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SLEEP DISORDERS AND SEVERITY OF ATTENTION-DEFICIT/HYPERACTIVITY DISORDER SYMPTOMS

TRANSTORNOS DO SONO E GRAVIDADE DOS SINTOMAS DO TRANSTORNO DE DÉFICIT DE ATENÇÃO/HIPERATIVIDADE

TRASTORNOS DEL SUEÑO Y GRAVEDAD DE LOS SÍNTOMAS DEL TRASTORNO POR DÉFICIT DE ATENCIÓN E HIPERACTIVIDAD

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ABSTRACT

Introduction: Sleep disturbances are highly prevalent among children and adolescents with attention-deficit/hyperactivity disorder (ADHD). Difficulties initiating sleep, delayed circadian timing, insufficient or irregular sleep, sleep-disordered breathing, restless legs syndrome, and medication-related sleep changes may intensify inattention, emotional dysregulation, oppositional behavior, and functional impairment. However, subjective sleep complaints are more consistent than objective polysomnographic abnormalities, and the direction of the relationship remains complex.

Objective: To critically examine the association between sleep disorders and ADHD symptom severity in children and adolescents and to evaluate whether sleep-directed interventions improve sleep, ADHD manifestations, cognition, behavior, and daily functioning.

Methods: A structured narrative review was conducted using PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library. Systematic reviews, meta-analyses, longitudinal studies, controlled sleep-manipulation experiments, randomized trials, and clinical guidelines were prioritized. Sleep phenotypes, measurement methods, directionality, medication effects, and treatment outcomes were analyzed separately.

Results: Children and adolescents with ADHD consistently exhibit more parent- and self-reported sleep problems than typically developing peers. Associations are strongest for sleep-onset difficulty, bedtime resistance, short or irregular sleep, difficult morning awakening, and daytime sleepiness. Actigraphy generally demonstrates longer sleep latency

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and lower sleep efficiency, whereas pooled polysomnographic findings are less consistent. Experimental sleep restriction aggravates parent-rated inattention, oppositionality, sleepiness, and affective instability, supporting a causal contribution of insufficient sleep to selected symptoms. Behavioral sleep interventions produce substantial improvement in sleep and modest reductions in ADHD symptom ratings. Melatonin improves sleep onset and circadian timing but has not consistently improved core ADHD symptoms. Adenotonsillectomy improves respiratory symptoms, sleepiness, quality of life, and caregiver-rated behavior in sleep-disordered breathing, although randomized trials have not shown superior objective attention or executive-function outcomes.

Conclusion: Sleep disturbance may mimic, amplify, result from, or coexist with ADHD. Its identification and treatment can remove a reversible component of impairment but should not be presented as a universal treatment for the neurodevelopmental disorder. Routine sleep screening, phenotype-specific investigation, medication review, and separate monitoring of nighttime and daytime outcomes should be integrated into ADHD care.

Keywords: Attention Deficit Disorder with Hyperactivity. Sleep Wake Disorders. Insomnia. Circadian Rhythm. Sleep Apnea Syndromes. Child. Adolescent.

RESUMO

Introdução: Os distúrbios do sono são altamente prevalentes entre crianças e adolescentes com transtorno de déficit de atenção/hiperatividade (TDAH). Dificuldades para iniciar o sono, atraso no ritmo circadiano, sono insuficiente ou irregular, distúrbios respiratórios do sono, síndrome das pernas inquietas e alterações do sono relacionadas ao uso de medicamentos podem intensificar a desatenção, a desregulação emocional, o comportamento opositor e o comprometimento funcional. Entretanto, as queixas subjetivas relacionadas ao sono são mais consistentes do que as alterações polissonográficas objetivas, e a direção dessa relação permanece complexa.

Objetivo: Examinar criticamente a associação entre os transtornos do sono e a gravidade dos sintomas do TDAH em crianças e adolescentes, bem como avaliar se as intervenções direcionadas ao sono melhoram o sono, as manifestações do TDAH, a cognição, o comportamento e o funcionamento diário.

Métodos: Foi realizada uma revisão narrativa estruturada utilizando as bases PubMed/MEDLINE, Scopus, Web of Science e Cochrane Library. Foram priorizadas revisões sistemáticas, metanálises, estudos longitudinais, experimentos controlados de manipulação do sono, ensaios randomizados e diretrizes clínicas. Os fenótipos do sono, os métodos de mensuração, a direção da associação, os efeitos dos medicamentos e os resultados dos tratamentos foram analisados separadamente.

Resultados: Crianças e adolescentes com TDAH apresentam consistentemente mais problemas de sono relatados pelos pais e pelos próprios indivíduos do que seus pares com desenvolvimento típico. As associações são mais fortes para dificuldades de iniciar o sono, resistência ao horário de dormir, sono curto ou irregular, dificuldade para acordar pela manhã e sonolência diurna. A actigrafia geralmente demonstra maior latência do sono e menor eficiência do sono, enquanto os achados polissonográficos agrupados são menos consistentes. A restrição experimental do sono agrava a desatenção, o comportamento opositor, a sonolência e a instabilidade afetiva avaliados pelos pais, sustentando uma

contribuição causal do sono insuficiente para determinados sintomas. As intervenções comportamentais para o sono produzem melhora substancial do sono e reduções modestas nas avaliações dos sintomas de TDAH. A melatonina melhora o início do sono e o ritmo circadiano, mas não tem demonstrado melhora consistente dos sintomas centrais do TDAH. A adenotonsilectomia melhora os sintomas respiratórios, a sonolência, a qualidade de vida e o comportamento avaliado pelos cuidadores em casos de distúrbios respiratórios do sono, embora ensaios randomizados não tenham demonstrado superioridade nos resultados objetivos de atenção ou de função executiva.

