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METHYLPHENIDATE AND SLEEP IN ADULTS WITH ADHD - A NARRATIVE REVIEW

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ABSTRACT

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental condition that is highly prevalent among children. However, the persistence of symptoms of this condition into the adulthood should not be dismissed. The worldwide prevalence of adult ADHD is estimated to affect about 2.5% to 3.4% of the adult population and is associated with severe behavioural, emotional and social disfuncions. Chronic sleep disturbances, most frequently insomnia and deleyed sleep-wake phase disorder, are among the most commonly reported symptoms in adult clinical presentation of ADHD.

In this paper we combined scientific evidence form most recent studies and research papers published between January 2016 and June 2026 concerning the effects of short- and long-acting methylphenidate formulations on sleep quality and sleep-onset latency in adults with ADHD. We searched across PubMed and Google Scholar databases in accordance with predefined methodological inclusion criteria to investigate the connection between stimuland-based pharmacotherapy and sleep efficiency.

Although stimulant pharmacotherapy may prolong sleep-onset latency by approximately 10 to 20 minutes in a subset of patients, clinical findings indicate that overall sleep duration and sleep efficiency generally remains unimpaired. Polysomnographic records further suggest that optimized methylphenidate dosing does not significantly disrupt the microstructure of physiological sleep architecture, including deep sleep (N3) and rapid eye movement (REM) stages. Moreover, modern modified release formulations demonstrate potential benefits in stabilizing circadian regulation, reducing nocturnal racing thoughts and mitigating the evening rebound of ADHD symptoms.

Ultimately, methylphenidate appears to have a promising safety profile regarding the sleep quality and architecture in adult population. Maintaining treatment adherence and improving patients' quality of life require the appropriate use of long-acting formulations, individualized dosage schedules, and consideration of patients' baseline chronotype.

KEYWORDS

ADHD, Attention-Deficit/Hyperactivity Disorder, Adults, Methylphenidate, Stimulants, Sleep Architecture, Sleep Quality, Sleep Onset, ADHD Pharmacotherapy

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1. Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a prevalent neurodevelopmental disorder that typically manifests first symptoms in early childhood. Until quite recently, it was commonly assumed that this condition rarely persisted in adult population. However, according to recent evidence, a substantial proportion of patients continue to experience symptoms throughout adolescence and into the adulthood. The global prevalence of adult ADHD is estimated on average to range from 2.5% to 5% of population. The clinical presentation of ADHD differs significantly between children and adults. While motor hyperactivity is a predominant feature in children, adults with ADHD more commonly experience disorganization, emotional dysregulation, internal restlessness, and chronic mind wandering. These symptoms can significantly impair patients' social, academic and occupational functioning. Furthermore, ADHD frequently co-occurs with other psychiatric disorders and mental health conditions; therefore early diagnosis and appropriate treatment are essential for improving patients' quality of life.

One of the most prominent features of the adult ADHD phenotype is chronic sleep disturbance. According to recent population-based analyses and multicentre registry studies (Ahlberg et al., 2023) difficulties with sleep initiation and maintenance affect between 60% and 80% of adults with diagnosed ADHD. Clinically, these patients often exceed the five-point threshold in the global score of the Pittsburgh Sleep Quality Index (PSQI), a well-established instrument used successfully to distinguish between "good" and "poor" sleepers with high sensitivity. This tendency appears to persist regardless of treatment status (Wigal et al., 2020).

This phenomenon arises from the complex pathophysiology of ADHD and involves both behavioural factors and specific molecular abnormalities, including a delayed phase of melatonin secretion (Coogan et al., 2019). This mechanism contributes to significant disturbances of endogenous circadian rhythms.

Contemporary therapeutic guidelines for adult ADHD rely primarily on pharmacotherapy, with psychostimulants — particularly methylphenidate hydrochloride — serving as first-line medications. Methylphenidate acts as a potent norepinephrine-dopamine reuptake inhibitor (NDRI), increasing the availability of these neurotransmitters in the synaptic cleft, predominantly within the prefrontal cortex and striatal regions. Numerous studies have demonstrated the efficacy of methylphenidate in alleviating the core axial symptoms of ADHD, including attention deficits and impulsivity). However, its direct impact on sleep quality and sleep architecture remain a subject of ongoing scientific debate, raising important clinical questions regarding the management of sleep disturbances in daily psychiatric practice.

