# Sleep, Cognition, and Behavioral Problems in School-Age Children: A Century of Research Meta-Analyzed

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Clear associations of sleep, cognitive performance, and behavioral problems have been demonstrated in meta-analyses of studies in adults. This meta-analysis is the first to systematically summarize all relevant studies reporting on sleep, cognition, and behavioral problems in healthy school-age children (5–12 years old) and incorporates 86 studies on 35,936 children. Sleep duration shows a significant positive relation with cognitive performance ($r = .08$, confidence interval [CI] [.06, .10]). Subsequent analyses on cognitive subdomains indicate specific associations of sleep duration with executive functioning ($r = .07$, CI [.02, .13]), with performance on tasks that address multiple cognitive domains ($r = .10$, CI = [.05, .16]), and with school performance ($r = .09$, CI [.06, .12]), but not with intelligence. Quite unlike typical findings in adults, sleep duration was not associated with sustained attention and memory. Methodological issues and brain developmental immaturities are proposed to underlie the marked differences. Shorter sleep duration is associated with more behavioral problems ($r = .09$, CI [.07, .11]). Subsequent analyses on subdomains of behavioral problems showed that the relation holds for both internalizing ($r = .09$, CI [.06, .12]) and externalizing behavioral problems ($r = .08$, CI [.06, .11]). Ancillary moderator analyses identified practices recommended to increase sensitivity of assessments and designs in future studies. In practical terms, the findings suggest that insufficient sleep in children is associated with deficits in higher-order and complex cognitive functions and an increase in behavioral problems. This is particularly relevant given society's tendency towards sleep curtailment.

*Keywords:* meta-analysis, sleep, cognition, behavioral problems, children

Over the past decades, sleep research has started to unravel the effects of sleep on daytime cognition and behavioral problems. Experimentally induced partial and total sleep deprivation in healthy adults can lead to a host of negative consequences within the affective, cognitive, and motor domains (Pilcher & Huffcutt, 1996), suggesting that an adequate amount of sleep is essential to maintain optimal daytime functioning. In a recent meta-analysis, Lim and Dinges (2010) aggregated past studies in adults addressing the consequences of total sleep deprivation on several cognitive domains and revealed considerable performance deficits in simple attention, moderate deficits in complex attention and working memory, and small detriments in short-term memory, whereas measures of mental processing speed and crystallized intelligence remained intact. At present, it is not clear whether sleep restriction similarly affects these domains in children, because a comprehensive meta-analysis is lacking. Both from the applied and fundamental scientific points of view, it is important and timely to fill this gap in knowledge.

From an applied point of view, studies spanning a full century indicate a disquieting reduction in the habitual sleep duration of children (Galland, Taylor, Elder, & Herbison, 2011; Iglowstein, Jenni, Molinari, & Largo, 2003; Terman & Hocking, 1913). It is important for parents, teachers, clinicians, and policy makers to know how this change of habits may be involved in cognitive and

behavioral problems in children. If, as is the case in adults, specific subdomains turn out to be more sensitive to sleep restriction, a focused applied approach would be to first evaluate the efficacy of sleep interventions in those children who both perform suboptimally in these domains as well as have relatively short sleep durations. Early detection and treatment may be important as it has been determined that detrimental effects of a period of curtailed or disturbed sleep in children could have more, and possibly irreversible, long-term consequences than is the case in adults (Beebe, 2011; Touchette et al., 2007), a contention that is supported by animal studies (e.g. Frank, Issa, & Stryker, 2001; Seugnet, Suzuki, Donlea, Gottschalk, & Shaw, 2011).

From a fundamental scientific point of view, studies in adults are now beginning to define likely candidates for the neurobiological mechanisms by which sleep restriction affects brain function, cognition and behavior. For example, as will be elaborated on in the next section of this introduction, likely mechanisms involved in the adverse effect of sleep deprivation on cognition are interference with synaptic scaling (Tononi & Cirelli, 2006) as well as with the reactivation (Wilson & McNaughton, 1994) and reorganization (Frankland & Bontempi, 2006; Takashima et al., 2006) of memory traces. Because brain structure, physiology, and function are different in children, defining differences and similarities in the effects of sleep restriction on cognition and behavioral problems along the developmental trajectory provides a unique opportunity to further elucidate these tentative neurobiological substrates of vulnerability to sleep restriction. This in turn will aid our understanding of the functions of sleep for the both the developing and mature brain.

The present meta-analysis, incorporating 86 studies on 35,936 children, aims to fill an important gap in our knowledge, by for the first time systematically summarizing all relevant studies reporting on sleep, cognition, and behavioral problems in healthy school-age children (5–12 years old). The current introduction first provides a concise background on the neurobiological underpinnings of the association of sleep with cognition and behavioral problems and then proceeds to introducing the development of sleep and finally its association with cognition and behavioral problems in children. The subsequent meta-analysis systematically addresses the following questions: First, is there an overall association of the duration and efficiency of sleep with cognition and behavioral problems? Second, do different cognitive and behavioral problem domains show differential sensitivity to sleep? Third, does this profile of differing sensitivities of associations in children match the profile previously described in scientific literature in adults? The fourth question addresses weaknesses in the literature on these associations in children as well as queries the characteristics of the assessment tools that most sensitively and robustly find associations of sleep with cognition and behavioral problems. Defining these is essential to optimize future research. The fifth question is how the findings in children fit in the proposed neurobiological mechanisms involved in the sensitivity of brain function to sleep. Addressing this association during a developmental stage that is characterized by marked quantitative and qualitative differences in brain structure and function provides an opportunity to critically evaluate the proposed mechanisms and substrates. Finally, in the present article, we will integrate the answers on these questions to provide a roadmap for the next wave of research on the importance of sleep for brain function, cognition, and behavioral problems in

children; what are the critical questions, and which assessment tools are best used to evaluate associations and intervention efficacy?

## The Role of Sleep in Cognition and Behavioral Problems: Hypotheses on Brain Mechanisms Involved

Why would nocturnal sleep be involved in daytime cognition and behavior? The present section briefly introduces four hypotheses, with different levels of elaborateness, on the supportive role of sleep in sustaining cognition, emotional processing, and behavior. All hypotheses propose an active role for sleep in the neuronal processing of information acquired during prior wakefulness, rather than merely providing rest or an absence of interference.

The *trace reactivation* or *replay* hypothesis (Born & Wilhelm, 2012; Hoffman & McNaughton, 2002; Sejnowski & Destexhe, 2000; Sutherland & McNaughton, 2000; Wilson & McNaughton, 1994) proposes that sleep aids memory consolidation through reactivation of traces of neuronal activity patterns that encoded information during the prior waking period. The reactivation is proposed to aid transfer of information: from temporary hippocampus-dependent storage to long-term hippocampus-independent neocortical storage. Whereas the hippocampus is initially essential to index (i.e., to provide "pointers" to) the coordinated patterns of activation of cortical modules, sleep is thought to promote the gradual strengthening of horizontal corticocortical connections that ultimately release memory traces from hippocampal involvement. The trace reactivation hypothesis has focused mainly on consolidation, enhancement, and reorganization of explicit memory processes that involve a dialogue between the hippocampus and the neocortex, but might apply as well to corticocortical connectivity of implicit memory traces, not involving hippocampal pointers.

The *synaptic homeostasis* hypothesis proposes that sleep is necessary to counterbalance the net increase in synaptic connectivity that occurs during wakefulness (Tononi & Cirelli, 2006). Waking information processing is associated with neuronal plasticity processes that adapt the synaptic strength of neuronal connections. Whereas synaptic strength can both increase and decrease, the former occurs more prominently during wake periods, leading to a net increases in synaptic strength by the end of the wake period. Sleep, and especially the slow cortical oscillation, a phasic electroencephalography (EEG) event typical of deep sleep, is proposed to be conductive to a general, homeostatic downscaling of synaptic strength. This is necessary to prevent saturation and preserve cost efficiency of the neuronal networks. The synaptic homeostasis hypothesis predicts that sleep deprivation will result in a synaptic overload of neocortical and limbic circuits, which could show in cognitive and emotional impairments.

Although not presently viewed as a formal hypothesis, converging evidence suggests a role of sleep in maintaining *functional integrity of the frontoparietal networks* that support sustained attention. Sustained attention refers to an essential requirement of most cognitive tasks: the capacity to remain attentive and respond to stimuli for a prolonged period of time. Neuroimaging studies in adults showed that sustained top–down attention requires activation of an extended, mostly frontoparietal, neuronal network that is compromised after sleep deprivation, most pronounced so in those adults whose performance suffers most (Chee & Tan, 2010; Drum-

mond et al., 2005; Weissman, Roberts, Visscher, & Woldorff, 2006). Attention can be maintained at a maximum level only for a limited duration. It typically waxes and wanes, and so does activity in brain networks regulating arousal and attention. Sleep deprivation aggravates this "state instability" of interleaved periods of normal versus attenuated performance and activation of attention networks (Chee et al., 2008; Doran, Van Dongen, & Dinges, 2001; Prado, Carp, & Weissman, 2011). Interestingly, the waning of attention network activity and performance can be preceded by waxing activation of the *default mode network* (DMN; Drummond et al., 2005; Eichele et al., 2008; Weissman et al., 2006) comprising frontal and posterior midline regions mostly but also parts of the hippocampus and medial temporal lobes (Biswal, Yetkin, Haughton, & Hyde, 1995; Raichle et al., 2001; Smith et al., 2009). Sleep deprivation affects the alternating long-distance neuronal interactions both between the attention networks and the DMN as well as within the DMN (Gujar, Yoo, Hu, & Walker, 2010). Imaging studies on the brain mechanisms underlying the detrimental effect of sleep deprivation on sustained attention in adults indicate a critical involvement of intrusions of DMN activity, as well as insufficient activation of the frontoparietal attention network that is necessary for sustained top–down control.

The recently emerging *overnight therapy* hypothesis (Walker & Van Der Helm, 2009) focuses more on the role of sleep in maintaining optimal emotional reactivity and emotional information processing, of which the derailment would show as internalizing and externalizing problems (Eisenberg et al., 2001). On the basis of a review of the current literature on adult studies, Walker and Van Der Helm (2009) proposed that sleep provides a window for a resetting of the neuronal systems involved in affect regulation and for the reprocessing of recent emotional experiences. These two processes result in maintenance of appropriate reactivity of limbic and associated autonomic networks. In support of this hypothesis, sleep-deprived adults show increased amygdala reactivity and an attenuation of control of the amygdala by the medial prefrontal cortex both involved in the regulation of emotion and the autonomic nervous system (Yoo, Hu, Gujar, Jolesz, & Walker, 2007). Walker and Van Der Helm (2009) proposed an important role for rapid eye movement (REM) sleep, a stage of sleep that occurs more prominently at the end of the sleep period and may thus be curtailed considerably in case of restricted sleep duration.

These four hypotheses are not necessarily mutually exclusive or exhaustive. They are, furthermore, subject to ongoing discussion, elaboration, and refinement (e.g., Diekelmann & Born, 2010; Stickgold, Whidbee, Schirmer, Patel, & Hobson, 2000). The hypotheses received support from data on the effects of sleep and sleep deprivation in adults, yet inconsistencies and complications remain. Researchers' understanding of the mechanisms and the validity of the hypotheses could profit from studies on the effects of sleep and sleep deprivation in children, for children are in a very different developmental stage with respect to brain structure, physiology, and function. Of relevance to the hypotheses mentioned previously, first, synaptic scaling is thought to be more abundant in children than in adults (Huttenlocher, 1979; Huttenlocher & de Courten, 1987; Paus, Keshavan, & Giedd, 2008), and it may not be limited to occurring predominantly during sleep, as is suggested to be the case in adults (Tononi & Cirelli, 2006). Second, the process of memory trace reactivation in adults involves connections between the hippocampus and neocortex, notably the medial prefron-

tal cortex, while these connections are still immature in children (Abrahám et al., 2010; Benes, Turtle, Khan, & Farol, 1994; Fair et al., 2008; Kelly et al., 2009; Power, Fair, Schlaggar, & Petersen, 2010). Third, the involvement of the frontoparietal network in the effects of sleep deprivation on sustained attention in adults may likewise be different in children, because this network is also still quite immature (for reviews, see Daniels, Frewen, McKinnon, & Lanius, 2011; Uddin, Supekar, & Menon, 2010). A critical question therefore is whether there are differences in the effects of sleep restriction on cognition along the developmental trajectory, and if so, how they impact hypotheses on the tentative neurobiological substrates of the involvement of sleep in cognition and behavioral problems? In order to properly address these questions, we will provide brief introductions on the childhood development of sleep and its association with cognition and behavioral problems.

## Childhood Development of Sleep

What are the characteristics of sleep, and how do they develop? The most frequently investigated sleep parameters are total sleep duration and sleep efficiency, where the latter refers to the percentage of the total amount of time spent in bed that a person is actually asleep. Infants spend approximately 60% of each day asleep, during which time they assume a characteristic position and lose responsiveness to environmental input. Sleep duration declines to 40% of the day in early adolescence and 33% in adults (Iglowstein et al., 2003; Ohayon, Carskadon, Guilleminault, & Vitiello, 2004). Sleep profoundly changes brain activity, and this process is thought to be essential for the maintenance of optimal cognitive and emotional functioning. Interindividual differences in sleep duration are influenced by variation in sleep need, sleep problems, and sleep restriction through exogenous factors (e.g., limit-setting problems, presence of a television or computer in the bedroom) or endogenous factors (intrinsic sleep disorders, genetic predisposition; Jenni, Molinari, Caflisch, & Largo, 2007; Pesonen et al., 2010). Boys on average sleep somewhat less than girls (Buckhalt, El-Sheikh, & Keller, 2007; Russo, Bruni, Lucidi, Ferri, & Violani, 2007). Sleep efficiency, defined as the percentage of time in bed actually spent asleep, is typically higher than 95% in school-age children and remains near 90% until adults reach the age of about 50 years (Montgomery-Downs, O'Brien, Gulliver, & Gozal, 2006; Ohayon et al., 2004).

In child studies, sleep parameters are most easily estimated using questionnaires or sleep diaries, or alternatively by using actigraphy, the long-term recording of wrist activity from which sleep and wake states can be estimated (Cole, Kripke, Gruen, Mullaney, & Gillin, 1992; Sadeh, Alster, Urbach, & Lavie, 1989, Sadeh, Lavie, Scher, Tirosh, & Epstein, 1991). The gold standard for the assessment of sleep, however, remains polysomnography (PSG), which reveals electrophysiologically distinct stages of brain activity during sleep from the electroencephalogram (EEG), electromyogram (EMG) and electrooculogram (EOG). These stages have historically been divided into one stage of rapid eye movement (REM) sleep and four stages (Stages 1–4) of nonrapid eye movement (NREM) of increasing sleep depth with the deeper sleep Stages 3 and 4 collectively referred to as *slow wave sleep* (Rechtschaffen & Kales, 1968). The recently introduced consensus sleep classification discriminates only three stages of NREM

sleep: lighter sleep Stages N1 and N2, and deeper sleep Stage N3 (Iber, Ancoli-Israel, Chesson, & Quan, 2007). REM sleep constitutes 50% of total sleep time during early infancy, declining to 5%–20% in older children and adults (Hoban, 2010). The duration of the ultradian cycles of alternating REM and NREM sleep states is substantially shorter in infants (50–60 min) than in adults (90–100 min).