Conclusão: Os distúrbios do sono podem mimetizar, intensificar, resultar do TDAH ou coexistir com o transtorno. Sua identificação e tratamento podem eliminar um componente reversível do comprometimento funcional, mas não devem ser apresentados como tratamento universal para o transtorno do neurodesenvolvimento. A triagem rotineira do sono, a investigação específica de acordo com o fenótipo, a revisão do uso de medicamentos e o monitoramento separado dos resultados noturnos e diurnos devem ser integrados ao cuidado de pessoas com TDAH.

Palavras-chave: Transtorno de Déficit de Atenção/Hiperatividade. Transtornos do Sono-Vigília. Insônia. Ritmo Circadiano. Síndromes da Apneia do Sono. Criança. Adolescente.

RESUMEN

Introducción: Los trastornos del sueño son altamente prevalentes entre los niños y adolescentes con trastorno por déficit de atención e hiperactividad (TDAH). Las dificultades para iniciar el sueño, el retraso del ritmo circadiano, el sueño insuficiente o irregular, los trastornos respiratorios del sueño, el síndrome de piernas inquietas y las alteraciones del sueño relacionadas con la medicación pueden intensificar la falta de atención, la desregulación emocional, el comportamiento oposicionista y el deterioro funcional. Sin embargo, las quejas subjetivas relacionadas con el sueño son más consistentes que las alteraciones polisonográficas objetivas, y la dirección de esta relación sigue siendo compleja.

Objetivo: Examinar críticamente la asociación entre los trastornos del sueño y la gravedad de los síntomas del TDAH en niños y adolescentes, así como evaluar si las intervenciones dirigidas al sueño mejoran el sueño, las manifestaciones del TDAH, la cognición, el comportamiento y el funcionamiento cotidiano.

Métodos: Se realizó una revisión narrativa estructurada utilizando las bases de datos PubMed/MEDLINE, Scopus, Web of Science y Cochrane Library. Se priorizaron revisiones sistemáticas, metanálisis, estudios longitudinales, experimentos controlados de manipulación del sueño, ensayos aleatorizados y guías clínicas. Los fenotipos del sueño, los métodos de medición, la dirección de la asociación, los efectos de los medicamentos y los resultados del tratamiento se analizaron por separado.

Resultados: Los niños y adolescentes con TDAH presentan de manera consistente más problemas de sueño, según los informes de los padres y de los propios individuos, que sus pares con desarrollo típico. Las asociaciones son más fuertes para las dificultades para iniciar el sueño, la resistencia a la hora de acostarse, el sueño corto o irregular, la dificultad para despertarse por la mañana y la somnolencia diurna. La actigrafía generalmente demuestra una mayor latencia del sueño y una menor eficiencia del sueño, mientras que los hallazgos polisomnográficos agrupados son menos consistentes. La restricción experimental

del sueño agrava la falta de atención, el comportamiento oposicionista, la somnolencia y la inestabilidad afectiva evaluados por los padres, lo que respalda una contribución causal del sueño insuficiente a determinados síntomas. Las intervenciones conductuales para el sueño producen una mejora sustancial del sueño y reducciones modestas en las evaluaciones de los síntomas del TDAH. La melatonina mejora el inicio del sueño y el ritmo circadiano, pero no ha demostrado de manera consistente una mejoría de los síntomas centrales del TDAH. La adenotonsilectomía mejora los síntomas respiratorios, la somnolencia, la calidad de vida y el comportamiento evaluado por los cuidadores en casos de trastornos respiratorios del sueño, aunque los ensayos aleatorizados no han demostrado resultados objetivos superiores en atención o función ejecutiva.

Conclusión: Los trastornos del sueño pueden imitar, intensificar, ser consecuencia del TDAH o coexistir con el trastorno. Su identificación y tratamiento pueden eliminar un componente reversible del deterioro funcional, pero no deben presentarse como un tratamiento universal para el trastorno del neurodesarrollo. La evaluación sistemática del sueño, la investigación específica según el fenotipo, la revisión de la medicación y el seguimiento separado de los resultados nocturnos y diurnos deben integrarse en la atención de las personas con TDAH.

Palabras clave: Trastorno por Déficit de Atención e Hiperactividad. Trastornos del Sueño-Vigilia. Insomnio. Ritmo Circadiano. Síndromes de la Apnea del Sueño. Niño. Adolescente.

