

# The Impact of Inadequate Sleep on Children's Daytime Cognitive Function

Ronald E. Dahl

**This report describes the relationship between sleep and cognitive function in children. As reviewed, the empiric data to directly address the effects of sleep loss or disruption on children's cognitive function are quite sparse. However, a wide range of clinical and observational data support a general picture that inadequate sleep results in tiredness, difficulties with focussed attention, low threshold to express negative affect (irritability and easy frustration), and difficulty modulating impulses and emotions. In some cases these symptoms may resemble attention deficit hyperactivity disorder. These findings are discussed in relation to a model of sleep loss influencing prefrontal cortex including executive functions involved in the control of attention and emotions.**

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**T**HERE IS INCREASING evidence that children often reveal cognitive, emotional, and behavioral manifestations of inadequate sleep without showing frank sleepiness. This appears to represent a bit of a paradox. That is, many toddlers and early school-aged children respond to short-term sleep loss with what appears to be the opposite of sleepiness, that can include, irritability, crankiness, low-frustration tolerance, and short attention span. Numerous clinicians and clinical investigators have commented on how some sleep deprived children can manifest cognitive and behavioral changes that can mimic attention deficit hyperactivity disorder (ADHD).<sup>1-3</sup> Similarly, parents often describe their very young children "bouncing off the walls" when they miss a needed nap or are up too late. Objective measures of sleepiness (such as the Multiple Sleep Latency Test) have also revealed that before puberty, children often show less daytime sleepiness than adolescents or adults.<sup>4,5</sup>

As will be presented in this report, however, a broader conceptual framework of cognitive and behavioral changes following sleep loss may be convergent with an alteration at the highest level of cortical integration. Although there is a scant amount of data from controlled studies in young subjects, circumstantial and clinical data, as well as extrapolation from adult and animal

research, will be used to present a model focused on prefrontal cortex.

## SLEEP, SLEEPINESS, AND DEVELOPMENT

It may be best to begin with basic questions. What is sleep? Why is it important for cognitive and behavioral function? Why is it particularly important early in development? Although sleep appears to be ubiquitous across species and necessary for survival, the actual physiological function of sleep remains a complete mystery. Most common-sense conceptualizations of the function of sleep, as a period of simple rest or replenishment, fail to address the essence of sleep on a behavioral level that includes a categorical diminishment in awareness and responsiveness to the external environment. During the deepest nonrapid eye movement sleep (stages 3 and 4), there is nearly complete loss of awareness and virtual inability to respond rapidly to stimuli in the external environment.<sup>6,7</sup> Why does the brain require long intervals of relative unresponsiveness? This is even more puzzling when one considers the evolutionary perspective regarding adaptation pressures in the human ancestral environment. For example, early hominids living in an open savannah region were surrounded by large carnivorous nocturnal predators and virtually no safe sleeping sites.<sup>8</sup> Why then would the human brain have evolved a regulatory system that shuts down awareness and responsiveness for most of the night?

Whatever the function of sleep, it appears to be particularly important during early brain development. In fact, it can be argued that sleep is the primary activity of the brain during early maturation. By age 2 years, the average child has spent 10,000 hours asleep and about 7,500

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*From the Western Psychiatric Institute & Clinic, University of Pittsburgh, School of Medicine, Department of Psychiatry, Pittsburgh, PA.*

*Supported by MH41712-10, National Institute of Health, Rockville, MD.*

*Address reprint requests to Ronald E. Dahl, MD, 3811 O'Hara St, Pittsburgh, PA 15213-2593.*

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hours in all waking activities combined. Over the next 3 years (ages 2 to 5 years), there will be an approximate even balance between sleep and wakefulness. Thus, by early school age, a typical child has spent more time asleep than in all social interactions, environmental explorations, eating, playing, or any other waking activities.

One of the experimental approaches to investigate the basic function of sleep has been to try to take it away and examine the consequences. This approach has been used in basic animal research as well as clinical studies. It has been shown through an elegant series of very well-controlled experiments,<sup>9,10</sup> that rats deprived of sleep can survive approximately as long as they can without food. Unfortunately the precise nature of the mortality has not been divulged through these experiments. These studies have shown that both total sleep deprivation and paradoxical sleep deprivation (believed to be equivalent to REM sleep) are lethal in rats.