The sleep EEG furthermore shows characteristic phasic events, notably the spindles and slow waves that reflect oscillations within the thalamocortical system. The sleep spindle, a waxing and waning of a prominent 11- to 15-Hz oscillation that lasts for 0.5–1.5 s, is seen mostly in Stage 2 sleep. Slow waves of 0.5–4.0 Hz occur either as isolated *K* complexes during Stage 2 sleep or in more continuous sequences during Stages 3 and 4. Both spindles and slow waves have been suggested to represent key phenomena involved in the role of sleep for maintenance of brain function and cognition. They may facilitate synaptic downscaling (Tononi & Cirelli, 2006) or reactivate memory traces (Wilson & McNaughton, 1994), as described by two of the previously mentioned hypotheses.

Both spindles and slow waves develop during the first 6 months of life (de Weerd & van den Bossche, 2003). Whereas sleep duration drops considerably during the period that children go to primary school (from 5–6 to 11–12 years old), the expression of slow waves increases and peaks in early adolescence at a level higher than seen in adults (Feinberg, 1982). After the age of about 11 or 12 years, slow wave activity initially declines steeply, followed by a slower ongoing decline with aging (Campbell & Feinberg, 2009).

## Sleep, Cognition, and Behavioral Problems in Childhood

Does sleep matter for cognitive performance and behavioral problems in childhood? Most research on the relation of sleep to cognition and behavioral problems has focused on adults; the topic has not yet been explored to the same extent in normal healthy school-age children, although it received considerable attention in children with diagnosed sleep disorders. Since the present meta-analysis focuses on the normal population of healthy children, it may suffice to summarize that both cognitive and behavioral problems have been shown in children suffering from sleep-disordered breathing (Ebert & Drake, 2004; Gozal, 1998; Kaemingk et al., 2003; Kheirandish & Gozal, 2006), insomnia (Velten-Schurian, Hautzinger, Poets, & Schlarb, 2010) and periodic limb movement disorder (Crabtree, Ivanenko, O'Brien, & Gozal, 2003).

As mentioned before, sleep duration in healthy children shows considerable interindividual variability. This variability is associated with variability in cognitive functioning and behavioral problems. Cross-sectional studies in community samples suggest relations of the quality or duration of sleep with cognitive measures, such as executive functioning (Sadeh, Gruber, & Raviv, 2002), intelligence (Busby & Pivik, 1983), and academic grades (Buckhalt, El-Sheikh, Keller, & Kelly, 2009; El-Sheikh, Buckhalt, Keller, Cummings, & Acebo, 2007). Short sleep duration has also been linked to behavioral problems (Aronen, Paavonen, Fjallberg, Soininen, & Torronen, 2000; Lavigne et al., 1999; Paavonen, Porkka-Heiskanen, & Lahikainen, 2009). Some studies, however, did not find any associations of sleep quantity or quality with

behavioral problems or cognitive outcomes such as academic achievements (Eliasson, Eliasson, King, Gould & Eliasson, 2002; Loessl et al., 2008; Mayes, Calhoun, Bixler, & Vgontzas, 2008; Terman & Hocking, 1913).

All studies mentioned were nonexperimental; that is, neither quantity nor quality of sleep was experimentally manipulated. Hence, the reported relations are not necessarily causal: a low sleep quantity or quality may either be a cause or consequence of cognitive or behavioral problems, or both outcomes could result from common underlying causes. Experimental sleep manipulation studies, necessary to determine directionality, have thus far been scarce in children. In a group of 10- to 14-year-olds, one night of restricting the allowed time in bed to 5 hr affected executive functions (verbal creativity and abstract thinking) but not sustained attention or explicit memory (Randazzo, Muehlbach, Schweitzer, & Walsh, 1998). In other studies restricting time in bed to 4 hr for a single night, investigators found no effects on cognitive tasks in a group of 8- to 15-year-olds (Carskadon, Harvey, & Dement, 1981a; Fallone, Acebo, Arnedt, Seifer, & Carskadon, 2001), whereas an increase in inattentive behavior but not hyperactive behavior was found in a group of 8- to 15-year-olds (Fallone et al., 2001). Seven consecutive nights of restricting time in bed to 6.5 hr increased academic problems in children ages 6–12 years (Fallone, Acebo, Seifer, & Carskadon, 2005). A single night of total sleep deprivation in 12- to 15-year-olds revealed that participants' post sleep-deprivation performance became highly variable, with significant decrements seen on a mathematical problem-solving task and a word memory task (Carskadon, Harvey, & Dement, 1981b). The first experimental study on accumulative effects of repeated modest sleep restriction versus sleep extension in 9- to 12-year-old children (Sadeh, Gruber, & Raviv, 2003) showed a positive association of sleep duration with performance on a digit forwards memory task, a continuous performance task, and a simple reaction time task.

The contribution of sleep specifically to memory consolidation can be examined by comparing memory retrieval following a period of sleep to memory retrieval following a similar duration of wakefulness. This study design, applied to children between the ages of 9 and 12 years, demonstrated a significant sleep-dependent consolidation advantage on a verbal memory task (Backhaus, Hoeckesfeld, Born, Hohagen, & Junghanns, 2008). In contrast, a study in a similar age group reported worse consolidation of a serial reaction time task following sleep than after wakefulness—opposite to the findings reported for the adult comparison group (Fischer, Wilhelm, & Born, 2007). Differential effects of sleep on the consolidation of explicit versus implicit memory have been noted and confirmed in a number of child studies (Diekelmann, Wilhelm, & Born, 2009; Prehn-Kristensen et al., 2009; Wilhelm, Diekelmann, & Born, 2008).

In conclusion, cognitive and behavioral impairments have been reported in children with sleep disorders and in children after experimental sleep restriction as well as those children described as "short sleepers." Relatively few studies seem to have addressed the importance of sleep quality (usually measured with sleep efficiency), rather than quantity (sleep duration), for cognitive and behavioral impairments in children. The results of the studies are moreover equivocal, and certain cognitive and behavioral problem subdomains seem differentially affected by sleep loss in children as compared to adults. As it stands, several narrative reviews have

appraised the childhood literature and investigated relations among sleep, sleep restriction, and cognition in children (for example, Beebe, 2011). These reviews concluded that sleep is important for learning and memory (Curcio, Ferrara, & De Gennaro, 2006; Hill, Hogan, & Karmiloff-Smith, 2007), in particular for memory encoding, working memory, and long-term memory consolidation (Kopasz et al., 2010). Thus far, however, only a single meta-analysis has been performed in which individual study outcomes were statistically aggregated (Dewald, Meijer, Oort, Kerkhof, & Bogels, 2010). This meta-analysis was limited to observational studies of school performance and excluded experimental studies and other measures of cognition or behavioral problems. The report concluded that better sleep was related to better school grades. School performance outcomes, however, provide only a limited, unspecified view on the spectrum of cognition and behavioral problems. As yet, no complete systematic meta-analysis on the association of sleep, cognition, and behavioral problems in children has been performed in which all outcomes across different domains of cognition and behavioral problems are objectively aggregated, and the effects of sleep on these outcomes have been objectively specified. More important, thus far, the entire range of cognition and behavioral problems measured in relation to sleep in children has not yet been examined within separate subdomains and meta-analyzed individually. It would be of great interest to determine how sleep relates to different cognitive domains such as sustained attention, executive functioning, and memory, and to investigate potential differences between internalizing and externalizing behavioral problems.

The current meta-analysis aims to take all relevant studies into account in order to determine whether a reduced sleep quantity or quality in healthy children relates to impaired cognition and increased behavioral problems. Given the past inconsistencies in findings, it appears timely to determine the status of and gaps in the current knowledge to pave the way for further research on the topic. The meta-analysis is a particularly well-suited tool for this purpose, as it allows for a systematic aggregation of all past results reported in literature, as well as for a comparison between different characteristics of studies in order to explain discrepancies. In particular, the present meta-analysis aims to evaluate the overall relation of sleep with daytime cognitive functioning and behavioral problems in school-age children. For this purpose, we separated all outcomes into two dimensions. The first dimension comprises cognitive outcomes—usually quantified as performance on a psychological task to estimate information processes such as sustained attention, executive functioning, and memory, but also including intelligence and school grades. The second dimension comprises outcomes referring to problematic behavior—usually obtained by parent or teacher questionnaire designed to estimate behavioral problems (e.g., negative mood, anxiety, hyperactivity, or aggressive behavior). Furthermore, the present meta-analysis aims to determine whether the subdomains of cognition and behavioral problems differ with respect to the strength of their association with sleep. We hypothesized that sleep duration and quality in children are positively associated with cognitive performance and negatively with behavioral problems, and we expected these relations to extend across the same subdomains as reported in adults. Finally, as past studies have been inconsistent in their findings, our goal in the present meta-analysis was to explore possible systematic reasons for the heterogeneity of results. By comparing and contrasting different study designs, we hoped to identify the most sensitive methodology in assessing the relation of sleep, cognition, and behavioral problems.

## Method

In order to warrant optimal methodology, we performed the meta-analysis according to the guidelines of the Meta-analysis Of Observational Studies in Epidemiology (MOOSE) group (Stroup et al., 2000). For a brief overview of the history of the meta-analysis as a research tool and an extensive background to the state-of-the-art procedures recommended for meta-analyses in the developmental field—as applied in the current meta-analysis—we refer to Van IJzendoorn, Bakermans-Kranenburg, & Alink, (2011).

### Literature Search

An extensive literature search was performed, incorporating four different search strategies (Cooper, Hedges, & Valentine, 2009; Mullen, 1989). The initial search was performed using electronic databases: Thomson Reuters (formerly ISI) Web of Knowledge, PsycINFO, and National Center for Biotechnology Information MEDLINE. The keywords adopted for this search were *sleep*, *sleep disorders–circadian rhythm*, *sleep deprivation*, *sleep disorders–sleep initiation and maintenance disorders*, or *sleep disorders–intrinsic*, *dyssomnias*, combined with *child* or *child–preschool,* and combined with *cognition, cognition disorders, neuropsychology, behavior, memory, behavior control, neuropsychological tests,* or *neurobehavioral manifestations.* We excluded *review*, *epilepsy*, *mental retardation*, *apnea*, *sleep apnea central*, *sleep apnea syndromes*, *sleep apnea obstructive*, *snoring*, and *tonsillectomy*. To ensure we captured all internalizing and externalizing behavioral problem variables, we conducted two separate searches on *sleep* combined with *child* and *internalizing*, and *sleep* combined with *child* and *externalizing*. The search was finished in December 2010. Additionally, online thesis databases were searched. Second, a comprehensive search of sleep-related conference proceedings was performed for the annual meeting of the Associated Professional Sleep Societies, the bi-yearly Congress of the European Sleep Research Society (2004 onwards), and the first Congress of the International Pediatric Sleep Association (2010). We did not include data reported in abstracts that were subsequently published in full papers. The third method entailed contacting experts in the field, known as the *invisible college*, which included a request at the fifth annual Conference on Pediatric Sleep Medicine in October 2009. Finally, all reference lists of relevant studies were checked for further suitable studies. In those instances where we found a relevant foreign-language article, we searched for a corresponding English-language article. All collected studies were investigated for relevance according to the following inclusion and exclusion criteria:

(a) The study must contain a quantitative measure of sleep. Both sleep duration (time in bed, total sleep period, or actual sleep time; definitions provided in the following) and sleep efficiency outcomes were accepted as independent variables. Sleep duration estimates were included if an actual estimate of sleep quantity (in hours and/or minutes) was provided; studies reporting mere qualitative judgments on whether sleep length was sufficient or not were excluded. Time in bed was defined as the amount of time

between getting into bed and getting out of bed. The total sleep period was defined as the amount of time between falling asleep and final awakening. Both time in bed and total sleep period could be either subjectively estimated or objective measured using actigraphy or polysomnography. Actual sleep time was defined as total sleep period minus the amount of time spent awake in this period, as estimated using actigraphy or polysomnography. Sleep efficiency was defined as the proportion of the total amount of time spent in bed that the participant was asleep and was included only if this quantitative estimate was obtained by actigraphy or polysomnography. Sleep efficiency and sleep duration are the widely used and standardized measures in pediatric sleep research. Other sleep parameters are less frequently reported and their operationalization varies considerably across different studies.

(b) The study must contain at least one measure of cognition or behavioral problems. All outcomes were classified into one of two dimensions: cognition (i.e., experimental task performance and school performance outcomes such as intelligence scores, memory task performance, school grades, or national curriculum assessments) and behavioral problems (i.e., observations of behavioral problems such as observations of hyperactivity–impulsivity, oppositional–aggressive behavior, attention problems, social problems, withdrawn behavior, negative mood, anxiety, or problem behavior at school).

(c) The study must statistically relate the outcomes on cognition or behavioral problems to the outcomes on sleep.

(d) The study's participants must be children. We defined childhood as the period between 5 and 12 years of age. Due to a lack of indisputable developmentally appropriate cutoff ages for childhood, we also included those studies with age ranges containing both our ages of interest and surrounding ages. The cutoff age of 5 years was chosen as this generally reflects the age that children start school, and thus they are exposed to equal daytime structure and often no longer nap during the day (Thorleifsdottir, Bjornsson, Benediktsdottir, Gislason, & Kristbjarnarson, 2002). The cutoff age of 12 years was chosen to reflect the onset of adolescence, a rough starting point of puberty. Additionally, this age was chosen as it is prior to the transfer to secondary education with its changing environmental demands and prior to the onset of the sleep phase delay that typically occurs in mid- to late puberty (Carskadon, Vieira, & Acebo, 1993).

(e) The study's group of interest was healthy children, that is, children not diagnosed with physical, neurological, developmental, psychiatric, or sleep disorders. Often studies failed to mention details of their screening procedure, in which case we chose to include the study. If a study included both a clinical group and a healthy control group that met our criteria, we only included the results obtained in the latter group if these were reported separately.

(f) The study must be reported in the English language.

(g) Case reports and review articles were excluded.

The literature search resulted in 86 studies, including 35,936 children: 32 studies reported only on cognition, 27 studies reported only on behavioral problems, and 27 studies reported on both cognition and behavioral problems. Figure 1 provides an overview of the entire literature search procedure and the number of suitable studies located through each of the four search strategies. All studies were retrieved through online databases or the libraries of the authors' respective institutes or were obtained from the authors directly.

## Data Coding

The resulting 86 studies were coded on characteristics of participants, methods, procedures, and results in a specifically developed coding spreadsheet. The coding procedure was undertaken with the aim of answering the following questions: First, are there individual characteristics such as age and gender that determine the strength of the association of sleep with cognition and behavioral problems? Second, are there characteristics of the assessment and procedural methodology that make studies systematically more or less sensitive to find an association of sleep with cognition and behavioral problems? Third, do different cognitive and behavioral problem domains show a different strength of the association with sleep?

