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SLEEP DURATION, OBESITY, AND INSULIN RESISTANCE IN CHILDREN AND ADOLESCENTS

DURAÇÃO DO SONO, OBESIDADE E RESISTÊNCIA À INSULINA EM CRIANÇAS E ADOLESCENTES

DURACIÓN DEL SUEÑO, OBESIDAD Y RESISTENCIA A LA INSULINA EN NIÑOS Y ADOLESCENTES

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Natália Bóscolo Gutierrez Borges[1], Ana Beatriz Finamore Horta[2], Beatriz Rolim Mendes Baptista[3], Leonardo Batista de Souza Zanguettin[4], Carolina Machado Ferreira[5], Mariana Poiani dos Santos Capalti[6], Ana Amélia Teixeira Alcantara[7], Kellen Cristina Kamimura Barbosa Silva[8]

ABSTRACT

Introduction: Insufficient sleep, delayed sleep timing, and night-to-night irregularity are common during childhood and adolescence. These exposures coincide with critical periods of adiposity gain, pubertal insulin resistance, circadian reorganization, and increasing autonomy over food, activity, and digital-media use. Short sleep is consistently associated with obesity, but the independent relationship with insulin resistance and the reversibility of metabolic risk remain less certain.

Objective: To critically synthesize evidence linking sleep duration with obesity and insulin resistance in children and adolescents, distinguish duration from timing and regularity, quantify the principal observational and experimental effects, and translate the evidence into clinically useful assessment and management.

Methods: A structured narrative review of PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library was conducted through August 31, 2026. Pediatric systematic reviews, meta-analyses, prospective cohorts, objective sleep studies, randomized sleep-manipulation experiments, and sleep-focused obesity-prevention or treatment trials were prioritized. Total and central adiposity were analyzed separately from fasting insulin, homeostatic model assessment of insulin resistance, oral-glucose-tolerance-test indices, and incident insulin resistance.

Results and Discussion: Prospective meta-analyses consistently associate short sleep with later overweight or obesity, with pooled relative risks generally between 1.30 and 1.58 and a stronger signal in school-aged children than in adolescents. Experimental restriction increases energy-dense food intake, dietary glycemic load, and eating in the absence of hunger, providing a plausible behavioral pathway. Sleep-extension trials reliably increase

[1] Universidade Federal do Triângulo Mineiro (UFTM). E-mail: naboscolo@gmail.com
[2] Faculdade de Medicina do ABC (FMABC). E-mail: abfhorta@gmail.com
[3] Instituto de Assistência Médica ao Servidor Público Estadual (IAMSPE). E-mail: Beatrizsrmb@gmail.com
[4] Universidade de Marília (UNIMAR). E-mail: Leonardozanguettin@gmail.com
[5] Universidade Federal do Triângulo Mineiro (UFTM). E-mail: d202020193@uftm.edu.br
[6] Faculdade de Medicina de São José do Rio Preto (FAMERP). E-mail: mrcapalti@gmail.com
[7] Faculdade de Medicina de São José do Rio Preto (FAMERP). E-mail: anamelialc@gmail.com
[8] Universidade Federal do Triângulo Mineiro (UFTM).

sleep, but effects on body mass index are small, heterogeneous, and frequently absent in intention-to-treat analyses. Evidence concerning insulin resistance is more complex: cross-sectional associations often attenuate after adjustment for adiposity and puberty, whereas a two-year actigraphy cohort found that sleeping at least one hour below age recommendations was associated with 2.74-fold higher odds of developing insulin resistance. In a randomized crossover trial of adolescents with obesity and type 2 diabetes risk, one week of sleep extension increased oral-glucose-tolerance-test-derived insulin sensitivity by 20% compared with habitual and restricted sleep.

Conclusion: Adequate and regular sleep should be considered a fundamental component of pediatric metabolic health, but not a stand-alone obesity treatment. Evidence supports a modest prospective contribution to obesity and a potentially reversible effect on insulin sensitivity in high-risk adolescents. Clinical care should assess sleep duration, timing, variability, obstructive sleep apnea, pubertal stage, and adiposity together, while future trials should evaluate sustained metabolic effects using objective sleep measurements and robust glucose phenotyping.

Keywords: Sleep Duration. Pediatric Obesity. Insulin Resistance. Circadian Rhythm. Child. Adolescent.

RESUMO

Introdução: O sono insuficiente, o atraso no horário de dormir e a irregularidade entre as noites são comuns durante a infância e a adolescência. Essas exposições coincidem com períodos críticos de ganho de adiposidade, resistência à insulina durante a puberdade, reorganização circadiana e aumento da autonomia em relação à alimentação, à atividade física e ao uso de mídias digitais. O sono curto está consistentemente associado à obesidade, mas a relação independente com a resistência à insulina e a reversibilidade do risco metabólico permanecem menos esclarecidas.

Objetivo: Sintetizar criticamente as evidências que relacionam a duração do sono à obesidade e à resistência à insulina em crianças e adolescentes, distinguindo duração, horário e regularidade do sono, quantificar os principais efeitos observacionais e experimentais e traduzir as evidências em estratégias de avaliação e manejo clinicamente úteis.

Métodos: Foi realizada uma revisão narrativa estruturada nas bases PubMed/MEDLINE, Scopus, Web of Science e Cochrane Library até 31 de agosto de 2026. Foram priorizadas revisões sistemáticas pediátricas, metanálises, coortes prospectivas, estudos objetivos do sono, experimentos randomizados de manipulação do sono e ensaios de prevenção ou tratamento da obesidade com foco no sono. A adiposidade total e central foi analisada separadamente da insulina de jejum, do modelo de avaliação da homeostase da resistência à insulina, dos índices derivados do teste oral de tolerância à glicose e da incidência de resistência à insulina.

Resultados e Discussão: Metanálises prospectivas associam consistentemente o sono curto ao sobrepeso ou à obesidade subsequente, com riscos relativos agrupados geralmente entre 1,30 e 1,58 e uma associação mais forte em crianças em idade escolar do que em adolescentes. A restrição experimental do sono aumenta o consumo de alimentos energeticamente densos, a carga glicêmica da dieta e a ingestão alimentar na ausência de

fome, fornecendo uma via comportamental plausível. Os ensaios de extensão do sono aumentam de forma consistente a duração do sono, mas os efeitos sobre o índice de massa corporal são pequenos, heterogêneos e frequentemente ausentes nas análises por intenção de tratar. As evidências relativas à resistência à insulina são mais complexas: as associações transversais frequentemente diminuem após o ajuste para adiposidade e puberdade, enquanto uma coorte de dois anos baseada em actigrafia constatou que dormir pelo menos uma hora abaixo das recomendações para a idade esteve associado a uma probabilidade 2,74 vezes maior de desenvolver resistência à insulina. Em um ensaio clínico randomizado e cruzado com adolescentes com obesidade e risco de diabetes tipo 2, uma semana de extensão do sono aumentou em 20% a sensibilidade à insulina derivada do teste oral de tolerância à glicose, em comparação com o sono habitual e o sono restrito.

