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Chapter 2

Sleep and Cognition in Developmental Age

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1. Introduction

Cognitive development is the construction of thought processes, including remembering, problem solving, and decision-making, from childhood to adulthood. Moreover, it refers to how a person perceives, thinks, and gains understanding of his or her world through the interaction of genetic and learned factors. Among the areas of cognitive development are information processing, intelligence, reasoning, language development, and memory.

Historically, the cognitive development of children has been studied in a variety of ways. The oldest is through the Intelligence Quotient (IQ) based on the concept of "mental age" according to which the scores of a child of average intelligence match his or her age, while a gifted child's performance is comparable to that of an older child, and a slow learner's scores are similar to those of a younger child. IQ tests are used worldwide, but they have come under increasing criticism for defining intelligence too narrowly and for being biased with regard to race and gender.

Therefore, the study and knowledge of the various exogenous/environmental factors that could influence the cognitive development could be considered mandatory for the comprehension of the childhood general developing.

2. Sleep and cognitive processes in children

In healthy children, disturbed sleep has been associated with behavioural impairments (e.g., hyperactivity, aggression, anxiety, etc.) [1-7] and reduced neurocognitive performance (e.g., lower IQ, impaired memory, reduced academic performance, reduced attentive ability, etc.) [1,2,6,8].

Among pediatric age, growing evidence suggests the role of sleep habits as disturbing factor for influencing the cognitive development. The sleep disturbance can impact cognition as shown by the negative effects of sleep breathing disorders both in adults [9] and children [10] and as pinpointed by studies on the interaction between specific sleep stages in declarative memory functioning [11] and learning disabilities [12].

Since 1980, [13]more studies have suggested that sleep is associated with IQ levels in children, although the underlying mechanism remains still unknown. Studies involving children and adults have identified a significant relationship between poor or insufficient sleep and decreased cognitive capacity [14-26]. Furthermore, longer habitual sleep duration in healthy school-aged participants has been associated with better performance on measures of perceptual reasoning and overall IQ [23]. These findings reveal an association between sleep duration and performance on IQ tests. Actually, the DSM-5 [27] highlighted the importance of general mental abilities and of the adaptive functioning, beyond the IQ scores for the assessment of individual cognition. In this light, the social and practical domains appear to be more relevant, although the role of sleep cannot be excluded by these aspects, as showed by reports among subjects affected by intellectual disabilities (28-32).

Several Authors have proposed that sleep spindles may physiologically underpin intelligence or high-level general mental ability [33-35]. Sleep spindles are a feature of (predominantly) stage 2 Non-Rapid Eye Movement (NREM) sleep, and are characterized by recurrent and brief bursts of spindle-like EEG activity.

Moreover, spindles may be classified as either slow (10–13 Hz) or fast (13–16 Hz), with different EEG scalp topographies [36], and are both co-active also with hemodynamic responses in different cortical regions [37] and playing a strong role in the reprocessing of previously encoded information [38].Moreover, Mednick et al in 2013 have been showed as the pharmacological increase induction in spindle occurrence tend to improve memory, providing even stronger support for a mechanistic role of spindles in memory consolidation [39]. Specifically, retention of verbal informations are related to spindles recorded over frontal brain regions [40], while parietal spindles correlate with spatial memory [41].

In general, in humans the maturation of cognitive skills seems to be linked to a subsequent period of slow-wave activity (SWA), that undergoes maturation in parallel with cortical morphology [42,43] and sleep spindles (10–14 Hz) directly involved in synaptic remodeling, leading to alterations in synaptic strength and synchronized neuronal firing [44-47]

On the other hand, sleep spindle frequency in healthy school-age children seems to be negatively associated with performance on the working memory and perceptual reasoning modules of the Wechsler Intelligence Scale for Children-IV (WISC-IV) [48]. Moreover, lower sleep spindle frequency may be associated with better performance on the Intelligence perceptual reasoning and working memory WISC-IV scales, although sleep spindle amplitude, duration and density could be not directly associated with performance on the IQ test. [48]

In general in paediatric age children, sleep disturbances have been often considered as the epiphenomena of an underlying maturational disorder leading to cognitive impairments. However, cortical maturation and sleep-dependent mechanisms of brain plasticity seem to

follow similar developmental trajectories, suggesting closer interactions between these two dynamic processes [42,43,49,50]. In this light, abnormal sleep activity in children might be a causal, or at least a contributing factor in cognitive and learning impairments [51,52].