There has also been extensive clinical literature examining the effects of sleep deprivation in adults. These studies have included total sleep deprivation, partial sleep deprivation, and differential sleep stage deprivation (depriving the subject of REM or delta sleep specifically). Studies in adults have shown that the cognitive performance deficits following sleep deprivation are highly influenced by a variety of factors, including: the length of the test, the knowledge of the results, self-pacing versus experimenter pacing, difficulty or complexity of the task, proficiency in performing the task, and the degree of short-term memory required to perform the task.<sup>11</sup>

Although a full discussion of these issues is beyond the scope of this report, the highlights of that work have been summarized in a meta analytic review of the effects of sleep deprivation on cognitive and physical performance in adults by Pilcher and Huffcutt.<sup>12</sup> The results of that meta analysis indicated that: (1) cognitive performance was more effected by sleep deprivation than motor performance; (2) mood was much more influenced than either cognitive or motor performance; (3) in general, performance levels decreased in sleep deprived subjects as the length of the task increased; and (4) there were a great many individual differences

in sensitivity to sleep deprivation effects. In addition, that meta analysis emphasized the power of partial sleep deprivation to diminish performance.

Other comprehensive reviews of the cognitive effects of sleep deprivation in humans have emphasized the importance of lapses in vigilance and attention,<sup>11,12,13</sup> and the central influence of compensatory efforts on motivation.<sup>14</sup> That is, the relative ability to temporarily affect or buffer the deficits from sleep deprivation through increased effort or motivation. As will be presented in greater detail in the last section, Horne and Pettitt<sup>14</sup> has emphasized prefrontal cortex (PFC) influences and divergent thinking as particularly sensitive to sleep deprivation effects in adult humans.

#### INADEQUATE SLEEP AND SLEEPINESS IN CHILDREN

There are surprisingly few data addressing the specific effects of inadequate sleep and sleepiness on daytime function in children. However, there is a wide spectrum of clinical observations and descriptions of children with insufficient or disrupted sleep. Although there are clearly individual differences in response to inadequate sleep, the general pattern of results includes: difficulties with focussed attention, irritability, emotional lability, and low-threshold for frustration and distress. The similarity of these "sleepiness" symptoms to attention deficit disorder symptoms has received comment by numerous investigators and clinicians. One of the first to make the association between child sleepiness and attention deficit was Navalet et al, who found a high proportion of adults diagnosed with narcolepsy reporting attention deficit disorders as children.<sup>15</sup> Another group of investigators found that a high proportion of children with fragmented sleep secondary to obstructive sleep apnea syndrome exhibited attention deficit/hyperactivity symptoms that reversed upon treatment of the sleep fragmentation because of sleep disordered breathing.<sup>3</sup> Picchietti and Walters<sup>2</sup> have reported ADHD symptoms secondary to sleep disruption from periodic limb movements. Dahl et al reported a case where a behaviorally based sleep disorder was contributing significantly to objectively mea-

sured ADHD symptoms in an experimental setting.<sup>1</sup>

Viewed from a broader perspective, however, the children showing deficits in focused attention and executive modulation of drives, impulses, and emotions are not fundamentally different from adult sleep deprivation models. For example, the majority of adults throughout the world use some type of stimulant (usually coffee or tea) to help offset the effects of insufficient sleep. The resulting increase in focused attention and stabilization of mood appears to contribute significantly to the popularity of caffeinated beverages. Thus, it should not be surprising that children with ADHD type symptoms from insufficient sleep have also been reported to show clinical improvement in these domains of symptoms with stimulant medication.

Experimental, controlled studies of sleep deprivation in children, however, are quite limited. A study by Anders and Roffwarg attempted selective interruption and deprivation of sleep in the human newborn.<sup>16</sup> The investigators report "the results indicated selective sleep stage deprivation by manual awakenings was impossible to achieve during the newborn period. During the disturbance the interrupted sleep stage showed considerable 'tenacity'." They did note that nonrapid eye movement sleep disruption showed a greater tendency for preferential recovery than REM sleep. Behavioral changes were not noted in this study.