Conclusão: O sono adequado e regular deve ser considerado um componente fundamental da saúde metabólica pediátrica, mas não como tratamento isolado para a obesidade. As evidências sustentam uma contribuição prospectiva modesta para a obesidade e um efeito potencialmente reversível sobre a sensibilidade à insulina em adolescentes de alto risco. A assistência clínica deve avaliar conjuntamente a duração do sono, o horário de dormir, a variabilidade, a apneia obstrutiva do sono, o estágio puberal e a adiposidade, enquanto estudos futuros devem avaliar os efeitos metabólicos sustentados utilizando medidas objetivas do sono e uma caracterização robusta da glicemia.

Palavras-chave: Duração do Sono. Obesidade Pediátrica. Resistência à Insulina. Ritmo Circadiano. Criança. Adolescente.

RESUMEN

Introducción: El sueño insuficiente, el retraso en el horario de sueño y la irregularidad de una noche a otra son frecuentes durante la infancia y la adolescencia. Estas exposiciones coinciden con períodos críticos de aumento de la adiposidad, resistencia a la insulina durante la pubertad, reorganización circadiana y creciente autonomía respecto de la alimentación, la actividad física y el uso de medios digitales. El sueño de corta duración se asocia de manera consistente con la obesidad, pero la relación independiente con la resistencia a la insulina y la reversibilidad del riesgo metabólico siguen siendo menos claras.

Objetivo: Sintetizar críticamente la evidencia que relaciona la duración del sueño con la obesidad y la resistencia a la insulina en niños y adolescentes, distinguir la duración del horario y la regularidad del sueño, cuantificar los principales efectos observacionales y experimentales y traducir la evidencia en estrategias de evaluación y manejo clínicamente útiles.

Métodos: Se realizó una revisión narrativa estructurada de PubMed/MEDLINE, Scopus, Web of Science y Cochrane Library hasta el 31 de agosto de 2026. Se priorizaron revisiones sistemáticas pediátricas, metanálisis, cohortes prospectivas, estudios objetivos del sueño, experimentos aleatorizados de manipulación del sueño y ensayos de prevención o tratamiento de la obesidad centrados en el sueño. La adiposidad total y central se analizaron por separado de la insulina en ayunas, el modelo de evaluación de la homeostasis de la resistencia a la insulina, los índices derivados de la prueba oral de tolerancia a la glucosa y la incidencia de resistencia a la insulina.

Resultados y Discusión: Los metanálisis prospectivos asocian de manera consistente el sueño de corta duración con el sobrepeso o la obesidad posteriores, con riesgos relativos agrupados generalmente entre 1,30 y 1,58 y una asociación más fuerte en los niños en edad escolar que en los adolescentes. La restricción experimental del sueño aumenta el consumo de alimentos de alta densidad energética, la carga glucémica de la dieta y la ingesta en ausencia de hambre, lo que proporciona una vía conductual plausible. Los ensayos de extensión del sueño aumentan de manera consistente la duración del sueño, pero los efectos sobre el índice de masa corporal son pequeños, heterogéneos y con frecuencia están ausentes en los análisis por intención de tratar. La evidencia relacionada con la resistencia a la insulina es más compleja: las asociaciones transversales suelen atenuarse después del ajuste por adiposidad y pubertad, mientras que una cohorte de dos años basada en actigrafía encontró que dormir al menos una hora por debajo de las recomendaciones para la edad se asociaba con una probabilidad 2,74 veces mayor de desarrollar resistencia a la insulina. En un ensayo aleatorizado y cruzado con adolescentes con obesidad y riesgo de diabetes tipo 2, una semana de extensión del sueño aumentó en un 20% la sensibilidad a la insulina derivada de la prueba oral de tolerancia a la glucosa, en comparación con el sueño habitual y el sueño restringido.

Conclusión: Un sueño adecuado y regular debe considerarse un componente fundamental de la salud metabólica pediátrica, pero no un tratamiento aislado para la obesidad. La evidencia respalda una contribución prospectiva modesta a la obesidad y un efecto potencialmente reversible sobre la sensibilidad a la insulina en adolescentes de alto riesgo. La atención clínica debe evaluar conjuntamente la duración del sueño, el horario de sueño, la variabilidad, la apnea obstructiva del sueño, el estadio puberal y la adiposidad, mientras que futuros ensayos deberán evaluar los efectos metabólicos sostenidos mediante mediciones objetivas del sueño y una caracterización rigurosa de la glucemia.

Palabras clave: Duración del Sueño. Obesidad Pediátrica. Resistencia a la Insulina. Ritmo Circadiano. Niño. Adolescente.

1 INTRODUCTION

Sleep is a biologically active period that coordinates endocrine secretion, autonomic balance, appetite regulation, substrate utilization, memory, and behavior. The American Academy of Sleep Medicine recommends 9–12 hours of sleep per 24 hours for children aged 6–12 years and 8–10 hours for adolescents aged 13–18 years. These ranges describe sleep durations associated with optimal health and should not be interpreted as diagnostic thresholds that automatically establish causation in an individual child.[1]

Pediatric obesity is a chronic, heterogeneous disease influenced by biological susceptibility, social conditions, the food environment, physical activity, medication, neurodevelopment, and family systems. Current clinical guidance recommends comprehensive assessment of sleep and obesity-related comorbidities because insufficient sleep and obstructive sleep apnea (OSA) frequently coexist with excess adiposity and may reinforce cardiometabolic risk. Sleep assessment should therefore complement rather than replace nutritional, behavioral, medical, and social evaluation.[2]

The epidemiological literature is extensive but methodologically heterogeneous. A systematic review of 141 articles representing 110 independent samples and 592,215 participants concluded that longer sleep was favorably associated with adiposity, emotional regulation, academic achievement, quality of life, and several other health indicators. Associations with cardiometabolic biomarkers were less consistent, and most studies relied on self- or parent-reported sleep duration.[3]