Specifically, converging evidence demonstrated that sleep plays a critical role in the 'evolution' of memories [53]. In fact, once encoded, sleep-dependent memory processing can not only stabilize memories – a process classically referred to as memory consolidation – but can also enhance them and integrate them into existing memory networks, extracting key elements for retention, abstracting the gist from multi-item memories, discovering the rules governing such collections of item memories, and even modifying them in ways that facilitate the subsequent discovery of creative insights [53].

Conversely, in the declarative memory domain (i.e. the memory for facts and events, usually verbalizable and explicit), beneficial effects of post-learning sleep on performance have been highlighted using mostly verbal (word pairs) and visuospatial (e.g. memory for pictures or objects' location, virtual navigation) hippocampus-dependent learning tasks. For instance, cued recall of learned pairs of words was consistently shown better after post-learning sleep than after a similar period of time spent awake [54-61]. Additionally, sleep may help protecting recently learned memories against retroactive interference due to the acquisition of a novel and related verbal material [62,63]. Therefore, sleep would participate in memory consolidation processes more than by merely protecting novel memories from ongoing, non-specific daytime interferences and memory decay as previously advocated [64]. Likewise in the non-declarative or procedural memory domain, beneficial effects of post-learning sleep have been evidenced for the consolidation of novel skills and habits, although results are more controversial. Sleep-dependent improvements in visual discrimination skills have been consistently demonstrated using the texture discrimination task (TDT) [65-70]. Furthermore in this task, performance deteriorates over repeated practice sessions within a day reflecting the saturation of the underlying neural circuits, unless sleep is allowed between sessions [71].

In this light, performance stabilizes or even increases depending on the duration of the sleep episode and the availability of slow wave sleep (SWS) and rapid eye movement (REM) sleep [72].

A most characteristic electrophysiological feature of non-rapid eye movement (NREM) sleep is the slow oscillation, visible on scalp electroencephalography (EEG) as a biphasic wave of high amplitude and a fundamental frequency of around 1 Hz [73]. This slow oscillation is the result of the alternation of periods of extended synchronization and desynchronization of the membrane potentials of numerous cerebral cortical neurons [74].

During the hyperpolarized phase, often called "down state", neurons remain silent for up to a few hundred milliseconds. During the depolarized phase, also called "up state", neuronal spike activity takes place, often including burst firing [74]. The "up states" seem to be associated with complex and widespread neuronal network activity throughout the brain [75], including high-frequency oscillations. Especially these oscillations, and their coalescence with

slow oscillations, have been implicated in network communication and systems consolidation of memory traces [76-79].

During the up states of slow oscillations, newly encoded memory representations are thought to be reactivated and redistributed, enabling a shift from temporary storage to long-term storage. Crucial for the dynamic formation of neuronal ensembles and altering of the synaptic connections during the up state is the co-occurring thalamo-cortical and cortico-cortical neuronal activity in higher frequency bands, notably the 10–15 Hz sleep spindles [80] and the >30 Hz gamma oscillations [81-83].

Over past decades, it has been evidenced that sleep can contribute to the consolidation of declarative memories in children. How and whether sleep helps in consolidating verbal and non-verbal procedural skills in this population remains a matter of debate and deserves further investigations. Dedicated studies combining comprehensive behavioural measures, neurophysiological and/or neuroimaging recordings in healthy and pathological populations are crucially needed to unravel the mechanisms underlying the evolution of sleep-dependent memory consolidation processes during childhood. Moreover, we could speculate that neurophysiological and neuroimaging investigations may contribute to enlighten the pathophysiological associations linking abnormal sleep patterns, cognitive disturbances and impaired sleep-dependent plasticity processes throughout the developmental phase. These investigations should be conducted in parallel with the study of pathological conditions in which children present abnormal sleep patterns and cognitive deficits, such as, for a few instances, ADHD, specific language impairments and epileptic syndromes. In this framework, comparing the development of sleep-dependent plasticity markers [84] in children with or without cognitive disorders, and how this evolution interacts with cognitive functioning and/or cortical maturation, constitutes a promising field of research to understand the pathophysiological conditions subtending the long-term disruption of cerebral plasticity processes involved in memory consolidation during sleep [84].