One study by Carskadon et al<sup>17</sup> looked at acutely restricting sleep to 4 hours on a single night in nine children aged 11 to 13 years. No significant effects were seen on abbreviated versions of a Wilkinson Addition Test and a Williams Word Memory Test or a listening vigilance task. In a second study, however, with slightly older children (12 subjects ages 11.7 to 14.6 years) 1 night of total sleep deprivation<sup>18</sup> was examined, that showed a marked tendency for impairment in all performance measures during the sleep deprivation. Statistical significance was reached for number of words recalled in the Williams Word Memory test and number of problems attempted in the Wilkinson Addition Test comparing sleep deprived with baseline measures. It is also important to understand that the subjects in these studies were

carefully selected to be free of any sleep problems, volunteered for these studies, obtained optimal sleep in the period prior to the sleep studies, and received a great deal of support, special attention, and were highly motivated to complete the studies. All of these factors may have contributed to diminishing the effects of sleep deprivation compared with the general population. Further, the degree of sleep deprivation was relatively modest, particularly given other studies that suggested that cumulative sleep restriction in older adolescents resulted in much larger effects.<sup>19</sup>

At puberty, there appears to be a shift in the expression of sleepiness following mild to moderate sleep loss.<sup>5</sup> That is, adolescents in Tanner stages IV and V showed much greater sleepiness on multiple sleep latency test measures and had a greater tendency to sleep until late, in the morning, even if they obtained as much sleep as they had at prepubertal stages.

If sleepiness results in short periods of sleep, it may produce retrograde amnesia, ie, a failure of memory consolidation. Specifically, it impairs the transfer of information from short-term storage into long-term storage (Fig 1).

Additional information regarding the relationship between inadequate sleep and daytime cognitive function comes from the clinical literature examining treatment of inadequate or fragmented sleep. These include improvement in cognitive function, school performance, and social interactions following treatment of obstructive sleep apnea syndrome.<sup>3</sup> Another very interesting and well-controlled study by Minde et al,<sup>19</sup> examined the treatment of young toddlers and preschool age children with significant sleep problems. In the Minde et al study, objectively scored videotape monitoring of child

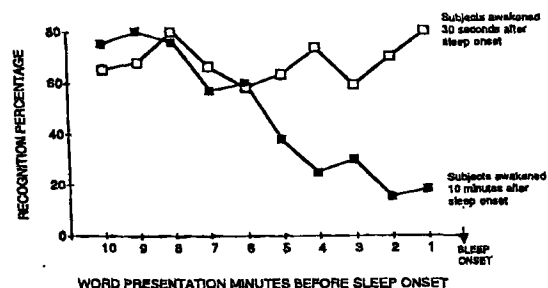


Fig 1. Memory is impaired by sleep onset as illustrated in this graph. (Reprinted with permission.<sup>32</sup>)

behavior and mother-child interactions showed that treating sleep disturbances resulted in the significant improvement in the behavior of the children, but no significant changes in the mother's behavior. The specific area of deficits that showed improvement following treatment in the sleep disturbed children were: attentional and social skills scores, infant dysregulation, irritability, and negative behavior. The results of these studies are consistent with a model that following the treatment of the sleep disturbances, the increased and improved sleep resulted in greater self control and modulatory behaviors by the children. The absence of significant changes in videotape quantification of maternal behaviors during daytime feeding interaction suggests the changes were secondary to improved self-control in the child following improved sleep.

In addition to clinical studies, however, a few clinical vignettes may help to illustrate changes following treatment of inadequate sleep.