This contrast represents one of the central interpretive problems in the field. Evidence relating sleep duration to obesity is comparatively consistent, whereas evidence concerning insulin resistance depends more heavily on measurement method, pubertal stage, adiposity, and adjustment for confounding. A positive association with obesity should not automatically be used to infer an independent effect on insulin sensitivity.[3]

Prospective studies reduce, but do not eliminate, reverse causation. A meta-analysis of longitudinal pediatric cohorts found that short sleep predicted subsequent overweight or obesity, with age-stratified relative risks of 1.40 during infancy, 1.57 during early and middle childhood, 2.23 among children aged 9 to younger than 12 years, and 1.30 among adolescents. Each additional hour of sleep was associated with only a small reduction in subsequent body mass index (BMI) or BMI z score, emphasizing that the population association is meaningful but the expected individual weight effect is modest.[4]

Another longitudinal meta-analysis of 11 studies and 24,821 participants estimated more than twice the odds of overweight or obesity among short sleepers. Differences in age, short-sleep definitions, follow-up, baseline adiposity, and publication bias likely explain part of the variation across pooled estimates. A contemporary interpretation should therefore avoid presenting one relative risk as universal and should instead describe a gradient according to developmental stage, chronicity, sleep timing, and regularity.[5]

Sleep duration is only one dimension of sleep health. A systematic review of multiple pediatric sleep dimensions found that duration demonstrated the most consistent association with obesity, while timing, regularity, efficiency, and daytime sleepiness were promising but less uniformly measured. Late timing may increase exposure to evening food and artificial light, whereas irregular timing may reflect social jetlag, unstable routines, or compensatory sleep.[6]

Long sleep also requires cautious interpretation. Extended reported duration may represent recovery from prior sleep deprivation, chronic illness, depression, medication use, socioeconomic adversity, or misclassification of time in bed as actual sleep. The observation of a U-shaped association in some studies does not establish that long sleep itself is metabolically harmful.[6]

The cardiometabolic question is narrower and more difficult. A systematic review found strong pediatric evidence for associations between inadequate sleep, adiposity, and elevated blood pressure, some evidence for insulin resistance, and inconsistent findings for lipids, inflammation, and metabolic syndrome.[7] A focused review of inadequate sleep and type 2 diabetes similarly identified mixed results across 23 studies, with several associations attenuating after adjustment for adiposity, pubertal maturation, sex, or age.[8]

Experimental manipulation of sleep duration provides biological plausibility but remains limited by small samples and short intervention periods. Sleep restriction may alter food choice, appetite regulation, circadian alignment, autonomic activity, and insulin action before measurable changes in body fat occur. Conversely, sleep extension trials have consistently increased sleep but have not produced a uniform or substantial reduction in body weight.[7,8]

This review therefore addresses four related but nonidentical questions: whether short sleep predicts later obesity; whether sleep timing and variability add information beyond average duration; whether sleep is independently associated with insulin resistance after accounting for adiposity and puberty; and whether increasing sleep changes dietary intake,

body weight, or insulin sensitivity. Maintaining these distinctions prevents stronger obesity evidence from being inappropriately interpreted as proof of diabetes prevention.

2 OBJECTIVES

2.1 PRIMARY OBJECTIVE

To evaluate the relationship between sleep duration, obesity, and insulin resistance in children and adolescents.

2.2 SECONDARY OBJECTIVES

1. To quantify prospective associations between short sleep and incident overweight, obesity, BMI, central adiposity, and body fat.
2. To determine whether sleep timing, night-to-night variability, social jetlag, or catch-up sleep modifies the association between sleep duration and adiposity.
3. To evaluate associations with fasting insulin, homeostatic model assessment of insulin resistance (HOMA-IR), oral-glucose-tolerance-test indices, and incident insulin resistance.
4. To examine the experimental effects of sleep restriction or extension on energy intake, food selection, appetite-related behavior, body weight, and insulin sensitivity.
5. To assess whether sleep-focused interventions prevent or treat pediatric obesity and identify the populations most likely to derive metabolic benefit.
6. To provide a clinical framework integrating sleep duration, timing, regularity, obesity severity, pubertal development, OSA, and diabetes risk.

3 METHODOLOGY

3.1 REVIEW DESIGN

A structured narrative review was undertaken because the available evidence includes prospective epidemiological research, cross-sectional objective sleep studies, experimental feeding investigations, randomized crossover sleep manipulation, and multicomponent obesity-prevention trials with heterogeneous exposures and outcomes.

3.2 INFORMATION SOURCES AND SEARCH STRATEGY

PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library were searched from database inception through August 31, 2026. Search concepts included

combinations of “child,” “adolescent,” “sleep duration,” “short sleep,” “sleep extension,” “sleep restriction,” “bedtime,” “sleep timing,” “sleep variability,” “social jetlag,” “catch-up sleep,” “obesity,” “adiposity,” “body mass index,” “waist circumference,” “insulin resistance,” “HOMA-IR,” “Matsuda index,” “glucose tolerance,” and “type 2 diabetes.”

Reference lists and linked reports from major systematic reviews, meta-analyses, clinical guidelines, and randomized trials were examined. Pediatric systematic reviews, prospective cohorts, studies using actigraphy or polysomnography, controlled sleep-manipulation experiments, and randomized interventions were prioritized.

Adult-only investigations, case reports, and studies without an interpretable sleep or metabolic outcome were excluded from the principal synthesis.

3.3 EXPOSURE AND OUTCOME FRAMEWORK

Sleep exposure was classified as:

1. Average sleep duration.
2. Duration relative to age-specific recommendations.
3. Weekday–weekend variability.
4. Sleep midpoint and bedtime.
5. Social jetlag and compensatory sleep.
6. Experimentally restricted or extended sleep.
7. Sleep-focused behavioral intervention.

Metabolic outcomes were divided into:

1. Total adiposity, including BMI, BMI z score, overweight, and obesity.
2. Central adiposity, including waist circumference, waist-to-height ratio, and visceral fat.
3. Dietary or appetite-related mediators.
4. Fasting glucose and insulin.
5. HOMA-IR.
6. Oral-glucose-tolerance-test-derived insulin sensitivity.
7. Incident or persistent insulin resistance.

3.4 EVIDENCE APPRAISAL

Observational studies were evaluated according to prospective temporal order, objective sleep measurement, repeated adiposity or metabolic outcomes, pubertal staging,

and adjustment for baseline BMI, physical activity, dietary intake, socioeconomic context, and OSA.

Randomized studies were assessed according to allocation method, achieved sleep separation, adherence, intention-to-treat analysis, intervention contamination, attrition, follow-up duration, and multiplicity of outcomes.