1 INTRODUCTION

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental condition characterized by persistent and developmentally inappropriate inattention and/or hyperactivity–impulsivity that causes impairment in more than one setting. Its consequences may include academic difficulties, unstable peer relationships, family conflict, emotional dysregulation, accidents, and reduced quality of life. Although ADHD is not primarily a sleep disorder, sleep is closely connected to the cognitive and behavioral processes affected by the condition, including sustained attention, inhibitory control, working memory, motivation, reward processing, and emotional regulation.[1]

Clinical guidelines recommend that ADHD assessment include investigation of coexisting developmental, emotional, behavioral, and physical conditions. Sleep disorders are particularly important because they may produce daytime manifestations that resemble ADHD, intensify symptoms in a child with established ADHD, reduce the apparent effectiveness of treatment, or emerge as adverse effects of pharmacological therapy. A child can also have both ADHD and an independent sleep disorder, and improvement of one condition does not necessarily eliminate the other.[2]

Sleep complaints are frequent in clinical ADHD populations. In a study of 239 school-aged children with ADHD, 28.5% had mild sleep problems and 44.8% had moderate or severe problems. More severe sleep disturbance was associated with poorer psychosocial quality of life, impaired morning and evening routines, school lateness or absence, worse caregiver mental health, and disruption of parental employment. Despite this burden, fewer than half of caregivers reported that sleep had been discussed with the treating pediatrician.[3]

A foundational meta-analysis involving 722 children with ADHD and 638 controls found that ADHD was associated with greater bedtime resistance, difficulty initiating sleep, night waking, difficult morning awakening, sleep-disordered breathing, and daytime sleepiness. Objective differences were more selective: actigraphy identified longer sleep latency and shorter sleep duration, while polysomnography identified lower sleep efficiency and more sleep-stage shifts. The results established an important methodological distinction: subjective sleep problems are consistently reported, but ADHD is not associated with a single or universal polysomnographic profile.[4]

Subsequent research has reinforced this heterogeneity. A systematic review of 148 studies and more than 42,000 children concluded that disturbances involving sleep continuity, duration, circadian timing, movement during sleep, and daytime alertness are common in

ADHD. However, results varied according to medication status, psychiatric comorbidity, age, measurement method, and the definition of sleep disturbance.[5] A more recent meta-analysis of 45 studies, including 1,622 children and adolescents with ADHD and 2,013 typically developing participants, found longer sleep latency and moderately lower sleep efficiency when sleep was measured by actigraphy. Corresponding differences measured by polysomnography were not statistically significant.[6]

This subjective–objective discrepancy should not be interpreted as evidence that family reports are invalid. Questionnaires capture bedtime conflict, inconsistent routines, dependence on caregivers for sleep onset, difficult waking, and the functional burden experienced by the family. Actigraphy estimates sleep–wake patterns across several nights in the child's natural environment, but it infers sleep from movement and may misclassify quiet wakefulness. Polysomnography directly measures sleep physiology but is generally limited to one or two nights in an unfamiliar laboratory. Each method therefore evaluates a different component of the sleep phenotype.[4–7]

The relationship between ADHD and sleep is also bidirectional. Executive dysfunction, impulsivity, bedtime procrastination, emotional dysregulation, sensory sensitivity, and difficulty disengaging from stimulating activities can delay sleep. Conversely, insufficient or fragmented sleep can impair attention, emotional control, motivation, and morning functioning. Anxiety, depression, autism spectrum disorder, oppositional behavior, obesity, excessive digital-media exposure, family stress, and irregular daily schedules may influence both sleep and ADHD symptom ratings.[5,7]

The clinically relevant question is therefore not whether ADHD or sleep disturbance is the sole cause of the other. Instead, it is necessary to identify the sleep phenotype, determine its temporal relationship with daytime symptoms and treatment, and estimate how much functional impairment is potentially reversible. This review evaluates the principal sleep disorders and sleep characteristics associated with ADHD severity and critically examines the effects of sleep-directed interventions.

2 OBJECTIVES

2.1 PRIMARY OBJECTIVE

To evaluate the association between sleep disorders or disturbed sleep characteristics and the severity of ADHD symptoms in children and adolescents.

2.2 SECONDARY OBJECTIVES

1. To compare subjective sleep findings with results obtained through sleep diaries, actigraphy, and polysomnography.
2. To evaluate longitudinal and experimental evidence concerning the direction and possible causality of the association.
3. To distinguish behavioral insomnia and delayed circadian timing from insufficient sleep, sleep-disordered breathing, restless legs syndrome, and periodic limb movements during sleep.
4. To assess the effects of stimulant and nonstimulant medications on sleep.
5. To examine whether behavioral treatment, sleep extension, melatonin, and treatment of sleep-disordered breathing improve ADHD symptoms and associated functional outcomes.
6. To propose a phenotype-based clinical approach to sleep assessment in children and adolescents with ADHD.

3 METHODOLOGY

3.1 REVIEW DESIGN

A structured narrative review was conducted because the available evidence encompasses epidemiological studies, clinical cohorts, objective sleep investigations, longitudinal analyses, experimental sleep manipulation, pharmacological studies, and disorder-specific randomized trials. The substantial heterogeneity of exposures, instruments, populations, and outcomes did not support a new quantitative meta-analysis.

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 “attention-deficit/hyperactivity disorder,” “ADHD,” “child,” “adolescent,” “sleep,” “insomnia,” “sleep duration,” “sleep restriction,” “circadian rhythm,” “chronotype,” “melatonin,” “actigraphy,” “polysomnography,” “sleep-disordered breathing,” “obstructive sleep apnea,” “restless legs syndrome,” “periodic limb movements,” “stimulant,” and “behavioral sleep intervention.”

