



Sleep and Memory in Children

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Abstract

Purpose of Review This short review article aims at emphasizing interesting and important new insights about investigating sleep and memory in children aged between 6 and 13 years (middle childhood).

Recent Findings That sleep in comparison to wakefulness benefits the consolidation of memories is well established—especially for the adult population. However, the underlying theoretical frameworks trying to explain the benefits of sleep for memory still strive for more substantiated findings including biological and physiological correlates.

Summary Based on the most recent literature about sleep-related memory consolidation and its physiological markers during middle childhood, this article provides a review and highlights recent updates in this field.

Keywords Middle childhood · Sleep · Memory consolidation · Sleep spindles · Slow oscillations

Introduction

Relationships between sleep and memory processes in the adult brain are nowadays well established. However, there are recent controversial discussions about the robustness of sleep-mediated memory benefits, which should be considered whenever investigating hypotheses in this research field. Cordi and Rasch [1••] argue that more attempts to replicate and meta-analytic approaches together with higher standards for reproducible science are critical to advance the field of sleep and memory. Besides this upcoming debate in the current adult sleep and memory literature, it still remains unclear, whether and how sleep plays a role in the development of memory in children. It has to be noted that it is important to explicitly differentiate between studies [1••] examining associations between sleep physiology and general cognitive abilities (e.g., intelligence, learning ability) indicating a trait from [2] studies comparing how sleep physiology affects overnight memory consolidation, indicating a state. It is well known that sleep is the brain's main activity during the early years: until

school age, a child has spent more time asleep than in social interactions, exploring the environment, eating, or any other waking activity [2, 3]. Further, it is well established that developing brains need a considerable amount of sleep each day and that sleep promotes neural plasticity and thereby memory processes—especially the consolidation of memories (i.e., a process that makes memories stronger and less vulnerable to interference). A meta-analysis by Astill and colleagues [4] suggests that insufficient sleep in children (5–12 years) is associated with deficits in higher-order and complex cognitive functions like memory and an increase in behavioral problems. In a recent longitudinal study by Seegers and colleagues [5], parents ($N = 1192$) reported their children's nocturnal sleep duration annually from ages 2.5 to 10 years and it was found that short persistent sleep duration is associated with poor vocabulary performance in middle childhood. In the above studies, the fact that sleep quality and quantity are positively related to general cognitive abilities irrespective of whether learning occurred before sleep indicates the general—trait-like—nature of this association. Besides those studies examining associations between general cognitive ability and sleep physiology (trait), there is growing evidence that children like adults improve after periods of sleep (during the night but also during the day, i.e., after naps) on declarative memory tasks [6–11, 12••, 13••]. However, these reported sleep-related gains in memory performance are typically unrelated to any sleep parameters, as polysomnography was not recorded during children's sleep. To give a very recent example, Peiffer and colleagues [12••] trained children (7–12 years)

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and adults (20–30 years) in a declarative task, where they had to learn new associations between non-objects and their functions. Retrieval performance was tested during an immediate and a delayed retrieval session separated by either a retention interval containing sleep or wakefulness. Sleep led to stabilized memory retrieval performance only in children, not in adults, whereas no age-related difference was observed after a similar period of wakefulness. The authors concluded that this effect might be related to more abundant and deeper sleep during childhood. Another very interesting behavioral study by Prehn-Kirstensen and colleagues [14] showed that sleep prevents the forgetting of reward-associated memory representations. They found an indication that this effect is more pronounced in children than in adults. It has to be noted—like for other studies investigating sleep-related memory in children—that besides these very interesting behavioral effects, the authors of both publications did not record polysomnography during sleep and therefore could not quantify sleep on a physiological level, or search for potential relationships with overnight changes in memory performance. However, studies like these, comparing populations of adults with that of children, are rare but are critical to understanding whether and how these processes differ in adults as compared to children. In addition to studies reporting solely behavioral results, there is second category of empirical studies, which apply polysomnography in children. These studies mainly report results of a purely correlative nature, where sleep parameters are correlated with learning and memory performance to uncover the underlying neurophysiological correlates of the beneficial effect of sleep for memory consolidation. One very recent study [15••] addressed the association of sleep electroencephalography features with executive functions as well as planning and problem-solving skills by application of the “Tower of Hanoi” task in children aged between 9.5 and 12.8 years. Their results indicated a stronger performance improvement across wake in children with more stage N2 sleep and less slow-wave sleep. Stronger improvements across sleep were related to higher slow sleep spindle density and to reduced fast sleep spindle density, duration, and power. However, as noted by the authors themselves, it remains unknown whether these findings are specific to the acquisition of planning and problem-solving skills (state), or that they apply to a more general cognitive factor (trait). Furthermore, there is an increasing number of a third category of studies that actively manipulate sleep in order to observe neurocognitive and memory consequences [16–18, 19••]. These studies applied different schedules of sleep restriction and their findings in general suggest that sleep quality and quantity are consistently related to children’s and adolescent’s memory performance and that sleep deficits are often associated with poor declarative and procedural memory consolidation. Randazzo and colleagues

