Cognitive, Behavioral, and Functional Consequences of Inadequate Sleep in Children and Adolescents

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The premise that inadequate sleep can cause problems with cognition, behavior, or other aspects of daytime functioning has been long discussed in Western culture. Relevant writings date back to antiquity, and once technological advances allowed for a popular press, it disseminated definitively worded advice on proper sleep patterns for children. Physicians and the lay public could not be blamed, however, for a healthy dose of skepticism. After all, the advice seemed to change among investigators, and it did not take much digging to see that it was based on thinly veiled personal observations and opinions. Over the past several decades, however, scientific data have largely replaced subjective observations and personal speculation and have confirmed that at least some of early observers’ impressions were correct. This article reviews the available data, and then outlines why that evidence is particularly concerning from a developmental perspective.

METHODOLOGICAL CONSIDERATIONS

This review focuses on data collected in children and adolescents. There is a large and well-developed literature on sleep deprivation in adults that can provide initial
guidance for pediatric research, but cannot be extrapolated to children without studying children, for several reasons. Whereas the large majority of adult experimental studies have examined the impact of 1 to 2 nights of complete sleep deprivation, it is reasonable to question how these findings generalize to children, whose overall sleep need is greater and who far more often experience chronic partial sleep restriction than total sleep deprivation. Moreover, circadian rhythms shift developmentally, and adolescence brings tremendous changes in sleep physiology, particularly within the lower slow-wave electroencephalographic (EEG) frequency ranges, that may alter the response to sleep restriction. Finally, the contexts in which children must function differ substantially from adults. Adult research findings on how experimental sleep deprivation affects truck drivers, medical residents, or other professionals are important, but these findings provide only a rough guess as to the effect of chronic sleep restriction on classroom behaviors or learning, the development of new driving skills, or behavioral and social functioning in developing children.

Studying children and adolescents poses methodological challenges. The primary research design for adult sleep deprivation studies, in which participants stay in lab to allow for greater control of sleep and activity schedules, is less of an option for child researchers because parents are often reluctant to leave their children in the care of unfamiliar adults, participants may be resistant to a prolonged stay away from home, and children’s sleep may be particularly subject to disruption in an unfamiliar environment. Further, children are a vulnerable population, for whom additional protections against risk may be required. Although it seems that any health effects of short-term sleep restriction are reversible with 1 to 2 nights of recovery sleep, researchers must consider the risk of events that could occur during sleep restriction (eg, a poor school grade, auto accident for young drivers) and attempt to mitigate that risk. As a result, there have been few experimental studies.

High-quality studies using multiple research designs are needed because each has strengths and weaknesses. Correlational, case-control, and quasi-experimental studies lend themselves well to assessing the real-world associations between sleep and daytime function, often in impressively large samples that promote subgroup analyses and generalization of findings. However, the potential for uncontrolled confounding factors (eg, parent work schedules, parenting styles, family structure, child daytime activities/habits, teen employment) limits causal inferences, and measurements of both sleep and outcome variables are often imprecise. In contrast, experimental studies allow for more confident attribution of causality and more measurement precision, but the conclusions can be limited by small samples and by methods and measures that do not map cleanly onto real-world circumstances.

Like research designs, different cognitive and behavioral assessment techniques have complementary strengths and weaknesses. Questionnaires have the advantages of easy administration, low cost, and ready assessment of real-world functioning, but are prone to reporter biases. Office-based standardized neuropsychological tests avoid such bias and can parse out specific cognitive skills. However, some domains of functioning, particularly attention and executive functioning (eg, planning, organization, mood, and behavior regulation), are difficult to assess in an office-based testing environment. Direct systematic rating of child behaviors by trained observers can provide an objective perspective in applied settings but is logistically very difficult; the 2 studies that have used such techniques in the sleep-behavior literature used simulated classrooms rather than embedding raters in subjects’ schools.

In the end, there is no single best way to study the effect of sleep on the daytime functioning of children and adolescents. The best conclusions tend to be drawn from an accumulation of studies with complementary strengths and weaknesses.
The largest research base that links sleep in children to daytime functioning comes from correlational studies in epidemiologic samples. As summarized in recent major reviews, children’s quantity and/or quality of sleep repeatedly has been shown to correlate with their levels of daytime sleepiness and performance at school. The strength of that association may vary by student age and sex; one recent meta-analysis of sleep and school functioning reported that studies of younger children, particularly those that enrolled more boys, tended to show the largest effects. To some degree, research in this area could be criticized for an overreliance on parent- or self-report of sleep and academic performance. However, such reports correlate well with objective measures, and importantly, the association between sleep and academic functioning has been reported even when both constructs were measured objectively.