Coding categories can be found in Table 1 and were used in subsequent analyses. In those instances where the study failed to clarify sufficiently the methodology used, we classified the study in the "unknown" category. This was often the case for conference abstracts, which contained less detailed information than the published articles. Upon completion of coding, the range of measurement tools of cognition and behavioral problems was examined, to
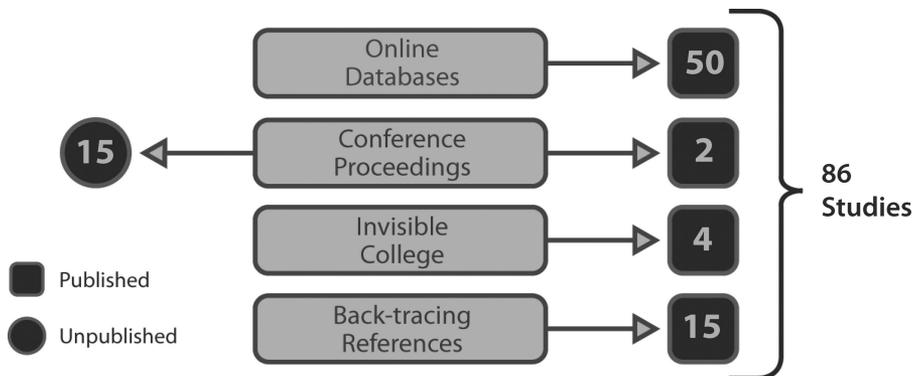


*Figure 1.* Schematic overview of literature search method. Four different search methods were used, locating 15 unpublished and 71 published studies that met all selection criteria; 86 studies were included in the meta-analysis.

Table 1
*Coding System for Studies on Sleep, Cognition and Behavioral Problems*

| Variable | Coding system |
| --- | --- |
| Study characteristics | |
|   Daytime functioning | |
|     Dimension | a = Cognition |
| | b = Behavioral problems |
|     Daytime functioning assessment tool | The tool (task/questionnaire) used to measure cognition and/or behavioral problems[a] |
|   Nighttime sleep | |
|     Outcome | a = Sleep duration |
| | b = Sleep efficiency |
|      If sleep duration: precision of estimate? | a = Time in bed |
| | b = Total sleep period |
| | c = Actual sleep time |
| | d = Unknown estimate[b] |
|      If sleep duration: type of sleep restriction? | a = Cross-sectional variation in sleep duration |
| | b = Wake-sleep comparison |
| | c = Imposed sleep restriction |
|       No. of nights of imposed sleep restriction | a = 1 night (experimental sleep restriction) |
| | b = 2 or more nights (experimental sleep restriction) |
| | c = Retrospective (nonexperimental imposed sleep restriction) |
| | d = Unknown number of nights[b] |
|     Sleep assessment tool | a = Subjective report |
| | b = Actigraphy |
| | c = Polysomnography |
| | d = Unknown tool[b] |
|     No. of nights included for the sleep estimate | No. of nights of sleep measured |
| Participant characteristics | |
|   Sample size | Total sample size for which results are reported |
|   Age of children | Average age of sample for which results are reported |
|   Gender distribution | a = Male (100% boys) |
| | b = Mixed (30–70% of each gender) |
| | c = Female (100% girls) |
| | d = Unknown distribution[b] |
| Publication bias characteristics | |
|   Publication type | a = Journal article |
| | b = Other publication (conference proceeding, thesis) |

[a] Upon completion of coding, we examined the range of daytime functioning assessment tools and determined the different subdomains of cognition and behavioral problems these tools measured. Subsequently, we split all daytime functioning variables into seven cognitive subdomains (sustained attention, executive functioning, multiple-domain cognitive functioning, explicit memory, implicit memory, intelligence, and school grades) and three behavioral problems subdomains (internalizing behavioral problems, externalizing behavioral problems, and mixed behavioral problems).   [b] In those instances where the study failed to clarify the methodology used, we classified the study in the "unknown" category.

determine which subdomains of cognition and behavioral problems these tools measured. Subsequently, all outcome measures in the cognitive dimension were categorized into one of seven subdomains. It should be realized that any categorization scheme has its pros and cons, and fuzzy boundaries are inevitable in any effort to map the performance at specific tasks on underlying psychological concepts (that are subject to changes themselves), let alone on neurobiological substrates. We therefore chose to adhere as close as possible to the categorizations most often applied in the handbooks on neuropsychology, child neuropsychology, and cognitive neuroscience. Gold standards are, respectively, Lezak's (1976) *Neuropsychological Assessment*, Baron's (2004) *Neuropsychological Evaluation of the Child*, and Gazzaniga, Ivry, and Mangun's (1998) *Cognitive Neuroscience: The Biology of the Mind*. Their categorization was followed insofar as possible with only marginal adaptations. First, we had to skip the motor–

perception category because it was not addressed in an appropriate number of studies on sleep in children. Second, we added school performance because it represents an integrated performance measure of great practical value used in numerous studies. Third, we split the memory category into an implicit and an explicit one, following the extensive literature on sleep and learning in adults that indicate categorical differences between these two, both with respect to sleep and sleep stage sensitivity as well as with respect to the neurobiological mechanisms involved. Finally, we had to define a "miscellaneous" category for those tasks that seemed to involve multiple categories without one standing out clearly.

**Cognitive subdomains.** The seven cognitive subdomains were as follows:

*Sustained attention.* Sustained attention refers to the capacity to remain attentive and respond to stimuli for a prolonged period of time. Typical performance outcome measures are reaction times

and lapses (nonresponses). This category comprised all sustained attention tasks, including measures of simple responses that are to be sustained over substantial lengths of time. Tasks included the listening attention task, the serial alternation task, the oddball task, the children's test battery for the assessment of attention, the Gordon Diagnostic System vigilance task, the test of everyday attention of children, the test of variables of attention, the attentional networks test, the Bourdon–Vos test, the psychomotor vigilance task, the steer clear task, the continuous performance test, and a simple reaction time task.

*Executive functioning.*    The cognitive domain of executive functioning comprises inhibitory control, working memory, and cognitive flexibility (Miyake et al., 2000). In more general terms, the domain refers to the functions necessary for an individual to adapt to changing situations that require creativity, flexibility, self-control, and discipline (Diamond & Lee, 2011). This category included measures of response inhibition (e.g., the Gordon Diagnostic System's inhibition task or the go-no-go task); working memory (e.g., the digit span forwards and/or backwards, the n-back task, or the Sternberg memory scanning task); planning (the Developmental NEuroPSYchological Assessment's tower task, the Beery–Buktenica Developmental Test of Visual-Motor Integration); set-shifting (the Wisconsin Card Sorting Test or the Children's Category Test); creative thinking (the Torrance Tests of Creative Thinking); and composite working memory scores (e.g., the Working Memory Index in the Wechsler Intelligence Scale for Children [WISC]).

*Multiple-domain cognitive functioning.*    This category included tasks that clearly require integration of multiple cognitive domains although not traditionally labeled as tasks to measure executive functioning. Examples are the Wilkinson Addition Test, the Woodcock–Johnson Test, the Differential Ability Scales' general cognitive ability outcome, Raven's Standard Progressive Matrices, the Porteus Maze Test, the Wide Range Achievement Test, the Peabody Picture Vocabulary Test, and subtests of incomplete intelligence assessment, such as the WISC's block design, or a simple question on a child's intelligence level.

*Explicit memory.*    The first of two commonly discriminated types of memory that involve different neuronal networks is explicit memory, the knowledge of facts and events (Squire & Zola, 1996). Explicit memory has been linked to the hippocampus and the surrounding medial temporal lobe cortex (Squire, Stark, & Clark, 2004). This category comprised tasks that require participants to learn and retrieve explicit information. The category included a word-pair association task, the Williams Scale for the Measurement of Memory (Williams Word Memory Task), verbal learning tasks such as the California Verbal Learning Test, and the Neurobehavioral Evaluation Systems' Serial Digit Learning task, the Narrative Memory subtest of the Developmental NEuroPSYchological Assessment for Children, the emotional memory task, and a two-dimensional object location task.

*Implicit memory.*    The second of the two types of memory is implicit memory, the unconscious memory of procedures and skills, which has been linked to the basal ganglia and cerebellum (Doyon et al., 2009). This category comprised tasks that require participants to learn and reproduce implicit skills or procedures. Examples are a finger thumb opposition task, a sequence finger-tapping task, the mirror tracing task, and the serial reaction time task.

*Intelligence.*    Intelligence is meant to assess general cognitive capabilities. This category included only total intelligence quotient (IQ) scores, not subtest scores, of intelligence tests such as the WISC, the Wechsler Abbreviated Scale of Intelligence, and the Wechsler Preschool and Primary Scale of Intelligence. In those articles reporting both total IQ scores and subtest scores, only the total IQ score was included in the meta-analysis as a measure of intelligence. In those articles reporting only subtest scores, these subtest outcomes would be assigned to the relevant cognitive subdomain category and thus would not be included in this total intelligence category.

*School performance.*    This category consisted of integrative measures such school grades or national curriculum assessments.

**Behavioral problem domain.**    Outcome measures in the behavioral problem dimension were categorized into one of three subdomains:

*Internalizing behavioral problems.*    These concern actions or behaviors where negative emotions or feelings are directed inwards, including social withdrawal, depression, anxiety, and feelings of worthlessness. The word *internalizing* refers to the observation that a child usually sees himself or herself as the reason of problems or failures. This category consisted of actions or behaviors where children direct emotions or feelings inward (e.g., withdrawn behavior, unhappiness, fearfulness, or anxiety).

*Externalizing behavioral problems.*    These concern actions or behaviors where negative emotions or feelings are directed outward into disruptive actions or behavior, including rule breaking and irritable behavior. The word *externalizing* refers to the observation that a child usually "acts out" problems, which leads to destructive, aggressive, argumentative, or antisocial actions. This category consisted of action or behaviors where children direct emotions or feelings outward into disruptive actions or behavior (e.g., hyperactive behavior, aggression, or delinquency).

*Mixed behavioral problems.*    This category contained total behavioral problem outcomes that could not be separated into internalizing and externalizing behavioral problem subscores.

Two coders (the first and second authors) coded all articles blind to each other's categorizing. To assess intercoder reliability, we calculated agreement rates for all coding categories; Cohen's kappa was computed only for the categorical variables. Omissions or partly correct observations were calculated as errors, resulting in a conservative estimate of agreement. The overall agreement rate was 92%. All disagreements in coding were reviewed by both coders and resolved.

## Data Analyses

Meta-analyses were performed using Comprehensive Meta-Analysis (Biostat, Englewood, NJ) Version 2.0 (Borenstein, Hedges, Higgins, & Rothstein, 2005). The statistical results extracted from all studies were converted into Pearson's *r* correlations and subsequently transformed to Fisher's *z*. The latter effect size statistic shows a better distribution, especially concerning the standard error (Lipsey & Wilson, 2001; Mullen, 1989). Effects in the hypothesized direction (i.e., a positive relation between sleep and cognition and a negative relation between sleep and behavioral problems) were given a positive sign (e.g., longer sleep duration relates to better cognitive performance, or shorter sleep relates to more behavioral problems). Effects indicating a relation opposite

to that hypothesized were given a negative sign. Those studies reporting a mere nonsignificance of results, but no actual statistics, were assigned a conservative nonsignificant effect size (calculated using the study's sample size and $p = .50$). For those studies reporting only a subset of (significant) outcomes, this conservative nonsignificant effect size was assigned to the remaining—nonsignificant—variables.

Meta-analytic data analysis requires an independence of effect sizes; therefore, when studies reported multiple effect sizes, these were combined to independent effect sizes by intrastudy meta-analyses (Cooper et al., 2009; Mullen, 1989). This resulted in a final 29 studies reporting only on cognition, 23 studies reported only on behavioral problems, and 24 studies reported on both cognition and behavioral problems. To control for the dependency of outcomes, we performed a hierarchical procedure of successive within-study meta-analyses. For example, when outcomes were reported for multiple (associated) sleep duration estimates (i.e., time in bed, total sleep period, and actual sleep time), these were meta-analyzed to produce an independent effect size prior to the overall analyses. In subsequent moderator analyses, we compared the effect sizes obtained using different sleep duration estimates.

When longitudinal data were reported, where possible, only the first measurement was included. When both total sleep (24-hour based, including daytime naps) and nighttime sleep (merely at night) were reported, only the latter was included because data on daytime napping were missing in most studies, likely due to the rapid decline in napping after age 4. Thus by opting for the nighttime sleep outcome only, we facilitated a standardization of sleep duration measures. When studies reported both pure and partial (including covariates) associations, only the former was included. When studies reported both associations of sleep, cognition, and/or behavioral problems, as well as (post hoc) group comparisons, we included only the former; grouping criteria were substantially inconsistent between different studies and therefore deemed too arbitrary. Where possible, questionnaire total scores were included in the overall analyses rather than subscale scores. When results were reported for different age groups, we included only those age groups that matched our criteria; independent relevant age groups were included as separate effect sizes. When multiple articles reported on the same participant group, outcomes were meta-analyzed to independent effect sizes that were suitable for the overall analyses. When multiple articles reported outcomes on the same variables, these results would be included only once; if the results were identical, we would pick the first publication's results; if the results were only slightly different (e.g., due to a slightly different sample size), we would chose the larger participant group. When different variables with slightly different characteristics (e.g. sample size) were combined by within-study meta-analysis, the resultant effect size was given the most conservative estimate (e.g., the smaller sample size).

The Fisher $z$-transformed effect sizes were examined for outliers as defined as standardized $z$ values exceeding $\pm 3.26$ (Tabachnick & Fidell, 2001), a procedure recommended by Mullen (1989). No outliers were detected; thus, no additional effect size adjustments were necessary for further calculations. Additionally, all sample sizes were inspected for outliers. Two studies, reporting on the same participant group, showed an extremely large sample size exceeding the outlier cutoff $z$ value (Yen, King, & Tang, 2010; Yen, Ko, Yen, & Cheng, 2008). These outlying values were

subsequently Winsorized, and the Winsorized values were used in further data analysis to prevent the results being unduly determined by these outliers (Hampel, Ronchetti, Rousseeuw, & Stahel, 1986).

Four overall meta-analyses were performed to compute four main effect sizes, each assessing the overall relation between sleep and cognition and/or behavioral problems. First, all effect sizes were categorized according to the dimension they related to: cognition or behavioral problems. Next, within each dimension, a categorization was made between the effect sizes relating to sleep duration and those relating to sleep efficiency. The resulting four categories of data were meta-analyzed separately: cognition and sleep duration, cognition and sleep efficiency, behavioral problems and sleep duration, and behavioral problems and sleep efficiency. Funnel plots were created to determine whether the four main data sets were affected by publication bias. A funnel plot is a visual representation of all studies' effect sizes against their standard errors (often plotted as 1/standard error, or precision). In an ideal situation, containing no publication bias, the plot would be funnel shaped, with smaller samples showing larger variation in effect size and larger samples showing less variation in effect size due to the smaller influence of random variation (Duval & Tweedie, 2000a; Sutton, Duval, Tweedie, Abrams, & Jones, 2000). For each of the four main meta-analyses, we used the "trim and fill" method to test the effects of adding possible missing studies to balance the funnel (Duval & Tweedie, 2000a, 2000b). We reported the four overall effect size values both unadjusted, and adjusted for publication bias.