HOMA-IR was considered a feasible epidemiological surrogate rather than a universal diagnostic test. Insulin physiology changes substantially during puberty, and no single HOMA-IR cutoff has been validated across all pediatric ages, sexes, pubertal stages, and populations.

4 RESULTS

4.1 EVIDENCE PROFILE

The evidence base included large prospective meta-analyses of incident obesity, systematic reviews of multidimensional sleep and cardiometabolic health, actigraphy-based cohorts, controlled sleep-restriction feeding studies, randomized sleep-extension trials, preschool obesity-prevention interventions, and a randomized crossover trial with oral-glucose-tolerance testing in adolescents at elevated risk of type 2 diabetes.

Evidence was strongest for the prospective relationship between short sleep and subsequent adiposity. Evidence was intermediate for behavioral and dietary pathways and less mature for the long-term modification of insulin resistance.

4.2 DEVELOPMENTAL GRADIENT

The association between short sleep and obesity was generally stronger in preschool and school-aged children than in older adolescents. Younger children typically have greater parental control of routines, less autonomy over food and media, and larger differences between adequate and inadequate sleep relative to physiological need.[4–6]

During adolescence, biological circadian delay interacts with early school start times, academic demands, employment, social activity, and digital-media use. Short sleep becomes common across the population, reducing the contrast between exposed and comparison groups. Adolescents also acquire greater independence over food selection, bedtime, caffeine intake, and physical activity.[4–6]

Puberty independently reduces insulin sensitivity. This transient physiological change may resolve in healthy adolescents but can interact with obesity and genetic susceptibility to

produce sustained dysglycemia. Studies that apply a single HOMA-IR cutoff without pubertal staging risk classifying physiological variation as disease or obscuring a true sleep-related effect.[7,8]

Table 1

Quantitative observational and prospective evidence for adiposity and insulin resistance

Study	Sample	Sleep exposure	Outcome	Principal quantitative finding	Interpretation
Miller et al., 2018[4]	Prospective meta-analysis of pediatric cohorts	Short versus longer sleep	Incident overweight or obesity; BMI	RR 1.40 at 0–<3 years; 1.57 at 3–<9 years; 2.23 at 9–<12 years; and 1.30 at 12–18 years. Each additional hour was associated with BMI z-score change of −0.03	Consistent temporal association, but the expected individual BMI effect per additional hour was small
Fatima et al., 2015[5]	11 longitudinal studies; 24,821 participants	Short versus normal sleep	Incident overweight or obesity	Pooled OR 2.15, 95% CI 1.64–2.81	Large relative association with heterogeneous exposure definitions and residual confounding
Jansen et al., 2018[9]	528 adolescents aged 9–17 years; 6–7 days of actigraphy	Duration and variability phenotype	BMI z score and obesity prevalence	Insufficient-stable sleep was associated with BMI z score +0.68 and obesity prevalence ratio 2.54 compared with sufficient-stable sleep	Consistently insufficient sleep carried the strongest adiposity signal
Matthews et al., 2012[18]	245 healthy adolescents	Diary and actigraphic sleep duration	HOMA-IR	Shorter sleep was associated with higher HOMA-IR after adjustment for age, race, sex, BMI, and waist circumference	Independent cross-sectional signal without temporal confirmation
Chen et al., 2021[19]	384 adolescents; seven-day actigraphy	Weekday duration and sleep midpoint	Log HOMA-IR	Weekday sleep-duration β −0.049; weekend midpoint β 0.0486; associations were stronger in girls	Duration and timing contributed independently in a cross-sectional model

Study	Sample	Sleep exposure	Outcome	Principal quantitative finding	Interpretation
Jansen et al., 2023[20]	362 adolescents; seven-day actigraphy and approximately two-year follow-up	Duration relative to recommendations and sleep midpoint	Development of insulin resistance	Sleeping ≥1 hour below recommendations: OR 2.74; latest versus earliest sleep midpoint: OR 2.63	Prospective association independent of adiposity change, but confidence intervals were wide
Alves et al., 2022[21]	62 children aged 7–11 years; actigraphy and OGTT	Total sleep time	Matsuda insulin sensitivity and adiposity	Longer sleep predicted better insulin sensitivity; body fat mediated 62% of the association	Adiposity appeared to be an important pathway in preadolescent children
Beunders et al., 2023[22]	894 children aged 8–11 years; nine-night actigraphy	Duration, fragmentation, and 24-hour rhythm	Fat mass, visceral fat, glucose, and cardiometabolic risk	Greater intradaily variability was associated with slightly higher fat mass and visceral fat in boys, without a consistent overall cardiometabolic association	Rhythm disruption may identify adiposity differences without a universal metabolic effect

BMI: body mass index; CI: confidence interval; HOMA-IR: homeostatic model assessment of insulin resistance; OGTT: oral glucose tolerance test; OR: odds ratio; RR: relative risk.

5 RESULTS AND DISCUSSION

5.1 SHORT SLEEP AND SUBSEQUENT OBESITY

The most consistent finding across pediatric sleep research is that shorter sleep precedes greater adiposity. In the major prospective meta-analysis, the direction of association was consistent across developmental stages, although its magnitude varied substantially.[4] The particularly large estimate among children aged 9 to younger than 12 years should not be interpreted as a sudden biological change at a particular birthday.

Differences in the number of studies, short-sleep thresholds, baseline ages, follow-up periods, and adjustment strategies can generate apparently sharp subgroup gradients. The more defensible conclusion is that chronic short sleep is associated with a modest-to-moderate increase in obesity risk across childhood and adolescence.[4]

A separate meta-analysis of 11 longitudinal studies estimated an odds ratio of 2.15 for subsequent overweight or obesity among short sleepers.[5] Odds ratios may appear larger than risk ratios when outcomes are common, and the included studies differed substantially in exposure definitions and follow-up. Another prospective meta-analysis reported a relative risk of approximately 1.45, which decreased to approximately 1.30 after influential heterogeneous cohorts were excluded.[24]

These findings support consistency in the direction of the association but do not support one universal numerical estimate. The magnitude is likely modified by age, baseline adiposity, socioeconomic context, sleep timing, physical activity, and chronicity of exposure.

The continuous effect estimate provides important clinical context. Each additional hour of sleep was associated with only approximately 0.03 lower units of subsequent BMI z score.[4] This effect is considerably smaller than the improvement expected from an effective comprehensive obesity intervention in an individual child.

