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Review Article

The Bidirectional Relationship Between Sleep Disturbances and ADHD: A Narrative Review of Behavioral, Cognitive, and Neurobiological Mechanisms

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Abstract

ADHD and sleep disturbances represent one of the most clinically significant yet incompletely understood comorbid relationships in contemporary psychiatry, in part because much of the previous literature has treated sleep disturbance as a downstream consequence of ADHD rather than as a potential contributing factor to symptom expression. This review presents an integrative bidirectional model that brings together behavioral, cognitive, and neurobiological findings on the ADHD–sleep relationship across the lifespan. A structured narrative synthesis was undertaken, informed by a systematic literature search of five bibliographic databases covering January 2010 to December 2024, with included studies organized according to a three-tier mechanistic framework (behavioral, cognitive, neurobiological) and a corresponding bidirectional model. Across pediatric, adolescent, and adult cohorts, sleep problems are reported in approximately 25–80% of individuals with ADHD, depending heavily on the operational definition and assessment modality used; sleep-onset latency is increased by roughly 15–30 minutes relative to controls in several studies; experimental sleep loss is associated with moderate-to-large cognitive decrements (Hedges' g ≈ −0.32 to −0.78) in general-population samples, with preliminary evidence that individuals with high ADHD symptom loads may be disproportionately affected; and dim-light melatonin onset is delayed by approximately 1–1.5 hours, alongside reported associations with circadian clock-gene polymorphisms of modest and heterogeneous effect. The review offers an organizing framework for identifying modifiable sleep-related targets in ADHD case formulation and for designing chronotype-informed, non-pharmacological interventions, while explicitly acknowledging the predominantly correlational nature of the underlying evidence.

Keywords

Attention-deficit/hyperactivity disorder, Sleep disturbance, Bidirectional relationship, Circadian rhythm, Non-pharmacological intervention

1. Introduction

Attention-deficit/hyperactivity disorder (ADHD) is among the most common neurodevelopmental disorders worldwide. Epidemiological analyses of Global Burden of Disease data indicate an increasing trend in age-standardized prevalence and disability-adjusted life years (DALYs) across 204 countries from 1990 to 2019 (Cortese et al., 2023). This upward trend should be interpreted with caution: it likely reflects, at least in part, broadening diagnostic criteria, increased clinical awareness, and improved case ascertainment and reporting, rather than a straightforward rise in underlying impairment. Beyond the core features of inattention, hyperactivity, and impulsivity, contemporary accounts have increasingly conceptualized ADHD as extending across the 24-hour cycle—a framing that, while heuristically useful, applies to a subset rather than to all individuals, given the marked heterogeneity of ADHD presentations. Within this broader view, the disorder is associated with behavioral features such as bedtime struggles and disrupted sleep–wake cycles; cognitive features including inattention, working-memory impairment, and executive dysfunction that are themselves partly sleep-dependent; and neurobiological features involving prefrontal–striatal circuitry that may be modulated by circadian and homeostatic dysregulation (Becker, 2020). The clinical relevance of these overlapping features is heightened by their apparent bidirectionality: insufficient or poor-quality sleep can exacerbate core ADHD symptoms, plausibly via reduced prefrontal functioning, whereas the neurobiology of ADHD may predispose to delayed sleep phase and other circadian-rhythm disturbances, potentially establishing a self-reinforcing cycle (Faraone et al., 2021).

Substantial progress has been made in characterizing the descriptive epidemiology of this comorbidity, with meta-analyses and expert syntheses documenting elevated subjective and, to a lesser degree, objective sleep disturbances in children, adolescents, and adults with ADHD relative to matched controls. Although some authors have described sleep disturbance as highly characteristic of ADHD, not all individuals with ADHD exhibit clinically significant sleep problems, and this heterogeneity should be kept in view. The predominant approach in this literature has nonetheless been largely unidirectional, treating sleep disturbance as a consequence of the disorder rather than as a potential contributing factor to symptom expression, with the behavioral, cognitive, and neurobiological evidence strands often examined in isolation rather than integrated within a coherent explanatory framework.

This narrative review aims to address this gap by assembling convergent evidence on the

bidirectional interaction between sleep problems and ADHD across behavioral, cognitive, and neurobiological levels, proposing an integrated account of this association, appraising the methodological imbalance of the current evidence base, and outlining candidate treatment strategies.

