introduced bias against certain studies, particularly studies with null findings (that often go unpublished). Due to the complexity of the research question, findings could not be stratified by youth developmental level and needed to include studies that varied in whether/how they controlled for confounders, both of which could influence results. Finally, given the broad scope of this review, it was necessary to group nuanced predictor and outcome variables to succinctly present information, which could have potentially obscured fine-grained effects.

## Conclusions

Overall, mechanistic studies designed to identify weight-related factors driving the pediatric sleep/obesity relationship have generally not delivered on initial excitement. To date, there are no unifying patterns of findings that fully explain how sleep impacts weight status in youth. Results from this review highlighted issues with risk of bias in this literature, as well as considerable variability in the relationships between markers of sleep and most weight-related factors. Because of widely discrepant findings, the literature is vulnerable to selective citation, so researchers are cautioned to be discerning when reading or citing this literature, as well as when they are designing future studies. The majority of the studies included in this review were cross-sectional and group-based. There is a clear need for more experimental, longitudinal, with-in subjects, bi-directional, and novel cross-sectional studies. Although there has been much improvement in the measurement of physical activity, future studies would benefit from more rigorous measurement of various markers of sleep and dietary intake. Finally, although studies of sleep and dietary intake or altered feeding behavior are likely too heterogeneous for formal

meta-analysis, it would be reasonable to pursue a meta-analysis in the area of sleep and physical activity.

**Practice points**

- Despite meta-analytic evidence of a pediatric sleep/obesity link, the field still does not understand driving mechanisms. The literature is more mixed than consistent.
- The few exceptions included: 1) consistent experimental (but not cross-sectional) evidence that restricting time in bed increases caloric intake, 2) cross-sectional results linking longer sleep with higher dietary quality, and 3) cross-sectional findings suggesting that shorter and later sleep is related to more sedentary activity, screen time, and breakfast-skipping.
- Risk of bias is high in most observational studies, with particular concerns about confounding and shared method variance.
- Encouragingly, experimental studies and objective sleep/physical activity measurement have increased in the past 10 y. This points to the feasibility of using high-quality, creative methods to assess these relationships.
- For the field to move forward, effort and resources will need to be invested into conducting high-quality studies that address methodological issues.

**Research agenda**

Specific areas warranting further inquiry include:

- Experimental studies that investigate effect of manipulating sleep duration or timing on desire for sugary/sweet foods or beverages, overall dietary quality, breakfast-skipping, and sedentary/screen time.
- Meta-analysis within the area of sleep and physical activity.

All future empirical research studies should:

- Employ rigorous measurement of sleep and outcome variables, being mindful to use different reporters or devices for each.
- Prospectively collect data across multiple days, rather than rely on retrospective report.
- Investigate sleep variables beyond just sleep duration (e.g. timing, variability, quality).

## Conflicts of interest

None.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.smrv.2019.04.010.

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