Sleep restores daytime deficits in procedural memory in children with attention-deficit/hyperactivity disorder

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A B S T R A C T

Sleep supports the consolidation of declarative and procedural memory. While prefrontal cortex (PFC) activity supports the consolidation of declarative memory during sleep, opposite effects of PFC activity are reported with respect to the consolidation of procedural memory during sleep. Patients with attention-deficit/hyperactivity disorder (ADHD) are characterised by a prefrontal hypoactivity. Therefore, we hypothesised that children with ADHD benefit from sleep with respect to procedural memory more than healthy children. Sixteen children with ADHD and 16 healthy controls (aged 9–12) participated in this study. A modification of the serial-reaction-time task was conducted. In the sleep condition, learning took place in the evening and retrieval after a night of sleep, whereas in the wake condition learning took place in the morning and retrieval in the evening without sleep. Children with ADHD showed an improvement in motor skills after sleep compared to the wake condition. Sleep-associated gain in reaction times was positively correlated with the amount of sleep stage 4 and REM-density in ADHD. As expected, sleep did not benefit motor performance in the group of healthy children. These data suggest that sleep in ADHD normalizes deficits in procedural memory observed during daytime. It is discussed whether in patients with ADHD attenuated prefrontal control enables sleep-dependent gains in motor skills by reducing the competitive interference between explicit and implicit components within a motor task.

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1. Introduction

Attention-deficit/hyperactivity disorder (ADHD) is one of the most frequent psychiatric disorders in childhood and adolescence. It is characterised by patterns of inattention, impulsiveness, and hyperactivity and symptoms often persist into adulthood (American Psychiatric Association, 2000; Huber, Ghilardi, Massimini, & Tononi, 2004). ADHD symptoms are caused by inherent alterations in structure and function of frontostriatal brain regions (Brennan & Arnsten, 2008; Castellanos & Tannock, 2002; Zang et al., 2005). Patients with ADHD display deficits in executive functions as well as in declarative and procedural memory performance (Adi-Japha, Fox, & Karni, 2011; Bailey, Lorch, Milich, & Charnigo, 2009; Dige, Maahr, & Backenroth-Ohsako, 2010; Holdnack, Moberg, Arnold, Gur, & Gur, 1995; Krauel et al., 2007; Lorch, Milich, Flake, Ohlendorf, & Little, 2010; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). However, poor sleep quality can also lead to ADHD-like
symptoms of inattention or hyperactivity in healthy children (Gruber et al., 2007, 2011; Paavonen et al., 2009; Steenari et al., 2003) and these symptoms were suggested to be related to temporary dysfunctions of frontostriatal loops (Killgore, 2010). While reports of altered sleep architecture in ADHD are inconsistent, a reduced subjective sleep quality has been frequently observed in ADHD (Cortese, Faraone, Konofal, & Lecendreux, 2009; Konofal, Lecendreux, & Cortese, 2010; Owens, 2009). Since low sleep quality can hamper “online” memory performance (such as encoding or retrieval) (Robillard, Prince, Boissionneault, Filipini, & Carrier, 2011), one might expect that memory performance after sleep is deteriorated in ADHD.

Sleep, however, serves to restoration or daytime alertness (Siegel, 2005) thereby enabling optimal preconditions for processes of encoding and retrieval. Sleep remarkably fosters the “offline” consolidation of newly acquired memories (Diekelmann & Born, 2010; Stickgold, 2005; Walker, 2008). The prefrontal cortex (PFC) plays a critical role in the “offline” consolidation during sleep of both declarative and procedural memories, but in various ways: On the one hand, prefrontal generated slow oscillations during sleep foster the consolidation of declarative memory (Marshall, Helgdöttir, Mölle, & Born, 2006; Marshall, Mölle, Hallschmid, & Born, 2004). Recently we reported a reduced consolidation of declarative memory in young patients with ADHD (Prehn-Kristensen et al., 2011a). Noteworthy, this deficit could not be ascribed to insufficient sleep quality or changes in sleep architecture. Rather, it was proposed to be related to a reduced function of prefrontal mediated slow oscillations during sleep. On the other hand, reduced PFC activity coincided with the gain in procedural memories after sleep in healthy adults (Fischer, Nitschke, Melchert, Erdmann, & Born, 2005), and adult patients with lesions in the PFC displayed a better procedural memory performance after sleep compared to healthy controls (Beldarrain, Astorgano, Gonzalez, & Garcia-Monco, 2008). The latter findings suggest that patients with ADHD, who are characterised by a hypofrontality (Rubia et al., 1999; Zang et al., 2007, 2005), should benefit from sleep with respect to motor performance.

