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REVIEW ARTICLE

# Understanding the Impact of Stimulants on Sleep in ADHD: Evidence from Systematic Assessment of Sleep in Adults

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## Abstract

Stimulants are widely prescribed to manage attention-deficit/hyperactivity disorder (ADHD) in adults. Stimulants promote wakefulness and can produce insomnia side effects. We hypothesized that systematic studies of sleep effects would reveal patterns of sleep impairment that may be important for clinicians to monitor and manage. We conducted a review and analysis of studies that measured sleep systematically during stimulant treatment in adults. We identified nine studies that met our search criteria, including four double-blind placebo-controlled studies. All studies recorded self-report subjective sleep quality data, three studies collected actigraphy data, and three studies collected polysomnography data. One study found better subjective sleep quality under open-label treatment conditions. Both polysomnography studies found improvement in aspects of sleep patterns. Two of the actigraphy studies suggested that adults receiving stimulant treatment may have less movement during sleep, and one showed reduction in amount of sleep. Further research could inform best practices for maintaining sleep quality during stimulant treatment.

## Key Points

Research suggests that stimulant treatments for attention-deficit/hyperactivity disorder (ADHD) are associated with higher rates of insomnia.

Studies suggest that, on average, adults with ADHD receiving extended-release formulations of stimulants, compared with those receiving placebo, report similar sleep experiences and a similar likelihood of shifts in the quality of their sleep.

Comprehensive treatment of adults with ADHD should include monitoring for the overall impact of stimulant treatment on sleep/wake balance and adjust treatment accordingly. Further research may confirm if delay of sleep onset and refreshingness of sleep are particularly important indicators of sleep impact to measure.

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## 1 Introduction

Methylphenidate and amphetamine salt stimulants are widely prescribed to manage the inattentive and impulsive/hyperactive symptoms of attention-deficit/hyperactivity disorder (ADHD), among other applications. Regulatory agencies have approved a range of medications for ADHD, which use a variety of delivery systems to deliver methylphenidate or amphetamine salt enantiomers. These include products designed to mitigate ADHD symptoms during waking hours with a single administration. However, stimulant agents increase wakefulness, so poorly timed delivery of these agents could infringe on adequate patterns or quality of sleep [1]. Inadequate sleep duration, timing, or quality can all have negative impacts on physical and mental wellbeing, but the impact may be particularly burdensome for individuals who already have self-regulation challenges because of ADHD. Ideally, treatments that reduce the self-regulation challenges of patients with ADHD during the day would also allow for, or even facilitate, refreshing sleep.

The core symptoms of ADHD may contribute to behavior patterns and activity schedules that compromise healthy sleep patterns [1]. By definition, ADHD in adults results in impaired function in two or more settings across school, work, or domestic domains because of inattentive or impulsive/hyperactive traits [2]. Inefficient performance of activities of daily living in adults with ADHD can include

compromised self-care in the domestic setting [3]. Because adults with ADHD often take longer to complete tasks and are easily side-tracked to more interesting or less mentally effortful tasks, they often have challenges finishing activities of the day and transitioning to bed. Adults with untreated ADHD also often report having unsettled minds as they attempt to initiate sleep, and their insomnia may occur in part from awareness of unaddressed priorities. Executive function challenges beyond attention and moment-to-moment behavior control are common in this population, including problems with planning, time awareness, and task prioritization. These additional functional challenges can further compromise good sleep hygiene patterns, such as adherence to wind-down activities before sleep [3].

Although adults with ADHD may often experience sleep problems because of ADHD-related self-care challenges, these problems can also occur because of the mood or anxiety disorders that are commonly comorbid with ADHD. However, studies that included systematic characterization of the presence of comorbidity suggested that sleep complaints occurred at very high rates independent of the presence of comorbid mental health conditions [4, 5]. Circadian rhythm disorders may be particularly prominent in individuals with ADHD, although it is hard to disentangle whether this reflects a true comorbidity or a secondary effect of ADHD behavioral patterns [6, 7].

