Sleep and Alertness in Children With Attention-Deficit/Hyperactivity Disorder: A Systematic Review of the Literature

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Study Objective: To review evidence on sleep and alertness in children with attention-deficit/hyperactivity disorder (ADHD) controlling for potential confounding factors.

Methods: A PubMed search. Studies using ADHD diagnostic criteria other than DSM-III-R or IV and studies not excluding or controlling for psychiatric comorbidity or medication status were not included in the review. Results from objective studies were combined using meta-analysis.

Results: From the 46 studies located, 13 were retained. With regard to objective studies, the proportion of subjects who fell asleep during the Multiple Sleep Latency Test, the number of movements in sleep, and the apnea-hypopnea index were significantly higher in children with ADHD than in controls. We found no significant differences in other objective parameters (sleep-onset latency; number of stage changes; percentages of stage 1 sleep, stage 2 sleep, slow-wave sleep, or rapid eye movement sleep; rapid eye movement sleep latency; and sleep efficiency). Limited evidence from subjective studies suggests no significant differences in sleep-onset difficulties and bedtime resistance between children with ADHD and controls, after controlling for comorbidity and medication status. Data on sleep duration, night and morning awakenings, and parasomnias are still very limited.

Conclusion: Results from our systematic review suggest that children with ADHD have higher daytime sleepiness, more movements in sleep, and higher apnea-hypopnea indexes compared with controls. Given the limited number of studies controlling for confounding factors, further subjective and objective studies are needed to better understand alterations in sleep and alertness in children with ADHD.

Keywords: Sleep, alertness, ADHD, children, adolescents, meta-analysis

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INTRODUCTION

ATTENTION-DEFICIT/HYPERACTIVITY DISORDER (ADHD) IS ONE OF THE MOST COMMON CHILDHOOD NEUROPSYCHIATRIC DISORDERS, ESTIMATED TO AFFECT 5% TO 10% OF SCHOOL-AGED CHILDREN.1,2 According to the Diagnostic and Statistical Manual of Mental Disorders-Fourth edition (DSM-IV),3 ADHD is characterized by a chronic pattern of inattention, impulsivity/hyperactivity, or both. Onset before the age of 7 and impaired functioning in 2 or more settings are required for the diagnosis.3

In recent years, there has been growing interest in sleep alterations associated with ADHD. This seems to reflect clinicians’ concern for better evaluation and treatment of those children with ADHD who may present with significant disturbances of sleep-wake patterns. Researchers have analyzed sleep and alertness in children with ADHD using both subjective and objective studies. The former have been based on questionnaires filled out by the children or their parents; the latter have used techniques such as polysomnography, actigraphy, infrared video camera, and the Multiple Sleep Latency Test (MSLT). Results from both subjective and objective studies have been inconsistent: while some authors have reported significant differences between children with ADHD and controls, others have failed to replicate these findings.4,5 Attempts to draw overall conclusions are hampered by the differing inclusion criteria of these studies. In particular, there are 3 important sources of variations that may account for some of the discrepancies seen in the results.

Firstly, the methods used to evaluate ADHD differed across studies, ranging from simple evaluation of symptoms of hyperactivity/impulsivity and inattention to more rigorous application of the diagnostic criteria listed in the various editions of the DSM (DSM-II, III, III-R, IV). It is only through the application of standardized criteria that one can arrive at an appropriate diagnosis of ADHD. However, the use of DSM-II and III criteria to assess the relationship between sleep-alertness disorders and ADHD does have some limitations. DSM-II was not based on empirical data and explicit diagnostic criteria.4 DSM-III (but not III-R or IV) included restless sleep as a defining characteristic of attention deficit disorder,5 so that studies conducted with DSM-III may have been confounded by the use of sleep disturbance as a diagnostic criterion. Therefore, the use of DSM-III-R and DSM-IV criteria minimizes subject-selection bias.