2. Literature Review

2.1. Epidemiological Evidence on ADHD and Sleep Problems

Over the past decade, epidemiological research has consistently identified sleep disturbance as among the most frequently reported co-occurring problems in ADHD, with prevalence estimates exceeding those observed in typically developing peers. It is important, however, to specify what is being counted: estimates vary substantially depending on whether one refers to formally diagnosed sleep disorders or to broader subjective sleep complaints, and prevalence figures should be read in light of this distinction. In a systematic review and meta-analysis of adolescents comprising 2,465 individuals with ADHD and 18,417 controls across thirteen case-control studies, adolescents with ADHD reported significantly greater subjective sleep complaints and excessive daytime sleepiness, together with smaller but detectable polysomnographic differences, indicating a meaningful sleep-related burden in adolescent ADHD (Marten et al., 2023). Comparable patterns are observed in adults: pooling subjective and objective data across studies, several self-reported sleep variables and selected actigraphic indices—such as increased sleep latency and reduced sleep efficiency—distinguish adults with ADHD from matched controls, whereas polysomnographic differences remain comparatively subtle (Díaz-Román et al., 2018). The recurring discrepancy between pronounced subjective ratings and modest objective deviations, summarized in Table 1, admits several explanations that should be weighed before invoking underlying pathology, including reporting and recall bias, sleep-state misperception, parental-report bias in pediatric samples, and methodological variability across measurement modalities. With these caveats in mind, the modality-dependent pattern is most parsimoniously interpreted as reflecting a combination of genuine sleep difficulty and measurement-related factors, rather than either alone.

Table 1

Prevalence of Sleep Problems and Common Assessment Tools in ADHD Populations

Age group	Reported prevalence range	Predominant phenotypes	Common assessment tools
Children (6–12 yr)	25–50%	Bedtime resistance, prolonged sleep latency, night waking	Parent-report (CSHQ), actigraphy, PSG
Adolescents (13–17 yr)	40–70%	Delayed sleep phase, daytime sleepiness, short sleep duration	Self-report (PSQI, SDSC), actigraphy
Adults (≥18 yr)	60–80%	Insomnia, delayed sleep phase, fragmented sleep	PSQI, ISI, actigraphy, PSG

Note. Prevalence ranges synthesised from multiple cross-sectional and meta-analytic sources and vary with operational definition and assessment modality. CSHQ = Children's Sleep Habits Questionnaire; PSQI = Pittsburgh Sleep Quality Index; SDSC = Sleep Disturbance Scale for Children; ISI = Insomnia Severity Index; PSG = polysomnography.

2.2. Phenotypic Spectrum and Assessment Frameworks of Sleep Disorders in ADHD

The sleep disorders observed in ADHD do not constitute a unitary phenotype but span a wide range, including insomnia, delayed sleep phase syndrome, sleep-disordered breathing, parasomnias, and movement disorders. Restless legs syndrome is particularly notable, with meta-analytic data showing elevated bidirectional co-occurrence and suggesting shared underlying mechanisms involving dopaminergic and iron-metabolism pathways (Migueis et al., 2023). Proper characterization of these phenotypes nonetheless demands methodological care, because modality-specific discrepancies are substantial: an analysis of objective sleep continuity in children and adolescents with ADHD, drawing on forty-five studies involving 1,622 patients and 2,013 controls, found fewer differences than parent- or self-report measures imply, with actigraphy and polysomnography frequently detecting no significant effect even amid considerable subjective burden (Liang et al., 2023). This pattern has clear clinical significance: reliance on any single modality risks either over- or under-estimating the true impact of sleep disturbance. Accordingly, multimodal characterization—combining subjective measures, actigraphy, and, where feasible, polysomnography—is preferable for robust phenotype definition, although we recognize that full multimodal assessment is not always feasible in routine research or clinical settings and that the optimal assessment battery remains to be established by consensus.