Because sleep problems are common in adults with ADHD, and because poor sleep compromises function, prescribers of stimulants for adults with ADHD should monitor their impact on sleep. Clinical trials of stimulant agents in adults with ADHD revealed evidence that they are likely to produce insomnia. For example, US FDA registration studies of extended-release amphetamine products found high insomnia rates: 27% for extended-release mixed amphetamine salts versus 13% for placebo [8]; 27% for lisdexamfetamine versus 8% for placebo [9]; and 31% for mixed salts of a single-entity amphetamine product versus 8% for placebo [10]. Elevated rates of insomnia were also reported in clinical trials of extended-release formulations of methylphenidate when compared with placebo. For example, a recent evaluation of a 16-h-release formulation of methylphenidate revealed higher rates of insomnia (15.8 vs. 3.8% with placebo) and initial insomnia (6.1 vs. 1.3% with placebo) [11]. It is important to note that these registration studies are conducted using forced-dose regimens that may exaggerate the impacts of treatment compared with the dose-optimization treatment regimens used in clinical care. However, rates of insomnia are also reported during dose-optimized protocols, such as the rate of 20.7% reported from the open-label extension of the study on the 16-h-release formulation of methylphenidate [12].

Methylphenidate and amphetamine are thought to act by modulating dopamine and norepinephrine levels via blockade of reuptake. In the case of amphetamines, increased release of neurotransmitters also appears to occur [13]. Their wake-promoting effects may mimic the modulating effect of catecholamine pathways, one of several neurobiological circuits that contribute to sleep/wake states [14]. Animal studies have indicated that a complex array of neurotransmitters participate in sleep/wake regulation and stages of sleep [15], and norepinephrine—released from brainstem regions to a broad array of cortical regions—has long been implicated as a factor in the modulation of both sleep/wake and rapid eye movement states [16]. In rodents, the locus coeruleus, which supplies norepinephrine to higher brain regions, fires in a pattern correlating with sleep arousal states [17].

However, evidence indicates that pathways relevant to dopamine also contribute to levels of wakefulness. This evidence is mixed and based on a small literature suggesting there may be less variation in dopamine pathway activity than in noradrenergic and other neurotransmitter activity between electroencephalogram-based sleep/wake states, that selective dopamine reuptake blockers can produce arousal, and that the wake-promoting effects of dopamine may depend on dopamine transporter activity [18, 19]. Evidence also indicates that catecholaminergic modulation via stimulant exposure could also affect the pattern of sleep stages. Animal research has shown that amphetamine suppresses rapid eye movement and slow-wave sleep [20]. Anecdotally, many patients report rebound tiredness as a result of cessation of amphetamines, offering further evidence of a shift in sleep/wake equilibrium during stimulant treatment. In addition, animal studies have suggested that methylphenidate may affect circadian patterns at the level of genetic expression [21].

Methylphenidate and amphetamine products are thus likely to have mixed and varied effects on sleep and daytime function in adults with ADHD. We conducted a review to understand the current level of evidence for the kinds of sleep problems that clinicians may expect these agents to produce as they attempt to treat ADHD. We hypothesized that studies which systematically measured sleep using scales or objective measures would show higher rates of sleep problems and might inform treatment choices and monitoring practices.

## 2 Method

The methods of this review are reported according to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. The relevant studies were

found via a search of PubMed (last accessed in September 2021).