Reference lists of relevant systematic reviews, meta-analyses, randomized trials, and clinical guidelines were examined to identify additional studies. Recent evidence was

prioritized, while older landmark studies were retained when they established an important clinical or mechanistic principle.

3.3 ELIGIBILITY AND EVIDENCE PRIORITIZATION

Studies involving children or adolescents with diagnosed ADHD or prospectively measured ADHD symptoms were considered. Systematic reviews, meta-analyses, longitudinal cohorts, controlled sleep-manipulation studies, and randomized clinical trials received the greatest weight. Cross-sectional studies were included when they provided relevant prevalence, severity-gradient, or objective sleep data.

Adult-only studies, isolated case reports, and reports without interpretable sleep or ADHD-related outcomes were not used as principal evidence. Studies of sleep-disordered breathing were included when behavioral, attentional, executive, or ADHD-related outcomes were reported, even when participants were not selected specifically for ADHD.

3.4 EXPOSURE AND OUTCOME CLASSIFICATION

Sleep exposures were classified into six groups:

1. Behavioral insomnia and bedtime resistance.
2. Delayed circadian timing.
3. Insufficient, irregular, or experimentally restricted sleep.
4. Sleep-disordered breathing, including obstructive sleep apnea.
5. Restless legs syndrome and periodic limb movements during sleep.
6. Medication-associated sleep changes.

Outcomes included total ADHD symptom severity, inattentive symptoms, hyperactive–impulsive symptoms, oppositional behavior, emotional dysregulation, daytime sleepiness, executive function, objective attention, academic performance, quality of life, and family functioning.

3.5 CRITICAL APPRAISAL

Observational studies were interpreted with attention to reverse causation, shared-rater bias, medication use, psychiatric comorbidity, obesity, pubertal development, socioeconomic context, digital-media use, and family routines. Intervention studies were evaluated according to randomization, masking, adherence, objective confirmation of sleep

change, attrition, outcome-rater blinding, and consistency between subjective and objective results. Statistical association was not interpreted as proof of causality, and improvement in caregiver-rated behavior was not considered equivalent to normalization of objective cognitive performance.

4 RESULTS

4.1 GENERAL EVIDENCE PATTERN

The most consistent finding was a higher burden of subjective sleep disturbance among children and adolescents with ADHD. Sleep-onset difficulty, bedtime resistance, insufficient sleep, difficult morning awakening, and daytime sleepiness were the manifestations most frequently associated with greater functional impairment. In specialist ADHD services, clinically elevated sleep-problem scores have been reported in more than 80% of participants, and a greater number of sleep-problem domains has been associated with greater ADHD severity.[8]

A clinical study of 136 children and adolescents with ADHD compared 42 patients with sleep disturbance with 94 patients without it. The sleep-disturbance group had worse inattention, academic skills, adaptive functioning, emotional regulation, and stress-related symptoms. Several associations persisted after adjustment for demographic and clinical variables, suggesting that sleep disturbance identifies a particularly vulnerable ADHD phenotype.[9]

Objective evidence was less uniform. Actigraphy more consistently demonstrated prolonged sleep latency and reduced sleep efficiency, whereas pooled polysomnographic differences were not significant.[6] This pattern suggests that problems arising across multiple nights in the home environment may not be fully reproduced by a short laboratory recording.

Table 1

Principal quantitative evidence relating sleep disturbance to ADHD symptoms and functioning

Study	Sample	Sleep exposure or intervention	Principal result	Interpretation
Sung et al., 2008[3]	239 children with ADHD	Parent-reported sleep problems	28.5% had mild and 44.8% had moderate/severe sleep problems	Sleep problems were associated with substantial child and family impairment

Study	Sample	Sleep exposure or intervention	Principal result	Interpretation
Cortese et al., 2009[4]	16 studies; 722 ADHD and 638 controls	Questionnaires, actigraphy, polysomnography, sleep-latency testing	Broad subjective differences; smaller and more selective objective differences	ADHD is not characterized by one universal physiological sleep profile
Liang et al., 2023[6]	45 studies; 1,622 ADHD and 2,013 controls	Actigraphy and polysomnography	Actigraphy showed longer latency and lower efficiency; pooled polysomnographic differences were nonsignificant	Measurement method materially changes the observed association
Bond et al., 2024[8]	Specialist ADHD service	Children's Sleep Habits Questionnaire	84% exceeded the clinical cutoff and 64% had at least two sleep-problem domains	Greater sleep disturbance was associated with greater ADHD severity
Becker et al., 2019[10]	162 ADHD and 140 controls	Ratings, diaries, and approximately two weeks of actigraphy	Adjusted odds of clinically elevated parent-rated sleep disturbance were 6.20 times higher in ADHD	Multimethod evidence supports greater sleep and daytime-sleepiness burden
Becker et al., 2019[12]	72 enrolled; 48 adherent adolescents with ADHD	One week with 6.5-hour versus 9.5-hour sleep opportunity	Restriction increased parent-rated inattention and oppositionality and increased sleepiness	Experimental evidence supports a causal contribution to selected symptoms
Van der Heijden et al., 2007[21]	105 medication-free children with ADHD and sleep-onset insomnia	Melatonin versus placebo for four weeks	Sleep onset advanced and total sleep increased, without significant behavioral or cognitive improvement	Melatonin treats sleep timing, not the core ADHD disorder
Hiscock et al., 2015[23]	244 children with ADHD	Behavioral sleep intervention versus usual care	ADHD symptom difference −2.9 at three months and −3.7 at six months; number needed to treat for moderate/severe sleep problems was 3.9	Sleep improvement was clinically meaningful; ADHD benefit was modest
CHAT, 2013[17]	464 children with obstructive sleep apnea	Early adenotonsillectomy versus watchful waiting	Polysomnographic normalization occurred in 79% versus 46%; no significant advantage in the primary attention/executive outcome	Respiratory and behavioral benefits exceeded objective cognitive benefit
PATS, 2023[18]	459 children with mild sleep-disordered breathing	Early adenotonsillectomy versus watchful waiting	No significant improvement in primary executive-function or attention outcomes	Symptoms, behavior, and quality of life improved despite neutral cognitive primary outcomes