From a pharmacological perspective, stimulants may induce insomnia as an adverse effect resulting from prolonged stimulation of the reticular activating system. This phenomenon is particularly pronounced with the use of immediate-release (IR) formulations, which require multiple daily doses and may contribute to an evening rebound effect of ADHD symptoms. Consequently, recent clinical research has increasingly focused in recent years on modern extended-release and modified-release formulations, such as osmotic release oral systems (OROS) or multilayered controlled-release capsules (PRC-063) (Zhu et al., 2023; Goodman et al., 2017). In the context of methylphenidate's effects on sleep, the concept of the "stimulant paradox" has emerged as an important topic of chronofarmacotherapy (Jaeschke and Sułkowska, 2025). Although methylphenidate may prolong sleep-onset latency or reduce subjective sleep efficiency in a subset of patients, it paradoxically may also attenuate racing thoughts, decrease evening anxiety, and facilitate the circadian rhythm stabilization in certain individuals.

Understanding how optimized methylphenidate dosing influences both subjective sleep measures (assessed using sleep diaries and the PSQI) and objective sleep parameters (evaluated through actigraphic and polysomnographic studies) is crucial for improving the overall quality of life of these patients.

This paper provides a systematic synthesis of the most recent scientific evidence published between January 2016 and June 2026 evaluating the impact of short- and long-acting methylphenidate formulations on the quality, duration, and physiological architecture of sleep in adult patients with ADHD. The analysis incorporates both subjective patient-reported outcomes and objective laboratory findings, with particular emphasis on the clinical implications for optimizing treatment and dosing strategies.

2. Methodology

We searched two electronic databases – PubMed as the primary database and Google Scholar as a supplementary database – to identify most recent and comprehensive evidence regarding the impact of methylphenidate pharmacotherapy on sleep quality and sleep architecture in adult population with Attention-Deficit/Hyperactivity Disorder (ADHD). We limited our research to most recent publications including the time period between January 2016 and June 2026.

In PubMed database, the following search strategy was used: ("ADHD" OR "attention deficit hyperactivity disorder") AND "adults" AND "sleep quality" and "methylphenidate". In Google Scholar, a simplified search string was applied: ADHD adults sleep quality methylphenidate. We sorted the search results by relevance, and manually screened the first ten pages (100 items) to ensure methodological rigor and comprehensive coverage of the available literature.

We based the selection of the publications on predefined substantive and methodological criteria. Only articles published in English were included.

Inclusion criteria:

- Population: Adult patients (over 18 years of age) with a formal clinical diagnosis of ADHD according to DSM-IV, DSM-5, ICD-10, or ICD-11 criteria.
- Intervention: Pharmacological treatment using methylphenidate, including both immediate-release (IR) formulations and modern modified- or extended-release formulations (e.g., OROS and PRC-063). Comparative studies involving non-stimulant medications (atomoxetine) were also permitted.
- Outcomes: Assessment of sleep parameters using objective methods (polysomnography [PSG] and actigraphy) or validated subjective measures (e.g., the Pittsburgh Sleep Quality Index [PSQI], insomnia rating scales, prospective sleep diaries). Population-based and epidemiological studies evaluating the baseline prevalence of sleep disorders and related comorbidities in adults with ADHD were also considered for inclusion when relevant.
- Study design: Randomized controlled trials (RCTs), cohort studies, prospective open-label studies, systematic reviews, and meta-analyses.

Exclusion criteria:

- Studies conducted exclusively in paediatric or adolescent populations (under 18 years of age).
- Publications evaluating only non-pharmacological interventions (e.g., cognitive behavioural therapy [CBT], neurofeedback) without specifically assessing the effects of methylphenidate.
- Studies involving polypharmacy in which methylphenidate was administered concomitantly with substances known to affect sleep architecture (e.g., selective serotonin reuptake inhibitors [SSRIs]).
- Studies that did not evaluate sleep-related outcomes (e.g., studies evaluating only daytime functioning and productivity).
- Opinion pieces, conference abstracts, editorials, commentaries, and unpublished doctoral or master's dissertations that had not undergone scientific peer review).