### 2.1 Review Inclusion Criteria

We conducted a PubMed literature search with the following search terms: (sleep) AND (psychostimulants OR stimulants) AND (adhd OR attention deficit disorder OR attention deficit hyperactivity disorder) NOT (children). We also reviewed the reference lists of the reports that we found. Studies were included if they evaluated the effect of stimulant treatment on sleep in adults with ADHD using at least one objective measure or sleep-specific scale. We excluded studies that only reported adverse events of insomnia. We allowed the inclusion of studies with a mix of outcome measures, such as actigraphic variables (e.g., physical activity during sleep), polysomnography, and validated sleep scales. Conference abstracts were eligible for inclusion. Studies were included if they evaluated a drug classified as a psychostimulant (e.g., lisdexamfetamine, mixed amphetamine salts, and methylphenidate). Studies had to include participants who had ADHD, but no exclusions based on type of ADHD or method of diagnosis were made. Studies had to include only adults aged ≥ 18 years; no other exclusionary criteria were set for the study participants.

The second author (DW) independently screened abstracts found through the original PubMed search and identified studies of interest. The first author (CS) also independently screened abstracts and chose which were sufficient to be included in our review. There were no disagreements between screeners. Relevant data (sample size, intervention studied, study design, notable outcomes) were gathered from all studies and are included in Table 1.

## 3 Results

In total, 92 potentially relevant records were identified by our search of PubMed (last accessed 10 September 2021); 16 of these met initial inclusion criteria. We conducted a detailed assessment of these records and excluded nine: two did not include a stimulant intervention, one was an abstract of another study included here (which we accessed separately from the original search), and six did not include a report on sleep endpoints. Nine records are included in this review. The additional studies included [22, 23] met our eligibility criteria and were cited in other papers found in the literature search (see Fig. 1).

The studies in Table 1 include five double-blind prospective placebo-controlled studies [11, 23, 25, 26, 28], a post hoc analysis of data from two of these studies supplemented with additional data [27], a two-group comparison study [24], and three open-label studies [22, 24, 29]. The majority of studies included participants exposed to methylphenidate to treat ADHD, but lisdexamfetamine, mixed amphetamine salts, and dextroamphetamine treatments were also represented.

The double-blind studies offered the most scientifically meaningful evidence among the studies we found for stimulant treatment effects on sleep. The average study duration of these studies was 5 weeks. One of the double-blind studies used lisdexamfetamine as an intervention [23], one used mixed amphetamine salts [26], and three used some variation of methylphenidate [11, 25, 28]. These studies had large numbers of participants, except for the study by Boonstra et al. [25], in which 31 participants received methylphenidate. Two of the studies had positive findings for subjective impact of psychostimulants on sleep under double-blind conditions, with improvement in the Pittsburgh Sleep Quality Index (PSQI) subscale report of daytime function [23] and in daytime alertness on the Epworth Sleepiness Scale (ESS) [28]. The actigraphy study conducted under double-blind conditions found evidence of more restful sleep [25] but also later bedtime, slower sleep onset, and shorter sleep duration. The largest double-blind analysis, a post hoc evaluation of sleep effects of lisdexamfetamine and mixed amphetamine salts, explored self-reported change from good to bad sleep status as measured by the PSQI, or vice versa [27]. No significant change in sleep quality status was found in that analysis or in the post hoc analysis of an extended formulation of methylphenidate [11].

The design of studies presenting unblinded exposure data was heterogeneous. Overall improvement in global sleep quality as measured by the PSQI during open-label treatment with dexmethylphenidate extended-release was also reported in one study [11]. A small open-treatment study [24] found that the treated subjects had several improvements in their sleep as measured by actigraphy. In a similar open-treatment study [22], subjects' polysomnographic measures of sleep improved as compared with their matched counterparts. Lastly, a small pilot open-label polysomnographic study found that methylphenidate was associated with an increased amount of time spent in stage 2 sleep [29].