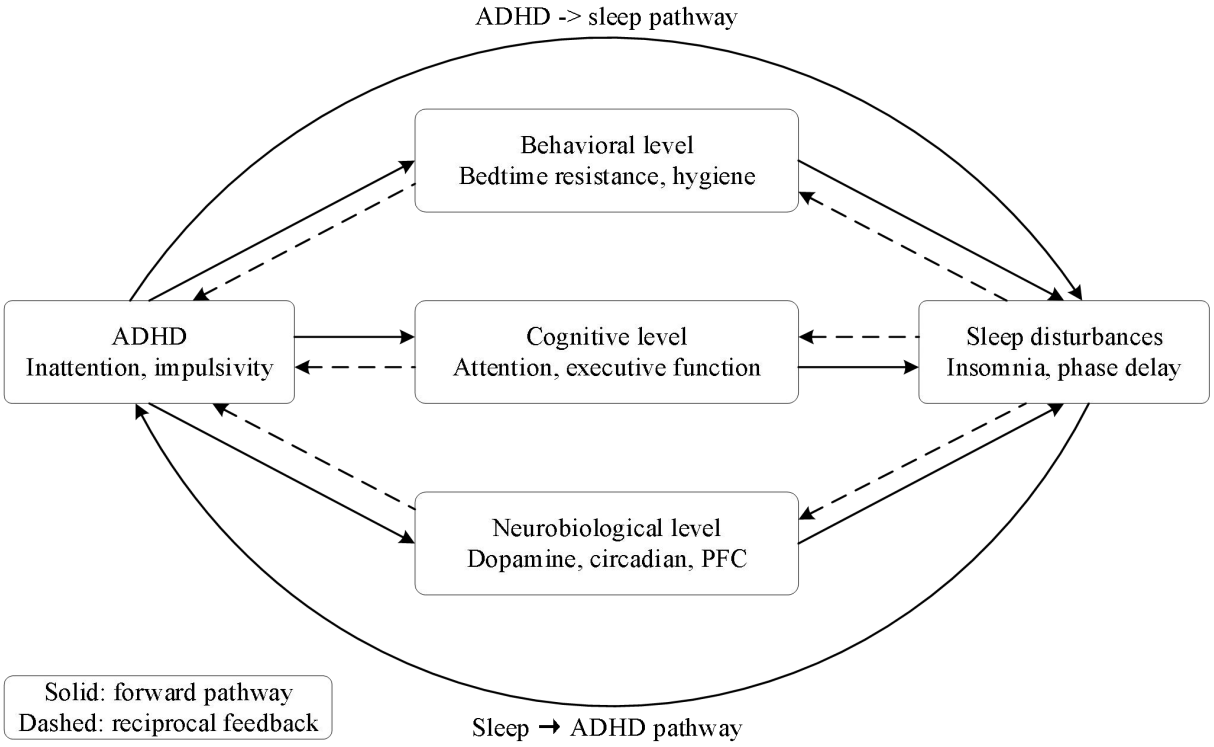
2.3. Major Theoretical Models of the ADHD–Sleep Relationship

Models of the ADHD–sleep relationship have evolved from unidirectional toward bidirectional integration, with circadian-rhythm disruption increasingly treated as a primary rather than a secondary issue. The hypothesis that chronic circadian delay is a core feature rather than a mere by-product of ADHD—supported by reports of delayed dim-light melatonin onset, higher rates of evening chronotype, and associations with circadian clock-gene polymorphisms—suggests that circadian factors warrant closer attention in diagnostic formulation, though the genetic associations in particular are of modest and inconsistently replicated effect and should not be overstated (Bijlenga et al., 2019). Building on this perspective, the integrative framework summarized in Figure 1 articulates how dopaminergic hypofunction, circadian disruption, and prefrontal–striatal vulnerability may interact. In the ADHD-to-sleep direction, dopaminergic and arousal-regulatory atypicalities, together with behavioral difficulties around bedtime, can delay sleep onset and shift circadian phase; in the

sleep-to-ADHD direction, circadian misalignment and curtailed or fragmented sleep can degrade prefrontally mediated attention and executive control, thereby amplifying daytime symptoms. This reciprocal formulation reframes sleep disruption not merely as a co-occurring condition to be treated separately but as a potentially intrinsic component of the disorder's expression—an emphasis that may be particularly relevant when pharmacotherapy incompletely addresses sleep-related impairment.

Figure 1

Conceptual Framework of the Bidirectional Relationship Between ADHD and Sleep Disturbances



3. Data and Methods

3.1. Search Strategy and Information Sources

This review adopts a structured narrative synthesis approach, which is well suited to integrating heterogeneous evidence across behavioral, cognitive, and neuroscientific domains without imposing the homogeneity assumptions required for quantitative meta-analysis (Sukhera, 2022). To enhance transparency and reduce selection bias, the narrative synthesis was supported by a systematic literature search; we emphasize, however, that the present work is a narrative rather than a systematic review, and the search procedures below are reported to document coverage rather than to support quantitative pooling. Searches were conducted across five bibliographic databases—PubMed, Web of Science, PsycINFO, Scopus, and Embase—selected for their complementary coverage of psychiatry, neuroscience, sleep medicine, and developmental psychology. The search window spanned January 2010 to December 2024, with deliberate emphasis on studies published between 2020 and 2024 to