Even so, findings have not been universal. Mayes and colleagues recently suggested that sleep is minimally associated with academic knowledge. This study relied heavily on office-based tests of academic knowledge, which are only partial predictors of classroom performance. Classroom performance also depends on skills that are difficult to test in the office, including sustained attention, behavior regulation, planning, and organization. Indeed, school-identified learning problems did significantly correlate with poor parent-reported sleep quality in the Mayes and colleagues’ study. However, this effect disappeared after statistically covarying for symptoms of attention-deficit/hyperactivity disorder (ADHD), which led to the speculation that any apparent link between sleep and learning problems could be because of the confounding effects of ADHD. Alternatively, however, symptoms of ADHD may not represent confounding factors but the mechanism by which poor sleep quality was linked to learning problems.

Indeed, inadequate sleep has been linked to difficulties with attention, impulse control, and behavior regulation, with potential consequences that extend beyond the classroom. Poor sleep quality is associated with crash risk in teen drivers, and short sleep has been linked to accidental injuries in young children and adolescents, as well as risk-taking behaviors in adolescents. Because poor regulation of attention and behavior are the key features of ADHD, it has been concluded that a subgroup of children with primary sleep problems may be misdiagnosed with ADHD. However, the relationship between ADHD and sleep is complex, and the reader is referred to the relevant article elsewhere in this issue and several other recent reviews for detailed coverage.

The links between sleep and other psychiatric diagnoses, including depression and anxiety disorders, are similarly complex and extend beyond the current discussion. Briefly, sleep problems are disproportionately present in many psychiatric conditions, and the direction of causation seems to be reciprocal rather than unidirectional. There is also evidence that the presence or severity of sleep disturbance predicts psychiatric symptom severity and functional impairment. However, it is difficult to know how to apply this information to the general population; although sleep disruption seems to be linked to mood, studies have yielded mixed associations between sleep duration and emotional functioning. Indeed, within one study, parent-reported mood problems and behavior problems had variable associations with sleep duration, depending on the source of information on behavior/mood (parent-vs self-report) and sleep duration (parent vs actigraphy).

Of greater interest are studies of daytime functioning of children and adolescents with obstructive sleep apnea (OSA), a largely treatable disorder in which the upper airway is chronically and/or repeatedly obstructed during sleep. As reviewed by
OSA has been linked to poor classroom grades, sleepiness, inattention, hyperactivity, oppositional behaviors, and mood dysregulation (but not ongoing mood disturbance) in the vast majority of relevant studies, most of which have targeted children aged 5 to 12 years. Beebe and colleagues recently extended those findings through the adolescent years. Office-based tests of intelligence have yielded inconsistent results in children with OSA, with the most consistent evidence of IQ deficits during the preschool and early grade-school years. Other tests of cognition have yielded mixed results, but there is some evidence of poor scores on tests of attention, executive functioning, and learning/memory in children with OSA. OSA is of particular interest in this article not only because a large number of studies have examined behavioral outcomes of children with this condition but also because treatments focus on the airway and would not be expected to affect behavior in a manner independent of sleep. Nonrandomized studies have shown improved daytime functioning following surgical intervention for uncomplicated OSA, bolstering the suggestion that OSA is causally related to daytime dysfunction. However, the results of the childhood adenotonsillectomy (CHAT) study are awaited, an ongoing large randomized adenotonsillectomy trial for OSA with blinded outcome measures.

There have been studies that have linked other treatable sleep conditions, most notably restless legs syndrome (RLS) and periodic limb movement disorder, to daytime dysfunction, particularly to hyperactivity/impulsivity and inattention. However, compared with OSA, studies have been few, causal implications are unclear, and intervention data are difficult to interpret because the relevant sleep treatments can also directly affect daytime functioning. For more information on RLS, see the article by Durmer and colleagues elsewhere in this issue.

Outside OSA, only a handful of correlational and case-controlled studies of pediatric sleep have used objective measures of cognitive functioning. In 2 studies, poor quality sleep was significantly associated with poor attention, working memory, and/or impulse control. A recent study linked objectively defined short sleep with lower IQ test scores. However, other studies have reported either (1) no relationship between sleep duration and IQ or (2) an association only for males and on selected aspects of intelligence. Two studies of large sample size on young children have arrived at conflicting results with respect to overall cognitive functioning and sleep.