Based on the results that reduced PFC activity is associated with gaining in motor skill after sleep, and that ADHD is linked to a PFC hypocoactivity, we hypothesised that, in children with ADHD, sleep enhances memory consolidation of motor skills. We assume that healthy children do not show any sleep-associated gain in motor performance, as we reported in former studies (Fischer, Wilhelm, & Born, 2007; Prehn-Kristensen et al., 2009; Wilhelm, Diekelmann, & Born, 2008). In ADHD, sleep quality seems to be more affected by sleep laboratory environment than in healthy controls (Philipsen et al., 2005; Prihodova et al., 2010). Therefore, we conducted sleep measurements at children’s home.

2. Methods

2.1. Participants

ADHD patients and controls were recruited by announcements in our outpatient clinic and in local schools. All participants and their parents were interviewed with a German translation of the Revised Schedule for Affective Disorders and Schizophrenia for School-Age Children: Present and Lifetime Version (K-SADS-PL; Delmo et al., 2000; Kaufman et al., 1997). A standard parent-reported questionnaire (the Child Behaviour Checklist, CBCL; Achenbach, 1991) was filled out by parents in order to assess psychiatric symptoms of their children. ADHD patients were excluded, if they displayed any comorbidity apart from oppositional defiant disorder (ODD). Controls were excluded if they displayed any psychiatric abnormalities. Further exclusion criteria for all participants were below-average intelligence quotient (IQ < 85) as measured by Culture Fair Intelligence Test Revised Vision (CFT-R; Weiß, 2006), significant memory impairment as measured by Diagnosticum für Cerebralschädigung (DCS, cut-off score: < 16th percentile of the reference sample; Lamberti & Weidlich, 1999), or sleep-related disorders as assessed with an adaptation of the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Due to these criteria, 12 children with ADHD and 4 controls were excluded after the diagnostic session. Another four children and one control child dropped out due to motivational reasons.

The remaining 16 ADHD patients (aged 9–12) and 16 healthy controls (aged 9–12) had at least an average intelligence quotient and no significant memory impairments. Children with ADHD and typically developing controls had normal or corrected-to-normal vision and did not differ significantly in age (p = .227). IQ (p = .459) or DCS score (p = .8, Table 1). All children in the experimental condition met the criteria for ADHD according to DSM IV TR (American Psychiatric Association, 2000), eight suffered from inattentive type and another eight from combined type. Four patients with ADHD additionally exhibited an oppositional defiant disorder (ODD). Twelve patients took methylphenidate but discontinued medication 48 h (approximately twelve half-lives) prior to each experimental condition. Controls did not exhibit any psychiatric symptoms. Parents of the ADHD group reported more attention problems in their children than parents of healthy controls (p < .001, Table 1).

All participants and their parents gave written informed consent, and participants were reimbursed with a voucher for their participation. The study was approved by the ethics committee of the medical faculty of the University of Kiel and followed the ethical standards of the Helsinki Declaration.

2.2. Procedural memory task

The procedural memory task (i.e. button-box task) required the participants to press eight buttons affixed to a response box that were consecutively flashed up according to a repeating 8-elements sequence. Participants were told to use their non-dominant hand (handedness was measured according to Oldfield, 1971). Buttons were arranged in two rows, each consisting of four buttons (see Fig. 1). Participants were told to press the button which currently flashed up as fast as possible.
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