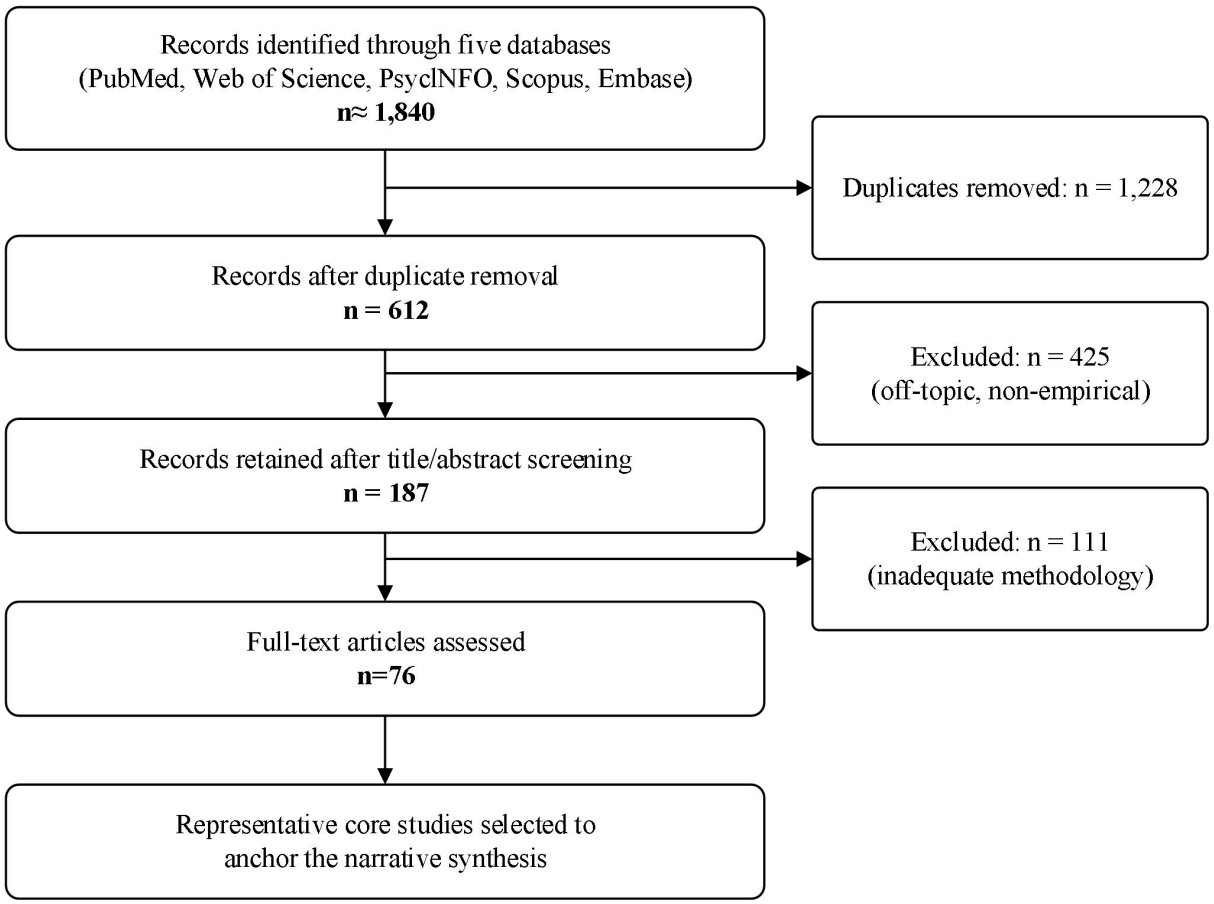
reflect the rapid methodological evolution of the field. Search terms combined ADHD diagnostic terminology with sleep-related terms (e.g., insomnia, delayed sleep phase, restless legs syndrome, sleep-disordered breathing) and assessment-related terms (e.g., polysomnography, actigraphy).