[16] randomly assigned children between the ages of 10 and 14 years to either an experimental sleep restriction group (5 h time in bed) or a control group (11 h time in bed). Higher cognitive functions in children, such as verbal creativity and abstract thinking, were impaired already after a single night of restricted sleep. More recently, the authors of the very elegant “Need for Sleep Study” [17, 19••] implemented a parallel-group design where 56 adolescents (15–19 years) were randomly assigned to a week of either 5 or 9 h of time in bed for sleep each night as part of a 14-day protocol conducted at a boarding school in Singapore.

The protocol consisted of three nights of baseline sleep, seven nights of sleep manipulation, and three nights of recovery sleep. Memory performance was assessed by a declarative learning task (40 word pairs). Besides manipulating time in bed, the authors further investigated different learning strategies: word pairs were either presented over four consecutive days (i.e., spaced items), or all at once during one single study session (i.e., massed items), with total study time kept constant across conditions. Recall performance was examined 0 h, 24 h, and 120 h after all items were studied. In general, recall of massed items was impaired by a greater amount in adolescents exposed to sleep restriction. In contrast, cued recall performance on spaced items was similar between sleep groups. These findings demonstrate the importance of combining good study strategies as well as good sleep habits to optimize memory outcomes. A further way to experimentally manipulate memory consolidation during sleep is called “targeted memory reactivation,” which has been described, e.g., by Rasch and colleagues [20] (2007) in adults. They found that presenting odor cues during learning of object locations in a declarative task (2D object-location) and re-presenting the same odor cues during slow wave sleep improves memory consolidation. These findings were recently replicated in a field study in a regular school setting investigating children aged between 10 and 11 years [21••]. Their results replicate previous findings in adults and show that the odor effect also works outside the laboratory settings. However, they did not monitor sleep objectively, but continuous cueing at night obviously produces similar effect sizes as in the study of Rasch and colleagues [20] with selective cueing in specific sleep stages. Taken together, there is an increasing body of evidence, investigating sleep-related memory consolidation during childhood, however, still strikingly little is known about the specific functions of sleep physiology for the quality and quantity of memory during childhood, which requires more of the abovementioned third category of studies, experimentally manipulating sleep and memory consolidation as well as longitudinal approaches to investigating developmental changes of these relationships.

Sleep in Children

Starting in the utero and continuing through childhood but also during adolescence and adulthood, environmental and social activities control the timing and duration of sleep (and wakefulness), which underlies our circadian rhythm of approximately 24 h. Timing of light exposure controls the timing of melatonin secretion and thereby the timing of sleep. Awakening time, timing, and brightness of artificial lighting after sunset and before sunrise, exercise, feeding schedule, and social interactions influence the child's timing of sleep propensity [22]. Regarding the sleep architecture of children, we know that children show distinctly different sleep patterns than adults: total sleep time, sleep efficiency, NREM3, and also REM sleep are higher during childhood, while percentage of NREM2 increases with age. Furthermore, REM latency is negatively correlated with age [23]. Besides these changes at the macroscopic level of sleep architecture, there are further differences at the level of cortical oscillations unique to the sleeping brain.

