EDITORIAL

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Editorial: Time to Wake Up: Appreciating the Role of Sleep in Attention-Deficit/Hyperactivity Disorder

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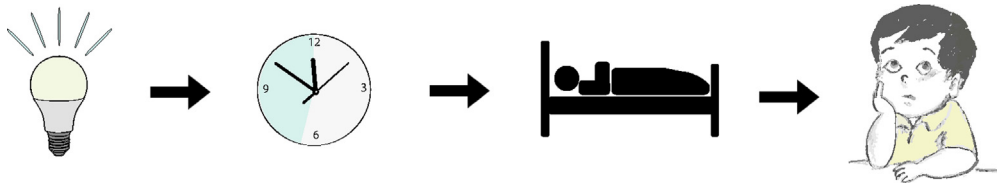
We spend approximately a third of our lives sleeping, and many *DSM-IV* and *DSM-5* disorders have sleep problems listed as diagnostic features and are often considered "comorbidities." However, very little is known about the role sleep problems play in the *etiology* of psychiatric disorders such as attention-deficit/hyperactivity disorder (ADHD).

To appreciate such a role, it is important to distinguish between "sleep deprivation"—a full night without sleep that clearly affects daily functioning—and "sleep restriction"—receiving less sleep than what is considered normal for a given age. When the sleep of healthy adults was experimentally *restricted* to 6 hours per night for 2 consecutive weeks, the adults' attention and concentration gradually decreased. Interestingly, when asked about their functioning after this period, they reported no complaints, although their objective attentional performance was at the same level as that of healthy adults who were fully *sleep deprived* for 2 nights.[1] Another study showed that even after subsequently sleeping 8 hours for 7 nights, attention was still not fully recovered from 5 days of sleep restriction to 4 hours per night,[2] suggesting the profound impact of restricting sleep. In other words, "sleeping-in on weekends" is not sufficient to fully recover from restricted sleep during the week. From this perspective, it is interesting to note that 21st century children sleep an average of 75 minutes less than they did a century ago.[3] Studies using electroencephalography have substantiated this trend by showing that, throughout the past decade, increasing signs of drowsiness are exhibited in healthy children's brain activity.[4] When considering these observations, it becomes evident that sleep indeed plays a fundamental role in daily functioning, and that it could play a role in the development of complaints that are observed in ADHD.

The article by Becker *et al.*[5] in this issue addresses the role of sleep restriction in ADHD symptomatology. We very much appreciate how they characterize the relation between sleep restriction and ADHD symptomatology in a more causal way than previous attempts have done in this population. Becker *et al.* subjected adolescents with ADHD to a crossover design, with a 1-week sleep-restriction phase, in which sleep was restricted to 6.5 hours per night, and a 1-week sleep-extension phase, in which sleep was extended to 9.5 hours per night. Their results associated sleep restriction with aggravated ADHD symptomatology, such as increased inattention, oppositional behavior, and sluggish cognitive tempo. Interestingly, the significant aggravation was found only for parent-rated inattention, but not for self-rated inattention or attentional performance on a continuous performance task. These findings are largely in line with the results reported by van Dongen *et al.*,[1] in which participants were unaware of cognitive deficits observed after 2 weeks of sleep restriction, explaining why no effects were reported by Becker *et al.* on *self-rated* inattention (ie, unawareness), but were found on *parent-rated* inattention. Furthermore, Becker *et al.* did not find an effect of sleep restriction on objective attentional performance (continuous performance task), which might be explained by their effective restriction of 1.6 hours for 5 consecutive days, which is a shorter and lesser restriction than induced experimentally by van Dongen *et al.* In addition, knowing that patients with ADHD could already have some sleep problems[6] compared with a healthy population, this population could be subject to ceiling effects with respect to the negative effect of sleep restriction on attention. In line with this, the average sleep duration in the stabilization phase in the study by Becker *et al.* was 6.5 hours, whereas 9 hours would be considered a "normal" sleep duration for this population. Despite the limited restriction in a population that already had a short sleep duration, strong effects on parent-rated inattention were found between sleep extension and sleep restriction.

Alternatively, experimentally inducing sleep restriction as Becker *et al.*[5] did also can be induced by an "unhealthy"

FIGURE 1 Pathway of LED Light Exposure



Note: A pathway is depicted in which LED light exposure positively influences the amount of sleep-onset delay, which in turn negatively influences the amount of received sleep, which ultimately leads to worsening of attention. Please note color figures are available online.

naturalistic environment. Recently, more and more research has focused on the impact of blue-light exposure in the evening. The human circadian system aligns mostly to the presence or absence of light. At the time when daylight intensity diminishes, and its color spectrum shifts from blue to red, our major circadian clock (suprachiasmatic nucleus) signals the pineal gland to produce melatonin, the "sleep hormone." However, when humans are artificially exposed to blue light during evening hours (ie, modern devices such as tablets, smartphones, and LED lights), the circadian clock will not signal the pineal gland, thereby delaying the production of melatonin.[6] Such a delayed melatonin production also will delay sleep onset. In our current society, "zeitgebers" (eg, daylight) are weakened (eg, by artificial light), and obligatory activities in the morning (eg, school or work) oblige us to wake up in the morning at a fixed time. Therefore, delayed sleep onset is associated with shorter sleep duration,[7] making the trend of decreased sleep duration in 21st century children[3] with increasing signs of drowsiness[4] more obvious in light (!) of more blue-light exposure in the evening.

The influence of shorter sleep duration (ie, sleep restriction) as studied by Becker *et al.* can be seen as part of a larger pathway that has relevance for ADHD symptomatology. This proposed pathway is visualized in Figure 1 (for further review and references, see the study by Bijlenga *et al.*[6]).

Although Becker *et al.* demonstrate a causal link between shorter sleep duration and parent-rated inattention as the last part in this pathway, future studies should investigate other links of the pathway in relation to ADHD. In addition to the current study by Becker *et al.* investigating the aggravation of symptoms in adolescents with ADHD, investigating healthy adolescents could help establish to what degree sleep restriction would result in reaching the clinical cutoff criteria for ADHD.

Becker *et al.*[5] propose that sleep should be assessed in adolescents with ADHD and that it might be important to directly target or treat sleep problems in ADHD interventions. According to the *DSM-5,* for every diagnosis made, other explanations for the symptoms should be ruled out. In line with Becker *et al.*, we propose clinicians incorporate assessments that quantify sleep and sleep problems, thereby ruling those out as the sole cause of ADHD symptoms. For the subgroup in which inattention and/or hyperactivity-impulsivity problems are primarily explained by underlying sleep problems, a diagnostic presentation category referred to as ADHD-SOM (derived from "somnus," ie, sleep) could be added to embed this group in current clinical practice and research (for further detail, see Bijlenga *et al.*[6]). Considering sleep problems in patients with ADHD will bring us closer to offering treatment to patients that closely matches the individual etiology of their symptoms, thereby optimizing outcomes.

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