Moreover, the well known relationship between sleep and cognition in all ages of life suggests a key role of sleep in cognitive impairment conditions such as mental retardation [85-89], borderline intellectual functioning [32], learning [12,90], memory [91,92] and executive functions disabilities [93-95]. The approaching to the intellectual disabilities could be difficult, particularly in developmental age. In this framework sleep neuropshysiology may help the knowledge and comprehension for the functional interrelationships between the cerebral areas.

In general, the decreasing of sleep efficiency and decreased REM ratio were reported as characteristic neurophysiological signs in several developmental disabilities like Down syndrome [96,97], autism [96], Angelman syndrome [98] and in ADHD [99].

Moreover, lower sleep efficiency, higher WASO, increases in NREM sleep EEG (relative) delta and region-dependent decreases in sigma/high frequency activities were reported in subjects with Asperger syndrome [100].

Finally, reduced total sleep time, decreased sleep efficiency percentage, higher WASO, increases in frontally measured NREM sleep EEG delta power and SWS time, as well as region-

dependent decreases in sigma power and reduced REM sleep percent were reported in Williams syndrome [101]. Thus, several papers are reporting similar sleep-EEG alterations in different conditions affecting intellectual functioning.

3. From childhood to adulthood: Differences and similarities in the developmental course

The differences between children and adults are legion, and how they approach and learn from new situations is clearly one of them. Purely psychological studies, ranging from the work of Piaget in the 1950s and 1960s to the ongoing work of Spelke and Carey [102], have focused on the developmental trajectory of learning capacities and the dependence of each incremental improvement on the ones preceding it. Other studies focused on the continuing development of the cerebral cortex as key to changes in learning style and intellectual development [103]. In their recent study, Wilhelm et al. suggest that at least some of the differences in how adults and children process newly acquired information result from age-dependent differences in the forms of sleep-dependent processing applied to such memories [104]. Specifically, their findings suggest that children, 8–11 years of age, show greater sleep-dependent extraction of explicit, or declarative, knowledge of the rules that govern an implicit procedural task than do adults, 18–35 years old [104].

In general, not every memory undergoes all of these forms of sleep-dependent processing, and the mechanisms that determine which ones are employed for a given memory remain poorly understood. [104].

A possible explanation of this age difference in declarative knowledge is found in the structure of children' sleep. Children not only obtained significantly more sleep than the adults (9.8 vs 6.5 hr), but spent more than twice as much of that time in deep, slow wave sleep (SWS; 39% vs 17%; 217 vs 64 min)). [105].

The suggestion that increased SWS in children might lead to better extraction or maintenance of declarative as opposed to non-declarative (e.g., procedural) knowledge has its counterpart in the suggestion found in a recent report [106] that further decreases in SWS with aging might underlie the difficulty to retain new declarative memories experienced by the elderly.

Even childhood naps may be part of this story. Among 15-month-old infants, only those who napped after a learning task retained knowledge of it the next morning [107]. Moreover, they suggested that the developmental changes in sleep architecture, with more naps, SWS, and REM sleep in children than adults, reflects parallel changes in how sleep guides the evolution of memories across the life cycle, in part enhancing explicit fact memory in children, but more abstract knowledge in adults. Perhaps sleep makes children smarter, but adults wiser [105].