First, children compared to adults show an increase in EEG delta (0.5–4 Hz) and theta (4–8 Hz) activity [24–26] and second, the morphology of slow oscillation (SO) as well as sleep spindles changes considerably throughout development. Slow wave activity becomes most pronounced during adolescence where about 40% of NREM3 sleep is lost. According to our own data [27••] and also others [24, 25, 28–30], this prominent age-dependent change in slow wave activity is reflected in declines in power, amplitude, and steepness of slope. Interestingly, sleep slow waves are traveling waves and are thus a marker for brain connectivity. Schoch et al. [31] very recently investigated across-night dynamics of traveling slow waves during sleep in children from preschool age (2–5 years) to young adolescence (9–17 years). By using high-density EEG during sleep, they elegantly demonstrated that brain connectivity undergoes across-night dynamics specific to maturational periods. SO propagation distance decreased across a night of sleep, which was dependent on age and most prevalent in preschool children. The authors propose that these changes represent important milestones in maturational brain processes and that slow waves are therefore important markers for neurodevelopment, directly involved in human brain development processes (for a review see [32••]). Besides SO, it is well known that sleep spindle number, density, duration, intra-spindle frequency, and local distribution change with age [23, 29, 33–41]. Spindle oscillations are a prominent feature of NREM2 sleep, consisting of 7–14 Hz (0.5–3 s) waxing and waning field potentials. They are electrographic landmarks for the transition from wake to sleep and they are associated with loss of perceptual awareness. There is profound knowledge that they are generated by thalamic and cortico-thalamic networks, superimposed to delta activity or temporally locked to a K-complex (for review [42–44]). Based on

frequency and topography, sleep spindles can be distinguished in frontal slow spindles (11–13 Hz) and centro-parietal fast spindles (13–15 Hz) with presumed distinct underlying generators [45–47]. Both spindle types seem to share common thalamic activation; however, slow spindles are associated with activation in the superior frontal gyrus and sources in the frontal cortex, while fast spindles appeared to originate in the precuneus with mesial frontal, sensorimotor area, and hippocampal activations [45, 47]. The notion of two different sleep spindle types is already corroborated by their developmental trajectory. In an early cross-sectional study [48] (age range 4–24 years), frontal spindle frequency was shown to increase rapidly during early adolescence with an abrupt decrease in spectral power. In contrast, centro-parietal spindles showed a linear frequency increase and marginal power changes across adolescence.

A more recent longitudinal study by Campbell and Feinberg [49] supports these findings. The spectral peak of sigma frequency (11–15 Hz, i.e., the frequency band corresponding to sleep spindles) has been found to shift linearly from childhood to adolescence (6–18 years of age), indicating spindle frequency acceleration. Whereas the lower sigma frequency power (11–12.8 Hz) rapidly declined from 12 years onwards, higher sigma frequency power (13.4–14.4 Hz) steadily increased. This indicates different developmental trajectories of slow and fast spindles. Further cross-sectional studies showed that spindle density (number of sleep spindles per time epoch) increased and reached a maximum at 16 years of age but subsequently decreased from early adulthood (20–69 years of age) onwards [34, 35]. Furthermore, not only the independent development of the morphology of these two most prominent sleep oscillations but also the coupling of these oscillatory hallmarks of sleep changes considerably throughout maturation. Using a longitudinal study design spanning from childhood (9.5 ± 0.8 years) to adolescence (16 ± 0.9 years), we found that SO-spindle coupling strength increases during maturation and most fascinating, this increase indicated enhanced memory consolidation from childhood to adolescence [27••]. Taken together, there is accumulating knowledge about the physiology of sleep also during childhood; however, there are still some significant ingredients missing before we can identify the developmental role and function of sleep for memory consolidation and general memory performance especially in children.