Next, we performed an in-depth analysis to examine which aspects of cognitive functioning and behavioral problems in particular related to sleep. The averaged effect sizes obtained for the different cognitive subdomains were compared to one another to determine whether all subdomains of cognitive functioning related to sleep equally. Two meta-analyses were performed per cognitive subdomain, one for sleep duration and one for sleep efficiency. Similarly, two meta-analyses were performed on the averaged effect size per behavioral problem subdomain, one for sleep duration and one for sleep efficiency, and subsequently the subdomains were compared with one another.

Finally, we performed moderator analyses in order to investigate whether specific characteristics of a study determined its sensitivity to detect an association of sleep with cognition or behavioral problems. The first five moderator analyses related to study characteristics:

- First, the studies were assigned to one of three different categories according to the *type of sleep restriction* experienced by the participants: (a) cross-sectional variation studies (i.e., no imposed sleep restriction; in these studies, participants slept their regular hours at night), (b) wake–sleep comparison studies (i.e., within-subject contrasts in which participants' performance was measured following a period of nighttime sleep as compared with following a period of daytime wakefulness; the latter could be viewed as normal daytime sleep restriction), and (c) imposed sleep restriction studies (i.e., nighttime sleep curtailment induced experimentally or through a real-life sleep-restricting factor, as for instance, an early school start time).
- Subsequently, the imposed sleep restriction studies were assigned to subcategories according to the *length of imposed sleep restriction*: a single night of experimental sleep restriction, multi-

ple (2 or more) nights of experimental sleep restriction, retrospective quasi-experimental sleep restriction through a known real-life sleep-restricting factor, or an unknown length of sleep restriction. The retrospective category included for example comparisons of children with different school start times, where earlier start times were associated with shorter sleep durations.

• For the third moderator analysis, studies were assigned to one of four categories according to the *precision of the sleep duration estimate* (i.e., "time in bed," "total sleep period," "actual sleep time," or an "unknown" sleep duration estimate).

• For the fourth moderator analysis, studies were assigned to one of four categories according to the *sleep assessment tool* they incorporated: subjective sleep estimates reported in a questionnaire or a sleep diary, sleep estimates derived from activity patterns recorded by a wrist-worn actigraph, quantitative polysomnographic sleep assessment (i.e., the recording of multiple electrophysiological signals throughout the sleep period), or an unknown sleep assessment tool.

• For the fifth moderator analysis, studies were compared on the continuous variable *number of nights included for the sleep estimate*. Studies with an undefined number of nights because of measuring a retrospective sleep judgment were scored as concerning a conservative 1-month period. Studies lacking any information on the number of nights included for the sleep estimate were assigned a conservative estimate fitting with the study's sleep assessment tool.

Two moderator analyses related to participant characteristics:

• The sixth moderator analysis investigated *gender* effects by assigning each study to one of four gender distribution categories: male (100% boys), mixed (30%–70% of each gender), female (100% girls), and unknown distribution; there were no studies with 1%–29% of either gender.

• For the seventh moderator analysis, studies were compared on the continuous variable *mean age*. Studies lacking mean age information were assigned the midpoint of the reported age-range as a predictor in the meta-regression.

Finally, one moderator analysis related to publication bias: The eighth moderator analysis investigated whether *publication type* was of any influence, by comparing not-yet published studies (e.g., conference abstracts and dissertations) with those studies reported in published peer-reviewed articles.

All analyses and significance tests were performed using random effects models. Whereas fixed effects models assume that the observed effect size reflects the population effect plus random error due to subject-level sampling error (Cooper et al., 2009; Lipsey & Wilson, 2001; Rosenthal, 1995), random effects models additionally include between-study variance due to differences in operationalization of variables (Hedges & Olkin, 1985; Lipsey & Wilson, 2001; Rosenthal, 1995). We used the random effects model throughout this article in order to be conservative, as recommended by Borenstein et al. (2005). Homogeneity of variance of effect sizes is generally tested using the $Q$ statistic (Borenstein et al., 2005). A significant $Q$ value indicates heterogeneity of a specific set of studies. We included the $Q$ statistic in the results tables to show the homogeneity of each category. In our investigation of categorical moderators, we compared the different categories using the between-study $Q$ statistic. When moderator categories' effect sizes were not independently calculated (i.e., different categories contained different outcomes obtained from the same participants), the 85% confidence intervals surrounding the calculated average effect sizes were compared as an exploratory test of whether effect sizes significantly differed from one another. An absence of overlap between 85% confidence intervals was considered a statistically significant difference under a random effects model (Goldstein & Healy, 1995; Van IJzendoorn, Juffer, & Poelhuis, 2005). To allow for reliable estimates of meta-analyzed effect sizes, we required a minimum of four studies per moderator category (Bakermans-Kranenburg, Van IJzendoorn, & Juffer, 2003). Meta-analytic outcomes of categories with fewer than four studies are deemed uninformative. When comparing different moderator categories by between-study $Q$ statistics, we included only those categories that were large enough (containing four or more studies). In regards to the two continuous moderators, we performed meta-regression using the methods of moments computation. The resultant slope reflects the change in effect size that occurs with each 1-point increase on the continuous moderator's scale.

## Results

### Overall Effects

**Sleep and cognition.**

*Sleep duration.* The combined effect size for the relation between cognition and sleep duration revealed a significant positive association ($r = .08$, 85% confidence interval [85% CI] [.06, .10]) in a heterogeneous set (as indicated by the significant $Q$ value) of 52 studies: total number of participants ($N$) = 24,454, $Q(51) = 122.58$, $p < .01$ (see Figure 2 and Table 2). Adjusted for publication bias, this relation remained significant ($r = .06$, 85% CI [.03, .09]. The in-depth analysis of cognitive subdomains revealed significant associations of sleep duration with executive functioning (number of studies [$k$] = 14, $r = .07$, 85% CI [.02, .13]), multiple-domain cognitive functioning ($k = 11$, $r = .10$, 85% CI [.05, .16]), and school performance ($k = 20$, $r = .09$, 85% CI [.06, .12]). Sleep duration was not significantly related to sustained attention, explicit memory, implicit memory, or intelligence. The 85% CIs overlapped; thus, no significant difference between cognitive subdomains' relation with sleep could be concluded.

*Sleep efficiency.* The combined effect size for the relation between cognition and sleep efficiency did not reveal a significant association ($r = .12$, 85% CI [.03, .21]) in a heterogeneous set (as indicated by the significant $Q$ value) of 10 studies: $N = 1,207$, $Q(9) = 31.06$, $p < .01$ (see Figure 3 and Table 2). Adjusted for publication bias, this relation remained nonsignificant ($r = .12$, 85% CI [−.002, .24]). The in-depth analysis of cognitive subdomains revealed that sustained attention and executive functioning did not relate to sleep efficiency, and the remaining cognitive subdomains were reported too infrequently to obtain a reliable estimate: multiple-domain cognitive functioning ($k = 3$), explicit memory ($k = 3$), implicit memory ($k = 0$), intelligence ($k = 3$), and school performance ($k = 3$).

**Sleep and behavioral problems.**

*Sleep duration.* The combined effect size for the relation between behavioral problems and sleep duration revealed a signif-
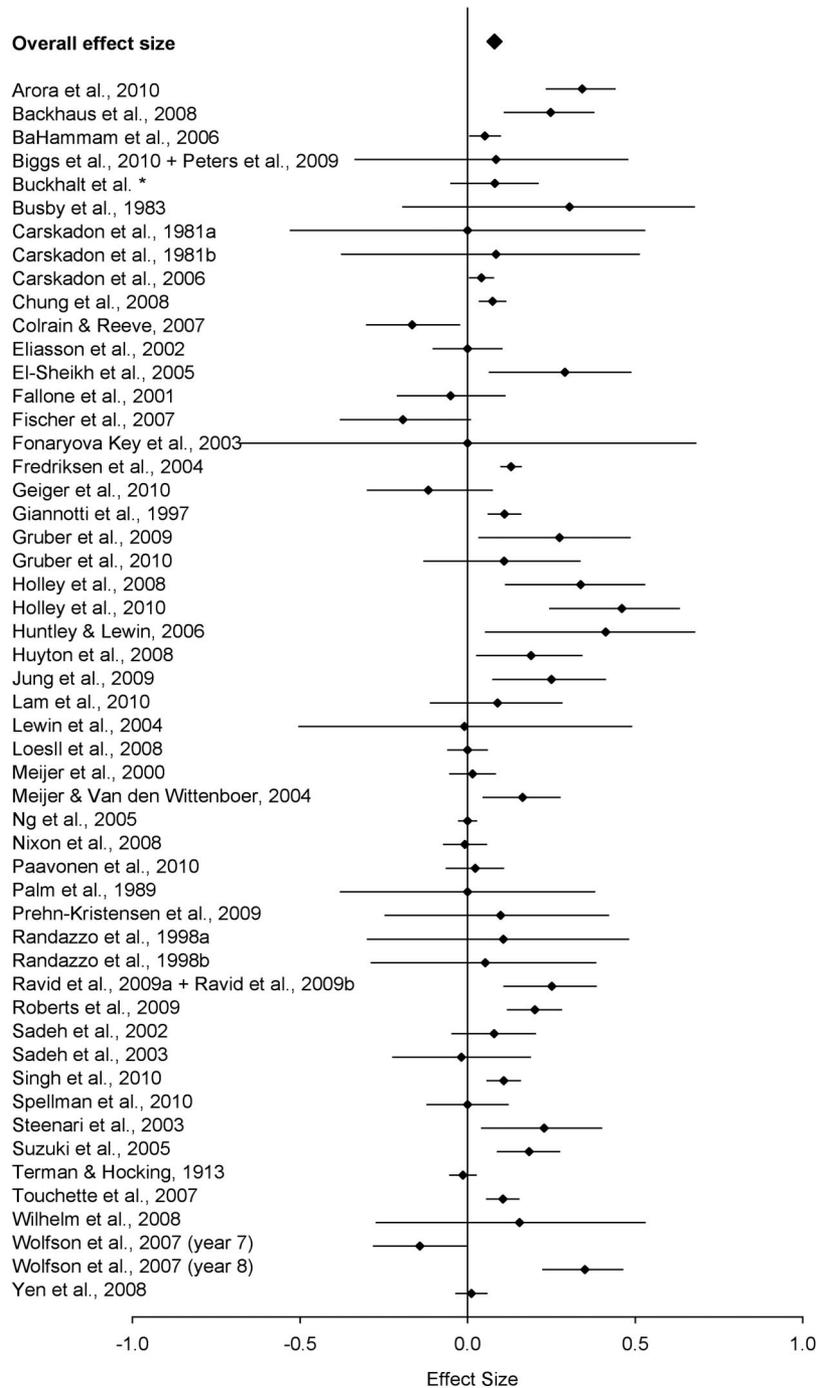
*Figure 2.* Forest plot showing all studies relating children's sleep duration to their cognitive performance. Effect sizes (Pearson's *r* correlations) and 85% confidence intervals are plotted. A positive effect size reflects a correlation in the hypothesized direction (i.e., longer sleep duration relates to better cognitive performance). In those instances where multiple studies reported on the same participant group, these outcomes were meta-analyzed prior to the overall analysis to ensure an independence of effect sizes; this is shown by a + sign. Full references appear in the Appendix. * Indicates Buckhalt et al., 2007 + Buckhalt et al., 2009 + El-Sheikh, Buckhalt, Keller, et al., 2007 + Keller et al., 2008.

Table 2
*Meta-Analyses of Studies Relating Sleep to Cognitive Performance*

| | Cognition | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Sleep duration | | | | | Sleep efficiency | | | | |
| Study characteristics | $k$ | $n$ | $r$ | 85% CI | $Q$ | $k$ | $n$ | $r$ | 85% CI | $Q$ |
| Dimension | | | | | | | | | | |
| Cognition | 52 | 24,454 | .08** | [.06, .10] | 122.58** | 10 | 1,207 | .12 | [.03, .21] | 31.06** |
| Subdomains | | | | | | | | | | |
| Sustained attention | 15 | 996 | .02 | [−.05, .08] | 7.94 | 4 | 587 | −.03 | [−.15, .09] | 4.86 |
| Executive functions | 14 | 2,390 | .07* | [.02, .13] | 16.68 | 5 | 1,063 | .03 | [−.06, .12] | 8.37 |
| Multiple-domain cognition | 11 | 3,066 | .10* | [.05, .16] | 33.08** | 3[a] | 868 | .07 | [−.04, .17] | 2.71 |
| Explicit memory | 11 | 615 | .07 | [.00, .14] | 16.69 | 3[a] | 837 | .00 | [−.11, .10] | 1.88 |
| Implicit memory | 4 | 67 | −.06 | [−.18, .06] | 8.49* | | | | | |
| Intelligence | 6 | 787 | .10 | [.02, .18] | 21.02** | 3[a] | 571 | .12 | [−.01, .24] | 27.00** |
| School performance | 20 | 21,157 | .09** | [.06, .12] | 84.66** | 3[a] | 259 | .15 | [.02, .28] | 0.20 |

*Note.* A positive effect size reflects a correlation in the hypothesized direction (i.e., longer sleep duration or higher sleep efficiency relates to better cognitive performance). $k$ = no. of studies; $n$ = total no. of participants; $r$ = effect size; CI = confidence interval; $Q$ statistic reflects the within-category heterogeneity ($df = k − 1$).
[a] Category contains fewer than four studies; thus the results must be interpreted cautiously.
* $p < .05$.    ** $p < .01$.

icant positive association ($r = .09$, 85% CI [.07, .11]) in a heterogeneous set (as indicated by the significant $Q$ value) of 47 studies: $N = 30,938$, $Q(46) = 215.37$, $p < .01$ (see Figure 4 and Table 3). Adjusted for publication bias, this relation remained significant ($r = .08$, 85% CI [.05, .11]). The in-depth analysis of subdomains of behavioral problem revealed a significant positive association for both internalizing behavioral problems ($k = 37$, $r = .09$, 85% CI [.06, .12]) and externalizing behavioral problems ($k = 38$, $r = .08$, 85% CI [.06, .11]). The 85% CIs overlapped; thus, no significant difference between behavioral problem subdomains' relation with sleep could be concluded.

*Sleep efficiency.* The combined effect size for the relation between behavioral problems and sleep efficiency did not reveal a

significant association ($r = .13$, 85% CI [−.01, .26]) in a heterogeneous set (as indicated by the significant $Q$ value) of seven studies: $N = 967$, $Q(6) = 40.66$, $p < .01$ (see Figure 5 and Table 3). Adjusted for publication bias, this relation remained nonsignificant ($r = .13$, 85% CI [−.06, .30]). Both internalizing and externalizing subdomains of behavioral problems were not significantly related to sleep efficiency, the 85% CIs of these subdomains overlapped.