Secondly, few studies have excluded or controlled for the effect of psychiatric comorbidity. Comorbid disorders frequently associated with ADHD, such as oppositional defiant disorder, depressive disorders, and anxiety disorders,1 may account for some of the sleep disturbances found in ADHD. It has been reported that

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depressive disorders may be associated with bedtime resistance, delayed sleep onset, problems with sleep maintenance, and excessive daytime sleepiness in children. Polysomnographic studies have found alterations in sleep architecture and reduced number of body movements in depressed adolescents and, although less consistently, in children. Children and adolescents with anxiety disorders have presented in subjective studies with sleep-onset delay, bedtime resistance, problems with sleep maintenance, and sleep-related involuntary movements; in objective studies, anxiety disorders have been associated with prolonged sleep-onset time, alterations in sleep architecture, and higher density of periodic legs movements. Children with oppositional defiant disorder have presented with bedtime resistance and difficulty in sleep onset in subjective studies. Psychiatric comorbidity is therefore likely to have had a significant influence on the results of the studies comparing sleep and alertness in children with ADHD versus control subjects.

Thirdly, some studies did not exclude or control for medication status. Since pharmacologic treatment may affect sleep and alertness, the assessment of nonmedicated subjects appears more suitable to evaluate the relationship between sleep-alertness alterations and ADHD.

Although not all children with ADHD may present with sleep-alertness disturbances, an examination of possible specific sleep-alertness alterations in a subgroup of subjects, after controlling for potential confounders, may allow a better understanding of the pathophysiology of ADHD as well as of the potential therapeutic implications in the clinical management of children with ADHD.

To our knowledge, no systematic review or meta-analysis of sleep-alertness disorders in children with ADHD, controlling for the 3 above-mentioned potential confounding factors, is available. Therefore, the aims of this work were to review the literature on sleep-alertness problems in children with ADHD and to combine the results of the studies that controlled for the 3 mentioned confounding factors.

METHODS

We performed a PubMed search from 1987 (when the American Psychiatric Association first published the DSM-III-R) to October 2005, using the following key words in various combinations: ADHD, attention-deficit/hyperactivity disorder, attention, hyperactivity, sleep, alertness, and children. References from each relevant paper, including 11 existing reviews of the literature, were examined to determine if any relevant studies had been missed during the database searches. We did not include case reports or descriptive reports without data analysis. We excluded all studies that had not been performed using rigorous clinical interviews according to DSM-III-R or DSM-IV criteria. Studies that examined sleep in medicated subjects were not included.

The most frequently studied parameters from subjective and objective studies were considered for our analysis. We specifically analyzed 9 items from subjective studies: (1) bedtime resistance, (2) sleep-onset difficulties, (3) night awakenings, (4) restless sleep, (5) sleep duration, (6) difficulty with morning awakenings, (7) excessive daytime sleepiness, (8) parasomnias (other than sleep restlessness), and (9) sleep-disordered breathing (SDB). Eleven parameters from objective studies were included in our analysis: (1) sleep-onset latency (SOL), (2) number of stage changes, (3) percentage of stage 1, (4) percentage of stage 2, (5) percentage of slow-wave sleep (SWS), (6) REM latency, (7) percentage of REM, (8) sleep efficiency, (9) proportion of subjects who fell asleep during the MSLT, (10) number of movements in sleep, and the (11) apnea-hypopnea index (AHI).

We did not consider data from studies that failed to exclude or control for the following relevant psychiatric comorbid disorders: oppositional defiant disorder in the analysis of bedtime resistance and difficulties in sleep onset, depressive and anxiety disorders in the analysis of bedtime resistance, sleep-onset difficulties, night awakenings, restless sleep and movements in sleep, sleep duration, difficulty with morning awakenings, excessive daytime sleepiness, and parameters of sleep architecture.

We contacted authors when data presented in their studies were incomplete.

All these subjective and objective parameters were considered for inclusion in a formal meta-analysis. This type of analysis allows results from different studies to be combined, thereby providing a more precise estimate of differences between children with ADHD and controls. However, it was difficult to combine subjective parameters because results were presented variously as proportions, as ordered categories, and as continuous variables, as was the objective parameter “number of movements in sleep.” These were therefore summarized descriptively. Study results for the remaining 10 objective parameters were combined in meta-analyses using a fixed-effects model, with variances assumed to be different across studies. Effect size was presented overall and for each study with 95% confidence intervals and was defined as the absolute mean difference between children with ADHD and controls for continuous variables and the log odds ratio for the single binary variable. Absolute mean difference was preferred to standardized mean difference so that effect size could be presented in recognized units but could not be used for the analysis of SOL owing to particularly high heteroscedasticity (different variances) in SOL between studies. Sensitivity analyses considered standardized mean differences instead of absolute mean differences for all continuous variables, relative risk instead of odds ratio for the binary variable, and the use of random-effects models.