Sleep should therefore be considered one modifiable component of energy balance and metabolic regulation rather than a sufficient explanation for pediatric obesity. Its population impact may nevertheless be meaningful because insufficient sleep is common and influences other health and behavioral outcomes.

Residual confounding remains likely. Caregiver employment, household schedules, neighborhood safety and noise, bedroom crowding, food insecurity, digital access, parental obesity, mental health, and physical activity may affect both sleep and adiposity. Prospective adjustment reduces but cannot eliminate these influences.

Reverse causation is also plausible. Existing obesity may shorten or fragment sleep through OSA, gastroesophageal reflux, discomfort, depression, reduced activity, and medication. Bidirectionality should consequently be expected rather than treated solely as a methodological problem.

5.2 SLEEP REGULARITY, TIMING, AND CATCH-UP SLEEP

Average sleep duration can conceal clinically distinct patterns. In 528 adolescents who underwent nearly one week of actigraphy, those with consistently insufficient sleep had a BMI z score 0.68 units higher than adolescents with sufficient and stable sleep. Their obesity prevalence was approximately 2.54 times greater.[9]

Adolescents with insufficient but variable sleep had a smaller BMI z-score difference of 0.30. This finding raises the possibility that nights of compensatory sleep may attenuate

part of the adiposity association. However, the cross-sectional design cannot determine whether catch-up sleep was protective or merely represented different family schedules, school demands, or unmeasured health characteristics.[9]

An earlier study of 308 school-aged children found that shorter and less regular sleep clustered with higher BMI and less favorable glucose, insulin, lipid, and inflammatory profiles.[10] The lowest obesity prevalence occurred among children who slept longer and maintained more stable schedules.

These findings helped shift the field from one average nightly duration toward multidimensional sleep health. Nevertheless, studies have not consistently used the same metric for sleep regularity, variability, or social jetlag, limiting direct comparisons.

Late sleep timing may influence metabolic risk independently of sleep duration because eating, light exposure, and physical activity occur at a biologically different circadian phase. A 2024 meta-analysis of 12 observational studies and 57,728 participants found that late bedtime was associated with obesity, with a pooled odds ratio of 1.27.[25]

The late-bedtime estimate was smaller than many short-sleep estimates, but it persisted in studies that adjusted for sleep duration. The association cannot fully distinguish cultural scheduling and family routines from endogenous circadian delay.

Weekday and weekend sleep should therefore be recorded separately. An adolescent sleeping six hours on school nights and eleven hours on weekends is not physiologically equivalent to one sleeping 8.5 hours every night, even if their weekly average appears similar.

Large weekend extensions indicate accumulated sleep debt and social jetlag. Compensatory sleep may reduce part of the acute sleep deficit but can delay circadian timing and make sleep initiation more difficult before the next school day. Regular adequate sleep remains preferable to alternating restriction and recovery.

5.3 EXPERIMENTAL EFFECTS ON ENERGY INTAKE AND FOOD CHOICE

Controlled sleep manipulation strengthens causal inference by demonstrating changes in eating behavior before substantial body-fat accumulation occurs. In a crossover trial of 37 children aged 8–11 years, extending and restricting time in bed by 1.5 hours created an achieved sleep-period difference of approximately two hours and 21 minutes.[11]

During the longer-sleep condition, children consumed approximately 134 fewer kilocalories per day and weighed 0.22 kg less. Fasting leptin was lower, while ghrelin did not

differ significantly.[11] The short-term weight change likely reflected energy balance, fluid distribution, and gastrointestinal contents rather than durable fat loss.

Among 41 adolescents aged 14–16 years, a crossover comparison of 6.5 versus 10 hours in bed produced an achieved sleep difference of 2.53 hours.[12] Restricted sleep increased dietary glycemic index, glycemic load, and the consumption of desserts and sweets. Differences in total calorie and carbohydrate intake showed trends but were less definitive.

The Daily Rest, Eating, and Activity Monitoring (DREAM) randomized crossover trial evaluated a more realistic one-hour bedtime manipulation in 105 children aged 8–12 years. During sleep restriction, energy from noncore foods increased by approximately 416 kJ per day.[13]

Among participants who achieved the prespecified sleep difference, total energy increased by 361 kJ per day, noncore-food energy by 504 kJ, and ultraprocessed-food energy by 523 kJ. Emotional overeating also increased modestly.[13] These results suggest that food quality and eating behavior may change before total daily energy intake demonstrates a large effect.

Several mechanisms may operate simultaneously. Additional waking time provides more opportunity to eat, while fatigue can reduce executive control over food selection. Late sleep schedules move eating toward the biological night, when glucose tolerance may be less favorable.

Sleep restriction may also reduce spontaneous activity even when planned exercise remains unchanged. Changes in leptin, ghrelin, cortisol, sympathetic activity, and reward processing have been proposed, but pediatric findings are not sufficiently consistent to support a single hormonal explanation.

5.4 SLEEP EXTENSION AND WEIGHT-RELATED OUTCOMES

Intervention evidence is more restrained than observational evidence. A meta-analysis of preschool studies found that four of five sleep-containing interventions improved weight-related outcomes. Pooled differences were −0.27 kg/m² for BMI and −0.07 for BMI z score.[14]

These effects were small, and the interventions frequently included other behavioral components. Improvements cannot therefore be attributed exclusively to sleep.

A randomized trial involving 78 children aged 8–11 years with habitual sleep of 9.5 hours or less used behavioral strategies to extend sleep. The intervention increased the sleep

period by approximately 40 minutes per night compared with controls, and 52% of intervention participants achieved at least 30 additional minutes compared with 15% of controls.[15]

Intention-to-treat differences in diet, BMI, and BMI z score were not statistically significant. Across both groups, children who actually gained at least 30 minutes demonstrated a 0.31 kg/m² lower BMI change and a 0.07-unit lower BMI z-score change.[15] Because this comparison was based on achieved sleep rather than randomized allocation, it remained vulnerable to healthy-adherer bias.

A 2024 meta-analysis of eight pediatric sleep lifestyle interventions involving 2,231 participants with overweight or obesity found no significant pooled BMI benefit. The effect size was 0.18, with a 95% confidence interval from −0.04 to 0.40, and statistical heterogeneity was substantial.[16]

Programs differed in participant age, intervention intensity, baseline sleep deficiency, obesity-treatment context, and whether sleep was the primary target. Most studies also had at least some risk-of-bias concerns. The pooled result does not support sleep extension as an isolated weight-loss treatment.