In summary, correlational and case-control studies have yielded good evidence of the association between inadequate sleep and disturbances in children’s behavior and attention regulation, daytime sleepiness, academic performance, and, to the extent that it has been explored, executive functioning. However, the possibility of uncontrolled confounding factors limits the degree to which causal inferences can be drawn from these studies.

**QUASI-EXPERIMENTAL STUDIES**

As summarized in Table 1, there have been several quasi-experimental studies in which scientists have carefully observed that in middle- and high-school students, sleep duration was systematically influenced by school start times. In an impressive illustration of how public policy can affect health, starting school later in the morning is associated with students getting more sleep, regardless of whether comparing across schools (between groups), within a group of students over time, or within individuals over time. The link between more sleep and later school start time is primarily when students awaken; bedtimes are relatively unchanged. Not surprisingly, later start times are also associated with less subjective and physiologic
Finally, later start times seem to be associated with improved enrollment stability, better attendance among the least stable students, less tardiness, fewer teen driving accidents, and slightly fewer sick days and depressive symptoms. Limitations of many of these studies include an unknown risk for uncontrolled confounding factors (eg, school management, historical events, child development), reliance on self-report of daytime functioning, and samples consisting of superhealthy individuals who may not reflect the general population. Importantly, evidence of academic gains or improvements on standardized test scores is sparse, and none of these studies has yet demonstrated that students learn more with later school start times.

EXPERIMENTAL STUDIES

Since 1896, hundreds of publications have documented the impact of experimental sleep deprivation or restriction on adults' sleep-wake regulation, affect regulation, cognitive performance, real-world functioning (eg, driving), and neuronal activity. In contrast, as of late 2010, there had been only 7 analogous published studies of pediatric populations, all in print since 1980 (Table 2). These studies so far allow for 5 broad conclusions.

First, compared with when they are well-rested, sleep-deprived children fall asleep more easily during the day, self-report sleepiness, and look sleepier. Second, children are less attentive when sleep deprived. It has been difficult to demonstrate this conclusion on formal attention tests, but shortened sleep results in visibly more inattentive behaviors, whether reported by individuals who are not blind to sleep condition, teachers who were likely blind to sleep condition, or observers whose blind state was rigorously maintained. Third, no experimental study has yet shown that sleep restriction induces hyperactivity, impulsivity, or other externalizing behaviors in children, despite the correlational and case-control evidence that these changes should occur. Fourth, there is some evidence that depriving children of sleep affects their higher-level cognitive skills. One study documented diminished creativity and reasoning skills following a single night of shortened sleep, but overall study results were mixed. Another study reported that sleep-deprived adolescents showed diminished higher-level executive functioning skills according to parent- and self-report forms. Fifth, there is evidence that the impact of sleep deprivation is substantial enough to result in real-world impairment. Sleepiness and inattention in real-world settings have been reported by teachers, parents, and subjects. One study also found deficits in executive functioning as applied to daily life, and 2 studies reported learning difficulties in a simulated or real classroom.