To characterize potential sources of bias in these studies, we inspected the funding sources and methods of ascertainment. Five of the studies [11, 23, 26–28] had a named industry sponsor, whereas the rest did not report industry funding. One of these [27] was itself a post hoc analysis of two studies [23, 26] that were funded by industry. Although exact recruitment sources were not mentioned, such large multicenter studies are typically a mix of community- and clinic-recruited subjects. Four other studies specifically recruited subjects from a clinic setting [22, 24, 25, 29]. The

**Table 1** Studies of stimulant treatment for ADHD in adults that included sleep/wake measurement

| Study | Population analyzed | Drug formulation | Study design | Sleep findings associated with stimulant treatment |
|---|---|---|---|---|
| Kooij et al. [24] | $N = 16$ (8 ADHD, 8 control) | MPH and dextroamphetamine | 3-week open-label treatment | Reported sleep quality improved ($p = 0.02$); actigraphy demonstrated improved sleep quality ($p < 0.02$), and lower activity level ($p < 0.01$), vs. an eight-person control group |
| Boonstra et al. [25] | $N = 72$ (33 ADHD (31 on MPH), 39 control) | MPH 0.5, 0.75, 1.0 mg/kg/day, four or five times daily | Baseline group comparison followed by 3-week dose optimization, double-blind crossover | Actigraphy demonstrated later bedtime ($p < 0.01$), later onset of sleep ($p < 0.05$), reduced total sleep duration ($p < 0.001$), fewer nighttime awakenings ($p < 0.001$), and longer periods of consolidated sleep in the ADHD group vs. non-ADHD controls |
| Spencer et al. [26] | $N = 272$ (137 MAS, 135 PL) | Triple-bead MAS | 7 weeks dose optimization, double blind | No difference in sleep quality measured by PSQI at any weekly time point |
| Sobanski et al. [22] | $N = 10$ | MPH mean 36.7 ± 11.2 mg/day | 21+ days of open-label dose optimization | Questionnaire showed better sleep restorative value ($p = 0.0174$) at endpoint vs. baseline. Polysomnography showed improved sleep efficiency ($p = 0.0354$) and shorter sleep onset latency ($p = 0.0237$) at endpoint vs. baseline |
| Adler et al. [23] | $N = 349$ (103 LDX 30 mg; 96 LDX 50 mg, 98 LDX 70 mg, 52 PL) | LDX 30, 50, 70 mg/day | 4 weeks forced-dose escalation, double blind | Treatment groups showed significant improvement in daytime functioning PSQI subscale vs. PL ($p < 0.0001$) from baseline to endpoint. No significant difference on global PSQI scores or overall sleep quality ($p = 0.1563$) |
| Surman et al. [27] | $N = 831$ (420 study 1, 411 study 2) | LDX 30, 50, 70 mg/day; triple-bead MAS: 25, 50, 75 mg/day | Post hoc analysis of data from Adler et al. [23] and Spencer et al. [26] | No significant PSQI change from good to bad sleep (defined as PSQI >5), or vice versa |
| Goodman et al. [28] | $N = 279$ (141 OROS MPH, 138 PL) | OROS-MPH: 18, 36, 54, 72 mg/day | 6 weeks dose optimization, double blind | ESS scores improved from baseline to endpoint significantly more with treatment than with PL ($p = 0.007$). No significant difference in PSQI overall or subscale scores or actigraphy measures |

**Table 1** (continued)

| Study | Population analyzed | Drug formulation | Study design | Sleep findings associated with stimulant treatment |
|---|---|---|---|---|
| Weiss et al. [11] | $N = 333$ (264 PRC-063, 69 PL) | PRC-063: 25, 45, 70, 100 mg/day | 4 weeks forced-dose escalation, double blind followed by 6 months open label | No significant PSQI change from good to bad sleep (defined as PSQI >5) or vice versa in controlled arm. Improvement in overall PSQI scores from baseline to endpoint during 6-month open-label arm ($p < 0.0001$), driven by mean sleep efficiency improvement. Quantitatively, more patients went from being bad sleepers to good sleepers than vice versa in the double-blind and open-label phases |
| Fredriksen et al. [29] | $N = 9$ | MPH-IR BID, TID, mean 43 mg | 42+ days open-label optimization | Polysomnography showed more time spent in stage 2 sleep ($p = 0.011$). ESS showed no significant change |