Sleep-Dependent Memory Consolidation

Since the pioneering work by Jenkins and Dallenbach [50] who claimed that recall in humans improved after an intervening night of sleep, a growing body of experimental evidence has shown the beneficial influence of sleep on memory

processes (for review, see [51]). Similar to human sleep, which is composed of NREM and REM sleep, human memory is also not a homogenous phenomenon. According to Squire [52], long-term memories belong to multiple memory systems, categorized in two main types: declarative, explicit memories, and non-declarative, implicit memories. Whereas declarative memory systems (i.e., accessible to conscious awareness) include episodic (personal events, e.g., “My cat Nelly likes sausages”) and semantic (general facts, e.g., “Paris is the capital city of France”) memories, non-declarative or implicit memory (without phenomenal awareness) contains procedural knowledge, motor learning, priming, classical conditioning, and non-associative learning (i.e., walking, speech production, playground skills like climbing). Brain structures like the hippocampus and prefrontal areas are responsible for forming and storing declarative memories, while for building long-term implicit motor memories, the prefrontal cortex and also basal ganglia and the basal-ganglia-thalamic network are required. Memory formation in general is established in three steps: (i) encoding, (ii) consolidation, and (iii) retrieval. After encoding, newly acquired memories are usually very fragile and prone to disruptions induced by the learning of other, especially interfering, information [53, 54].

Hence, in order to be able to accumulate knowledge (i.e., to constantly encode new information but simultaneously retain old memories), a post-learning process of memory stabilization or consolidation is required. While the acquisition and retrieval of new information occur mainly and most efficiently during wakefulness, there is ample evidence that memory consolidation (i.e., reactivation and redistribution of newly encoded memory from the temporary into the long-term store) primarily and most efficiently takes place during sleep, a period when information flow into the brain is strongly reduced (for review, see [55]). Newly acquired information is initially stored within a memory buffer, the hippocampus. During sleep, these memories become repeatedly reactivated and thereby gradually transferred into long-lasting memory networks. Specific networks are located within the neocortex, with the temporal dynamics of these reactivation phenomena being orchestrated by a fine-tuned interplay between fast field oscillations originating in hippocampal layers (i.e., sharp wave-ripple complexes [56, 57]), thalamo-cortically generated sleep spindles [45, 58], and cortical SOs (~0.75 Hz [59]). In recent decades, the vast number of studies supporting the beneficial role of sleep in memory has inspired several theories about the underlying mechanisms of sleep-associated memory consolidation. In early sleep and memory research, a main focus relied on “macroscopic” estimates of sleep, that is, the amount of NREM or REM sleep. Earlier experimental evidence suggested that NREM sleep and REM sleep differentially modulate the consolidation of declarative and non-declarative memories, respectively (i.e., the dual process

hypothesis [60–62]). Further, there are also studies that indicate that the ordered succession of NREM sleep and REM sleep is necessary for the consolidation of memory traces, irrespective of the memory system (i.e., the double step hypothesis [63–65]). Today, however, specific sleep features and mechanisms, that is, the “microscopic” estimates of sleep, are regarded as increasingly important for different types of offline memory (re-)processing. Nowadays, two sleep and memory consolidation models have received the most attention: (1) the active system consolidation hypothesis (ASCO; for review, see [66]) and (2) the synaptic homeostasis hypothesis (SHY; for review, see [67]). Whereas ASCO asserts that memory consolidation during sleep is based on an active process that relies on covert memory reactivations during sleep, SHY assumes the beneficial effect of sleep on memory to be a passive epiphenomenon of a global synaptic downscaling occurring during sleep. According to ASCO, SOs during NREM sleep are the key player, as they are suggested to drive and group hippocampal memory reactivations into optimal time frames for cortical plasticity. SOs are generated within cortical networks [68] and occur in human sleep electroencephalogram with a mean spectral peak frequency of 0.7–0.8 Hz [69]. Every SO consists of a hyperpolarized “down”-state associated with a decreased cortical activity and a depolarized “up”-state characterized by an increased cortical activity [70, 71].