## Moderator Effects

### Sleep and cognition.
*Sleep duration.* The precision of the sleep duration estimate was the only categorical moderator to reveal a significant effect
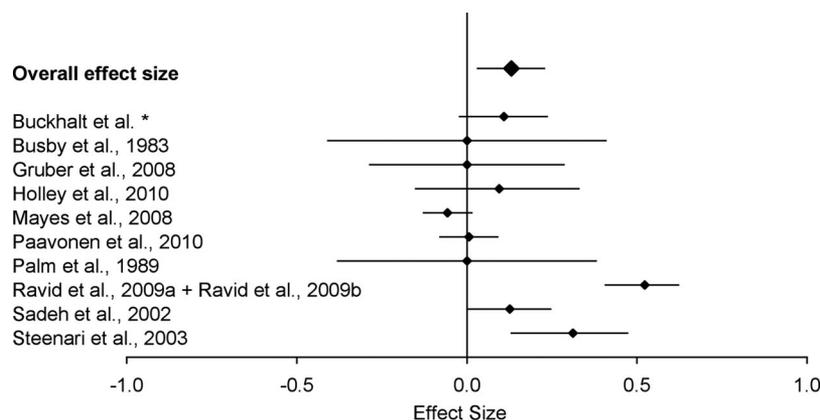


*Figure 3.* Forest plot showing all studies relating children's sleep efficiency to their cognitive performance. Effect sizes (Pearson's $r$ correlations) and 85% confidence intervals are plotted. A positive effect size reflects a correlation in the hypothesized direction (i.e., higher sleep efficiency relates to better cognitive performance). In those instances where multiple studies reported on the same participant group, these outcomes were meta-analyzed prior to the overall analysis to ensure an independence of effect sizes; this is shown by a + sign. Full references appear in the Appendix. Full references appear in the Appendix. * Indicates Buckhalt et al., 2007 + Buckhalt et al., 2009 + El-Sheikh, Buckhalt, Keller, et al., 2007 + Keller et al., 2008.
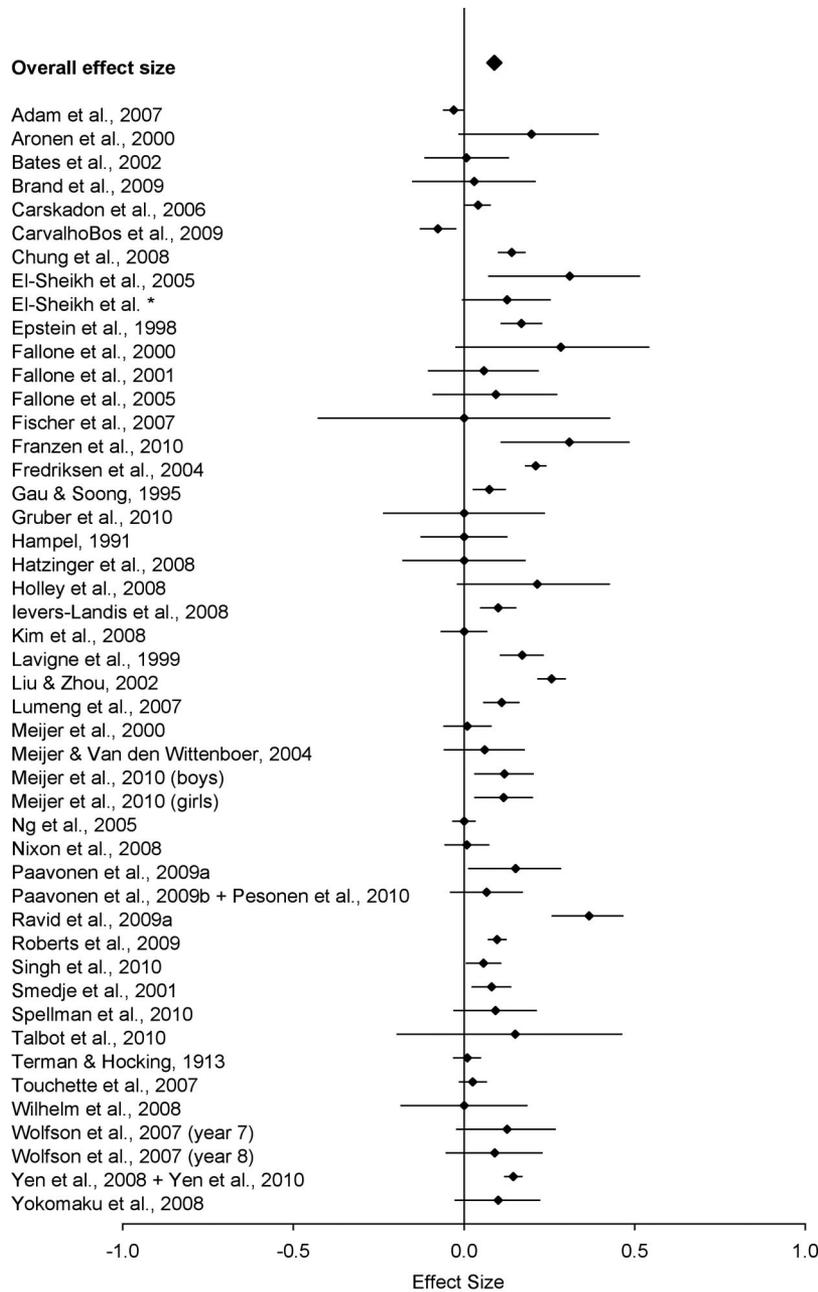
*Figure 4.* Forest plot showing all studies relating children's sleep duration to their behavioral problems. Effect sizes (Pearson's *r* correlations) and 85% confidence intervals are plotted. A positive effect size reflects a correlation in the hypothesized direction (i.e., longer sleep duration relates to less behavioral problems). In those instances where multiple studies reported on the same participant group, these outcomes were meta-analyzed prior to the overall analysis to ensure an independence of effect sizes; this is shown by a + sign. Full references appear in the Appendix. * Indicates El-Sheikh, Buckhalt, Keller, et al., 2007 + El-Sheikh, Buckhalt, Cummings, et al., 2007 + El-Sheikh, Erath, et al., 2007 + El-Sheikh, Kelly, Buckhalt, & Hinnant, 2010 + Keller et al., 2008.

(see Table 4). Studies using actual sleep time (*r* = .17, 85% CI [.12, .22]) showed the largest effect size. Together with "unknown" estimate of sleep duration (*r* = .16, 85% CI [.10, .22]), this subset of studies reported significantly larger effect sizes than studies using time in bed (*r* = .04, 85% CI [−.01, .08]) or

total sleep period (*r* = .07, 85% CI [.04, .09]). According to a meta-regression, the number of nights included for the sleep estimate just failed to reach significance (slope = .002, *p* = .08). Age did not significantly affect the relation (slope = −.01, *p* = .35).

Table 3

*Meta-Analyses of Studies Relating Sleep to Behavioral Problems*

| | Behavioral problems | | | | | | | | | |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| | Sleep duration | | | | | Sleep efficiency | | | | |
| Study characteristics | $k$ | $n$ | $r$ | 85% CI | $Q$ | $k$ | $n$ | $r$ | 85% CI | $Q$ |
| Dimension | | | | | | | | | | |
| Behavioral problems | 47 | 30,938 | .09** | [.07, .11] | 215.37** | 7 | 967 | .13 | [−.01, .26] | 40.66** |
| Subdomains | | | | | | | | | | |
| Internalizing problems | 37 | 25,335 | .09** | [.06, .12] | 287.93** | 6 | 555 | .13 | [−.02, .28] | 39.70** |
| Externalizing problems | 38 | 24,657 | .08** | [.06, .11] | 157.87** | 6 | 972 | .09 | [−.06, .24] | 32.94** |
| Mix | 2[a] | 559 | .05 | [−.13, .23] | 0.18 | | | | | |

*Note.* A positive effect size reflects a correlation in the hypothesized direction (i.e., longer sleep duration or higher sleep efficiency relates to less behavioral problems). $k$ = no. of studies; $n$ = total no. of participants; $r$ = effect size; CI = confidence interval; $Q$ statistic reflects the within-category heterogeneity ($df = k − 1$).

[a] Category contains fewer than four studies; thus the results must be interpreted cautiously.

* $p < .05$.  ** $p < .01$.

***Sleep efficiency.*** The sleep assessment tool moderator was the only moderator that could be examined, but it was not significant (see Table 4). However, the largest and only significant effect size ($r = .17$, 85% CI [.08, .27]) was found in those studies using actigraphy to register sleep efficiency. According to a meta-regression, the number of nights included for the sleep estimate did not significantly affect the relation between sleep efficiency and cognition (slope = .01, $p = .41$). Age just failed to reach significance (slope = −.09, $p = .05$), but four of the nine studies did not report an average age and had to be assigned an artificial estimate.

**Sleep and behavioral problems.**

***Sleep duration.*** None of the categorical moderators revealed a significant effect (see Table 5). Again, studies using actigraphy to assess sleep duration showed a highly significant effect size ($r = .14$, 85% CI [.08, .19]), whereas studies using polysomnography failed to show the expected association ($r = .01$, 85% CI [−.08, .10]). The strongest effect size was found in the subset of experimental sleep duration studies with more than 1 night of imposed sleep restriction ($r = .21$, 85% CI [.08, .33]), but only

four studies with 122 participants in total were included in this subset (see Table 5). In a meta-regression, the number of nights just failed to reach significance (.002, $p = .06$). Age did not significantly affect the relation (slope = .01, $p = .22$).

***Sleep efficiency.*** None of the categorical moderators could be examined (see Table 5). According to a meta-regression, the number of nights included for the sleep estimate revealed a significant positive slope (.07, $p < .05$), implying that the longer sleep was measured the larger the relation detected. Age did not significantly affect the relation between behavioral problems and sleep efficiency (slope = −.05, $p = .27$).

## Discussion

This discussion will start with brief answers to the specific questions raised in the introduction, followed by a more elaborate summary of the found domain-specific associations of sleep with cognition and behavioral problems in children and how these differ from previous findings in adults. Subsequently, we will address the
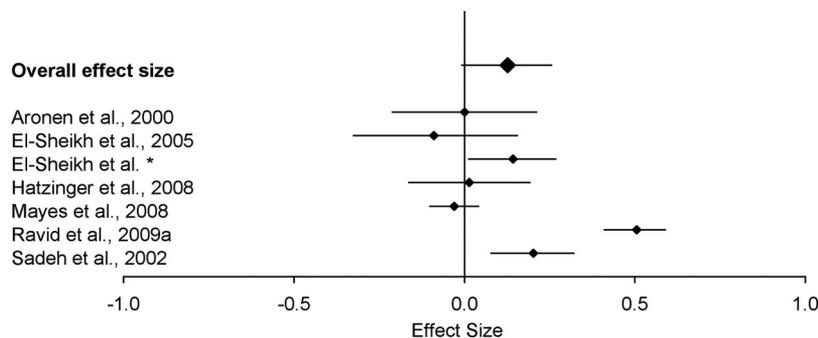


*Figure 5.* Forest plot showing all studies relating children's sleep efficiency to their behavioral problems. Effect sizes (Pearson's $r$ correlations) and 85% confidence intervals are plotted. A positive effect size reflects a correlation in the hypothesized direction (i.e., higher sleep efficiency relates to less behavioral problems). In those instances where multiple studies reported on the same participant group, these outcomes were meta-analyzed prior to the overall analysis to ensure an independence of effect sizes; this is shown by a + sign. Full references appear in the Appendix. * Indicates El-Sheikh, Buckhalt, Keller, et al., 2007 + El-Sheikh, Buckhalt, Cummings, et al., 2007 + El-Sheikh, Erath, et al., 2007 + El-Sheikh et al., 2010.

Table 4

*Moderator Analyses of Studies Relating Sleep to Cognitive Performance*

| | Cognition | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Sleep duration | | | | | | Sleep efficiency | | | | |
| Study characteristics | $k$ | $n$ | $r$ | 85% CI | $Q$ | $p$ | $k$ | $n$ | $r$ | 85% CI | $Q$ | $p$ |
| Type of sleep restriction | | | | | 0.37 | .83 | | | | | | |
| Cross-sectional variation | 32 | 22,916 | .08** | [.06, .11] | 88.38** | | 10 | 1,207 | .12 | [.03, .21] | 31.06** | |
| Wake-sleep comparison | 6 | 120 | .05 | [−.04, .13] | 13.71* | | | | | | | |
| Imposed sleep restriction | 14 | 1,418 | .07 | [.01, .13] | 19.40 | | | | | | | |
| Length of imposed sleep restriction | | | | | | | | | | | | |
| 1 night (experimental) | 7 | 159 | .05 | [−.09, .18] | 2.98 | | | | | | | |
| ≥2 nights (experimental) | 3[a] | 77 | .00 | [−.18, .19] | 0.06 | | | | | | | |
| Retrospective (nonexperimental) | 3[a] | 1,039 | .11* | [.03, .19] | 13.79** | | | | | | | |
| Unknown[b] | 1[a] | 143 | .00 | [−.15, .15] | 0.00 | | | | | | | |
| Precision of the sleep duration estimate | | | | | | | | | | | | |
| Time in bed | 11 | 4,458 | .04 | [−.01, .08] | 13.74 | | | | | | | |
| Total sleep period | 24 | 18,095 | .07** | [.04, .09] | 60.14** | | | | | | | |
| Actual sleep time | 20 | 907 | .17** | [.12, .22] | 22.80 | | | | | | | |
| Unknown[b] | 5 | 1,693 | .16** | [.10, .22] | 15.22** | | | | | | | |
| Sleep assessment tool | | | | | | | | | | | 2.95 | .09 |
| Subjective report | 22 | 22,474 | .08** | [.05, .11] | 86.10** | | | | | | | |
| Actigraphy | 18 | 1,768 | .09** | [.04, .13] | 20.96 | | 6 | 794 | .17** | [.08, .26] | 17.95** | |
| Polysomnography | 9 | 199 | .06 | [−.04, .15] | 8.75 | | 4 | 463 | −.03 | [−.18, .11] | 0.14 | |
| Unknown[b] | 4 | 73 | .04 | [−.08, .15] | 8.28* | | | | | | | |
| Gender | | | | | 1.00 | .32 | | | | | | |
| Male | 3[a] | 37 | .15 | [−.13, .41] | 0.33 | | 1[a] | 11 | .00 | [−.46, .46] | 0.00 | |
| Mixed | 39 | 20,370 | .08** | [.05, .10] | 85.43** | | 8 | 1,172 | .14 | [.04, .23] | 30.90** | |
| Female | 2[a] | 40 | −.13 | [−.29, .03] | 0.63 | | | | | | | |
| Unknown[b] | 8 | 4,007 | .11** | [.07, .16] | 30.88** | | 1[a] | 24 | .00 | [−.36, .36] | 0.00 | |
| Published article | | | | | 1.87 | .17 | | | | | | |
| No | 14 | 3,470 | .12** | [.07, .17] | 34.74** | | 2[a] | 61 | .05 | [−.20, .29] | 0.13 | |
| Yes | 38 | 20,984 | .07** | [.04, .09] | 86.00** | | 8 | 1,146 | .13 | [.03, .23] | 30.93** | |

*Note.* A positive effect size reflects a correlation in the hypothesized direction (i.e., longer sleep duration or higher sleep efficiency relates to better cognitive performance). $k$ = no. of studies; $n$ = total no. of participants; $r$ = effect size; CI = confidence interval; $Q$ statistic for a moderator reflects the between-categories contrast ($df$ = no. of categories − 1); when categories overlapped, comparisons between categories were based on 85% CIs. $Q$ statistic for a category reflects the within-category heterogeneity ($df = k − 1$).
[a] Category contains fewer than four studies; thus the results must be interpreted cautiously. [b] In those instances where the study failed to clarify the methodology used, we classified the study in the 'unknown" category. This category may contain different methodologies, and the results are thus less informative.
* $p < .05$. ** $p < .01$.

methodological issues and developmental mechanisms that could explain the different profiles of associations in children when compared with those of adults. To provide a roadmap for future research, we then discuss research recommendations, pointing out weaknesses in the literature and how they could be overcome to address, in future studies, the outstanding key questions listed in a final paragraph.