RESULTS

The PubMed search yielded 46 papers. Sixteen studies were excluded from our analysis because they were not conducted using rigorous clinical interviews according to ADHD-definition criteria of DSM-III-R or IV. We excluded a further 3 papers that presented case reports without data analysis. Seven studies examining subjects treated with medications affecting sleep were not included. Data from 5 studies that did not exclude or control for comorbid oppositional defiant disorder were not used in the analysis of bedtime resistance and difficulties in sleep onset. Five studies did not exclude or control for comorbidity with depressive or anxiety disorders; therefore, data from these studies relative to bedtime resistance, delayed sleep onset, problems with sleep maintenance, excessive daytime sleepiness, sleep architecture, total body movements, or periodic legs movements in sleep were not included in our analysis. Data on number of apneas per hour from the study by Andreou et al were excluded because the authors did not report findings in the control group. The recent papers by Van der Heijden et al and Sangal et al, although

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Table 1—Subjective studies on Sleep and Alertness in Nonmedicated Children With ADHD Versus Controls

<table>
<thead>
<tr>
<th>First author (year)</th>
<th>Subjects</th>
<th>Age, y*</th>
<th>Key results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marcotte (1998)</td>
<td>43 ADHD, 25 ADHD with comorbidities, 86 controls</td>
<td>8.5±1.7</td>
<td>No significant difference in sleep duration between children with ADHD and controls. Children with ADHD scored significantly higher, in the Sleepiness Scale and in the Breathing Problems Scale than controls (p &lt; .05).</td>
</tr>
<tr>
<td>Corkum (1999)</td>
<td>79 nonmedicated ADHD, 22 medicated ADHD, 35 clinical comparison, 36 nonclinical comparison</td>
<td>9.2±1.49</td>
<td>Dysomnia was related to confounding factor rather than ADHD. Combined subtype of ADHD was related to an increase in sleep-related involuntary movements (p &lt; .01). No group differences for parasomnias.</td>
</tr>
<tr>
<td>Mick (2000)</td>
<td>122 ADHD, 105 controls</td>
<td>14.6±3.1</td>
<td>After controlling for comorbidity and pharmacotherapy, no significant differences between children with ADHD and controls in bedtimes, sleep-onset difficulties, number of night awakenings, restless sleep, sleep duration, difficulty with morning awakenings, excessive daytime sleepiness, and parasomnias.</td>
</tr>
<tr>
<td>Owens (2000)</td>
<td>57 ADHD, 46 controls</td>
<td>7.5±1.6</td>
<td>Significantly higher number of night awakenings (p &lt; .05), number of parasomnias (including restless sleep and moving during sleep) (p &lt; .01), sleep duration (p &lt; .001), and daytime sleepiness (p &lt; .05) in children with ADHD compared with controls.</td>
</tr>
<tr>
<td>LeBourgeois (2004)</td>
<td>45 ADHD, 29 controls</td>
<td>9.8±2.8</td>
<td>Children with ADHD were more likely to have chronic snoring, but this difference was not statistically significant.</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD. ADHD refers to attention-deficit/hyperactivity disorder.

The results on sleep-disordered breathing were not included here because subjects with a symptom complex highly suggestive of obstructive sleep apnea were excluded from this study.

**Objective Studies**

The key results of the objective studies are summarized in Table 2. No individual study reported differences in SOL or in sleep-architecture parameters (number of stage changes, percentage of stage 1 sleep, percentage of stage 2 sleep, percentage of SWS, REM latency, percentage of REM, sleep efficiency). Number of movements in sleep was significantly higher in children with ADHD compared with controls in all 3 studies in which this was assessed.38,41,42 Both studies that used MSLT43,45 reported significantly reduced mean sleep latency in children with ADHD in comparison with the control group, and significantly higher proportions of children with ADHD fell asleep during the MSLT compared with controls. Two44,45 of the 3 studies43,44,45 that measured the AHI found significantly higher values in children with ADHD compared with controls.