*ADHD* attention-deficit/hyperactivity disorder, *BID* twice daily, *ESS* Epworth Sleepiness Scale, *LDX* lisdexamfetamine, *MAS* mixed amphetamine salts, *MPH* methylphenidate, *MPH-IR* methylphenidate immediate release, *OROS MPH* osmotic-release oral methylphenidate, *PL* placebo, *PRC-063* extended-release methylphenidate, *PSQI* Pittsburgh Sleep Quality Index, *TID* three times daily

rest of the studies did not specify whether recruitment was from a specialty clinic or the community. The information reported in the studies we reviewed was insufficient for us to understand the extent to which participants with sleep problems in sleep assessment may have been preferentially included or excluded.

Extended searching identified an additional study that was similar to those that met our inclusion criteria but that we could not include because it did not separate the effects of stimulant treatment from those of atomoxetine, which also was administered in some of the study population. In this cohort study, the circadian rhythms of 22 non-ADHD controls were compared with those of both 17 participants with ADHD receiving stimulant or atomoxetine and 17 unmedicated participants with ADHD. The researchers compared the sleep/wake actigraphy patterns and ex vivo measurements of fibroblast cultures to assess rhythms of circadian gene expression and found that medicated patients with ADHD had significantly lower sleep efficiency and more nocturnal awakenings than both other cohorts. Because some of the participants were receiving atomoxetine, we could not determine whether these results were attributable to stimulant exposure [21].

## 4 Discussion

### 4.1 State of Knowledge of the Impact of Stimulants on Sleep

Our literature review did not find robust support for our hypothesis that stimulant pharmacotherapy produces sleep impairment in adults with ADHD. Only one of the nine studies that met our search criteria found evidence of worse sleep parameters—bedtime, sleep onset, and sleep duration—during methylphenidate treatment [25]. This study is one of two actigraphy studies, and the other was small and used different methods, and may not have replicated the same findings for those reasons [24]. We found weak evidence for the opposite of our hypothesis—better sleep—across the studies in our review, as one study suggested improved sleep quality on retrospective report during open-label treatment with an extended-release form of methylphenidate [11]. Actigraphy studies have suggested that adults receiving stimulant treatment may have less movement during sleep [24, 25, 29]. Two polysomnography studies associated stimulant treatment with improvements in different aspects of sleep patterns [22, 29]. We also found weak evidence for improvements in wakefulness in two double-blind controlled studies [23, 28] and one open study [22].

However, the strongest replicated scientific finding we found comes from two controlled clinical trials of extended-release methylphenidate and two of extended-release

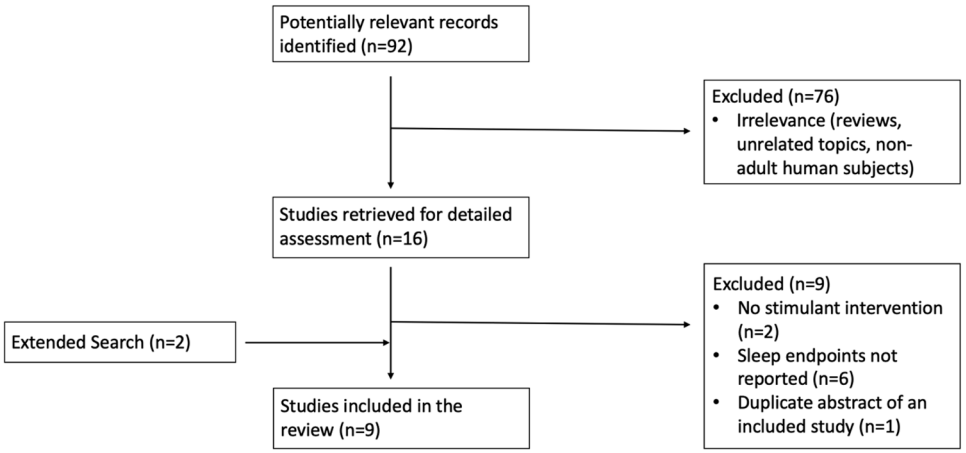


**Fig. 1** Flow diagram of the study selection process

amphetamine salt formulations was that a subjective impact on sleep quality is not detectable vs placebo on average in larger studies using retrospective self-report questionnaires [11, 23, 26–28].