These findings are important because they infer that inadequate sleep causes daytime deficits. However, to date, there simply have been too few studies to answer many important questions. In children, there is little to no knowledge (1) of how circadian rhythms interact with sleep restriction to affect functioning, (2) of the response to sleep restriction as it accumulates over time, and (3) on whether and why some individuals are more vulnerable to sleep restriction. Moreover, the existing studies have generally had limited statistical power because of small sample sizes and/or the use of between-subjects research designs. The samples also have been largely composed of superhealthy individuals, with limited ethnic and socioeconomic diversity. Young children have been overlooked entirely in published work, although this age range may show unique symptoms (eg, greater impulsivity). In addition, although it is clear that sleep restriction results in impairments that extend beyond simple
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<th>Analytic Design</th>
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<tr>
<td>Dexter et al</td>
<td>10th–11th</td>
<td>Between groups</td>
<td>2 nearby schools serving students with similar demographics, one starting at 7:50 AM and the other at 8:35 AM</td>
<td>Students at the school that started at 7:50 AM reported significantly less sleep and a trend toward more sleepiness. Sleepiness ratings at both schools approached the pathologically sleepy range</td>
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<td>Wolfson et al</td>
<td>7th–8th</td>
<td>Between groups</td>
<td>2 nearby schools serving students with similar demographics, one starting at 7:15 AM and the other at 8:37 AM</td>
<td>Students at the school that started at 7:15 AM reported significantly less sleep on school nights and greater sleepiness and had more school-documented tardiness. Reported class grades differed across schools for 8th graders, but not 7th graders</td>
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<td>Hansen et al</td>
<td>Incoming 9th grade honors students</td>
<td>Within group</td>
<td>Student sleep diaries the month before school started were compared with those completed at the first 2 wk of high school and again several months later</td>
<td>Weeknight sleep duration dropped dramatically from summer into the school year. It then lengthened a bit across the school year, but remained well less than summertime levels</td>
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<td>Carskadon</td>
<td>Students shifting from 9th to 10th grades</td>
<td>Within subjects</td>
<td>Students wore actigraphs in the spring of 9th grade at a school that started at 8:25 AM and again in the fall of 10th grade after transitioning to a school that started at 7:20 AM. Sleepiness was also assessed during each period</td>
<td>School night bedtimes did not change over time, but rise time became significantly earlier with the transition to the 7:20 AM start time, resulting in less sleep. Students’ physiologic sleepiness was higher during the second time point as well</td>
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<td>Study Source</td>
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<td>Wahlstrom$^{53,54}$</td>
<td>9th–12th</td>
<td>Mixed within group and between groups</td>
<td>7 schools in one district (district A) changed start time from 7:15–8:40 AM. Letter grades and enrollment data were compared before and after the schedule change. Also, after the school start time change, student-reported sleep and affect ratings were compared against analogous ratings in a nearby, demographically similar district that had a 7:30 AM start time (district B)</td>
<td>After start time change, students in district A changed schools less, and those who changed schools had higher attendance rates. There were no differences in overall student grades from before to after the start time change, but methodological issues complicated analyses. Focusing on the post-change period, compared with district B, students in district A had only slightly later bedtimes, but markedly later rise times, resulting in almost an hour more sleep per night. Also students in district A reported less daytime sleepiness, less tardiness due to oversleeping, and marginally fewer sick days and symptoms of depression</td>
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<td>Danner &amp; Phillips$^{55}$</td>
<td>9th–12th</td>
<td>Mixed within group and between groups</td>
<td>Students reported on sleep before and after start times were shifted from 7:30–8:30 AM in a countywide district. Motor vehicle crash data for 17–18-year-old adolescents in the county were contrasted with trends for other counties in the state</td>
<td>Compared with before the change in start times, after the start time had been changed, students’ school night sleep time increased significantly, weekend night sleep time decreased significantly, and motor vehicle crashes by teens in the county decreased. During the same period, teen crashes were stable or increased slightly elsewhere in the same state</td>
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### Table 2
Experimental studies of cognitive and behavioral effects of sleep restriction in children and adolescents