We proceeded the literature selection process according to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. In the initial step we combined results from both databases and removed the duplicates. Subsequently, we screened the titles and abstracts for eligibility according to the predefined inclusion criteria, resulting in the exclusion of publications involving inadequate population groups (e.g., paediatric population). Further, we examined full texts of potentially relevant articles and excluded several papers as a result of failing to meet our methodological requirements including insufficient quality of evidence, exclusive focus on non-pharmacological intervention or absence of relevant sleep-related outcomes.

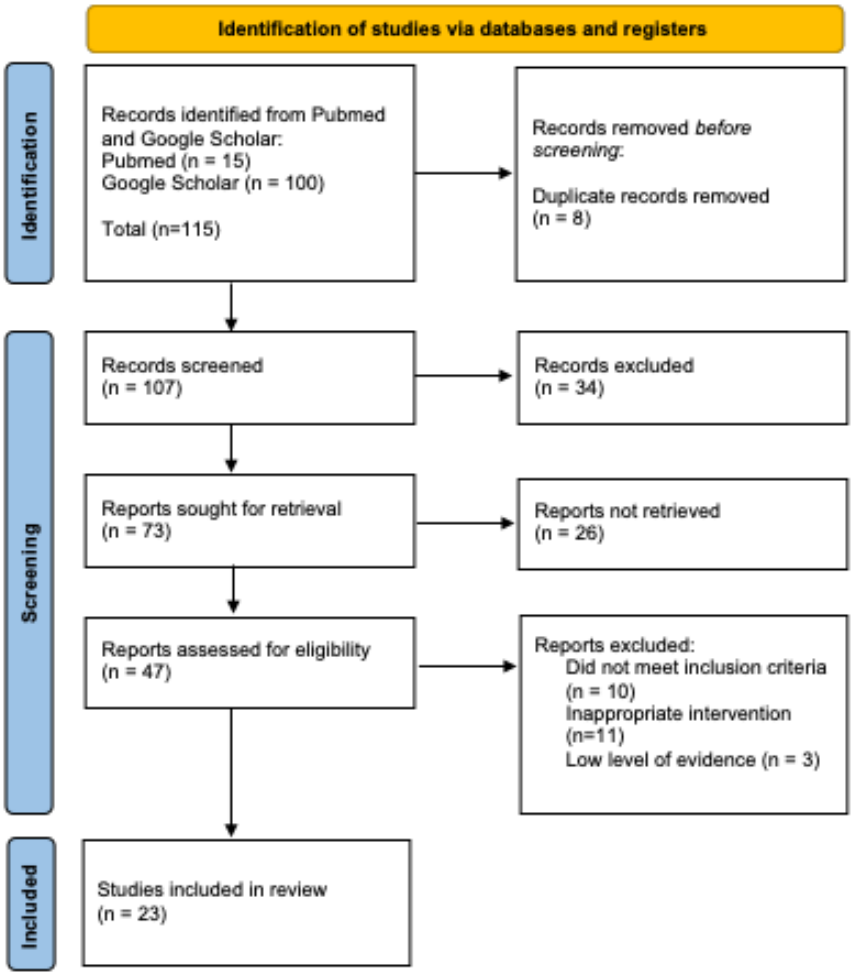

Fig. 1.

Ultimately, 23 scientific publications met all inclusion criteria and were included in the qualitive data synthesis presented in the Results section.

3. Results

3.1 Baseline Sleep Disturbances in Adults with ADHD

The reviewed literature consistently demonstrated that sleep disturbances are highly prevalent among adults with ADHD, regardless of pharmacological treatment status. The prevalence of sleep-related difficulties was estimated to affect between 60% and 80% of adults with ADHD, making sleep impairment one of the most prominent clinical features of the disorder. Large epidemiological studies reported significantly higher rates of insomnia, delayed sleep–wake phase disorder among adults with ADHD compared with the general population. Moreover, prescriptions for sleep medication were substantially more frequently reported in this population. (Ahlberg et al., 2023; Adamis et al., 2026).