Long-term findings further moderate the early promise. In the Prevention of Overweight in Infancy trial, an infancy sleep intervention produced lower BMI z scores at some intermediate assessments. At approximately 11 years, however, the adjusted BMI z-score difference was −0.16, with a confidence interval crossing no effect.[17]

The odds ratio for overweight or obesity was 0.85 and was also not statistically significant.[17] Early sleep support may benefit infant and family well-being, but a durable obesity-prevention effect has not been demonstrated.

The difference between prospective association and intervention effect is not contradictory. Short sleep may contribute to obesity while representing only one of many determinants. Correcting one exposure after obesity has developed does not necessarily produce substantial weight loss.

Table 2

Experimental and interventional evidence

Study	Population	Design and achieved sleep contrast	Outcomes	Principal quantitative result	Clinical interpretation
Hart et al., 2013[11]	37 children aged 8–11 years	Randomized crossover; approximately 2-hour-21-minute sleep difference	Energy intake, weight, leptin	Longer sleep: −134 kcal/day, −0.22 kg, and lower fasting leptin	Short-term causal effect on intake and scale weight, not evidence of durable fat loss
Beebe et al., 2013[12]	41 adolescents aged 14–16 years	Crossover; 6.5 versus 10 hours in bed	Dietary composition	Restriction increased glycemic index, glycemic load, and sweets consumption	Sleep loss altered food choice even when total-energy differences were less certain
Morrison et al., 2023[13]	105 children aged 8–12 years	Randomized crossover; bedtime one hour earlier versus later	Energy intake and eating behavior	Restriction increased noncore-food energy; per-protocol analysis demonstrated greater total, noncore, and ultraprocessed intake	Realistic sleep loss increased discretionary-food consumption
Miller et al., 2021[14]	Five preschool intervention studies	Sleep-containing prevention interventions	BMI and BMI z score	BMI −0.27 kg/m²; BMI z score −0.07	Small favorable effect with limited attribution to sleep alone
Hart et al., 2022[15]	78 children aged 8–11 years with short habitual sleep	Behavioral sleep extension versus control	Sleep, dietary intake, BMI	Sleep increased by approximately 40 minutes; no significant intention-to-treat BMI effect	Sleep extension was feasible, but randomized weight benefit was unproven
Liu et al., 2024[16]	Eight studies; 2,231 participants with overweight or obesity	Sleep lifestyle interventions	BMI	Effect size 0.18; 95% CI −0.04 to 0.40; I^2=83.73%	No significant pooled BMI benefit and substantial heterogeneity
Taylor et al., 2025[17]	512 children assessed at approximately 11 years	Infancy sleep intervention	BMI z score and overweight or obesity	BMI z score −0.16; overweight/obesity OR 0.85; both confidence intervals crossed no effect	Earlier benefit was not statistically sustained at 11 years
Dutil et al., 2024[23]	36 adolescents with obesity and type 2 diabetes risk	Randomized crossover of extended, habitual, and restricted sleep	OGTT-derived insulin sensitivity	Insulin sensitivity was 20% higher after extension than after habitual or restricted sleep	Strong short-term physiological signal in a selected high-risk population

BMI: body mass index; CI: confidence interval; OGTT: oral glucose tolerance test; OR: odds ratio.

5.5 INSULIN RESISTANCE: DIRECT EFFECT OR ADIPOSITY-MEDIATED ASSOCIATION?

Insulin resistance is not a fixed pediatric characteristic. It varies according to puberty, sex, body composition, ethnicity, recent illness, physical activity, and the laboratory method used for assessment.

Fasting HOMA-IR predominantly reflects hepatic insulin resistance and is practical for epidemiological studies. The Matsuda index integrates glucose and insulin responses during an oral glucose tolerance test and more closely reflects whole-body insulin sensitivity. The hyperinsulinemic–euglycemic clamp remains the reference method but is rarely feasible in large pediatric cohorts.

Apparently inconsistent studies may therefore be evaluating different physiological processes. A sleep association detected using HOMA-IR is not necessarily equivalent to one demonstrated using dynamic glucose testing.

In 245 healthy Black and White adolescents, shorter sleep measured through diaries and actigraphy was associated with higher HOMA-IR after adjustment for age, race, sex, BMI, and waist circumference.[18] Mean sleep duration was approximately 7.4 hours by diary and 6.4 hours by actigraphy, demonstrating substantial sleep deficiency.

Sleep fragmentation was not associated with HOMA-IR, and long sleep did not demonstrate a clear U-shaped relationship. Because sleep and insulin were measured during the same developmental period, temporal direction could not be established.[18]

Cross-sectional actigraphy data from 384 adolescents demonstrated that shorter weekday sleep and later sleep midpoint were independently associated with higher log-transformed HOMA-IR.[19] The associations were stronger among girls.

Duration and timing may consequently represent partially independent metabolic exposures. However, sex-stratified findings require replication and were not confirmed through clamp-based measurement.

The strongest prospective observational study followed 362 adolescents for approximately two years. Participants sleeping at least one hour below age recommendations had 2.74 times the odds of developing insulin resistance.[20]

Those with the latest weekday sleep midpoint had 2.63 times the odds of developing insulin resistance compared with the earliest-timing group. Changes in adiposity did not statistically mediate these associations.[20]

The confidence intervals were wide and began near 1.0. Insulin resistance was also defined using puberty-specific HOMA-IR categories with imperfect sensitivity and specificity. The study supports temporal order but does not conclusively establish causality.

Preadolescent evidence suggests a stronger adiposity-mediated pathway. Among 62 children aged 7–11 years who underwent actigraphy and an oral glucose tolerance test, longer sleep was associated with better Matsuda insulin sensitivity.[21]

The relationship attenuated after adjustment for body-fat percentage, and body fat statistically mediated approximately 62% of the association.[21] The result is biologically coherent but cannot determine whether short sleep increased body fat or children with greater adiposity slept less.

A Generation R analysis of 894 children aged 8–11 years found limited evidence that sleep and 24-hour activity rhythms were associated with a combined cardiometabolic-risk profile.[22] Among boys, greater intradaily variability was associated with slightly higher fat-mass index and visceral fat.