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<thead>
<tr>
<th>Authors</th>
<th>Sample</th>
<th>Analytic Design</th>
<th>Comparison</th>
<th>Findings</th>
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<tr>
<td>Carskadon et al⁵⁹</td>
<td>9 subjects aged 11–13 y</td>
<td>Within subject, no crossover</td>
<td>Objective sleepiness and cognitive testing after a baseline night (10 h in bed), 1 night of 4 h in bed, and a recovery night (10 h in bed)</td>
<td>Objective sleepiness increased after sleep restriction. No significant changes across nights were noted on the cognitive tests, which included measures of complex addition, word learning/memory, and sustained auditory attention</td>
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<tr>
<td>Carskadon et al⁶⁰</td>
<td>12 subjects aged 11–14 y</td>
<td>Within subject, no crossover</td>
<td>Objective and subjective sleepiness and cognitive testing after baseline night (10 h in bed), 1 night of no sleep at all, and 2 recovery nights (10 h each)</td>
<td>After sleep deprivation, objective and subjective sleepiness increased and performance on all cognitive tests diminished, reaching significance on complex addition and word learning/memory, and showing trends on sustained attention tests</td>
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<td>Randazzo et al⁶³</td>
<td>16 subjects aged 10–14 y</td>
<td>Between subject</td>
<td>Cognitive test scores after random assignment to 11 h in bed or 5 h in bed for a single night</td>
<td>Significant effects on 3 indexes of creativity and a measure of concept formation/reasoning. No such effects were evident on a second measure of concept formation/reasoning, a test of verbal learning, or 7 other indexes of creativity</td>
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<td>Sadeh et al⁶¹</td>
<td>77 subjects aged 9–12 y</td>
<td>Mixed within &amp; between subject</td>
<td>Cognitive tests after 2 nights of normal sleep and 3 nights of a randomly assigned sleep condition (normal sleep duration ±1 h)</td>
<td>There were cross-condition effects for sleepiness and session-by-group interactions for reaction time and attention span, favoring the lengthened sleep condition. No such interactions were evident on tests of finger tapping speed, sustained attention, impulse control, working memory, or learning/memory</td>
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<tr>
<td>Study</td>
<td>Subjects</td>
<td>Design</td>
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<td>Fallone et al (^{11})</td>
<td>82 subjects aged 8–15 y</td>
<td>Between subjects</td>
<td>Cognitive testing, sleepiness, and behaviors in a simulated academic setting after one in an optimized sleep condition (10 h in bed) or restricted sleep condition (4 h in bed)</td>
<td>Observers rated those with restricted sleep as less attentive but not more hyper/impulsive. In the academic setting, subjects who had restricted sleep were sleepy but not hyper/impulsive. Self-report and objective sleepiness were higher in the restricted sleep condition. There were few cross-group effects on attention tests.</td>
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<td>Fallone et al (^{62})</td>
<td>74 subjects aged 6–12 y</td>
<td>Within subject, with crossover</td>
<td>Teacher behavior ratings while subjects underwent 3-wk protocol: baseline week (self-selected sleep duration), followed in counterbalanced order by optimized sleep (10+ h/night) vs sleep restriction (6.5–8 h/night)</td>
<td>Cross-condition effects were seen on teacher ratings of academic problems, sleepiness, and inattention; the worst ratings occurred during the restricted sleep condition, although otherwise the pattern of scores varied. There were no significant cross-condition effects on teacher ratings of hyperactivity/impulsivity, internalizing/mood issues, or oppositional/aggressive behaviors.</td>
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<td>Beebe et al (^{7,12,64})</td>
<td>19 subjects aged 13–16 y</td>
<td>Within subject, with crossover</td>
<td>Parent and subject ratings of behavior, simulated classroom performance, and EEG and fMRI assessments during a 3-wk protocol: baseline (self-selected sleep duration), followed in counterbalanced order by extended (10 h/night) vs restricted sleep (6.5 h/night)</td>
<td>Parents rated their teenage children as sleepier and having more problems with attention, oppositionality, behavior regulation, and metacognition; similar effects were also self-reported. Effects on reported hyperactivity/impulsivity were minimal. In the simulated classroom, learning was poorer and there was behavioral and EEG evidence of sleepiness/low arousal during sleep restriction. fMRI suggested compensatory neural mechanisms.</td>
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Abbreviation: fMRI, functional magnetic resonance imaging.
sleepiness, some constructs remain underexplored (eg, problem solving, memory) and others are unexplored but theoretically at risk (eg, affect regulation). Finally, only 1 pilot study so far has examined how pediatric sleep restriction affects waking neural activity.12,64

**ADDING A DEVELOPMENTAL CONTEXT**

The findings presented so far are important, but few address developmental issues that are particularly salient during childhood, such as the parallel development of the brain and its cognitive and behavioral functions, as well as unique contexts involving children.

Developmental changes are evident in the brain throughout the life span, but the most dramatic neurodevelopment occurs during childhood, guided by an interaction between genetic programming and environmental factors.65,66 Chronic or extreme exposure to stress or toxins during development can lead to aberrant neural connections, resulting in disruption of cognitive, behavioral, or emotional functioning.66 Inadequate sleep may be one such exposure; animal models have demonstrated that even short-term sleep deprivation can alter neural plasticity.67–73 Further, a rodent model of OSA suggests a developmental gradient, with particularly marked effects on the brain and learning during the period equivalent to early childhood.74,75 These findings are notable in light of human data that suggest that OSA has the greatest effect on behavioral functioning in boys younger than 8 years76 and on intelligence in preschool children.40 Inadequate sleep state can be prolonged in humans, so even low levels of inadequacy might result in changed neurodevelopmental patterns and trajectories over time. Notably, the anterior brain regions that show the most protracted development across childhood are also those that are thought to experience the greatest functional impact of sleep deprivation.77–79

There have been few publications on the neural response to sleep deprivation in children, but noninvasive technologies hold promise for providing additional data. Four relevant studies have been published. Magnetic resonance spectroscopy of school-aged children with severe OSA found region-specific chemical abnormalities suggestive of neuronal injury.80 Also, in young children with subclinical sleep-disordered breathing, altered neuronal processing of speech sounds has been detected via evoked response potentials, leading to the speculation that the brain may be attempting to compensate for sleep disturbance.81 A similar explanation was evoked to explain altered activation-deactivation patterns in attention-related brain regions of adolescents during sleep restriction.64 These adolescents also showed EEG slowing in a simulated classroom while sleep deprived.12 All these findings are preliminary, and at present, it is impossible to draw coherent conclusions because of the differences in sample size, research designs, and measures. However, these studies support the suggestion that inadequate sleep can substantively alter neural processing.