Meta-analytic studies provided by Díaz-Román et al. (2018) confirmed that adults with ADHD exhibit significantly poorer subjective sleep quality, longer sleep onset latency, increased nocturnal awakenings, and greater daytime sleepiness compared with healthy controls (Díaz-Román et al., 2018). Similarly, Coogan et al. (2019) identified substantial alterations in sleep–wake behaviour and circadian regulation, including delayed melatonin secretion rhythms and disrupted molecular circadian markers.

3.2 Subjective Assessment of Sleep Parameters Based on Questionnaires and Patient Diaries

Findings regarding the effects of methylphenidate on subjective sleep quality are heterogeneous. The majority of prospective studies reported either neutral or beneficial effects of methylphenidate treatment on subjective sleep quality.

Wigal et al. (2020) conducted a randomized, placebo-controlled crossover trial investigating impact of modern, multilayered formulation of methylphenidate hydrochloride (PRC-063) on subjective sleep parameters. The analysis of sleep quality using the Pittsburgh Sleep Quality Index (PSQI) showed statistically shorter sleep duration and lower sleep efficiency during the active PRC-063 treatment phase compared to the placebo phase. However, the mean sleep duration in both cohorts remained within the range of 6 to 7 hours per night. These findings led to conclusion that these changes were not clinically meaningful. Furthermore, the global PSQI score exceeded the 5-point threshold during both active treatment and placebo phase, confirming the characteristics of adults with ADHD as "poor sleepers", regardless of the stimulant treatment.

Several investigations suggested that improvements in ADHD symptom control may indirectly enhance sleep by reducing evening hyperactivity, racing thoughts, emotional dysregulation, and bedtime procrastination. Weiss et al. (2021) investigated a multilayer extended-release methylphenidate formulation (PRC-063) treatment and reported no clinically meaningful deterioration of subjective sleep outcomes during either the double-blind or open-label phases of treatment. Although treatment was associated with transient decline of sleep quality during dose optimization, long-term follow-up showed that sleep measures stabilized, with no clinically significant deterioration in overall sleep quality.

In a six-week prospective study, Low et al. (2023) observed significant improvements in self-reported sleep quality following methylphenidate initiation. Participants reported reductions in sleep-related complaints despite exposure to a stimulant medication traditionally associated with insomnia. Likewise, Adamis et al. (2026) observed that optimized pharmacotherapy was associated with stabilization of sleep patterns and reduced insomnia severity in a substantial proportion of patients.

Similar findings were reported by Cataldo et al. (2022), who found no significant worsening of daily sleep diary measures in adults receiving multilayer extended-release methylphenidate formulations. The stable release of methylphenidate minimizes evening fluctuations in dopamine levels, allowing patients to maintain predictable sleep onset times.

In a comparative study by Ni and Lin (2017), the effects of methylphenidate and the non-stimulant atomoxetine were compared. Authors showed that while atomoxetine exhibits a milder profile regarding sleep latency, methylphenidate was more effective at reducing the subjective sense of morning fatigue and improving daytime alertness, which secondarily contributed to a better evaluation of sleep quality.

However, insomnia still remains one of the most commonly reported adverse effects of stimulant treatment. Data synthesized in umbrella reviews and systematic evaluations indicate that a subset of patients experiences delayed sleep onset, reduced sleep duration, or worsening insomnia symptoms, particularly during treatment initiation or dose escalation (Gosling et al., 2025; Stein et al., 2022).

3.3 Objective Assessment of Sleep Architecture and Nocturnal Motility (PSG and Actigraphy

Evidence regarding the direct impact of methylphenidate on objective sleep architecture remains limited. The most informative data originate from polysomnographic investigations.

Fredriksen et al. (2021) conducted a pilot polysomnography study in adults with ADHD receiving methylphenidate treatment. This work directly monitored the sleep architecture of adult ADHD patients before and after the stabilization of methylphenidate treatment. Contrary to expectations, treatment was not associated with major disruptions of sleep architecture. The PSG results proved that an optimal dosage of methylphenidate does not induce pathological shifts in sleep stages. Changes in traditional polysomnographic variables, including total sleep time, sleep efficiency, and sleep-stage distribution, were generally modest. This study proved that stimulant pharmacotherapy does not disrupt the physiological microstructural pattern of sleep. Moreover, some participants demonstrated improvements in sleep continuity and reductions in nocturnal restlessness.