#### CLINICAL VIGNETTES

##### *Case 1: Sleep Disturbances in a Young Child Presenting With Out-of-control Behavior and Emotional Distress*

AB, a 16-month old girl, was brought in by her mother for chronic difficulties with night waking. For months, AB would awaken one to four times each night and would often require hours of interaction and struggles with her parents to get back to sleep. She was also resistant to daytime naps (except when falling asleep during car rides). In other aspects of her health, growth, and development she was completely normal. Her waking daytime behavior, however, showed extreme irritability, fussiness, and very low-tolerance for frustration. She cried frequently, and showed extreme negative distress to any blocked goal, and was difficult to console when upset or crying. Her attention span was very short and she often changed activities rapidly. AB's sleep difficulties responded well to a behavioral program focussed on self-comforting and the total amount of sleep increased from a baseline of 8 hours per 24 hours to 13 hours of sleep per 24 hours in the month following treatment. The change in daytime behavior was described as dramatic by multiple family members. Most notable were the decreases in negative and distressed emo-

tions, and an improved attention span. She was more easily consoled when upset, and was a more pleasant, happy child with a dramatic increase in positive social interactions. Multiple family members described her as "a totally different child," using phrases like "taming the monster."

The main point in this case is to show that insufficient or inadequate sleep can contribute to increased irritability, easy frustration, and difficulties with focused attention.

##### *Case 2: Sleep Disturbance Presenting With Depressive Symptomatology*

CD is a 14-year-old boy brought in for evaluation of depressed mood, lethargy, fatigue, loss of interest in activities, and significant deterioration in school performance. CD also had complained for 1 year of extreme difficulty falling asleep at night and on further evaluation, was found to be obtaining about 6 hours of sleep per night. Based on the symptoms of difficulty with sleep, depressed mood, loss of interest in activities, and worsened school performance, CD was thought to have a major depressive disorder. However, when CD's insomnia was effectively treated and the total amount of night time sleep increased to 9 hours per night, he showed a significant improvement in depressive symptoms.

The clinical point shown in this case is that insufficient sleep in an adolescent can result in mood deterioration, decreased energy, loss of interest in activities, and can mimic or exacerbate affective disorder symptomatology. However, this is a complex clinical issue because children and adolescents with a major depressive disorder also complain frequently about sleep disturbances.<sup>21</sup>

##### *Case 3: Sleep Onset Insomnia Exacerbating Daytime Mood and Behavior Problems in an Abused Child*

GH is an 11-year-old girl who had a history of sexual abuse 3 years in the past. The abuse had occurred at night time in her bed and led to traumatic associations with going to bed and going to sleep, resulting in chronic insomnia. She also showed a wide-range of daytime behavioral difficulties including easy distractibility, poor focused attention, and emotional lability.

Following moderately successful treatment of sleep onset insomnia (with both behavioral and pharmacological interventions), daytime behavioral and emotional symptoms improved significantly.

The point of these cases and descriptions is to show the wide range of cognitive, behavioral, and mood symptoms that can be associated with inadequate sleep. In the last section, a model is presented attempting to integrate this disparate array of information. This model focusses on the role of pre-frontal cortex (PFC) in organizing and modulating a wide range of cognitive, emotional, attentional processes, and a possible link to many of these sleep deprivation effects.

#### A MODEL OF SLEEP, SLEEP DEPRIVATION, AND PREFRONTAL CORTIX FUNCTIONS

Recently, Horne<sup>22</sup> reviewed and integrated information indicating that total sleep deprivation causes a pattern of deficits parallel to PFC neuropsychological anomalies, that are reversed after recovery sleep. The strongest effect of sleep deprivation is in effect, weakening or diminution of goal-directed behaviors. Literally, being "tired" is a subjective sense of difficulty initiating and maintaining behaviors related to long-term goals or effortful tasks. These deficits can be reversed by increasing extrinsic rewards (eg, monetary), by fear, or acute threat, whereby subjects can temporarily recruit greater effort to offset the effects of sleep deprivation. Further, longer sleep deprivation appears to cause greater impairment in goal-directed behavior and thus requires more compensatory effort or greater extrinsic rewards to offset "tiredness." Clearly, goal-directed, motivated behaviors are heavily influenced by PFC/executive control, particularly when the goals are abstract or complex. Moreover, specific neuropsychological assessments,<sup>14,22,23</sup> have shown that frontal tasks that require complex, divergent, or creative thinking (prefrontal functions) show the most pronounced sleep deprivation deficits (and the least buffering by increased subjective effort). Horne has reviewed other data for PFC impairment with sleep deprivation, including evidence for increased distractibility and decreased emotionality of speech. There is additional evidence, which includes: contingent negative variation (CNV) of cortical expectancy waves can be abolished by total sleep deprivation<sup>24,25</sup> and