In particular, the interaction of the three main rhythms of NREM sleep: (i) the thalamo-cortical spindles (7–14 Hz; 0.5–3 s), (ii) the hippocampal sharp wave ripples (100–250 Hz), and (iii) the cortical SOs (< 1 Hz) is thought to be critical for memory consolidation during sleep. Importantly, these three oscillations form a temporal hierarchy, where ripples and spindles are nested in SO peaks (especially in the depolarizing up-states characterized by increased cortical activity) with ripples also being locked to spindle troughs. This hierarchy likely constitutes an endogenous timing mechanism to ensure that the neocortical system is in an optimal state to consolidate new memories [72–75, 76•, 77–79]. In particular, the consolidation of declarative memory and also explicit and implicit motor sequence learning during sleep have been shown to rely on the hierarchical phase-locking of these rhythms (for reviews, see [55, 80]). Interestingly, both ASCO and SHY assign a key role to SOs regarding the beneficial effect of sleep on memory. However, SHY argues that slow oscillations are critical due to their involvement in synaptic downscaling and not based on an active role in reactivation. In short, SHY states the following: (i) learning occurs during wakefulness and is mainly accompanied by synaptic long-term potentiation. Hence, net synaptic strength is increased after a period of wakefulness; (ii) slow wave activity, which reflects the “energy” of the SO, changes as a function of the net synaptic weight that has accumulated during prior wakefulness [67, 81]. There is evidence that slow wave activity increases in

task-related cortical areas following learning [82–84], and decreases over the sensorimotor cortex following 12 h of daytime arm immobilization [85]; (iii) slow wave activity not only is a consequence of synaptic potentiation during wakefulness but also contributes to a generalized depression or downscaling of net synaptic strength during sleep. According to Tononi and Cirelli [86], downscaling reduces net synaptic weights back to a baseline level while preserving relative differences in connection strengths. It is proposed that synaptic downscaling might be promoted mainly by mechanisms involved in long-term depression and that this process is induced by the SO, possibly through the alternating sequence of cortical depolarization and hyperpolarization; (iv) synaptic downscaling is accompanied by several functional benefits, such as benefits for learning and memory. Given this knowledge, sleep seems to be a perfect candidate for shaping neuronal networks. More specifically, it is assumed that slow oscillations, sleep spindles, and sharp wave ripples are a functional correlate of the “quality” of thalamo- and/or hippocampo-cortical connectedness. In addition, these oscillations seem to operate and shape the interconnectivity between these brain areas and therefore seem to be biological markers indicating cognitive as well as memory performance because efficiently shaped thalamo- and/or hippocampo-cortical networks are of equal importance for both types of processing demands.

Sleep-Dependent Memory Consolidation in Children

As described earlier, the transition from childhood to adolescence is marked by considerable changes in sleep architecture, also especially affecting those sleep parameters involved in the SHY and ASCO model. Given the fact that children do not only have to memorize much more material but also sleep longer and more deeply than adults [23, 29], there is a strong expectation that sleep in children is important for memory consolidation. However, there is still a lack of research, investigating the effects of sleep on brain processes underlying the consolidation of memories in children, in spite of the close links between the development of sleep features (e.g., spindles or SO), brain maturation, and cognitive processes. An elegant study by Urbain et al. [87] recorded the brain activity of school-aged children (8.0–12.5 years) by magnetencephalography to explore the specific impact of diurnal sleep (90 min nap) on the neurophysiological processes during learning and consolidation of declarative memories (i.e., novel associations between unknown objects and their functions). Learning-dependent changes were observed within hippocampal and parahippocampal regions, followed by sleep-dependent changes in the prefrontal cortex, whereas no equivalent changes were observed after a similar period of wakefulness. Further, they found that learning-related