ADHD: attention-deficit/hyperactivity disorder; CHAT: Childhood Adenotonsillectomy Trial; PATS: Pediatric Adenotonsillectomy Trial for Snoring.

4.2 DIRECTIONALITY AND CAUSALITY

Longitudinal evidence supports a bidirectional relationship. ADHD-related dysregulation may predict subsequent sleep difficulty, while sleep disturbance may predict

later increases in ADHD symptoms, particularly inattention.[11] Nevertheless, longitudinal associations are generally modest and remain susceptible to time-varying confounding.

More direct evidence comes from experimental sleep restriction. In a randomized crossover study of adolescents with ADHD, a restricted sleep opportunity increased parent-rated inattention, oppositional behavior, daytime sleepiness, and sluggish cognitive symptoms.[12] A related analysis demonstrated poorer positive affect and greater negative affect during sleep restriction.[13] These experiments confirm that insufficient sleep can aggravate clinically relevant daytime manifestations, although they do not establish that sleep loss causes ADHD itself.

5 DISCUSSION

5.1 SLEEP DISTURBANCE AS A MIMIC, AMPLIFIER, CONSEQUENCE, OR COMORBIDITY

The relationship between sleep and ADHD cannot be represented by a single causal pathway. Sleep disturbance may function as:

- A mimic, when insufficient or fragmented sleep produces inattention, irritability, or behavioral disinhibition in a child without ADHD.
- An amplifier, when a sleep problem increases the severity of established ADHD.
- A consequence, when ADHD-related executive dysfunction and emotional dysregulation interfere with bedtime and sleep routines.
- A comorbidity, when ADHD and an independent sleep disorder coexist.
- A treatment effect, when medication changes sleep onset, duration, or daytime alertness.

These categories are not mutually exclusive. A child with ADHD may procrastinate at bedtime, use digital media late at night, receive a long-acting stimulant, and also have obstructive sleep apnea. Management therefore requires identification of each contributing factor rather than attribution of the entire presentation to a single diagnosis.

The distinction also has implications for outcome interpretation. If treatment improves sleep and reduces parent-rated inattention, the improvement confirms that part of the daytime impairment was sleep-sensitive. It does not prove that the original ADHD diagnosis was incorrect. Conversely, persistent ADHD symptoms after successful sleep treatment do not

indicate that the sleep intervention failed if sleep quality, morning functioning, and family well-being improved.

5.2 INSUFFICIENT AND IRREGULAR SLEEP

Adolescents with ADHD are especially vulnerable to insufficient sleep because pubertal circadian delay interacts with early school schedules, reduced parental supervision, bedtime procrastination, homework difficulties, social media, gaming, and variable medication effects. A multimethod study found shorter school-night sleep, more daytime sleepiness, and more difficulty initiating and maintaining sleep among adolescents with ADHD than among controls.[10]

The effects of sleep restriction are not uniform across symptom domains. In the crossover experiment conducted by Becker and colleagues, parent-rated inattention and oppositionality increased, but performance on a brief continuous-attention task did not significantly deteriorate.[12] Self-rated hyperactivity–impulsivity also did not increase and may decline when fatigue reduces overt motor activity. Thus, sleep deprivation may make a child less visibly active while simultaneously worsening attention, irritability, motivation, and morning functioning.

Irregular timing may add impairment independently of average sleep duration. Weekend catch-up sleep, all-night episodes, variable wake times, and long afternoon naps can perpetuate circadian delay. A single question about usual sleep duration is therefore insufficient. Weekday and weekend schedules, variability, naps, school start time, and the difference between time in bed and actual sleep should be documented.

Children aged 6–12 years are generally advised to obtain 9–12 hours of sleep per 24 hours, while adolescents aged 13–18 years are advised to obtain 8–10 hours.[27] These ranges are population recommendations rather than rigid requirements for every individual, but they provide a practical reference for identifying clearly inadequate sleep opportunity.

5.3 BEHAVIORAL INSOMNIA AND DELAYED CIRCADIAN TIMING

Behavioral insomnia includes inconsistent limit setting, parent-dependent sleep-onset associations, bedtime conflict, anxiety-related avoidance, and poorly structured routines. ADHD can reinforce these patterns through difficulty transitioning between activities, low tolerance of delayed reward, emotional escalation, and inconsistent adherence to family rules.