sleep deprivation causes a deterioration of imbedded figures test<sup>26</sup> that are believed to be PFC related. Horne also reviewed a study showing dramatic impairment of small group performance in a real-world test.<sup>26</sup> In this study, young military recruits working in small, highly motivated teams attempted strategic maneuvers during extended sleep deprivation. All teams were forced to withdraw before the end of the experiment (following 48 hours of sleep deprivation) because of an inability to perform tactically as a group. (In contrast, most studies of individual subjects find only modest deficits in cognitive performance following similar periods of sleep deprivation in laboratory situations.) Horne summarizes this evidence as showing that only certain types of complex tasks (involving PFC) show large deficits after sleep deprivation. In the model presented in this paper, this idea is extended to the hypothesis that tasks requiring the integration of cognitive challenges with social and emotional regulation (such as would be necessary to perform challenging maneuvers in small groups) are particularly prone to sleep deprivation impairment. That is, the highest level of integration (cognitive, social, and emotional processing) places the greatest modulatory demands on the PFC and is most sensitive to sleep deprivation effects.

A second common observation regarding human sleep deprivation, is variability or lability in emotional responses. These changes fit common experience regarding sleep deprivation: a general "disinhibition," such as periods of "silliness," loss of inhibitions, and erratic, impulsive behaviors. For example, sleep deprived adults in the study by Kollar et al<sup>27</sup> "periodically became hysterical and inane and did very childish things, ran wild, raced around the tables, and upset things." Bliss et al<sup>28</sup> reported episodes of giddy, childlike, uninhibited behaviors among sleep deprived adults. Sleep deprivation (or inadequate or disrupted night time sleep) in children has also been associated with decreased executive control, with daytime behaviors ("crankiness") reflecting impulsivity, distractibility, and emotional lability, in some cases resembling ADHD.<sup>1</sup>

One interpretation to summarize these findings, is that sleep deprivation (or inadequate or disrupted sleep) has particular effects on the cognitive modulation or integration of drives,

impulses, and emotions. Because the relative contribution of higher cognitive control undergoes significant changes with normal development across childhood and adolescence, these maturational changes in PFC/cognitive influences may account for some of the apparent age-related changes in the expression of sleep deprivation effects. This model may also be convergent with the well-established findings of transient paradoxical improvement in major depressive disorder following sleep deprivation. (For a further discussion of this model with a particular emphasis on affective regulation and the development of affective disorders, see Dahl et al.)<sup>29</sup>

### SUMMARY

Moving beyond these theoretical and conceptual approaches, however, there are a few general points relevant to inadequate sleep and cognitive function in children: (1) Despite wide-ranging clinical and observational data suggesting impaired control of attention and emotional lability, there are few controlled studies to address these important issues. (2) There is increasing evidence that many children with evidence of cognitive and behavioral changes from inadequate sleep may not appear frankly

sleepy from an initial clinical impression. (3) In addition to the relative lack of information concerning short-term effects of inadequate sleep, there is a complete absence of data to address the possibility of long-term effects of insufficient or inadequate sleep on cognitive development. (These issues may be more than theoretically interesting because there is increasing evidence that many children in our country are currently obtaining less sleep than in the past,<sup>30</sup> and less than in other cultures).<sup>31</sup> (4) Based on extrapolation from adult data and clinical observations, as well as circumstantial data, a model of decreased cognitive modulation of drives, impulses, and emotions (and less effortful control of attention) may be consistent with observations of sleep deprivation effects in children. The predictions based on this model include the likelihood that inadequate sleep could contribute to emotional and behavioral problems in some children. (5) There is clearly a need for additional well-controlled studies to address these important questions.

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