The lack of measured impact of stimulant treatment on sleep in ADHD exists in contrast to the clearly higher rate of adverse events of insomnia in clinical registration trials of stimulants. This may simply be because measuring spontaneously reported insomnia is different from measuring sleep experience, activity, and sleep stages. It can be imagined that a given individual could find that stimulants produce a mix of favorable and unfavorable shifts across these diverse measurements: someone could have insomnia from exposure to a stimulant but also might sleep more restfully once they fall asleep. We also may expect divergence between insomnia reports during the visits of a clinical trial that may reflect recent experience with a dose increase in the prior several days, and sleep experience evaluation using measures such as the PSQI, which query retrospectively about a longer period of time and may be more likely to reflect the pattern a person has settled into. Evidence for this divergence exists in analyses that found no association between more frequent shifts from good to bad or from bad to good sleep, yet found robust elevations in rates of sleep adverse events [11, 23, 26]. These studies compared active treatment and placebo and reported rates of sleep adverse events of 23.6 versus 5.1%, respectively, for a 16-h methylphenidate product [11], of 29 versus 8.9% for a 12-h mixed amphetamine salt product [26], and 19 versus 5% for lisdexamfetamine [23]. We note again that studies that include forced-dose titration might be expected to produce higher insomnia rates, whereas studies with dose optimization may produce lower rates.

This pattern of findings, with both overall lack of shifts in sleep experience reports and high rates of insomnia in controlled studies, could also be explained by the phenomenon of accommodation. Just as individuals often report reduced peripheral side effects of stimulants such as decreased appetite and dry mouth, there may also be accommodation of the sleep/wake effects of these agents. Some patients report sleep problems on initiation of stimulant treatment, or with dose increases, that resolve within several days. Many patients report more fatigue on stopping stimulants for a period of days than they recall experiencing before starting these agents, suggesting that the previous presence of a stimulant had effects on sleep/wake patterns. This could reflect behavioral changes—such as tolerating tired states better on stimulants—but there might also be accommodation in central nervous system arousal mechanisms in the presence of stimulants. To our knowledge, no systematic study has been conducted to allow us to separate out whether or how often such behavioral versus physiological changes occur.

### 4.2 Implications for Clinical Assessment and Treatment Monitoring

The literature we reviewed is reassuring that stimulant impact on sleep may be minimal during treatment for adults with ADHD, but one study we reviewed also gives a hint about aspects of sleep/wake impact that clinicians may want to monitor to minimize the impact of treatment. This study suggests that actigraphy might identify delayed bedtime, sleep onset, and sleep duration in some individuals receiving stimulant treatment [25]. We also suggest, as was done in one study [22], that clinicians could monitor for the improved restorative value of sleep as a positive outcome during comprehensive treatment. This metric is of particular interest because it was identified as a sleep trait that correlated with level of daily function in the National Comorbidity Survey-Replication analysis [30]. Clinicians may also want to pay particular attention to management of evening behavior routines that are affected by ADHD. Studies of sleep impact in adolescents, for example, suggested that stimulants could improve sleep indirectly by improving sleep preparation behavior or lower opposition to sleep [31].