Effect sizes with 95% confidence intervals from each individual study are shown in Figure 1, along with the pooled effect-size estimates obtained from the meta-analysis. The meta-analysis pooled estimates confirmed that there were no differences between children with ADHD and controls with respect to sleep-architecture parameters. There was a suggestion of higher SOL in children with ADHD, but the difference was not statistically significant. MSLT was analyzed in terms of the proportion of children who fell asleep, and the estimate of the combined log odds ratio was 2.16, equivalent to 8 times higher odds of falling asleep among children with ADHD compared with controls. Put another way, the pooled estimate of the relative risk of falling asleep during the MSLT for children with ADHD compared with controls was 1.57 (95% confidence interval: 1.23-2.00). The pooled result for the AHI was statistically significant at the 5% level, with children with ADHD having a mean about 1 point higher on the AHI scale compared with controls. Sensitivity analyses using random-effects models and standardized mean differences led to similar conclusions.

**DISCUSSION**

Our review identified only a rather limited number of studies in sleep problems in children with ADHD that controlled for potential confounding factors (13 studies). While further studies are still needed, our meta-analysis of objective parameters does at least allow more precise estimates than those of any individual study (the combined sample size of the 8 objective studies is 280 children with ADHD versus 228 controls).

Our results confirmed that SOL and sleep-architecture parameters (number of stage changes; percentages of stage 1, 2, slow-wave, and REM sleep; REM latency; and sleep efficiency) were not significantly different between children with ADHD and controls. Results from previous studies that found significant differences5,15 may thus have been accounted for, at least in part, by...
the confounding factors that we controlled in our analysis (inappropriate diagnostic criteria, comorbid psychiatric disorders, and medication status).

The pooled estimate for the odds of falling asleep during the MSLT was significantly higher among the children with ADHD, which was unsurprising because both individual studies included in this estimate were also significant. In addition, results from 2 subjective studies reported a significantly higher daytime sleepiness in children with ADHD, and this despite the known deficiencies of questionnaires filled out by parents in identifying their child’s sleepiness, which could be masked by hyperactivity. Both Lecendreux et al and Golan et al noted that their findings were consistent with the hypoarousal theory of ADHD, according to which children with ADHD are sleepier than controls and might use excessive motor activity as a strategy to stay awake and alert.

However, it should be recognized that the results on MSLT are based on just 2 studies, comparing a total of 64 children with ADHD versus 54 controls. Further, while the MSLT is considered the gold standard to assess alertness in adults, its use in children is controversial, particularly in those younger than 7 years. Although most of the subjects in the 2 studies that included MSLTs were over 7 years of age, it may not be appropriate to generalize these results to all children with ADHD. Given these limitations, further research is needed to better assess alertness and sleepiness in children with ADHD.

Number of movements in sleep was significantly higher in children with ADHD than in controls in all 3 objective studies that reported on this parameter. The finding of a significant association between ADHD and movements in sleep supports the notion that, at least in a subgroup of patients, ADHD may be a disorder with a 24-hour duration. This finding is consistent with the hypothesis that sleep disruption caused by movements in sleep may contribute to the daytime symptomatology of ADHD or that ADHD may contribute to nighttime movement. Alternatively, a third factor (such as a neurobiologic alteration) might contribute to both ADHD and movements in sleep.

Pooled results from 3 studies showed that children with ADHD had a significantly higher AHI than controls. The mean AHI