The primary question addressed by the current meta-analysis is whether sleep is associated with cognition and behavioral problems in children. The results show conclusively that shorter sleep is associated with worse cognitive functioning and more behavioral problems. The results regarding less efficient sleep were less conclusive but suggested a lack of association. The relations were not significantly affected by publication bias, as confirmed by funnel plot and trim-and-fill analyses and by a formal moderator analysis comparing not-yet published studies with published peer-reviewed articles. The second question, whether cognitive and behavioral problem domains are differentially sensitive to sleep, was also answered. In particular, shorter sleep is associated with worse school performance and executive and multiple-domain

cognitive functioning, as well as with more internalizing and externalizing behavioral problems. Sustained attention, intelligence, and performance on explicit and implicit memory tasks were not significantly associated with sleep duration. The third question addressed whether this profile of differential sensitivity matched the profile previously described in the scientific literature of studies on adults. Although a partial overlap was seen, child studies surprisingly failed to show significant associations of sleep with the cognitive domains of sustained attention and memory, while these may be the very two domains most robustly associated with sleep in adults. Methodological issues and developmental neurobiological differences—the fourth and fifth major questions posed in the present study—may be involved, as will be elaborated on later in this article. Integrating the answers on these questions, the present discussion concludes with a roadmap for the next wave of research on the importance of sleep for brain function, cognition, and behavioral problems in children. The conclusion tries to serve both fundamental and applied perspectives by defining the critical questions and the most sensitive assessment tools to evaluate associations and intervention efficacy.

Table 5

*Moderator Analyses of Studies Relating Sleep to Behavioral Problems*

| | Behavioral problems | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Sleep duration | | | | | | Sleep efficiency | | | | |
| Study characteristics | $k$ | $n$ | $r$ | 85% CI | $Q$ | $p$ | $k$ | $n$ | $r$ | 85% CI | $Q$ | $p$ |
| Type of sleep restriction | | | | | 0.75 | .39 | | | | | | |
|   Cross-sectional variation | 35 | 28,954 | .08** | [.06, .11] | 206.28** | | 7 | 967 | .13 | [−.01, .26] | 40.66** | |
|   Wake–sleep comparison | 2[a] | 27 | .00 | [−.19, .19] | 0.00 | | | | | | | |
|   Imposed sleep restriction | 10 | 1,957 | .12** | [.06, .18] | 7.43 | | | | | | | |
| Length of imposed sleep restriction | | | | | 1.48 | .22 | | | | | | |
|   1 night (experimental) | 1[a] | 82 | .06 | [.14, .25] | 0.00 | | | | | | | |
|   ≥2 nights (experimental) | 4 | 122 | .21* | [.08, .33] | 1.28 | | | | | | | |
|   Retrospective (nonexperimental) | 4 | 1,611 | .11* | [.04, .18] | 4.41 | | | | | | | |
|   Unknown[b] | 1[a] | 143 | .09 | [−.07, .25] | 0.00 | | | | | | | |
| Precision of the sleep duration estimate | | | | | | | | | | | | |
|   Time in bed | 12 | 8,129 | .08** | [.04, .12] | 42.63** | | | | | | | |
|   Total sleep period | 23 | 20,506 | .10** | [.07, .12] | 99.31** | | | | | | | |
|   Actual sleep time | 13 | 1,346 | .12** | [.06, .17] | 22.42* | | | | | | | |
|   Unknown[b] | 2[a] | 977 | .07 | [−.02, .16] | 0.15 | | | | | | | |
| Sleep assessment tool | | | | | 3.25 | .20 | | | | | | |
|   Subjective report | 29 | 28,873 | .08** | [.06, .11] | 187.63** | | | | | | | |
|   Actigraphy | 12 | 1,396 | .14** | [.08, .19] | 20.05* | | 5 | 488 | .19 | [.05, .32] | 21.93** | |
|   Polysomnography | 5 | 656 | .01 | [−.08, .10] | 0.24 | | 2[a] | 479 | −.01 | [−.22, .20] | 0.11 | |
|   Unknown[b] | 1[a] | 13 | .31 | [.08, .51] | 0.00 | | | | | | | |
| Gender | | | | | 0.07 | .79 | | | | | | |
|   Male | 2[a] | 704 | .02 | [−.08, .12] | 5.55* | | | | | | | |
|   Mixed | 38 | 25,792 | .09** | [.07, .12] | 171.58** | | 7 | 967 | .13 | [−.01, .26] | 40.66** | |
|   Female | 2[a] | 725 | .01 | [−.08, .11] | 6.33* | | | | | | | |
|   Unknown[b] | 6 | 4,120 | .08* | [.03, .14] | 15.04* | | | | | | | |
| Published article | | | | | 0.30 | .58 | | | | | | |
|   No | 8 | 3,269 | .07 | [.01, .13] | 7.49 | | | | | | | |
|   Yes | 39 | 27,669 | .09** | [.07, .12] | 202.55** | | 7 | 967 | .13 | [−.01, .26] | 40.66** | |

*Note.* A positive effect size reflects a correlation in the hypothesized direction (i.e., longer sleep duration or higher sleep efficiency relates to less behavioral problems). $k$ = no. of studies; $n$ = total no. of participants; $r$ = effect size; CI = confidence interval; $Q$ statistic for a moderator reflects the between-categories contrast ($df$ = no. of categories − 1); when categories overlapped, comparisons between categories were based on 85% CIs. $Q$ statistic for a category reflects the within-category heterogeneity ($df = k − 1$).
[a] Category contains fewer than four studies; thus the results must be interpreted cautiously.    [b] In those instances where the study failed to clarify the methodology used, we classified the study in the "unknown" category. This category may contain different methodologies, and the results are thus less informative.
* $p < .05$.    ** $p < .01$.

## Summary of Overall and Subdomain Findings in Children Versus Adults

The current meta-analysis reveals small but significant associations of sleep duration with cognition ($r = .08$) and behavioral problems ($r = .09$) in children. Sleep efficiency did not show significant associations. For the subdomains, the profile and strengths ($rs = .08–.10$) of associations differ from those previously summarized in meta-analyses on adult sleep deprivation studies ($rs = .06–.36$; adapted from Lim & Dinges, 2010; Pilcher & Huffcutt, 1996). The potential involvement of methodological issues and developmental mechanisms in the weaker association of sleep duration with different domains in children will be summarized in following text.

**Sustained attention.** The present meta-analysis did not find an association of sleep duration ($r = .02$, $p = .71$) or sleep efficiency ($r = −.03$, $p = .71$) with sustained attention in children, while this domain may be the most sensitive to sleep deprivation in adults (Lim & Dinges, 2010) and has even been proposed to be responsible for the performance degradation on other tasks (Lim &

Dinges, 2008, 2010; Philibert, 2005; Philip, Demotes-Mainard, Bourgeois, & Vincent, 1991). The most marked discrepancy between child and adult studies on the sensitivity of sustained attention to sleep justifies an elaborate discussion of this subdomain later. Critical questions are how methodological issues and developmental neurobiology may be involved in the discrepancy.

**Executive functioning.** The present meta-analysis found that curtailment of sleep length ($r = .07$), but not reduction of sleep efficiency ($r = .03$, $p = .62$), was associated with compromised executive functioning in children. As only few studies reported on sleep efficiency values, this result must be interpreted cautiously. The sleep duration finding is compatible with a recent meta-analysis in adults that showed a moderate effect of sleep deprivation on complex attention and working memory tasks (Lim & Dinges, 2010). Executive functioning and the prefrontal cortical circuits involved thus appear sensitive to sleep curtailment even early in development and quite robustly so, as has also been noted by others (Harrison & Horne, 1999, 2000; Horne, 1988; Jones & Harrison, 2001; Muzur, Pace-Schott, & Hobson, 2002). During

sleep, the prefrontal cortex shows the most prominent decrease in regional cerebral blood flow (Braun et al., 1997; Hofle et al., 1997; Maquet et al., 1990), which seems to have functional relevance since sleep deprivation affects the resting-state electroencephalographic signature of brain activity most prominently in prefrontal areas (Cajochen, Khalsa, Wyatt, Czeisler, & Dijk, 1999; Cajochen, Knoblauch, Krauchi, Renz, & Wirz-Justice, 2001; Finelli, Borbély, & Achermann, 2001). Functional brain imaging studies moreover reveal abnormal task activation in this area following sleep loss (Chee & Choo, 2004; Drummond et al., 2000; Horne, 1988). Our present findings, indicating the sensitivity of executive functioning to sleep restriction in children, indicate that the prefrontal cortical circuits should receive particular interest in future in-depth studies on the neurobiological underpinnings of the effects of sleep deprivation on cognition and behavioral problems.

**Multiple-domain cognitive functions.** The present meta-analysis found sleep curtailment in children also to be associated with compromised performance on tasks that less unequivocally belong to a single cognitive domain but rather require integration of multiple cognitive domains ($r = .10$). In adults, sleep loss was noted to be detrimental to divergent performance that requires mental flexibility and multitasking (Durmer & Dinges, 2005; Horne, 1988). Such integration typically involves the prefrontal circuitry. We therefore interpret the current findings as additional support for the conclusion of the previous paragraph, that prefrontal circuits deserve attention in future studies on the neurobiological underpinnings of the effects of sleep deprivation on cognition and behavioral problems.

**Memory.** The present meta-analysis did not find an association of sleep with memory in children (explicit memory, $r = .07$, $p = .14$; implicit memory, $r = -06$, $p = .48$). The lack of association cannot be directly compared with a recent meta-analysis in adults, because the latter did not include the cognitive domain of long-term memory (Lim & Dinges, 2010). Still, the findings are surprising given the numerous studies in adults that report a role of sleep in memory consolidation. Memory refers to a cognitive process that involves at least three steps: (a) initial learning or encoding, (b) consolidation and memory integration, and (c) remembering or recall. Sleep curtailment may interfere with each of these steps (e.g., Born, Rasch, & Gais, 2006; Diekelmann & Born, 2010; Fischer, Nitschke, Melchert, Erdmann, & Born, 2005; Maquet, 2001; Van Der Werf et al., 2009; Walker & Stickgold, 2004; Yoo, Hu, et al., 2007). Our results suggest that in childhood, methodological considerations or developmental aspects of brain structure and function impeded finding the selective supportive role of sleep for memory that is typically found in adult studies. These two possibilities will be addressed in more detail in the next section of the discussion.

**Intelligence.** Consistent with a recent meta-analysis in adults (Lim & Dinges, 2010), the present meta-analysis found no association of intelligence with sleep duration ($r = .10$, $p = .08$). In adults, intelligence correlates with sleep spindle activity, which may represent a signature of the structure and function of thalamocortical circuits (Fogel, Nader, Cote, & Smith, 2007; Schabus et al., 2006). Whereas intelligence is highly consistent across the life span (Gow et al., 2011), it remains to be evaluated whether its association with sleep spindles already exists in childhood.

**School performance.** Of considerable interest from an applied point of view is the fact that the present meta-analysis found sleep curtailment to be most strongly associated with compromised school performance in children ($r = .09$). The association may be this strong because school performance aggregates a child's performance over multiple cognitive domains and over an extended period of time. This makes the measure less sensitive to the fluctuations and measurement noise that occur in single brief tests that are usually applied in the other cognitive domains. A previous meta-analysis on the relation between sleep and school performance in 8- to 18-year old children and adolescents reported combined effect sizes of $r = .07$ for sleep duration estimates (17 studies) and $r = .10$ for sleep efficiency estimates (16 studies) (Dewald et al., 2010). Our subdomain analysis on school performance focused on a more homogeneous age range (5–12 years), adhered to the MOOSE guidelines (Stroup et al., 2000), included more studies reporting on sleep duration (20 studies), and less reporting on sleep efficiency (three studies); we included only those studies that quantitatively assessed sleep quality and not those that asked for subjective sleep quality estimates.

**Internalizing behavioral problems.** The present meta-analysis found that sleep length curtailment ($r = .09$), but not sleep efficiency reduction ($r = .13$, $p = .22$), was associated with internalizing behavioral problems. As only few studies reported on sleep efficiency values, this result must be interpreted cautiously. Sleep disturbances, anxiety, and depression are closely related in adults (American Psychiatric Association, 2000). The relation between sleep and emotional problems can be bidirectional, as emotional problems and (academic) stress also have a detrimental effect on sleep (Lund, Reider, Whiting, & Prichard, 2010). Experimental studies support the importance of appropriate sleep since curtailment alters emotional reactivity in adults (Franzen et al., 2010; Sterpenich et al., 2007; Yoo, Gujar, Hu, Jolesz, & Walker, 2007). Studies in children likewise support the overnight therapy hypothesis (Walker & Van der Helm, 2009) mentioned in the introduction, which proposes that sleep helps maintain emotional reactivity and reactivity of limbic and associated autonomic networks appropriately. The autonomic nervous system readout of skin conductance level is lower at baseline and more responsive to stress in children with sleep problems (El-Sheikh & Arsiwalla, 2011). The deviation is moreover associated with an increased vulnerability for the internalizing problems of depression and low self-esteem. Likewise, deviant heart rate variability at baseline and in response to a stressor, another index of suboptimal autonomic nervous system regulation, increases the risk of both externalizing and internalizing problems in children with sleep problems (El-Sheikh & Buckhalt, 2005; El-Sheikh, Erath, & Keller, 2007). These observations in combination with the findings of this meta-analysis warrant further evaluation of the involvement of emotional and autonomic regulation in the effect of sleep curtailment on behavioral problems in children.

**Externalizing behavioral problems.** The present meta-analysis found that sleep length curtailment ($r = .08$), but not sleep efficiency reduction ($r = .09$, $p = .37$), was associated with externalizing behavioral problems. As only few studies reported on sleep efficiency values, this result must be interpreted cautiously. Interestingly, the externalizing problems are thought to involve derailment in the prefrontal circuits supporting executive functioning (Huizinga & Smidts, 2011), thus once more indirectly supporting the sensitivity to sleep. It is important to note that in contrast to the lack of physical activity that characterizes sleep-deprived

adults (Schmid et al., 2009), sleep-deprived children may instead show hyperactivity (Aronen et al., 2000; Lavigne et al., 1999; Paavonen, Raikkonen, et al., 2009) even at a level resembling attention-deficit/hyperactivity disorder, a disorder also linked to dysfunctional prefrontal inhibitory control (cf. Boonstra et al., 2007). Dahl's (1996) developmental model proposes prefrontal immaturity to underlie the externalizing behavioral problems of sleep-deprived children. Our findings support a relation between sleep and externalizing behavioral problems in childhood, an association not commonly studied in adults.

## Why Are Associations Between Sleep and Cognition Different in Children?

**Methodological issues.**    Are there methodological differences that could mechanistically explain the different findings of the present meta-analysis in children as compared to previous ones in adults?