It also may be important for clinicians to monitor for and respond to interindividual differences in the sleep impact of stimulants. It is the first author's clinical impression that the duration of alertness effect reported by patients can differ from the duration of ADHD symptom improvement. Some patients report atypical alertness well after the focus benefit of stimulants has worn off. In contrast, some individuals do not experience much alerting effects from stimulants at all, saying they can still nap while the stimulants are active. Although it may be expected that individuals would have similar wakefulness response while on caffeine as while on stimulants, this is not always the case. Further, concomitant use of alerting or sympathomimetic agents can compound sleep impact or even obscure this impact by reducing daytime fatigue. Periodic cessation of stimulant treatment may help clarify the contribution of these factors and also help re-establish a baseline for treatment plan changes. However, it is also noteworthy that patients may be more comfortable if they taper off stimulants when stopping them over a few days, and this may avoid confounding withdrawal fatigue with an untreated natural state.

Sleep may be monitored with sleep diaries, interviews about sleep, or actual sleep measures. The widespread use of smartphones and wearable actigraphy devices offers opportunities for clinicians to characterize attributes of sleep without resorting to polysomnography. Regardless of the method used to monitor sleep effects of stimulant medication, it is important to make these methods as easy as possible for patients with ADHD to follow [1].

When clinicians attempt to address patients with both impaired wakefulness and ADHD, no comparative evidence exists to guide between choosing a stimulant versus other wake-promoting agents such as modafinil, armodafinil, or solriamfetol. Similarly, research to guide the treatment of sleep/wake problems that occur secondary to stimulant treatment is limited. Replicated exploration of how to treat sleep problems, in general, in individuals with ADHD is also scarce, although evidence has emerged for application of bright light therapy [1]. Behavioral interventions are effective at addressing many forms of insomnia, and a range of pharmacologic agents are available if these do not improve sleep. These include agents that may help mimic diurnal melatonin cues, sedative antihistaminergic agents, GABA-modulating sedative hypnotics, and agents with anxiolytic properties. We have argued that attention should be given to the next-day and sleep-quality effects of these approaches [1].

### 4.3 Areas of Interest for Further Research

Our review reassures that ADHD may often be treated with stimulants without clearly impacting sleep, but it remains clear that stimulants can produce insomnia. Patients will benefit from best practices that avoid insomnia or other compromise of sleep quality. Future research towards this goal could characterize pretreatment sleep characteristics, and how they change during treatment, to improve our ability to predict favorable sleep outcomes. Such research could help clinicians proceed with more confidence in their approach to patients with bedtime initiation behavior problems or sleep phase delays, which are common in adults with ADHD. Studies could also clarify when it is useful to address an emergent sleep/wake problem by changing to a different form of psychopharmacology for ADHD. Future studies ideally would use methods that optimize generalizability to clinical practice by accounting for or limiting selection/ascertainment biases and using methods that maximize the contribution of data about sleep impact independent of response to the intervention.

Even if stimulants lack the gross impact on sleep that subjects report in studies, they could affect—favorably or otherwise—sleep characteristics that influence daytime function or the occurrence of comorbidity. The way in which patients consume stimulants may vary significantly from how they are taken during short-term clinical trials, and future research could clarify the impact of variations in stimulant administration schedules on sleep/wake health. Studies might also clarify if there are treatment-associated changes in sleep characteristics that predict the best outcomes. Candidate characteristics to explore include circadian sleep phase, the restorative value of sleep, and sleep stage patterns. Sleep problems will often occur in patients independent of treatment effects, and—as we have explored previously [1]—it is important to understand which sleep treatments are most effective for adults with ADHD.

## 5 Conclusions

We found that sleep changes are not strongly associated with stimulant treatment, despite the known incidence of insomnia with stimulants and evidence that biological and functional sleep/wake changes could be created by these treatments. These findings are largely based on studies that measured sleep by retrospective, subjective self-report in individuals with low rates of comorbidity and used long-acting formulations of stimulants. Comprehensive care for adults with ADHD will benefit from continued attention to how sleep/wake patterns can be optimized during treatment and from further research towards optimum management of the comorbidity of sleep disorders and ADHD.

## Declarations

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