### Table 2—Objective Studies on Sleep and Alertness in Nonmedicated Children with ADHD Versus Controls

<table>
<thead>
<tr>
<th>First author (year)</th>
<th>Subjects</th>
<th>Age, ya</th>
<th>Key results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lecendreux (2000)</td>
<td></td>
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<tr>
<td>30 ADHD</td>
<td>7.8±1.6</td>
<td>No significant differences in SOL; stage changes; percentages of stage 1, stage 2, slow-wave, or REM sleep; REM latency; or sleep efficiency between children with ADHD and controls. Mean sleep latency during MSLT was significantly shorter for children with ADHD than controls. A significantly higher proportion of children with ADHD fell asleep during the MSLT than controls.</td>
<td></td>
</tr>
<tr>
<td>22 controls</td>
<td>8.4±1.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Konofal (2001)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>31 ADHD</td>
<td>7.8±1.6</td>
<td>No significant differences in SOL; stage changes; percentages of stage 1, stage 2, slow-wave, or REM sleep; REM latency; or sleep efficiency between children with ADHD and controls. Significantly higher number of movements in children with ADHD compared with controls (number of total upper limb movements, total lower limb movements, movements associated with behavioral awakenings, total number of all movements, percentage of time spent in motion and motionless).</td>
<td></td>
</tr>
<tr>
<td>21 controls</td>
<td>8.4±1.4</td>
<td></td>
<td></td>
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<tr>
<td>Huang (2004)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>88 ADHD</td>
<td>8.46±1.45</td>
<td>No significant differences in SOL; stage changes; percentages of stage 1, stage 2, slow-wave, or REM sleep; REM latency; or sleep efficiency between children with ADHD and controls.</td>
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</tr>
<tr>
<td>27 controls</td>
<td>9.00±1.96</td>
<td></td>
<td></td>
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<tr>
<td>Kirov (2004)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17 ADHD</td>
<td>11.2±2</td>
<td>No significant differences in SOL; stage changes; percentages of stage 1, stage 2, slow-wave, or REM sleep; REM latency; or sleep efficiency between children with ADHD and controls. Frequency of short movement-related epochs significantly increased in children with ADHD. Total movement time not significantly different between children with ADHD and controls.</td>
<td></td>
</tr>
<tr>
<td>17 controls</td>
<td>11.2±2.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gruber (2000)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>38 ADHD</td>
<td>9.6±2.7</td>
<td>No significant differences in SOL and sleep efficiency between children with ADHD and controls.</td>
<td></td>
</tr>
<tr>
<td>64 controls</td>
<td>9.4±1.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gruber (2004)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24 ADHD</td>
<td>8.94±1.25</td>
<td>No significant differences in SOL or sleep efficiency between children with ADHD and controls.</td>
<td></td>
</tr>
<tr>
<td>25 controls</td>
<td>8.83±1.01</td>
<td></td>
<td></td>
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<tr>
<td>Cooper (2004)</td>
<td></td>
<td></td>
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<tr>
<td>18 ADHD</td>
<td>10.5±3.0</td>
<td>No significant differences in AHI between children with ADHD and controls.</td>
<td></td>
</tr>
<tr>
<td>20 controls</td>
<td>10.0±3.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>34 ADHD</td>
<td>12.4±4.6</td>
<td>Mean sleep latency at MSLT was shorter in children with ADHD than in controls. Significantly higher proportion of children with ADHD than controls fell asleep during MSLT. The AHI was significantly higher in children with ADHD than controls.</td>
<td></td>
</tr>
<tr>
<td>32 controls</td>
<td>12.0±3.6</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Only studies rigorously using Diagnostic and Statistical Manual of Mental Disorders, Third Edition-Revised or Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition criteria were included. Studies that did not control for relevant comorbidities were not included. ADHD refers to attention-deficit/hyperactivity disorder; SOL, sleep-onset latency; REM, rapid eye movement; MSLT, Multiple Sleep Latency Test; AHI, apnea-hypopnea index; PLMI, periodic limb movement index.
problems that could mask hyperactivity. Further research should
be particularly common in children with ADHD, perhaps because
more-severe SDB may cause enough daytime sleepiness or other
problems that could mask hyperactivity. Further research should
advance the understanding of the mechanisms underlying the
link between symptoms of ADHD and apnea and hypopneas.
As suggested by Chervin et al,36 the effects of hypoxia or sleep-
fragmentation arousals associated with apnea and hypopneas
might explain the cause-and-effect relationship between SDB
and symptoms of ADHD. Specialized scoring of respiratory and
nonrespiratory arousals or computerized quantification of elec-

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Figure 1—Confidence interval plots of effect size from objective studies. Point estimates and 95% confidence intervals for effect size are given in terms of the absolute or standardized difference in means or the log odds ratio. Effect sizes greater than 0 indicate higher values in children with attention-deficit/hyperactivity disorder (ADHD) compared with controls. The results for individual studies are combined in an overall meta-analysis estimate of effect size, shown in the last line of each plot. Confidence intervals that exclude 0 can be considered statistically significant at the nominal 5% level.