These conclusions may be supported by the systematic review and meta-analysis by Diaz-Román et al. (2018) that synthesized findings from existing objective and subjective studies. Actigraphy analyses show that extended-release stimulants treatment may prolong objective sleep latency on average by 10 to 20 minutes compared to placebo. However, this meta-analysis confirms that Wake After Sleep Onset (WASO) and nocturnal motility (movement in bed) do not worsen. Furthermore, in some cases these factors may stabilize, correlating with reduction of nocturnal motor hyperactivity specific to the adult ADHD phenotype. These findings align with a systematic review by Surman (2022) based on objective actigraphy. Surman demonstrated that the effect of stimulants on sleep is highly individualized, and objective sleep disturbances following methylphenidate initiation largely depends on the patient's baseline sleep architecture prior to treatment.

Overall, current evidence does not support the hypothesis that methylphenidate causes substantial disturbances in sleep-stage composition. Instead, the available data suggest predominantly mild effects on sleep architecture, with considerable interindividual variability.

3.4 Circadian Rhythms and the Molecular Biological Clock

Several studies indicate that methylphenidate may influence circadian functioning beyond its effects on core ADHD symptoms. Coogan et al. (2019) demonstrated associations between medication status and normalization of molecular circadian rhythm markers. Researchers proved that methylphenidate influences the expression of biological clock genes. Moreover, they observed the resynchronization of the molecular profile, which may lead to circadian rhythm stabilization and increased morning activation. More recently, Adamis et al. (2026) found that pharmacological treatment was associated with improved circadian rhythm stability and reduced insomnia severity over time. The long-term use of methylphenidate resulted in a shift in patients' chronotypes toward a more normative direction, reducing the phenomenon of so-called "social jetlag".

The relationship between stimulant treatment and daytime sleepiness appears particularly noticeable. Haimov et al. (2026) reported shifts in the daytime sleepiness profile among young adults with ADHD receiving stimulant medication, suggesting improved regulation of alertness throughout the day. Preventing of afternoon energy crises resulted in natural and healthy accumulation of sleep pressure toward the end of the day. These findings may partly explain why some patients report improved nighttime sleep despite exposure to a psychostimulant.

I contrast, emerging evidence indicates that abrupt discontinuation of methylphenidate may negatively affect sleep regulation. Cueto et al. (2026) observed significant disturbances in rest–activity rhythms following medication withdrawal, suggesting that stable pharmacological management may play an important role in maintaining circadian organization in adults with ADHD.

Overall, the reviewed evidence indicates that although methylphenidate may modestly increase sleep-onset latency in some individuals, it does not appear to cause major disturbances in sleep architecture and may contribute to improved sleep quality through better control of core ADHD symptoms, especially after dosage optimalization.

4. Discussion

The results from most recent studies and publications shed new light on the impact of methylphenidate pharmacotherapy on sleep quality and architecture in adult patients with ADHD. Until quite recently it was commonly believed that methylphenidate treatment usually has a negative impact on nocturnal rest, because of its stimulant effect. The findings challenge the traditional assumption that stimulant treatment inevitably worsens sleep and instead support a more nuanced understanding of the relationship between ADHD pharmacotherapy and sleep regulation.

One of the most consistent findings across the reviewed studies is that sleep disturbances are highly prevalent in adults with ADHD regardless of medication status. Epidemiological and clinical investigations consistently demonstrate elevated rates of insomnia, delayed sleep phase syndrome, and poor sleep quality among adults with ADHD. Consequently, sleep impairment should be considered an intrinsic component of the adult ADHD phenotype rather than only a side effect of stimulant treatment.