The first issue concerns differences in the included fractions of *experimental versus observational studies*, precluding a straightforward comparison of our meta-analysis with a recent one conducted on studies in adults (Lim & Dinges, 2010). The meta-analysis on adults focused on studies that experimentally induced short-term sleep restriction. In the majority of the studies in children on the other hand, investigators observed naturally occurring variability in sleep duration. Thus, in contrast to studies in adults, more than half of the studies in children (68%) are of *correlative* nature, which limits conclusions about causality. The role of common underlying factors affecting both sleep and daytime functioning cannot be ruled out. Possible factors that can influence both sleep and functioning include neurobiological mechanisms such as autonomic nervous system functioning (El-Sheikh, Erath, et al., 2007) or temperament (M. Moore, Slane, Mindell, Burt, & Klump, 2011) as well as environmental factors such as parenting style (Owens & Jones, 2011). Moreover, short sleepers in the cross-sectional studies may represent a heterogeneous group: children who get less sleep than they need and children who just need less sleep. Because effects of sleep curtailment on cognition and behavioral problems can only be expected in the former group, effects will be diluted and effect sizes deflated. Whereas the meta-analysis on adults by Lim and Dinges (2010) included only studies reporting effects of acute sleep deprivation, sleep deprivation in child studies is seldom reported and, if so, is usually milder. Only one study applied total sleep deprivation (Carskadon et al., 1981b), possibly because of more stringent ethical rules for research on children.

Still, even in studies using the psychomotor vigilance task (PVT), reportedly the sustained attention task that is most sensitive to total sleep deprivation but also to chronic sleep curtailment (Banks & Dinges, 2007; Dinges et al., 1997) in adults, the effect of sleep restriction in children is surprisingly small (Peters et al., 2009). Here, a second set of methodological issues may be involved concerning systematic differences in *the way tasks can be performed*. Slow reaction times and lapses occur more frequently with increasing time on task (Doran et al., 2001). An issue that may arise in practice, especially in tasks that rely on top–down attentional control and lack bottom–up salience-induced promotion of attention, is that children may not be able to perform such a task without ongoing encouragements. Any such intervention,

although often necessary, interferes with the very intention and sensitivity of the task. Although tasks of prolonged duration have been applied in studies on children, reports are unclear as to whether children were monitored and encouraged to continue. It is not unlikely that this may have been the case, given the fact that sustaining attention for a prolonged period may be particularly difficult for children, even if well rested. Starting during the preschool years, the development of the ability to sustain attention is slow, notably so for simple reaction time tasks as compared with more interesting tasks (Ruff, Capozzoli, & Weissberg, 1998; Ruff & Lawson, 1990). A possible behavioral outcome measure that could be considered for future studies is to record the duration of involvement in the task before the first (and subsequent) encouragements have to be made. A related methodological consideration is that children may perform a task in a systematically different way and use other cognitive strategies. For instance, an often-used sequence finger-tapping task (Walker et al., 2003) elicits overt vocalization and is performed much slower by children than by adults, suggestive of a more hippocampus-based explicit learning strategy (Wilhelm et al., 2008).

A third set of methodological issues that may be involved in the large differences between reported sleep sensitivity of cognitive subdomains in adults and children concerns *floor/ceiling effects.* If, for example, children have less developed brain mechanisms to support prolonged attentional control to begin with (Supekar et al., 2010), as will be discussed later, the sustained attention capacities of well-rested kids leave little room for further attenuation by sleep restriction. Indeed, normative average reaction times in children are very long (Levy, 1980). For the PVT, average reaction times of well-rested children are even longer than those that are typical of sleep-deprived adults (e.g., Lamond et al., 2007; Peters et al., 2009).

Floor/ceiling effects may also be involved in our finding that cognitive and behavioral problem domains were associated with sleep duration but not with sleep efficiency. Sleep efficiency, the percentage of the total amount of time spent in bed that a person is actually asleep, is more than 95% in the majority of studies in school-age children and declines with age only after adolescence, and more strongly than any other sleep parameter (Ohayon et al., 2004). It may be that any variance within the very high range of more than 95% has little relevance to daytime functioning. Sleep efficiency is usually regarded as problematic only if it falls below a cutoff of 85%. Effects on daytime functioning may surface only in case of even lower values. For example, studies in older people report impaired cognitive performance mainly for those participants with very fragmented sleep–wake patterns and with sleep efficiency values of less than 70% (Blackwell et al., 2006; Oosterman, van Someren, Vogels, Van Harten, & Scherder, 2009). Our moderator analyses on sleep efficiency outcomes indicated that an association of sleep efficiency with cognitive performance or behavioral problems in children might surface only if, respectively, actigraphy and a more extended period of assessment are utilized. It should finally be noted that in total only 13 studies reporting on sleep efficiency could be included in the present meta-analysis, versus 74 on sleep duration, resulting in less statistical power to detect associations.

The fourth set of methodological issues that may be involved in the large differences between the association of cognition and sleep in children versus adults concerns *participant selection cri-*

*teria*. Heterogeneity of children participating may have introduced variance and deflated effect sizes if not captured using covariance or moderator analyses. For instance, while gender may influence the relation of sleep to cognition and behavioral problems in children, most studies failed to report separate outcomes for boys and girls, thus reducing the statistical power of the current meta-analytic investigation of gender and leaving possible gender differences within the pool of unexplained variance. Furthermore, several studies have shown that socioeconomic status and ethnicity significantly affect the relation of sleep to cognition and behavioral problems. Children of lower socioeconomic status that live in a more challenging home environment appear more sensitive to the effect of sleep curtailment on cognitive performance (Buckhalt et al., 2007; El-Sheikh, Buckhalt, Cummings, & Keller, 2007). A similar increased sensitivity was reported for African American children (Buckhalt et al., 2007). A deplorable lack of standard reporting methods in the studies meant that socioeconomic status and ethnic differences could not be included as moderators in the present meta-analysis, thus inflating unexplained variance. Finally, the studies in the current meta-analysis often included wide age ranges. Insofar as the effects of sleep on cognitive performance are age-dependent, even within the range of school-age children, this may have introduced more unexplained variance and deflated effect estimates. Although our meta-regression on age effects across studies did not reveal an effect of age, it should be noted that the age ranges among the meta-analyzed studies were at times quite wide.

A fifth category of methodological issues to be considered concerns the effect of the *environment and context* of task and sleep assessment. The structured environment of a laboratory setting may affect sleep, emotional state, motivation, and cognitive functioning differentially in adults and children. The fact that some naturalistic studies found significant associations between sleep measured at home and cognitive performance at school (e.g., Sadeh et al., 2002, 2003) suggest that a laboratory setting may obscure the detection of such association in children.

In summary, a direct comparison of effect sizes in adults and children is hampered by several methodological differences, which could underlie the smaller effect sizes we found in child studies. However, it is important to note that the methodological issues are mostly rather generic, not specific to a certain cognitive domain. Still, children's sleep did show an association comparable to that of adults with some domains, but not with other domains. Methodological differences do not provide an easy explanation that completely accounts for this dissociation. It therefore appears desirable to consider additional possible explanations and to present them side by side with the mentioned methodological ones. While acknowledging the importance of the latter, we would like to present a number of possible developmental neurobiological explanations, especially for the differential findings for sustained attention and memory.

**Developmental issues.**    Could developmental neurobiological issues be involved in the remarkable differences between children and adults in their association of sleep with sustained attention and memory? Theoretically, at least two principles should be considered if the sleep sensitivity that is present in adults cannot be demonstrated in children.

The first principle can be formulated in two ways. First, if the neuronal networks or mechanisms that are essential for the bene-ficial effects of sleep in adults are insufficiently developed, children *cannot yet fully benefit*. Secondly, if the neuronal networks or mechanisms that are sensitive to sleep deprivation in adults are likewise insufficiently developed, children *cannot yet fully suffer.* We will hypothesize that this principle may apply to the surprisingly lacking associations of sleep with sustained attention and memory in children and involves immaturity of hippocampal connectivity and long-range corticocortical connectivity especially along the anterior–posterior axis.

The second principle does not concern a developmental shortcoming, but rather the opposite: neurobiological mechanisms that are effective in early development, but less so or not at all in adults. We will argue for the possibility that in children some *benefits may not be limited sleep* but occur equally during wakefulness. We argue that such a principle may be involved in the lacking association of sleep with memory, as synaptic scaling might be much less restricted to the sleep period in children than it is in adults. Notably, this principle does not predict a deficiency but rather an advantage in children if memory consolidation could occur over periods of both wakefulness and sleep alike, rather than preferably over periods of sleep—as is the case in adults.

As briefly mentioned in the introduction, the brain undergoes profound changes during childhood. Macroscopically, its weight increases fourfold between birth and 10 years of age. Microscopically, it displays cell growth, cell proliferation, arborization, synaptogenesis, pruning, and an increase in white-matter volume due to myelination and axonal growth (Srinivasan, 1999). Even following the synaptic density peak in early infancy, school-age children still show exceptionally high levels of synaptic density. Many parts and connections of the brain have not yet fully matured, which is reflected in cognition and behavior. Children moreover sleep more than adults do, and their sleep expresses far more slow wave activity (Feinberg, 1982). These neurobiological differences render it likely that effects of sleep curtailment are different too.

Indeed, some of the neurobiological mechanisms that are thought to specifically underlie the sensitivity of specific cognitive subdomains to sleep curtailment in adults may not yet be in place in children. Immaturity may differentially affect the sensitivity of specific cognitive subdomains—depending on the maturational state of the subdomain's neurobiology—and its sensitivity to sleep curtailment. In the present section, we discuss possible developmental neurobiological causes underlying differential sensitivity to sleep in children for sustained attention and memory, cognitive domains that are sensitive to sleep in adults.

***Sustained attention.***    Might immaturity of a child's brain play a role in the striking absence of a robust association between sleep duration and sustained attention? As briefly discussed in the introduction, studies in adults indicate that sleep deprivation affects sustained attention tasks primarily by (a) intrusions of DMN activity that (b) disrupt the top–down attentional biasing activity of the frontoparietal network (Chee & Tan, 2010; Drummond et al., 2005). The extent to which sleep deprivation increases DMN activity and suppresses frontoparietal network activity determines the extent to which an individual's performance deteriorates (Chee & Tan, 2010; Drummond et al., 2005). Drummond et al. (2005) even suggested that the inappropriate DMN activation and the insufficient attentional network activation may serve as precursors to more overt signs of sleepiness such as eye closure and dozing

off. As noted before, these behaviors may be typical for sleepy adults, but less so for children, in whom a lack of sleep may rather be expressed as hyperactivity (cf. Boonstra et al., 2007). Given the essential involvement of long-range connectivity in these networks in modulating sustained attention, one might question to what extent these pathways are sufficiently developed in school-age children to attain the capacity of prolonged attention, even when well rested. Critical questions that can be asked are therefore: to what extent are children capable of (a) addressing the frontoparietal attention network to sustain attention and (b) expressing DMN intrusions that suppress activity in the frontoparietal attention network? To phrase it more specifically: to what extent are these networks developed and functional in children? If not yet in place, this may explain why children differ from adults with respect to the association of sustained attention and sleep duration and/or sleep efficiency.

Recent studies indeed showed structural and functional immaturity of these networks in children (e.g., Fair et al., 2008; Fransson et al., 2007; Supekar et al., 2010; for reviews, see Daniels et al., 2011; Uddin et al., 2010), especially for the longer range connections along the anterior to posterior axis (Dosenbach et al., 2010), including the frontal to parietal connectivity that is essential in top–down attentional control and is sensitive to disruption by DMN activity intrusion. Using resting-state functional magnetic resonance imaging (fMRI), Fair et al (2007) showed that, within the frontoparietal attention network, children lack functional connections from the dorsolateral prefrontal cortex to the intraparietal sulcus and inferior parietal lobule. Also, the DMN regions are only sparsely functionally connected at early school age (7–9 years old; Fair et al., 2008). Using multimodal imaging by combining resting-state fMRI, voxel-based morphometry, and diffusion tensor imaging-based tractography, Supekar et al. (2010) showed that posterior cingulate cortex–medial prefrontal cortex connectivity along the cingulum bundle is the most immature link in the DMN of children between the ages of 7 and 9 years as compared with young adults between the ages of 19 and 22 years—the latter being the typical age of college students involved in most reports on sustained vigilance and sleep in adults. Summarizing the findings, Daniels et al. (2011) concluded that, before the age of 9 years, anterior–posterior integration is absent or limited. Thus, it may well be that the developmentally immature underlying neurobiology prevents children from being able to benefit from sleep to promote sustained attention to reach the level attainable by well-rested adults.

*Explicit memory.* Might immaturity of a child's brain play a role in the surprising finding that children, unlike adults, do not show a robust association of sleep with memory consolidation? Explicit memory involves structures in the medial temporal lobe, including the hippocampus and parahippocampal cortex. As briefly described in the introduction, the replay hypothesis of memory consolidation during sleep presumes a gradual strengthening of the horizontal corticocortical connections to promote memory traces to be released from hippocampal involvement. Transfer from hippocampus to the medial prefrontal cortex seems particularly relevant (Frankland & Bontempi, 2006; Takashima et al., 2006). To what extent do children show maturity of (a) these horizontal corticocortical connections and of (b) connectivity between hippocampus and these corticocortically connected neuronal ensembles to transfer memory traces?

The answer to both questions is that there is a considerable immaturity. First, both structural and functional imaging studies—showing a gradual developmental increase in white-matter volume, myelination and intracortical maturity of axons—indicate that especially the large-scale architecture of feed-forward and feed-back corticocortical connections is still developing throughout late childhood and adolescence (e.g., J. K. Moore, 2002; Srinivasan, 1999). Thus, the functional integration of neural cell assemblies that support the memory traces when liberated from hippocampal involvement is immature. Second, with respect to hippocampal connectivity, even at the age of 11 years the density of hippocampus myelinated fibers has not reached the adult level (Abrahám et al., 2010). Benes et al. (1994) showed a 95% increase in myelin staining in the superior medullary lamina of the hippocampus between the first and second decades of life and suggested that part of this increase was due to developing connections with the anterior cingulate in the medial prefrontal cortex. Resting-state fMRI functional connectivity studies confirm immature patterns of this hippocampus–medial prefrontal cortex connection in children (Fair et al., 2008; Kelly et al., 2009; Power et al., 2010). All resting-state networks in the infant brain are characterized by immature or absent connectivity along the anterior–posterior direction (Fransson et al., 2007). This includes networks that are involved in memory in adults (i.e., the lateralized frontotemporal and frontoparietal networks including the middle frontal and orbital cortex, superior parietal cortex, middle temporal gyrus, and the posterior cingulate cortex; Damoiseaux et al., 2006).

Developmental studies on explicit memory confirm that the entire process of encoding, retention, and retrieval continues to develop throughout childhood. Whereas children as young as 3 or 4 years old are able to form episodic memories (Hayne & Imuta, 2011), performance on memory tasks continuously improves and does not resemble that of adults until the age of 11 years, but it continues to develop into young adulthood (cf. Menon, Boyett-Anderson, & Reiss, 2005).

There are a couple of interesting particularities of these immaturities with respect to the presumed role of sleep in explicit memory consolidation in adults. First, children show very high cue and context dependency—whereas sleep is proposed to be especially important for the integration of new knowledge into existing frameworks, thus making it less context dependent (Lewis & Durrant, 2011). Second, spatial memory is immature until children reach the age of 8 years (Overman, Pate, Moore, & Peuster, 1996), whereas it is one of the most robustly sleep-dependent types of explicit memory in adult humans and rats (Peigneux et al., 2004; Wilson & McNaughton, 1994).