Delayed circadian timing is different. The child or adolescent has a stable tendency to become sleepy late and may sleep relatively well when allowed a later schedule. Among medication-naïve children with ADHD and chronic sleep-onset insomnia, sleep onset and dim-light melatonin onset occurred approximately 45–50 minutes later than in children with ADHD without insomnia.[14] This finding supports a biological circadian component in at least a subgroup of patients.

Systematic evidence also associates ADHD with evening chronotype, delayed sleep timing, and altered circadian markers.[15] Nevertheless, not every child who resists bedtime has a delayed biological clock. Behavioral insomnia and circadian delay may coexist, but treatment emphasis differs. Limit-setting strategies are central to behavioral insomnia, whereas a stable wake time, morning light, reduced evening light, and appropriately timed melatonin are more relevant to circadian delay.

Screen exposure may contribute through several pathways: displacement of sleep time, psychological stimulation, difficulty disengaging, and evening light exposure. The association is also bidirectional because children with poor self-regulation may be more likely to use devices excessively. Device removal alone will not resolve anxiety, restless legs syndrome, obstructive breathing, or a markedly delayed circadian phase, but a media plan remains an important part of behavioral management.

5.4 SLEEP-DISORDERED BREATHING

Sleep-disordered breathing ranges from habitual snoring and increased upper-airway resistance to obstructive sleep apnea. Repeated respiratory obstruction may produce sleep fragmentation, intermittent hypoxemia, autonomic activation, and altered restorative sleep. These processes may contribute to inattention, irritability, emotional instability, learning problems, and hyperactive behavior.

A meta-analysis found an association between pediatric sleep-disordered breathing and ADHD symptoms and reported behavioral improvement after adenotonsillectomy.[16] However, observational surgical studies are vulnerable to expectation effects, regression to the mean, and caregiver-rater bias. Randomized trials provide a more cautious interpretation.

The Childhood Adenotonsillectomy Trial (CHAT) randomized 464 children with obstructive sleep apnea to early surgery or watchful waiting. Adenotonsillectomy produced greater improvements in respiratory symptoms, quality of life, caregiver-rated behavior, and polysomnographic findings. Polysomnographic normalization occurred in 79% of surgically

treated children and 46% of those assigned to watchful waiting. However, improvement in the primary objective attention and executive-function outcome did not differ significantly between groups.[17]

The Pediatric Adenotonsillectomy Trial for Snoring (PATS) evaluated 459 children with habitual snoring and mild sleep-disordered breathing. At 12 months, adenotonsillectomy did not significantly improve the primary measures of executive function or objective attention compared with watchful waiting. The surgical group nevertheless experienced greater improvement in behavior, sleepiness, symptoms, quality of life, and several cardiovascular measures.[18]

These findings should not be interpreted as evidence that surgery is ineffective. They indicate that the clearest benefits occur in respiratory symptoms, sleep-related quality of life, and caregiver-observed behavior rather than in short-term laboratory measures of attention. Adenotonsillectomy should therefore be recommended according to the respiratory indication and an individualized benefit–risk assessment, not as a universal treatment for ADHD.

Habitual snoring, witnessed pauses, gasping, labored breathing, unusual sleep positions, enuresis, morning headaches, dry mouth, tonsillar hypertrophy, obesity, and severe difficulty waking should prompt specific evaluation. Polysomnography is appropriate when obstructive sleep apnea is suspected and the result would affect management; it is not a routine diagnostic test for ADHD.

5.5 RESTLESS LEGS SYNDROME, PERIODIC LIMB MOVEMENTS, AND IRON STATUS

Restless legs syndrome (RLS) is characterized by an urge to move the legs, usually accompanied by uncomfortable sensations, beginning or worsening during rest, improving with movement, and predominating in the evening or at night. Young children may use nonspecific descriptions such as “growing pains,” “energy in the legs,” itching, insects, or the need to kick.

Periodic limb movements during sleep (PLMS) are repetitive movements documented by polysomnography. RLS is a clinical diagnosis, whereas PLMS is a physiological finding; the terms should not be used interchangeably. A systematic review and meta-analysis found RLS symptoms in approximately 11%–42.9% of children with ADHD, although prevalence varied widely because of diagnostic criteria, age, and referral setting.[19]

Iron status is relevant because iron contributes to dopaminergic function and reduced iron stores may aggravate RLS. However, iron supplementation should not be prescribed as

a nonspecific ADHD treatment. Ferritin and other iron studies are most appropriate when the history suggests RLS or PLMS, dietary deficiency, anemia, or another clinical risk. Treatment must consider the complete iron profile, inflammatory conditions, gastrointestinal adverse effects, and pediatric dosing safety.

5.6 MEDICATION-RELATED SLEEP EFFECTS

Stimulant medication can prolong sleep latency and reduce sleep duration in some children. A meta-analysis of objective sleep studies found that stimulant use was associated with longer sleep latency, lower sleep efficiency, and shorter total sleep time.[20] Greater dosing frequency was associated with longer latency, and effects differed among individuals.

Medication does not invariably worsen sleep. Effective treatment of evening impulsivity, emotional dysregulation, and rebound behavior can make bedtime easier. Conversely, an inadequately controlled afternoon rebound can create conflict and delay sleep even when the pharmacological effect has largely ended. The clinically relevant question is therefore whether sleep changed in a reproducible temporal relationship with medication initiation, dose adjustment, administration time, or formulation.