SOL refers to sleep-onset latency; Shifts, stage changes; ST1 %, percentage of stage 1 sleep; ST2 %, percentage of stage 2 sleep; SWS %, percentage of slow-wave sleep; REM, rapid eye movement latency; REM %, percentage of REM sleep; SE, sleep efficiency; MSLT, Multiple Sleep Latency Test; AHI, apnea-hypopnea index.
Evidence on sleep disorders and sleep problems in children with ADHD is still limited. The results of our systematic review of SDB were strong risk factors for future development or exacerbation of symptoms of hyperactivity. However, as noted by the authors, in this study, no clinician evaluations of ADHD were used. Therefore, further prospective studies using rigorous standardized evaluations of ADHD are needed to assess the cause-and-effect relationship between SDB and ADHD.

With regard to other sleep problems addressed in subjective studies, bedtime resistance and difficulties with sleep onset were not significantly different between children with ADHD and controls after controlling for the confounding factors, suggesting that medication status and comorbid psychiatric disorders may account (at least in part) for these sleep problems in most of the subjects. However, although children with ADHD taken together did not differ from controls, one cannot rule out that a subgroup of children with ADHD may present with significant difficulties with sleep onset. Van der Heijden et al. suggested that the endogenous circadian pacemaker may be fixated at a later phase than the desired sleep-wake schedule in children with ADHD and sleep-onset insomnia, independently from a comorbid oppositional defiant disorder. This preliminary finding deserves further investigation for its potential importance in understanding the pathophysiology underlying sleep-onset difficulties in children with ADHD.

More subjective studies are also needed in relation to sleep duration, night and morning awakenings, parasomnias, and SDB, given the paucity of data from studies that assessed these issues controlling for comorbidity and medication status and using rigorous diagnostic criteria for ADHD.

CONCLUSION

Evidence on sleep disorders and sleep problems in children with ADHD is still limited. The results of our systematic review indicated that children with ADHD fell asleep more often on MSLT and had increased motoricity and higher AHIs than controls. The other parameters examined in our review were not significantly different between children with ADHD and controls. Thus, the hypothesis that children with ADHD suffer from a wide range of sleep disorders and problems was not confirmed once the confounding factors of psychiatric comorbidity, medication status, and diagnostic criteria were controlled for. However, the reviewed studies (both the objective and the subjective ones) presented some methodologic limitations, and, therefore, the notion of “disturbed sleep” in ADHD needs a more-accurate investigation. We suggest that future research should address the issues summarized below in order to improve the assessment of potential specific patterns of sleep and alertness disturbances in children with ADHD.

With regard to subjective studies, most of the data available are based on small clinical samples. Epidemiologic studies are needed in order to assess the association between sleep-alertness alterations and ADHD in the general population using rigorous and standardized criteria for the assessment of this condition. In addition, questionnaires specifically targeted to assess sleep and alertness in school-aged children and adolescents should be developed and should include questions to be answered directly by the children or adolescents themselves, since they can provide data that can be complementary to information from parents.

Concerning objective measures, several methodologic issues need to be addressed. Polysomnography and MSLTs performed in laboratory settings provide artificial conditions that may facilitate sleep onset in children with ADHD by depriving them from usual environmental stimuli. Home recordings, during both weekends and weekdays, may represent a more appropriate method of evaluating sleep patterns, controlling for potential confounders related to laboratory recording. The use of complementary techniques could also be proposed: for instance, 1 week of high-sampling actigraphy coupled with polysomnography and MSLTs could provide more specific information on sleep-wake patterns taking into consideration the variability of ADHD symptoms across time. A careful analysis of microarousals and other spectral features of the electroencephalogram is needed to find possible specific sleep alterations. “Autonomic activation” (i.e., autonomic modification without electroencephalographic sign) also merits attention for its potential effects on daytime function. With regard to actigraphy, standardization in algorithms may facilitate more-precise comparisons between studies.

Given that the expression of sleep stages is related to maturational stages, future research should address sleep alterations in subjects with ADHD in relation to the developmental and pubertal phase. Finally, a better definition of potential specific sleep-alertness patterns in relation to ADHD subtypes (“predominant inattention,” “predominant hyperactive-impulsive,” and “combined”) and to the severity of ADHD symptoms may contribute to the understanding of the pathophysiology that underlies this complex disorder, suggesting more-accurate and specific evidence-based treatment strategies.

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