Several isolated associations involving awakening and glucose outcomes were inconsistent. These predominantly neutral metabolic results are important because they demonstrate that objective sleep differences do not invariably translate into measurable insulin resistance in generally healthy populations.[22]

5.6 EXPERIMENTAL EVIDENCE CONCERNING INSULIN SENSITIVITY

The Sleep Manipulation in Adolescents at Risk of Type 2 Diabetes (SMART2D) study provides the clearest pediatric experimental evidence. Thirty-six adolescents with obesity and additional diabetes risk factors completed a randomized crossover protocol following a habitual-sleep week.[23]

Increasing time in bed produced approximately one hour and two minutes more sleep than habitual. Restriction produced approximately one hour and 19 minutes less sleep. Following the extension week, Matsuda insulin sensitivity was 20% higher than after both habitual and restricted sleep.[23]

Several methodological features strengthened the inference. Each participant served as their own control, sleep change was confirmed by actigraphy, condition order was counterbalanced, and insulin sensitivity was derived from an oral glucose tolerance test.

The study nevertheless had important limitations. It was open label, included only 36 completers, and each experimental condition lasted one week. Participants had severe obesity and additional type 2 diabetes risk, limiting generalizability to the general pediatric population.

The absence of a significant difference between habitual and restricted sleep also deserves attention. Habitual sleep averaged approximately seven hours and 31 minutes, suggesting that participants may already have had restricted baseline sleep.

Additional restriction may not have substantially worsened an already constrained metabolic state, whereas extension permitted partial recovery. Carryover, adaptation, or nonlinear effects may also have influenced the results.

The trial does not establish whether improved insulin sensitivity persists for months, reduces glycated hemoglobin, prevents diabetes, or depends on changes in weight. It supports short-term physiological reversibility in a high-risk phenotype rather than a universal 20% benefit for all children.

For adolescents with obesity, habitual short sleep, and dysglycemia risk, sleep extension is therefore a reasonable component of comprehensive care. Established obesity or metabolic treatment should not be delayed while awaiting a response to sleep intervention.

5.7 BIOLOGICAL AND BEHAVIORAL MECHANISMS

Behavioral and physiological mechanisms likely operate together. Short sleep increases waking opportunities to eat and may impair executive control over food selection. Fatigue may decrease spontaneous physical activity even when structured exercise remains unchanged.

Late sleep shifts food intake toward the biological night, when glucose tolerance is less favorable. Irregular schedules may desynchronize central and peripheral circadian clocks in the liver, muscle, adipose tissue, and pancreas.

Recurrent sleep deficiency can also influence sympathetic activity, cortisol timing, inflammatory signaling, growth-hormone secretion, and reward processing. No single mechanism has been established as responsible for the entire relationship.

Adiposity functions as both a mediator and an effect modifier. Increased energy intake and reduced activity can increase fat mass, which subsequently promotes insulin resistance through ectopic lipid deposition, inflammation, and altered adipokine signaling.

Existing obesity may also increase vulnerability to the metabolic effects of sleep loss. It markedly increases the risk of OSA, which can independently affect insulin sensitivity and blood pressure.

A direct pathway from inadequate sleep to impaired insulin action can coexist with an indirect pathway mediated by adiposity. The relative importance of each pathway may change across development and should not be generalized from one age group to another.

5.8 OBSTRUCTIVE SLEEP APNEA AS A CONFOUNDING AND INTERACTING CONDITION

OSA must be distinguished from short sleep duration. A child may spend ten hours in bed while experiencing fragmented and intermittently hypoxemic sleep. Conversely, an adolescent may sleep seven consolidated hours without upper-airway obstruction but still have insufficient sleep opportunity.

Obesity increases the risk of pediatric OSA, and OSA may independently contribute to insulin resistance, elevated blood pressure, inflammation, and daytime fatigue. Failure to evaluate breathing symptoms may therefore create an apparently direct association between sleep duration and metabolic dysfunction.

Habitual snoring, witnessed apnea, gasping, labored breathing, unusual sleep positions, morning headache, enuresis, severe daytime sleepiness, and tonsillar hypertrophy should prompt disorder-specific evaluation. Sleep duration should be reassessed after treatment of OSA because improved sleep quality may change the required time in bed and daytime function.

5.9 CLINICAL ASSESSMENT

A clinically useful sleep history should be quantitative. Families should record bedtime, estimated sleep onset, wake time, nocturnal awakenings, naps, and weekday–weekend differences for at least one to two weeks.

Questions should address evening digital-media exposure, caffeine and energy drinks, late meals, homework, caregiver work schedules, bedroom environment, snoring, limb discomfort, mood, and medication use. Asking only “How many hours does the child sleep?” cannot identify delayed timing, variability, social jetlag, or fragmented sleep.

Anthropometric evaluation should include serial BMI percentile or BMI z score. Waist circumference, waist-to-height ratio, and blood pressure may provide additional information when clinically appropriate.

Metabolic investigation should be guided by obesity severity, family history, signs of insulin resistance, medication exposure, pubertal stage, and current pediatric recommendations rather than sleep duration alone.[2] HOMA-IR should not be treated as a universal diagnostic test independent of clinical context.

Management begins with sufficient sleep opportunity and a stable wake time. Bedtime should be moved gradually when the schedule is substantially delayed. Evening light and

stimulating media should be reduced, while morning light exposure and physical activity should be encouraged.

Caffeine should be avoided late in the day. Meals should be structured so that extending time in bed does not simply shift eating later into the evening. Adolescents should participate actively in planning because feasibility and autonomy substantially influence adherence.

Sleep intervention should be integrated into established pediatric obesity care. Comprehensive treatment may include family-based nutrition, physical activity, behavioral therapy, management of comorbidities, anti-obesity medication when indicated, and metabolic or bariatric evaluation for selected severe disease.

A successful sleep outcome may consist of 30–60 additional minutes of sleep, lower night-to-night variability, an earlier sleep midpoint, improved morning function, or resolution of breathing symptoms even if BMI does not immediately change. This avoids incorrectly classifying sleep treatment as unsuccessful when direct sleep-related benefits are clinically meaningful.