The expression of slow waves undergoes remarkable changes during development, both with respect to their topographical distribution [43, 108-110], as well as with respect to their amplitude [111-113]. The amplitude of slow oscillations increases during childhood to peak shortly before puberty [112]. Conversely, a steep drop occurs during adolescence, decelerating

at the age of about 17 years, after which the amplitude declines only slowly [111]. The amplitude of slow oscillations reflects the degree of synchronization by which cortical neurons switch between up and down states [88]. Although receiving much less attention, the capacity of a densely connected neuronal network to synchronize its activity may not only be reflected in the amplitude of slow oscillations, but might as well lead to more pronounced oscillations in frequency bands other than the 0.5–4 Hz range. Indeed, power in the theta (4–8 Hz) range declines across puberty and early adolescence [113]. Gaudreau et al. [114] investigated NREM sleep EEG power in a wider range of frequency bands across the age range of 6 to 60 years. They report a much higher absolute power of theta (4.0–7.75 Hz), alpha (8.0–12.0 Hz) and beta (15.25–31.0 Hz) in the group of children in the range of 6 to10 years, as compared to the groups of adolescents (range 14 to 16 years), young adults (range 19 to 29 years) and middle aged adults (range 36 to 60 years). The largest values for spindle-range power (12.25–15.0 Hz) were found in the adolescent group, suggestive of an inverted-U shape peaking somewhere between the age of about 10 years and late adolescence. Jenni and Carskadon [115] investigated developmental changes across the 0.6 to 25 Hz NREM-sleep power spectrum and found that children aged 9.6–12.9 years, as compared to children aged 11.8–15.9 years, had significantly higher absolute power not only in the low frequencies up to about 7 Hz, but also in the 12–13 Hz sigma range and 16–17 Hz low beta range. Recently, both Tarokh et al. [109, 110,116] and Baker et al. [117] applied within-subject follow-up design rather than the above-mentioned cross-sectional approaches, to confirm that changes in the sleep EEG across adolescence were not restricted to the lower frequency bands, neither to NREM sleep only. Across adolescence, the sleep EEG power decreases over a wide range of frequencies, up to the beta range for at least some derivations. In summary, the above mentioned developmental studies suggest that a wide range of cortical oscillations measured in the scalp EEG show their maximal signal to-noise ratio in late childhood, around the age of 11, where the signal of interest is the amplitude of the oscillations and the noise reflects the noise floor of scalp EEG assessment [118]. In contrast the gamma power increased on the rising slope and positive peak of the slow wave, with strongly suppression of both gamma and spindle activities during the negative peak, independently by external stiulation (i.e. acoustic) [118]

Recently, the topographic distribution of slow wave activity (SWA; EEG power between 0.75 and 4.5 Hz) during non-rapid eye movement (NREM) sleep was proposed to parallel cortical maturation from childhood through adolescence [43]. High density sleep EEG recordings in children and adolescents between 2 and 20 years of age showed that SWA exhibits a regional, age specific predominance with a developmental shift from occipital to frontal regions reaching frontal derivations only during adolescence. Strikingly, the local SWA maxima paralleled the time course of cortical gray matter [119, 120] and behavioural maturation [121] indicating that SWA may be a marker of brain maturation. This interpretation seems to be in line with the increasing number of reports showing a direct relationship between sleep slow waves and plastic cortical processes [122, 123]. More specifically, it has been hypothesized that wakefulness is associated with a significant increasing in synaptic strength, which is homeo-statically rebalanced during sleep. This hypothesis was confirmed in various species examin-ing markers of synaptic strength. A close relationship between SWA and cortical synapses has been proposed early on [124]. Although direct evidence is lacking, recent findings from animal

studies or humans using current in vivo measures for cortical structure and activity support the suggestion that synaptic strength is reflected in deep sleep slow waves [125]. Several studies have shown that slow wave characteristics (SWA, topography, slope, amplitude) are closely related to maturational alterations in the cortex [42,43,49,50]. Moreover, slow waves represent synchronized activity among cortical neurons, as shown by multiunit recordings in the rat [123]. Thus, neurons show synchronized activity, the larger is the amplitude of slow waves displayed by this network. Increased synchronization is achieved by stronger synaptic connections and/or a denser network (i.e. more connections) [125].

In general it has been stated that the SWA is related to cortical plasticity [123, 126-130] (e.g., a change in strength and/or number of synapses) not only occurs because of learning processes, but also in the course of brain maturation. In fact, it was shown that SWA is not equally distributed across the scalp in children and adolescents, but exhibits local age-specific maxima [43]. Furthermore, the location of maximal SWA seems to parallel the time course of cortical maturation along the posteroanterior axis [131]. Thus, the topography of SWA may reflect cortical plasticity during development [125].