Paralleling neurodevelopment, key functional skills develop across childhood. While academic skills are most easily appreciated as requiring learning, it is no exaggeration to state that every foundation skill necessary for adult functioning matures substantially during childhood and adolescence. To the degree that sleep deprivation affects a young child’s ability to engage with and learn from the environment—and the evidence reviewed earlier suggests that it does—maturation may be delayed or disrupted. Older children and adolescents may be vulnerable in other ways because their behaviors can have costly irreversible consequences.78 For example, there is a spike in accidental injuries during adolescence,78 and adolescent school underperformance
increases the odds of school dropout, failure to enroll in or complete college, adult mental illness or substance abuse, and low occupational attainment. If sleep quality or quantity affects injury risk or school success, as described previously, then the long-term legacy of pediatric sleep problems may be considerable.

In humans, it is neither feasible nor ethical to experimentally expose children to prolonged sleep restriction. However, longitudinal studies can examine the natural associations between inadequate sleep and later functioning. Over a dozen relevant studies have been published, and a well-replicated finding is that childhood sleep problems (variously defined) predict the development of anxiety and depressive symptoms over time, even after controlling for baseline mood difficulties and other potential confounds. This finding has been reported across time frames spanning preschool to midchildhood, preschool to midadolescence, midchildhood to late childhood, midchildhood to young adulthood, and adolescence to young adulthood. Longitudinal associations between sleep problems and externalizing behaviors, such as hyperactivity, aggression, or conduct, have tended to be weaker or less consistent, but also have been reported. Consistent with such associations, the presence of loud snoring, a hallmark symptom of OSA, in young children has been shown to predict later hyperactivity and poor school performance. Finally, sleep problems at the ages of 3 to 8 years have also been found to predict the early onset of substance use in adolescents.

However, such longitudinal relationships have not always been straightforward. One group has suggested that inadequate sleep has the greatest effects in children from homes of lower socioeconomic status. In several other studies, the initial presence of sleep problems was less important in predicting later functioning than whether these problems persisted or worsened over time. Such complexity has also been evident in studies of cognitive and learning outcomes. Especially among children from low-income homes, the presence of sleep problems in third grade predicts intellectual stagnation, whereas longer sleep predicts better reading development, over the following 2 years. Persistent or worsening sleep problems during childhood also predict poorer scores later on tests of executive functioning, but not on memory, nonverbal reasoning, vocabulary, or fine motor skills.

These longitudinal findings cannot prove causation but are consistent with a developmental model in which inadequate sleep is viewed as a noxious exposure that results, over time, in increased risk for adverse functional outcomes.

**SUMMARY**

Findings from studies that used complementary research methods have converged to strongly suggest that inadequate sleep quality and quantity are causally linked to sleepiness, inattention, and probably other cognitive and behavioral deficits that affect daytime functioning, with potential implications for long-term development. Important research questions remain, but the available data not only support the integration of sleep screening and interventions into routine clinical care (see related article elsewhere in this issue) but also support advocacy for public policy changes to improve the sleep of children and adolescents, with the goal of preventing long-term functional deficits.

As the pediatric sleep discipline moves forward, it is worth reflecting on other domains of public health that focus on prevention. When causes of adult conditions are earlier in development, treatments performed in adulthood are usually inefficient and not entirely efficacious; for example, skin cancer can be treated in adulthood but with mixed success and sometimes at great personal and societal cost. In
contrast, better understanding of the early effects of sun exposure during childhood has led to more effective primary prevention. Effective prevention has also emerged when childhood exposure to a suspected pathogen is studied to determine its deleterious mechanisms and lifetime effects. This was the case with inorganic lead exposure that, until studied in children, was tolerated at levels later shown to cause long-term adaptive deficits. In both of these examples, it is noteworthy that causal conclusions, and the effective preventive strategies that followed, were derived from a combination of translational, experimental, correlational, and longitudinal data. If similarly diverse data continue to bear out the developmental model described earlier, chronic sleep problems or sleep restriction might not be considered any more tolerable during childhood or adolescence than noxious serum lead levels or unregulated artificial tanning facilities.

REFERENCES