3.2. Eligibility Criteria and Screening Procedure

Records were considered for inclusion if they were English-language, peer-reviewed articles addressing the empirical or theoretical relationship between ADHD and sleep parameters in human participants of any age, with emphasis on systematic reviews, meta-analyses, and primary studies employing validated measurement tools. Editorials, conference abstracts lacking sufficient methodological detail, and case studies with fewer than ten participants were not prioritized. From an initial pool of approximately 1,840 records, 612 remained after duplicate removal; title and abstract screening yielded 187 records, and full-text assessment narrowed these to 76 articles. From this set, a core group of representative studies was selected to anchor the thematic synthesis, prioritizing the highest-quality and most directly relevant sources at each mechanistic level; consistent with the narrative format, the reference list reports these key sources rather than an exhaustive enumeration of every record consulted. The search and selection process is summarized in Figure 2.

Figure 2

Summary of the Literature Search and Selection Process



3.3. Analytical Approach to Narrative Synthesis

Thematic coding proceeded through iterative reading and inductive categorization. Each source was mapped onto a three-tier mechanistic framework distinguishing behavioral, cognitive, and neurobiological levels of evidence, and—where the source spoke to directionality—onto a bidirectional scheme distinguishing sleep-to-ADHD from ADHD-to-sleep pathways. Assignment to a mechanistic tier was based on the primary construct and outcome measures reported (for example, bedtime behavior and sleep-hygiene measures were coded as behavioral; attention, working-memory, and executive-function tasks as cognitive; and neurotransmitter, circadian, genetic, or neuroimaging measures as neurobiological). Because many studies span more than one level, sources were permitted to contribute to multiple tiers, and such overlap was tracked and resolved by reference to each study's principal aim. Reporting was guided by the SANRA criteria (Baethge et al., 2019); we note that SANRA is a tool for appraising the reporting quality of narrative reviews rather than a method for synthesizing evidence, and here it was used to structure and check the justification, aims, literature coverage, and referencing of the review rather than to govern study selection or data extraction.

4. Results

4.1. Behavioral Mechanisms: Bedtime Resistance and Daytime Dysregulation

The behavioral literature points to bedtime refusal and disorganized sleep–wake patterns as among the most replicable behavioral markers of ADHD-related sleep problems. Synthesized findings from a large body of pediatric work indicate that delayed sleep onset, evening preference, and bedtime resistance occur frequently enough to be regarded as characteristic behavioral markers rather than incidental associations with daytime symptoms (Bondopadhyay et al., 2022). The associated pattern appears bidirectional: pre-sleep cognitive arousal and difficulty disengaging from stimulating activities—features common in children with ADHD—are linked to sleep-onset delays of roughly 15–30 minutes relative to typically developing peers, while the resulting curtailment of sleep is, in turn, associated with greater next-day impulsivity and hyperactivity. Family-level factors may compound this cycle, as parental ADHD and insomnia symptoms have been associated with poorer child sleep hygiene independent of the child's own symptom level, indicating that behavioral influences operate at both individual and interactional levels.

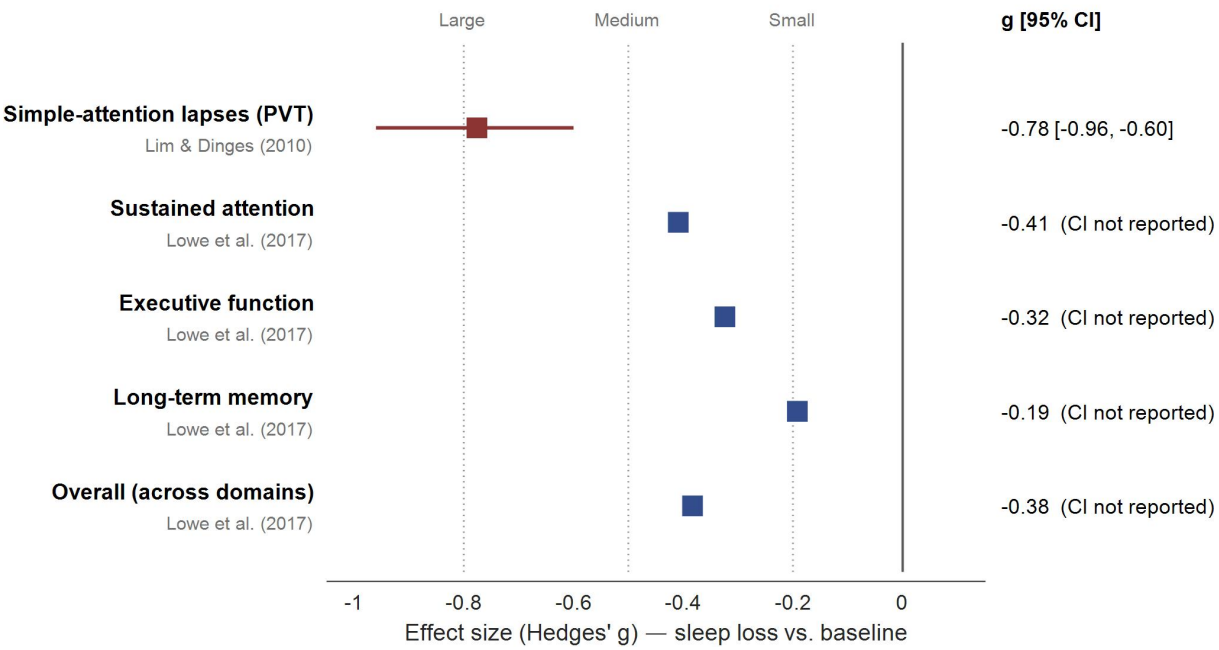
4.2. Cognitive Mechanisms: Attention, Executive Function, and Working Memory