activity in (para)hippocampal regions was correlated with increased slow wave sleep activity during the post-training nap, which was specific to the newly learned representations. A further very recent study by Sulkamo and colleagues (2019) investigated the relationships between local (occurring in only one channel: Fp1, Fp2, C3, or C4), bilateral, and diffuse (occurring in all four channels) spindles and neurobehavioral performance (Wechsler Intelligence Scale for Children; Developmental Neuropsychological Assessment; CogniSped) in 17 healthy children (8.9–10.8 years). The main result was that local spindles, and also more widespread central spindles, seem to be involved in the cognitive processes. Furthermore, this study highlights the methodological importance of age-adjusting frequency limits when investigating sleep spindles. One of our own studies [41] applied a longitudinal approach (across 7 years) to explore whether developmental changes in sleep spindle density can explain individual differences in sleep-dependent memory consolidation and general cognitive abilities. Mature spindle topography developed between initial (8–11 years) and follow-up recordings (14–18 years). Very fascinating, an enhancement in fast spindle density was associated with sleep-dependent word pair memory consolidation. Furthermore, fast spindle development predicted the difference in memory consolidation between initial and follow-up recordings, whereas slow spindle development correlated with cognitive abilities. Besides declarative sleep-related memory consolidation, we recently investigated the impact of sleep on complex gross-motor adaptation (i.e., riding an inverse steering bicycle) in children aged between 11 and 14 years. We combined a between-subjects (wake vs. sleep retention interval group) and a within-subject (learning vs. control task) design and did not find evidence for sleep-dependent motor memory consolidation. However, NREM2 slow sleep spindle activity was found to be related to overnight gains in performance accuracy. Additionally, decreases in tonic REM sleep duration indicated higher overnight improvements in accuracy. Regarding speed, an increase in tonic REM duration was favorable for higher overnight gains in riding time. Thus, although not yet detectable on the behavioral level, sleep also plays a role in the adaptation of gross-motor memory in children. In general, previous research investigating sleep-related memory processes in children mainly focused on the individual development of sleep physiology patterns like SOs and sleep spindles across brain maturation. As described above, it was demonstrated that these cardinal sleep oscillations undergo a substantial evolution in their defining features such as amplitude, frequency, distribution, and occurrence [27••, 34, 40, 48, 49, 88]. Interestingly, very recent findings indicate that especially the precise temporal coordination of SOs and sleep spindles—so-called SO-spindle-coupling—might be most important, as is its deteriorating over the lifespan, which might further contribute to age-related memory decline [89–91]. Very recently, we could foster this knowledge by our own longitudinal data, where we

investigated the maturation of this SO-spindle coupling from childhood (8–11 years) to adolescence (14–18 years). We found a significant enhancement of this coupling pattern until adolescence and most interestingly, this increase indicated enhanced declarative memory formation (i.e., word-pair associates) from childhood to adolescence. Our results, therefore, provided the first evidence based on physiology that improved coordination between SOs and spindles represents a biological marker for the development of a sleep-dependent memory network (Hahn et al. 2020), which has been earlier been discussed mainly from a theoretical standpoint within the ASCO theory [51, 55, 75]).

Conclusion

The research field investigating the beneficial effects of sleep for memory formation is rapidly growing, however, studies in children are particularly still needed to unravel the picture of sleep-dependent memory consolidation during human maturation. Despite our own experience regarding how hard it is to get ethical approval and to recruit highly motivated and compliant children (and parents), the implementation of longitudinal approaches as well as study designs actively manipulating sleep (e.g., sleep restriction or targeted memory reactivation) and memory (e.g., different learning strategies) in order to observe neurocognitive and memory consequences of reduced sleep quality and quantity are highly recommended. Furthermore, some basic methodological aspects have to be stressed, whenever analyzing sleep oscillations especially in children. Regarding sleep spindles age-adjusted frequency boundaries should be utilized and it has to be carefully considered which specific spindle parameters are utilized and how they are defined (sleep spindle number, density, amplitude, length, power, activity, or spectral peak frequency). Additionally, the variation of sleep spindle features between individuals, like for example gender differences, hormonal effects, and circadian rhythm variations (i.e., chronotype) have to be taken into account.

The same is true for analyzing slow oscillations and the combination of both sleep parameters (e.g., slow oscillation—sleep spindle coupling). Besides the physiological measures, also the assessment of memory consolidation needs to be chosen very carefully as this definitely affects the outcome of studies investigating sleep-related memory consolidation. Taken into consideration these methodological aspects whenever planning a study in this field, new insights into the basics of sleep-related memory consolidation could be provided and the understanding of the functional significance of sleep for cognition during childhood would be fostered.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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