The concept of the "stimulant paradox," recently discussed by Jaeschke and Sułkowska (2025), offers a useful framework for interpreting these findings. In healthy individuals, psychostimulants induce a state of cortical hyperactivation. However, in adults with ADHD, the baseline neurobiological state is characterized by a chronic deficit in dopaminergic transmission within the prefrontal cortex. This deficit manifests as racing thoughts, anxiety, internal restlessness, and the phenomenon of uncontrolled mind wandering, which often leads to prolonged sleep-initiation latency. The introduction of an optimal dose of methylphenidate restores neurochemical homeostasis, dampens cognitive chaos, and allows the patient to enter a state of relaxation necessary for falling asleep. This dual mechanism may explain why many patients report subjective improvements in sleep despite receiving stimulant medication.

The distinction between immediate-release and extended-release formulations appears clinically important. Studies evaluating OROS methylphenidate and PRC-063 consistently demonstrated favourable sleep outcomes, suggesting that modern delivery systems may reduce fluctuations in plasma drug concentrations and minimize evening symptom rebound. These findings support current recommendations favouring long-acting formulations for adult patients requiring all-day symptom control. Although definitive conclusions cannot yet be drawn, current evidence suggests that formulation selection may represent an important component of individualized sleep management strategies.

Another notable observation is the discrepancy between subjective and objective sleep measures. While some patients report longer sleep-onset latency or transient insomnia, polysomnographic studies generally demonstrate no substantial alterations in sleep architecture. Deep sleep and REM sleep appear largely preserved during optimized methylphenidate treatment. Adverse effects appear to be expressed primarily through changes in sleep continuity, circadian regulation, and subjective sleep experience. These observations are consistent with emerging evidence linking ADHD to disturbances in circadian biology rather than solely to insomnia-related mechanism. This finding is clinically reassuring given the importance of these sleep stages for memory consolidation, emotional processing, and neurocognitive functioning.

Another important observation is the substantial heterogeneity of treatment responses. Some patients experience improvements in sleep quality following treatment initiation, whereas others report worsening insomnia. Differences in dosage, formulation, timing of administration, baseline sleep characteristics, psychiatric comorbidities, and individual chronotype likely contribute to this variability. Recent longitudinal data emphasize the importance of considering comorbid anxiety disorders, mood disorders, and pre-existing sleep disorders when evaluating treatment outcomes

The reviewed literature also highlights the importance of individualized chronopharmacological approaches. Patient chronotype, timing of medication administration, comorbid psychiatric disorders, and baseline sleep disturbances may all influence treatment outcomes.

Future clinical protocols should increasingly incorporate chronobiological assessments to optimize both ADHD symptom management and sleep quality.

Limitations of the Analysed Studies

Several limitations should be acknowledged. First, substantial heterogeneity existed across studies regarding outcome measures, medication formulations, dosing strategies, and follow-up duration. Second, objective sleep assessments remain uncommon, with most studies relying on self-reported outcomes. Relatively few studies employed objective sleep assessments such as polysomnography. Furthermore, polysomnographic studies, while exceptionally precise, were conducted on small patient cohorts due to high costs and logistical difficulties associated with laboratory sleep registration. Third, long-term data extending

beyond one year of treatment remain limited. Finally, many studies were conducted in highly selected clinical populations, potentially limiting generalizability to routine psychiatric practice. There is also a distinct need for clearer differentiation of clinical ADHD subtypes in analyses, given that the baseline profile of circadian rhythm disturbances may differ significantly between them.

5. Conclusions

In conclusion, methylphenidate pharmacotherapy in the adult population with ADHD diagnosis exhibits a generally favourable safety profile that is neutral toward sleep architecture. Although a subset of patients may experience a minor prolongation of sleep-onset latency, clinically significant disruption of sleep architecture appears uncommon. In a subgroup of patients exhibiting evening racing thoughts, it can paradoxically facilitate calming and enhance satisfaction with nocturnal rest. The key to therapeutic success lies in shifting away from immediate-release formulations in favour of modern modified-release systems, strict adherence to morning administration schedules, and personalizing doses based on the patient's chronotype. Sleep disturbances in adults with ADHD should be treated as an integral component of the clinical picture of the disorder itself, rather than a priori as an adverse effect of properly managed stimulant pharmacotherapy.

Conflict of interests: No conflicts of interest to declare.

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