Regarding the effect of sleep deprivation, children demonstrate difficult behaviors when sleep deprived that can be stressful and impact quality of life for the entire family. Connecting sleep problems with daytime behavioral challenges may not be intuitive to parents. Adults manifest different symptoms when sleep deprived such as daytime sleepiness, psychomotor slowing and impairments in cognitive processing and memory [132]. In comparison, sleep deprivation in children is more likely to be associated with a range of emotional/behavioral disturbances, including problematic behaviors [133], attention problems [134-136], anxiety/depression [137], and hyperactivity [138, 139]. Brain maturation is a complex process [140] that begins prenatally with neural proliferation and migration and synapse formation continuing till two years of age. Myelination is an important process that begins prenatally as well but continues into adolescence with different systems myelinating at different times. The determinants of neurodevelopment and behavior rely on complex neural circuits that connect neural substrates to serve a specific function. The development of these neural circuits is still a mystery and influenced by genetic, sociocultural, medical and environmental factors [141]. The neuroanatomic substrates involved in neurobehavioral functioning span cortical, subcortical and brainstem regions and formulate complex networks which include the prefrontal cortex, amygdale and striatum. Executive functioning is highly localized to the prefrontal cortex. The amygdala is of great importance to emotional reactivity and affect and striatum to reward seeking behavior. Neuroimaging techniques reveal complex patterns of neuroanatomical functioning during specific sleep stages.

During NREM slow wave sleep, the brainstem, thalamus, basal ganglia, and prefrontal and temporal lobe regions all appear to undergo reduced activity [142]. In REM sleep, significant levels of activity are reported in the pontine tegmentum, thalamic nuclei, occipital cortex, mediobasal prefrontal lobes together with affect related regions including the amygdala, hippocampus, and anterior cingulate cortex [142]. The prefrontal cortex is relatively inactive all through sleep in contrast to its high activity during waking states [143]. This inactivity is reflected by the high voltage and slow brain wave oscillations in NREM sleep in the frontal

lobes, relative to other cortical regions, suggesting that the thalamocortical input is disabled and a lower level of metabolism in the frontal lobes during NREM sleep stages is present [144]. Several investigators have suggested that sleep is particularly important for restoring prefrontal cortical activity [145-148] however, this restorative process remains poorly understood. Neuroimaging studies showed profound effects of one night's total sleep deprivation on the blood flow to prefrontal areas which correspond to the deteriorations in daytime prefrontal task performance [149,150].

On the other hand, sleep deprivation also impacts neural circuitry underlying regulation of emotions, impulsivity and reward seeking behavior. Sleep deprived adult volunteers viewing emotional images have increased activation of the amygdala on functional neuroimaging yet weaker connection between the prefrontal cortex and the amygdala [151]. This scenario allows for uncontrolled, increased emotional response. Likewise, neurocognitive functions that involve the striatum and basal ganglia such as risk avoidance and responsiveness to rewards are also impacted by sleep deprivation. For instance, sleep deprived adults take greater risks and are less concerned about consequences of their behavior [152]. Such findings have also been noted in adolescents aged 11–13 years using functional magnetic resonance imaging (fMRI) and a guessing task with monetary rewards [153]. During reward anticipation, less activation in the caudate nucleus (part of the ventral striatum) was associated with reduced sleep time, later sleep onset time, and lower self-reported sleep quality. During reward outcome, less caudate activation was seen with later sleep onset time, earlier sleep offset time, and lower sleep quality. This findings suggested that sleep deprivation could contribute to low reactivity in reward-related brain areas in adolescents and may lead to compensatory increases in reward-driven behavior. Such findings have significant public health implications when one considers that reward seeking behaviors are associated with depressive symptoms, sensation seeking, and substance abuse in adolescents [137, 154].

4. Conclusions

The relationship between sleep and cognition is intriguing and not yet well understood.

Investigation into sleep habits in the young and the neurophysiological study of sleep (e.g. sleep macrostructure, microstructure, power spectra and CAP) may be considered as mandatory in the future for a better knowledge and comprehension of cognition development.

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