A medication review should include the drug, dose, release formulation, administration time, duration of benefit, rebound pattern, weekend use, appetite, caffeine consumption, naps, and baseline sleep before treatment. Potential responses include earlier administration, adjustment of the final dose, a different formulation, management of rebound symptoms, treatment of comorbid anxiety, or consideration of a nonstimulant.

Nonstimulants are not uniformly sleep-neutral. Atomoxetine can cause either somnolence or insomnia, while alpha-2 adrenergic agonists may cause sedation. Sedation should not automatically be interpreted as restoration of normal sleep architecture or adequate sleep duration. Treatment decisions should remain based on overall symptom control, functional benefit, adverse effects, and patient-specific priorities.

5.7 MELATONIN AND CIRCADIAN INTERVENTIONS

In a randomized trial of 105 medication-free children with ADHD and chronic sleep-onset insomnia, melatonin advanced sleep onset by approximately 27 minutes, while sleep onset became later in the placebo group. Total sleep time increased with melatonin and decreased with placebo. Despite these improvements, significant benefits were not observed for behavior, cognition, or quality of life.[21]

A smaller study of stimulant-treated children found that sleep hygiene produced an initial improvement and that melatonin provided an additional reduction in sleep-onset latency among nonresponders. The combined approach substantially improved sleep onset over follow-up, but ADHD symptoms did not significantly change.[22]

These findings support melatonin as a treatment for selected sleep-onset or circadian problems, not as a direct ADHD treatment. Timing is clinically important because melatonin has both chronobiotic and sleep-promoting effects. Administration at an inappropriate time may produce limited benefit or undesired phase shifting. Product quality, dose, adverse effects, other medications, and the indication should be reviewed by a qualified clinician.

5.8 BEHAVIORAL SLEEP INTERVENTIONS

Behavioral treatment has the strongest evidence when the sleep phenotype involves bedtime resistance, inconsistent routines, parent-dependent sleep onset, or insufficient sleep opportunity. Components may include a consistent wake time, a predictable wind-down routine, positive reinforcement, graduated limit setting, bedtime fading, management of anxiety, and a structured digital-media plan.

The most informative randomized trial enrolled 244 children with ADHD and sleep problems. The intervention consisted of two consultations addressing behavioral sleep strategies and sleep hygiene, followed by telephone support. ADHD symptom scores improved modestly at three and six months. Moderate or severe sleep problems at three months occurred in 30% of the intervention group and 56% of the control group, corresponding to a number needed to treat of approximately four.[23]

Mediation analysis suggested that improved sleep explained approximately half of the short-term ADHD benefit and one-third of the benefit at six months. This indicates that the intervention may also have improved family organization, parenting confidence, or treatment engagement. Importantly, the magnitude of improvement was greater for sleep than for core ADHD symptoms.

Systematic reviews support behavioral sleep interventions but emphasize small numbers of trials, heterogeneous treatment components, variable outcome instruments, and limited long-term evidence.[24,25] Behavioral intervention should consequently be presented as an evidence-based component of comprehensive care, not as a replacement for established ADHD treatment when clinically indicated.

Table 2

Phenotype-based assessment and management framework

Sleep phenotype	Key clinical indicators	Assessment	Initial management direction	Expected effect on ADHD-related impairment
Behavioral insomnia	Bedtime conflict, inconsistent routines, parent-dependent sleep onset	Clinical history and 1–2-week diary	Stable routine, behavioral reinforcement, limit setting, management of anxiety	Often improves bedtime behavior and family burden; core symptom benefit is generally modest
Delayed circadian timing	Reliable late sleep onset, difficult waking, better sleep on a later schedule	Diary; actigraphy in selected cases	Fixed wake time, morning light, reduced evening light, appropriately timed melatonin when indicated	May improve morning attention and sleepiness
Insufficient or irregular sleep	Short school-night sleep, weekend catch-up, all-nighters, long naps	Weekday/weekend diary; actigraphy if uncertainty remains	Increase sleep opportunity, stabilize timing, address devices and schedules	Experimental evidence supports improvement in inattention, oppositionality, and affect
Sleep-disordered breathing	Snoring, gasping, pauses, labored breathing, morning headache, enuresis	Pediatric sleep or otolaryngology evaluation; polysomnography when indicated	Disorder-specific treatment, including adenotonsillectomy when appropriate	Behavior and sleepiness may improve; objective attention benefit is not assured
RLS or PLMS	Evening leg discomfort relieved by movement, restless sleep, family history	Clinical criteria; iron studies and polysomnography when indicated	Correct documented iron deficiency and treat the confirmed movement disorder	May reduce sleep fragmentation; not a general ADHD treatment
Medication-associated disturbance	Sleep change after treatment initiation or dose/formulation change	Baseline and follow-up diary linked to dose timing	Adjust timing, dose, formulation, rebound management, or medication choice	Prevents avoidable worsening without unnecessarily undertreating ADHD

RLS: restless legs syndrome; PLMS: periodic limb movements during sleep.