Table 3

Phenotype-based clinical assessment and management

Sleep phenotype	Key clinical history	Measurement or confirmation	Initial management direction	Metabolic interpretation
Consistently short sleep	Sleep below age recommendations on school and free nights; difficult waking or daytime sleepiness	One- to two-week diary; actigraphy if history is unreliable	Increase sleep opportunity gradually and stabilize wake time	Most consistent prospective obesity signal; insulin effect may be greater in high-risk adolescents
Short school-night sleep with weekend catch-up	Large weekend extension and Sunday-night difficulty	Separate weekday and weekend duration and midpoint	Increase weekday sleep and avoid extreme weekend phase delay	Catch-up may reduce some acute sleep debt but does not replace regular adequate sleep
Late sleep timing	Late bedtime, late eating, evening light, adequate duration only on a late schedule	Diary or actigraphy; circadian testing only in selected cases	Fixed wake time, morning light, gradual phase advancement, and earlier meal timing	Late bedtime has a modest duration-independent association with obesity
Irregular sleep	Night-to-night variation, alternating schedules, or all-night episodes	Calculate variability across at least seven nights	Establish stable anchors for waking, meals, school preparation, and bedtime routine	Irregularity may indicate circadian disruption, although thresholds are not standardized
Obesity with suspected OSA	Snoring, apnea, gasping, morning headache,	Pediatric sleep assessment and	Treat OSA and obesity in parallel	OSA may independently

Sleep phenotype	Key clinical history	Measurement or confirmation	Initial management direction	Metabolic interpretation
	enuresis, or tonsillar hypertrophy	polysomnography when indicated		increase insulin and blood-pressure risk
Obesity or prediabetes with short sleep	Short sleep plus acanthosis, family history, dysglycemia, or severe obesity	Standard metabolic evaluation plus sleep diary or actigraphy	Add sleep extension to comprehensive metabolic care	SMART2D supports short-term insulin-sensitivity improvement in this high-risk phenotype
Apparently long sleep	Long time in bed, fatigue, depression, medication, chronic illness, or fragmented sleep	Distinguish true sleep from time in bed and evaluate underlying conditions	Treat the cause rather than restricting sleep to fit a population average	Evidence that long sleep independently causes pediatric metabolic disease is insufficient

HOMA-IR: homeostatic model assessment of insulin resistance; OSA: obstructive sleep apnea; SMART2D: Sleep Manipulation in Adolescents at Risk of Type 2 Diabetes.

5.10 CERTAINTY OF EVIDENCE AND RESEARCH PRIORITIES

Certainty is moderate that short sleep is prospectively associated with subsequent overweight or obesity. Consistency, temporal order, dose-related patterns, and experimental effects on food intake support a contributory role.

Certainty is lower regarding the magnitude of an independent effect because sleep definitions vary and residual social, behavioral, and medical confounding remains likely. The average BMI difference associated with each additional hour of sleep is small.

Certainty is low to moderate that sleep duration or timing contributes independently to insulin resistance. Several cross-sectional investigations and one prospective study support the association, but pubertal stage, adiposity, sex, and HOMA-IR definitions complicate interpretation.

SMART2D provides evidence of short-term improvement in oral-glucose-tolerance-test-derived insulin sensitivity among adolescents with severe obesity and diabetes risk.[23] Its limited sample, brief intervention, and selected population prevent broad generalization.

Future trials should enroll participants with objectively confirmed short sleep and specify whether duration, timing, or regularity is the principal target. Sleep differences should be maintained for months rather than days.

Outcomes should include body composition, waist circumference, visceral adiposity, oral-glucose-tolerance testing or clamp-derived physiology, glycated hemoglobin when appropriate, dietary timing, physical activity, OSA, and pubertal stage.

Randomization, blinded metabolic assessment, adherence reporting, and long-term follow-up will be essential. Trials should also determine whether metabolic improvements persist after active sleep intervention ends.

Equity must be incorporated into study design and clinical recommendations. Caregiver shift work, crowded housing, neighborhood noise, school start times, food insecurity, and digital demands can limit a family's ability to implement sleep recommendations.

Interventions should consequently evaluate school, community, and policy approaches in addition to individual sleep hygiene. A biologically plausible recommendation cannot be clinically effective if children and families cannot implement it.

6 LIMITATIONS

This was a structured narrative review rather than a prospectively registered systematic review with de novo meta-analysis. Although multiple databases, landmark reviews, and linked primary reports were examined, formal duplicate screening and a study-level risk-of-bias table were not undertaken.

Quantitative results were selected to demonstrate the evidence gradient and should not be interpreted as an exhaustive inventory. The underlying literature used heterogeneous definitions of short sleep and frequently combined time in bed with actual sleep.

Studies also varied in their assessment of naps, weekday and weekend schedules, sleep timing, variability, and circadian phase. Self- and parent-reported sleep may misclassify duration, whereas actigraphy infers sleep from movement and may classify quiet wakefulness as sleep.

Obesity outcomes ranged from BMI to directly measured fat mass and visceral adiposity. Insulin-related outcomes ranged from fasting surrogates to oral-glucose-tolerance-test indices, limiting direct comparison.

Residual confounding, reverse causation, puberty, sex differences, and OSA restricted observational inference. Intervention trials were generally small, short, behaviorally heterogeneous, and susceptible to differences in adherence.

Few trials were designed specifically to determine whether sleep intervention produces durable weight loss or prevents type 2 diabetes. Evidence remains limited for preschool children, children with normal weight but substantial diabetes risk, young people with disabilities, and populations facing major structural barriers to adequate sleep.

7 CONCLUSION

Short sleep is consistently associated with subsequent obesity in children and adolescents. Prospective estimates generally indicate a 30%–58% higher relative risk, with larger estimates in some school-aged cohorts, but the average BMI difference associated with an additional hour of sleep is small. Sleep is a contributory exposure rather than a sufficient explanation for pediatric obesity.

Sleep duration should not be interpreted in isolation. Late timing, irregular schedules, and large weekday–weekend differences may provide additional metabolic information and identify accumulated sleep debt. Catch-up sleep may reduce part of the acute deficit but does not replace regular adequate sleep, while apparently long sleep should prompt evaluation of its cause rather than automatic restriction.

Evidence concerning insulin resistance is promising but less certain. Adiposity mediates much of the association in some preadolescent populations, whereas short duration and late timing predicted incident insulin resistance independently of adiposity change in one adolescent cohort. A randomized crossover trial demonstrated 20% higher insulin sensitivity after one week of sleep extension in adolescents with severe obesity and type 2 diabetes risk, but its durability and generalizability remain unknown.

Clinical care should assess sleep opportunity, actual duration, timing, variability, daytime function, and OSA symptoms alongside serial adiposity and conventional metabolic risk. Sleep extension and schedule stabilization should be incorporated into comprehensive obesity or diabetes-risk management without delaying established nutritional, behavioral, pharmacological, or specialist interventions.

Future research should progress from broad correlations to phenotype-enriched, adequately powered, long-term trials using objective sleep measurement, standardized pubertal assessment, body composition, and robust glucose phenotyping. Until such evidence becomes available, the most defensible conclusion is that adequate and regular sleep supports metabolic health and may improve insulin sensitivity in vulnerable adolescents, but it is not a stand-alone cure for obesity or insulin resistance.

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