In summary, both cognitive and brain structural and functional observations in children suggest immaturity of the neurobiological substrate involved in replay and consolidation of explicit memories during sleep.

*Implicit memory.* In contrast to the hippocampus–cortical (and especially medial prefrontal cortex) dialogue involved in explicit memory, the implicit memory that underlies priming, conditioning, and the acquisition of skills involves parts of the striatum, cerebellum, and brain stem, which are functionally mature very early in life (Richmond & Nelson, 2007). Immaturity of these neurobiological substrates thus cannot be held responsible for a lack of sleep dependency of implicit learning in children. We suggest that both the cognitive profile and neurobiological mech-

anisms of the lack of sleep dependency in children differ considerably for explicit and implicit learning. In contrast to the immaturity of the hippocampus–cortical network, the network supporting implicit memory formation shows a high degree of maturity at birth, and indeed infants show a well-developed capacity for implicit learning. We propose that children have in fact superior capacities for implicit learning, because consolidation is not limited to occur preferentially overnight during sleep—as is the case in adults—but also during a period of wakefulness. Thus, we hypothesize that whereas adults have an advantage over children by being able to utilize sleep for consolidation of explicit memory, they also have a disadvantage by being more dependent on sleep for consolidation of implicit memory.

We suggest that, instead of a *lack* of skill improvement over sleep, children show improvements both over periods of sleep and over periods of wakefulness, resulting in a null difference in statistical tests where wake improvements serve as baseline. The brains of children may not be as dependent on sleep for network maintenance through synaptic downscaling as the brain of adults. Synaptic density in the cerebral cortex increases during infancy to reach a maximum at the age of 1 to 2 years. At this age, synaptic density is about 50% more than the mean adult density. A decline in synaptic density then sets in, lasting until the age of about 16 years (Huttenlocher, 1979; Huttenlocher & de Courten, 1987). Whereas it is commonly suggested that accelerated pruning of synapses occurs specifically during adolescence, human data actually indicate a gradual decrease in the number of synapses that begins in early childhood (Paus et al., 2008). Thus, throughout the age range aggregated in the present meta-analysis, developmental scaling of synaptic density can be presumed. The mechanism underlying the developmental reduction in synaptic density remains to be elucidated. Of note, hypotheses on mechanisms underlying the developmental reduction in synaptic density (e.g. Low & Cheng, 2006; Schafer & Stevens, 2010) seem unrelated to hypotheses on the underlying mechanisms of sleep-related downscaling of synaptic weights (Tononi & Cirelli, 2006).

A dialogue between these fields of expertise may yield critical hypotheses. At the extremes of the opposing possibilities—equal versus different mechanisms are involved in (a) developmental and (b) sleep-dependent synaptic downscaling—two hypotheses with opposing predictions may be put forward. The first hypothesis is that children show a net increase in synaptic density during wakefulness and downscaling is mostly limited to sleep, just like adults. As a consequence, downscaling must be even more prominent in the sleep of children than it is in the sleep of adults, in order to attain the net decrease in synaptic density across the childhood years, as compared with a relatively stable net synaptic density in adults. The second, alternative, hypothesis is that (a) developmental and (b) sleep-dependent synaptic downscaling rely on qualitatively different mechanisms. The developmental downscaling may not be dependent on sleep but may act on the brain continuously—thus, when awake and when asleep. A critical evaluation of the hypothesis on synaptic scaling during sleep indeed suggests that synaptic scaling may as well take place during wakefulness (Frank, in press). The extent to which this is possible may moreover change during development; qualitative differences in synaptic scaling efficiency exist between young and adult animals (e.g. Goel & Lee, 2007).

Insofar synaptic downscaling supports memory, these two alternative hypotheses are testable. In the first case, children would differ from adults by showing an enhanced sensitivity of memory consolidation to sleep. The present meta-analysis does not support this possibility. The second interpretation predicts that children show memory performance improvements over periods of wakefulness and sleep alike—unlike adults, who require sleep for these improvements to occur. In the second case, children would differ from adults by showing memory enhancement not only over periods of sleep, but also over periods of wakefulness. Indeed, we have preliminary support that this may be the case. Children show similar performance increments over periods of sleep and wakefulness (Schutte et al., 2008) on the motor sequence learning paradigm that most robustly shows selective sleep-dependent enhancement in adults (Walker & Stickgold, 2004). Similarly, children showed similar improvements on mirror-tracing performance over a period of wakefulness as over a period of sleep (Prehn-Kristensen et al., 2009). Finally, 9- and 12-year-old children show less susceptibility to daytime interference of a newly acquired motor memory than 17-year-olds, which supports the idea of childhood waking memory consolidation (Dorfberger, Adi-Japha, & Karni, 2007). It may thus be that children indeed have a more elaborate arsenal of brain mechanisms to support synaptic downscaling during sleep *and* wakefulness than is left in adults, whose mechanisms may have become much more dependent on sleep.

In summary, in addition to methodological considerations, developmental neurobiology may be involved in the differential effects of sleep curtailment in adults and children, especially for sustained attention and memory. We thus propose that the profile of associations of cognitive subdomains with sleep shows a developmental trajectory. This may even be the case within the age range of school-age children, as suggested by a longitudinal study in children 7 to 11 years old, which showed that the associations between sleep and cognition changed when they were reassessed 2 years later (Buckhalt et al., 2009).

## Research Recommendations

By examining the differences in study methodologies and the size of the relations that different designs detected through moderator analyses, we can provide useful suggestions that could both increase the sensitivity to detect sleep-dependent aspects of cognition and behavioral problems in future studies in children, but could also aid developmental research in general. A cautionary note is that the introduction of categories as additional variables in the moderator analyses reduces statistical power as compared to the overall meta-analysis.

The only significant moderator concerned *sleep assessment methodology*. The most precise estimate of sleep duration, the actual time slept, revealed the relationship between sleep and cognition most clearly. Its use—rather than, for instance, the total time spent in bed—should be recommended for future studies. Surprisingly, the meta-analysis suggests that polysomnography, the gold standard for sleep measurements, was unable to detect the relation of interest, whereas actigraphy could. The finding may be elucidated by the meta-regression analyses demonstrating the near-significant moderating effect of the number of nights included for the sleep estimate. Whereas most polysomnographic studies incorporated only 1 night of sleep measurement, actigraphic assessment

has the advantage that multiple nights can easily be assessed, thus effectively solving the problem of night-to-night variability (Van Someren, 2007) that also characterizes the sleep of children (Scholle et al., 2003). In agreement with a previous study that showed an increasing reliability of sleep estimates with the number of nights assessed (Van Someren, 2007), a relation with cognition and behavioral problems appears to be more readily detected for longer actigraphic sleep assessments. In addition, not only does actigraphy easily measure multiple nights, but also it obtains these measures of sleep in natural environments and captures the variance in sleep in these environments. For instance, when examining the experimental manipulation of sleep duration using actigraphy in the home environment, even modest changes in sleep duration affected cognitive performance (Sadeh et al., 2003). In contrast, polysomnography is typically obtained in a controlled laboratory environment and impacts several aspects of children adapting to this controlled environment.

Still, polysomnographic studies remain essential for a detailed quantification of sleep architecture and for an investigation into the involvement of different sleep stages and features such as sleep spindles or slow waves that are crucially involved in the role of sleep in cognitive function in adults (Born et al., 2006). Information on the relation of these features to cognition and behavioral problems in children is still minimal at present. Future studies should take advantage of the fact that technological developments now allow for home polysomnography. As compared with lab measurements, home polysomnography yields better sleep in children (Stores, Crawford, Selman, & Wiggs, 1998). Of note, also the manipulation of sleep has been applied successfully in the natural home environment. Thus, home manipulation and assessment studies prevent confounding by children's sensitivity to environmental variations, they may increase the sensitivity to detect the explored links, and they may add to the ecological validity of the findings.

The present meta-analysis revealed that child studies often lack detail of sleep quantification. Only few studies reported on sleep efficiency, which may have resulted in insufficient sensitivity to detect an association with cognition or behavioral problems. Cognitive performance was positively related to sleep efficiency in studies using the more sensitive actigraphic methodology. Too few studies reported on sleep architecture parameters. This is a highly desirable aim for future studies. For instance, the amount of time spent in different sleep stages has previously been linked with different aspects of memory consolidation (Diekelmann & Born, 2010), and sleep spindles have been linked to intelligence (Fogel et al., 2007; Schabus et al., 2006). A further examination of sleep spindles and slow waves is needed to better understand the role of sleep in cognition and behavioral problems in children.

In conclusion, when possible and of interest, ambulatory polysomnography will allow for in-depth analyses on the role of sleep. High-density EEG is recommended to explore the role of topographical immaturity of the anterior-to-posterior gradient of slow-wave expression over the cortex in children (Kurth et al., 2010). Since this set-up is not ubiquitous, a guideline for a minimal data set would include at least a week of actigraphy to estimate the average sleep time and to further explore variability measures, such as sleep schedules (Van Someren, 2007).

With respect to *cognition assessment methodology*, simple sustained attention tasks have been used relatively little in studies on the effect of sleep duration in children, whereas studies in adults suggest that they may tap the most fundamental process affected by sleep deprivation (Lim & Dinges, 2008, 2010). Optimized (Basner & Dinges, 2011) shorter tasks of simple sustained attention have become available and are recommended for use in child studies. Indeed, one study in children that applied partial sleep restriction to 5 hr for 1 night and used a most simple sustained attention task (PVT) lasting 5 min indeed did find a significant deterioration of performance (Peters et al., 2009). Thus, it may be advisable for future studies on cognitive effects of sleep in children to include a brief version of a most simple sustained attention task (like the PVT) rather than tasks of longer duration, more salient stimuli, or more response options.

With respect to the choice of *experimental versus observational protocols*, imposed sleep restriction is required to investigate directionality of the associations of sleep with cognition and behavioral problems. Large sample sizes will be required to evaluate differential effects of varying durations of allowed sleep and of different numbers of nights. Thus far, only one study has examined total sleep deprivation in children (Carskadon et al., 1981b). Based on adult studies, it makes sense to hypothesize that shorter sleep causes daytime cognitive and behavioral deficits, rather than vice versa. In an attempt to investigate this direction of causation, we performed a moderator analysis to compare the effects of varying amounts of sleep restriction. The results suggest that protocols applying multiple nights of sleep restriction are more likely to detect effects. However, because sleep restriction studies are presumed to strongly affect the child's well-being, the possibility to perform such studies is often discarded a priori by researchers, child care professionals, and parents for ethical and practical reasons, including the required involvement of parents or caregivers.

With respect to *inclusion criteria, analyses, and reporting*, investigations into the relation of sleep with cognition and behavioral problems would additionally benefit greatly from further examinations of gender differences, from standard reporting methods on socioeconomic status and ethnicity, and from a more in-depth investigation of developmental changes (i.e., aging from infancy to adolescence). As there may be developmental changes within the relation of sleep with cognition and behavioral problems, narrow age ranges may highlight effects that are concealed in studies incorporating a wide range of ages. In the current meta-analysis, we focused on the age range of 5 to 12 years to represent a relatively homogeneous group of children who had acquired the monophasic sleep pattern but had not yet experienced the typical delay in the circadian timing system that characterizes the onset of puberty (Carskadon, Acebo, & Jenni, 2004). However, to further explore how the relation of sleep to cognition and behavioral problems develops between the childhood years and the adult years, a future meta-analysis on a homogeneous group of adolescents would be invaluable. Equally important are longitudinal studies, of which the present meta-analysis found only nine reports. The suggestion that childhood sleep problems may result in cognitive impairments or behavioral problems at later ages (Touchette et al., 2007) warrants closer attention to the causes and consequences of sleep restriction in children. Longitudinal studies can provide essential information on how neuronal maturation and the relation of sleep to cognition and behavioral problems interact.

## Key Questions

The opportunities for methodological optimization of research may aid in addressing outstanding key questions on the relation of sleep to cognition and behavioral problems in childhood, and how these relations change with development:

• Which aspects of sleep in particular relate to cognition and behavioral problems in children—which sleep stages and which electrophysiological sleep features (e.g., sleep spindles, slow waves, or EEG time-frequency power bands)? More in-depth recordings of sleep are essential, if possible obtained with ambulatory high-density EEG.

• Which specific aspects of cognition and behavioral problems relate to sleep? It would be advantageous to use validated tasks that tap into cognitive function in greater detail, for instance, by using different types of memory tasks.

• What is the direction of causation in the relation of sleep to cognition and behavioral problems? And how exactly can we best describe the relation? For instance, adult studies have suggested an inverted U-shape function between sleep duration and life outcomes.

• Which neuronal structures in particular relate to the relation of sleep to cognition and behavioral problems in childhood? Modern brain imaging techniques—such as EEG, fMRI, and combinations thereof—will allow insight into the underlying neuronal structures and connectivity patterns.

• How does brain development parallel the development of sleep, cognition, and behavioral problems? Longitudinal study designs in which participants are followed up across the crucial childhood and adolescent years could reveal if and how they co-develop.

## Conclusions

The current meta-analysis provides the first comprehensive investigation of a century of studies on the relation of sleep to cognitive functioning and behavioral problems in school-age children and provides evidence for a small but significant association. In particular, shorter sleep is associated with worse executive and multiple-domain cognitive functioning and worse school performance. Shorter sleep duration also relates to more internalizing and more externalizing behavioral problems. Intelligence test scores appear a consistent trait not necessarily affected by variations in sleep duration. In marked contrast to adults, children's memory processes and the ability to sustain attention seem less dependent on sleep. We discussed how these surprisingly different results might be due to both methodological issues—differences between experimental and observational studies, different ways in which tasks are performed, floor/ceiling effects, participant selection criteria, and environment or context—and/or developmental neurobiological differences. In particular, the structure and function of a child's brain may impose limitations: processes (a) may not be able to benefit fully from sleep, (2) may not yet suffer fully from sleep restriction, and (c) may not yet be restricted to benefitting from sleep only but also may benefit equally well from wakefulness. We provide the following testable hypotheses:

• In childhood, the sleep dependency of sustained attention correlates with the maturation of anterior–posterior connectivity.

• In childhood, consolidation of synaptic-scaling-dependent learning over a period of wakefulness is better than in adults.

• In childhood, consolidation of trace reactivation-dependent learning over a period of sleep is worse than in adults.

• In childhood, the sleep dependency of memory consolidation correlates with the maturation of both anterior–posterior connectivity and hippocampus–medial prefrontal cortex connectivity.

• The neurobiological substrate of implicit learning matures earlier than the neurobiological substrate of explicit learning. Therefore, in children, memory consolidation over a period of time—whether or not including sleep—may be more easily detected in implicit memory paradigms.

It is expected that future studies that apply the here-provided methodological guidelines for improved sensitivity to examine the specifics of sleep, cognition, and behavioral problems in children will reveal fundamental insights that are highly relevant for our understanding of the brain, sleep, and childhood development. The suggestion that insufficient sleep in children affects cognitive performance and aggravates behavioral problems is of particular practical relevance, given the increasing tendency towards curtailment of their sleep.

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# Appendix

## Studies Used in the Meta-Analysis That Do Not Appear in Text

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