The cognitive consequences of sleep loss are well characterized in the general sleep literature, where deprivation and restriction produce a graded pattern of decrement, with sustained attention

typically most affected, followed by working memory and inhibitory control. Quantitatively, meta-analyses of predominantly healthy, non-ADHD samples report the largest decrements for sustained-attention measures ($g \approx -0.41$ in Lowe et al., 2017, rising to $g \approx -0.78$ for psychomotor-vigilance lapses in Lim & Dinges, 2010) and smaller but reliable effects on executive function ($g \approx -0.32$; Lowe et al., 2017). These benchmark estimates are derived from general-population studies and are presented here as an illustrative reference frame rather than as ADHD-specific effect sizes; their extrapolation to ADHD populations should therefore be treated as provisional. The most direct evidence bridging this general literature to ADHD comes from experimental work showing that individuals with high subclinical ADHD symptom loads exhibit greater executive-function vulnerability to sleep deprivation than matched lower-symptom controls (Floros et al., 2021). Taken together, these findings are consistent with a model in which sleep loss acts as an additive stressor on a pre-existing attentional and executive vulnerability, such that even modest sleep curtailment may push susceptible individuals across a functional threshold—although direct, adequately powered experimental studies in diagnosed ADHD samples remain comparatively scarce. The domain-specific pattern of effects drawn from the general-population meta-analyses is illustrated in Figure 3.

Figure 3

Effect Sizes of Sleep Restriction/Deprivation on Cognitive Domains: An Illustrative Synthesis



Note. Estimates are drawn from general-population meta-analyses (Lim & Dinges, 2010; Lowe et al., 2017) and are presented for illustration only; this figure does not represent a meta-analysis of ADHD samples. Negative g indicates cognitive decline under sleep loss; benchmarks per Cohen (1988): |g| = 0.2 small, 0.5 medium, 0.8 large.

4.3. Neurobiological Mechanisms: Neurotransmitters, Circadian Rhythm, and Brain Networks

At the neurobiological level, several interacting systems—rather than any single pathway—appear to mediate the behavioral and cognitive features of the comorbidity. Dopaminergic hypofunction in mesocortical and nigrostriatal pathways, central to dopaminergic accounts of ADHD, intersects with sleep–wake regulation through dopaminergic influences on the suprachiasmatic nucleus and melatonin secretion. Polymorphisms in CLOCK, BMAL1, PER2, and PER3 have been associated with both evening chronotype and adult ADHD symptoms, and dim-light melatonin onset is reported to be delayed by approximately 1–1.5 hours in adults with ADHD relative to controls; as noted above, these genetic associations are of modest and variable magnitude. Functional neuroimaging broadly converges with these findings, with reports of reduced prefrontal–striatal connectivity and altered default-mode-network deactivation in individuals with ADHD and co-occurring sleep problems.