5.9 CLINICAL ASSESSMENT

Sleep screening should occur during the initial ADHD evaluation, before and after relevant medication changes, and whenever treatment response is incomplete. A practical screen should investigate:

- Bedtime and wake time on school days and weekends.
- Sleep-onset latency and nighttime awakenings.
- Actual sleep opportunity and estimated sleep duration.
- Snoring, gasping, breathing pauses, and unusual sleep positions.
- Restless legs symptoms and repetitive nocturnal movements.
- Nightmares, parasomnias, nocturnal seizures, and enuresis.

- Daytime sleepiness, unplanned naps, and difficult morning awakening.
- Caffeine, energy drinks, medication timing, and evening digital-media use.
- Anxiety, depression, autism-related sensory problems, and family routines.

A one- to two-week sleep diary is often the most useful next step. Actigraphy is appropriate when schedules are uncertain, variability is clinically important, or an objective multi-night estimate would change management. Polysomnography should be reserved for a defined sleep-medicine indication such as suspected obstructive sleep apnea, clinically significant PLMS, unusual parasomnias, or possible nocturnal epilepsy.

Daytime outcomes should be measured separately at home and school. Parent-rated improvement may reflect better mornings, reduced conflict, and lower sleepiness, while teacher-rated symptoms may remain unchanged. Objective cognitive testing evaluates another level of function and should not be expected to reproduce every clinically meaningful behavioral improvement.

5.10 INTERPRETATION OF TREATMENT RESPONSE

The most consistent treatment gradient is that sleep interventions improve sleep more than they improve core ADHD symptoms. This is expected because sleep problems represent only one contributor to the clinical presentation. A successful intervention may reduce sleep-onset latency, morning irritability, daytime sleepiness, and family conflict while leaving clinically significant inattention or impulsivity that continues to require ADHD-specific treatment.

Outcome interpretation should therefore separate:

1. Improvement in the sleep disorder.
2. Improvement in caregiver-observed behavior.
3. Improvement in teacher-observed classroom functioning.
4. Improvement in objective attention or executive performance.
5. Change in the threshold for an ADHD diagnosis.

These outcomes are related but not interchangeable. Combining them into a single conclusion can exaggerate treatment effects or incorrectly classify a useful sleep intervention as ineffective.

6 LIMITATIONS

The available evidence is limited by considerable methodological heterogeneity. Studies use different definitions of insomnia, insufficient sleep, sleep-disordered breathing, RLS, and clinically relevant ADHD change. Sleep questionnaires, actigraphy algorithms, polysomnographic thresholds, and symptom scales also vary substantially.

Many studies are cross-sectional and rely on the same caregiver to rate both sleep and ADHD symptoms, increasing the risk of shared-method variance. Reverse causation is difficult to exclude, and several studies incompletely adjust for medication, anxiety, depression, autism spectrum disorder, oppositional behavior, obesity, pubertal development, socioeconomic conditions, screen exposure, and family routines.

Experimental and randomized evidence remains comparatively limited. Sleep-restriction experiments have relatively small samples and short intervention periods. Behavioral trials frequently cannot mask participants or caregivers, and surgical trials enroll children according to respiratory criteria rather than ADHD status. Neutral objective cognitive outcomes therefore cannot be generalized to every child with both ADHD and sleep-disordered breathing.

This review is a structured narrative synthesis rather than a prospectively registered systematic review with duplicate screening and a de novo meta-analysis. Its conclusions emphasize consistency across study designs, quantitative landmark evidence, clinical applicability, and the distinction between subjective and objective outcomes.

Long-term evidence is particularly scarce. Few studies determine whether sustained sleep improvement modifies academic achievement, injuries, emotional disorders, family functioning, or medication requirements over several years. Evidence is also limited for preschool children, girls, socioeconomically diverse populations, children with severe psychiatric comorbidity, and comparative strategies for melatonin timing and dose.

7 CONCLUSION

Sleep disorders and disturbed sleep are common in children and adolescents with ADHD and are associated with greater daytime and family impairment. The most consistent associations involve difficulty initiating sleep, bedtime resistance, insufficient or irregular sleep, difficult morning awakening, and daytime sleepiness. Subjective sleep differences are more robust than polysomnographic abnormalities.

The relationship is bidirectional and heterogeneous. ADHD-related executive and emotional dysregulation can destabilize sleep, while insufficient or fragmented sleep can aggravate inattention, oppositionality, emotional instability, and sleepiness. Experimental sleep restriction supports a causal contribution to selected symptoms but does not indicate that sleep loss is the primary cause of ADHD.

Treatment should be directed at the identified sleep phenotype. Behavioral intervention is appropriate for bedtime and routine problems; circadian strategies and carefully timed melatonin may improve delayed sleep onset; sleep-disordered breathing and restless legs syndrome require disorder-specific evaluation; and medication timing should be reviewed whenever sleep changes during ADHD treatment.

Improvement in sleep should be evaluated separately from changes in ADHD symptoms, school performance, and objective cognition. Treating a sleep disorder can remove a reversible component of impairment and clarify the residual ADHD phenotype, but it should not be presented as a universal substitute for evidence-based neurodevelopmental treatment.

Future research should combine longitudinal and experimental designs, objective verification of sleep change, blinded multi-informant ADHD outcomes, standardized sleep phenotypes, adequate follow-up, and prespecified mediation analyses. Current evidence supports routine sleep assessment and individualized management as essential components of comprehensive ADHD care.

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