Importantly, a dopamine-centered account, while foundational, is no longer sufficient on its own, and several additional systems have gained prominence in recent years. The orexin/hypocretin system, a hypothalamic regulator of arousal and sleep–wake stability, has been proposed as a contributor to the alertness deficits and sleep–wake instability observed in ADHD (Cortese, Konofal, & Lecendreux, 2008). Sleep-dependent glymphatic clearance has more recently been implicated: reduced glymphatic indices have been reported in both children and adults with ADHD and linked to cognitive performance, offering a candidate mechanism by which disrupted sleep could feed back onto neurocognitive function (Fang et al., 2025). Converging evidence also points to low-grade neuroinflammation and immune dysregulation, with systematic reviews and meta-analyses indicating altered peripheral inflammatory markers in ADHD (Anand et al., 2017; Misiak et al., 2022); notably, a prospective birth-cohort study found that inflammation partly accounted for the longitudinal association between early-childhood sleep problems and later ADHD, directly implicating an inflammatory pathway in the sleep–ADHD link (Morales-Muñoz et al., 2023). Finally, large-scale network accounts—particularly the triple-network model of salience, central-executive, and default-mode network interactions—provide a systems-level framework in which aberrant salience-network switching contributes to inattention and may be sensitive to sleep-related state fluctuations (Menon, 2011).

These strands suggest that no single mechanistic level is sufficient to explain the comorbidity. Rather, a cross-level account is required—one in which circadian misalignment, dopaminergic and arousal-regulatory atypicalities, impaired clearance and inflammatory processes, and large-scale network dysfunction act as mutually interacting contributors. We emphasize that the directionality of several of these links (for example, whether circadian misalignment exacerbates dopaminergic

dysfunction or the reverse) is inferred largely from cross-sectional and associational data and remains to be established by longitudinal and experimental work. The principal mechanistic strands are summarized in Table 2.

Table 2

Summary of Key Mechanistic Evidence Across Behavioral, Cognitive, and Neurobiological Levels

Mechanistic level	Primary findings	Representative measures	Directional implication
Behavioral	Elevated bedtime resistance, prolonged sleep-onset latency, eveningness preference, family-level transmission	CSHQ, sleep diary, actigraphy	Bidirectional; behavioral routines and daytime symptomatology reinforce one another
Cognitive	Graded decline in sustained attention, working memory, and inhibitory control under sleep loss (benchmarks from general-population studies)	N-back, Stroop, continuous performance tests	Predominantly sleep-to-ADHD; sleep restriction amplifies pre-existing attentional vulnerability
Neurobiological	Dopaminergic hypofunction, delayed DLMO (~1–1.5 h), CLOCK/BMAL1/PER variants (modest effect), reduced prefrontal–striatal connectivity; emerging orexin, glymphatic, inflammatory, and network contributions	DLMO sampling, fMRI, genotyping, inflammatory markers	Bidirectional; circadian, dopaminergic, and arousal systems mutually constrain one another

Note. CSHQ = Children's Sleep Habits Questionnaire; DLMO = dim-light melatonin onset; fMRI = functional magnetic resonance imaging. Emerging mechanisms (orexin/hypocretin, glymphatic clearance, neuroinflammation, large-scale network dysfunction) are discussed in Section 4.3.

4.4. Moderators and Sources of Heterogeneity: Subtype, Development, and Medication

The preceding synthesis treats ADHD at a relatively general level, but the ADHD–sleep relationship is moderated by several sources of clinical heterogeneity that warrant explicit consideration. First, ADHD presentations differ: the predominantly inattentive, predominantly hyperactive/impulsive, and combined presentations are not interchangeable with respect to sleep, and there are indications that sleep phenotypes—for example, daytime sleepiness versus sleep-onset insomnia—may differ across presentations, although the evidence is not yet definitive. Second, developmental stage shapes both the predominant sleep phenotype and the likely direction of effect: bedtime resistance and behavioral dysregulation are more salient in childhood, delayed sleep phase

and short sleep duration in adolescence, and insomnia and circadian delay in adulthood (see Table 1), and the balance of bidirectional influence may shift correspondingly across the lifespan. Third, and critically for interpretation, pharmacological treatment is a potential confounder: stimulant medications can themselves prolong sleep-onset latency and shorten sleep duration in some individuals, so an observed association between ADHD and sleep disturbance may reflect, in part, treatment effects rather than the disorder itself. Disentangling medication-related from disorder-intrinsic sleep disturbance—ideally using medication-naïve samples or within-subject titration designs—remains an important priority, as underscored by recent clinical updates on the management of sleep disturbance in ADHD (Cortese et al., 2024). These moderators imply that aggregate estimates should be interpreted as averages over heterogeneous subgroups, and that subgroup-sensitive designs are needed to refine the model.

5. Discussion

Synthesized across behavioral, cognitive, and neurobiological levels, the available evidence offers meaningful support for reconceptualizing the ADHD–sleep relationship as genuinely bidirectional, rather than as a unidirectional consequence of the disorder, with each level plausibly contributing to a self-reinforcing cycle. The experimental finding that individuals high in subclinical ADHD symptoms are especially susceptible to sleep-deprivation-related executive impairment is consistent with the cognitive evidence reviewed above (Floros et al., 2021). The circadian perspective adds an important dimension: the proposal that ADHD may be productively conceptualized as extending across the 24-hour cycle, grounded in clock-gene associations and dopaminergic–circadian interactions, has been advanced as a theoretical model and provides a rationale for chronotherapeutic approaches such as timed light exposure and melatonin supplementation (Korman et al., 2020). We stress, however, that this framing is a proposed model rather than an established biological consensus, and that the supporting genetic and circadian evidence remains heterogeneous; it is best regarded as a hypothesis-generating perspective and an area of ongoing debate. Clinically, these considerations bear on differential diagnosis, since sleep-related executive dysfunction may be mistaken for primary attentional deficit.

Several inconsistencies temper these conclusions. Objective polysomnographic findings are generally more modest than subjective reports would suggest—a discrepancy whose likely sources (reporting bias, sleep-state misperception, and modality-related measurement differences) were discussed in Section 2.1. The relationship appears bidirectional but may be asymmetric across development, with some interpretations of the literature suggesting a predominance of ADHD-to-sleep influence in childhood and of sleep-to-ADHD influence in adulthood; we emphasize that this developmental asymmetry is an interpretation of largely cross-sectional and heterogeneous evidence

rather than a conclusion established by longitudinal or meta-analytic data, and it requires direct longitudinal testing. The reported magnitude of melatonin-onset delay in adults also varies considerably across studies; this heterogeneity is itself informative and may stem from differences in study design, participant characteristics, medication status, chronotype distribution, and DLMO measurement protocols, which future work should explicitly model.

On the treatment side, non-pharmacological approaches have accumulated encouraging but still preliminary evidence. A modified cognitive-behavioral therapy for insomnia adapted for adult ADHD has shown improvements in insomnia symptoms and daytime functioning in feasibility and pilot work, suggesting a promising adjunct—particularly where pharmacotherapy alone does not address sleep-related impairment—though the strength of this recommendation should be tempered by the early-stage, often uncontrolled nature of the available studies (Jernelöv et al., 2019). Mindfulness-based and other behavioral interventions targeting pre-sleep arousal and sleep hygiene are additional candidate options; however, the current evidence base for these approaches in ADHD is limited and of variable quality, and they should be presented as plausible but not yet firmly established.

Several methodological limitations of the underlying literature warrant emphasis: the predominance of cross-sectional designs, which preclude causal inference; the relative scarcity of adult compared with pediatric studies; the limited use of objective sleep measures; and the shortage of longitudinal studies capable of testing bidirectional dynamics. Recent reviews accordingly recommend longitudinal, multimodal designs—combining actigraphy, polysomnography, and neuroimaging—together with experimental sleep manipulation and chronotype-, subtype-, and age-stratified treatment algorithms (Cortese et al., 2024).

6. Conclusion

This review applied a structured narrative synthesis, guided by SANRA reporting criteria and supported by a systematic search of five bibliographic databases, to integrate evidence on the ADHD–sleep relationship across children, adolescents, and adults, organizing sources by behavioral, cognitive, and neurobiological mechanism and by direction of effect. Across cohorts, sleep disturbance is reported in approximately 25–80% of individuals with ADHD (with estimates strongly dependent on definition and modality); sleep-onset latency is increased by roughly 15–30 minutes relative to controls in several studies; experimental sleep loss is associated with moderate-to-large cognitive decrements ($g \approx -0.32$ to -0.78) in general-population samples, with preliminary indications of heightened susceptibility among those with high ADHD symptom loads; and dim-light melatonin onset is delayed by approximately 1–1.5 hours, alongside modest and heterogeneous associations with CLOCK, BMAL1, and PER variants.

The contribution of this review lies in assembling these disparate evidence streams into a multi-level, bidirectional organizing framework—rather than a single established causal pathway—intended to help clinicians identify modifiable, sleep-related targets in ADHD case formulation and to orient translational researchers toward chronotype-informed interventions. Given the predominantly correlational and cross-sectional nature of the current evidence, several of the proposed cross-level links remain inferential, and longitudinal, multimodal, and subgroup-sensitive studies will be needed to test them directly.

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Ethics approval

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Consent to participate

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