Response: “Acute Impact of Immediate Release Methylphenidate Administered Three Times a Day on Sleep in Children with Attention-Deficit/Hyperactivity Disorder”

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We appreciate the opportunity to respond to the issues raised in the commentary regarding our recent publication in the Journal of Pediatric Psychology entitled “Acute Impact of Immediate Release Methylphenidate Administered Three Times a Day on Sleep in Children with Attention-Deficit/Hyperactivity Disorder.” A number of interesting points were made by the author of the commentary, some of which we are able to directly address and others which we addressed only in a speculative manner. Although research in the field of pediatric sleep is growing, there are still many questions that remain unanswered, particularly regarding the link between sleep and childhood psychopathology.

As the author of the commentary correctly points out, there is limited research examining the daytime consequences of sleep deprivation in children. However, it is important to highlight that the daytime sequelae of sleep deprivation in adults have been well documented in the literature. For adults, sleep deprivation has been shown to have significant consequences for mood, cognition and motor performance (for a review see Pilcher & Huffcutt, 1996). Executive attention, working memory, and divergent thinking involved in decision making are some of the specific cognitive abilities found to be most vulnerable to sleep deprivation (Durmer & Dinges, 2005).

An excellent review (Sadeh, 2007), published after submission of our manuscript, evaluates the research to date regarding the impact of sleep deprivation on children’s daytime functioning. Sadeh reviews the fairly large body of correlational research as well as the few experimental studies conducted which examine children’s response to sleep deprivation/sleep loss. Correlational research has demonstrated that poor sleep in children is associated with deficits in working memory, executive functioning, and attention, as well as poorer academic achievement, more behavioral problems, difficult temperament, increased negative mood, and poor emotional regulation. However, causal relationships cannot be inferred given the nature of this research. Sadeh notes that the results of the few published experimental studies, although not entirely consistent, have generally found that complex tasks (e.g., executive functioning, attention) are sensitive to sleep deprivation/restriction. The findings in children are similar to those found in the literature on adults. Based on this review, Sadeh concludes that: (a) there is converging evidence that sleep loss/poor sleep directly results in daytime sleepiness in children and (b) there is reasonable empirical support that sleepiness results in poorer neurobehavioral functioning (evidence is strongest for deficits in the areas of attention regulation, working memory, and executive functioning).

There is a growing body of research examining the relationship between attention-deficit/hyperactivity disorder (ADHD) and sleep problems. Some researchers hypothesize that ADHD can be the direct result of sleep deprivation (e.g., due to sleep apnea); whereas, other researchers suggest that ADHD symptoms may be exacerbated by coexisting sleep problems. We are not aware of any research that hypothesizes that ADHD symptoms are the result of too much sleep, as suggested by the author of the commentary. In fact, many studies utilizing parent report have found that children with ADHD sleep less than typically developing (TD) children. However, there are exceptions to this finding (both when sleep duration is measured subjectively and objectively). One such exception was from our previous study (Corkum, Tannock, Moldofsky, Hogg-Johnson, & Humphries, 2001), in which we compared sleep in medication naïve children with ADHD to children who were TD. We measured sleep using multiple measures, including a parent questionnaire, sleep diary, and actigraphy. A similar pattern was found across measures in that the ADHD group slept longer (17–23 min longer depending on the measure...
used) than did the TD group. In the article, we clearly indicated that this finding (longer sleep duration in the ADHD group) was not consistent with findings from previous research or from the overall findings in our review article (Corkum, Tannock, & Moldofsky, 1998) and we highlighted the need to replicate this result. It is important to note that in this study (Corkum et al., 2001), the ADHD group was not sleeping more than what would be considered developmentally appropriate. Rather, both groups of children (ADHD and TD) were sleeping less than would be expected based on developmental norms.

As we understand the points made in the commentary, the author suggests that children with ADHD may be sleeping too much. It is proposed that this excessive sleep may be caused by: (a) parents enforcing earlier/variable bedtimes and/or wake times or (b) an intrinsic need for less sleep in children with ADHD. Our current study (Corkum, Panton, Ironside, McPherson, & Williams, 2008) is a within subjects design, so we are not able to directly compare our ADHD sample to a TD sample. However, if we compared the sleep duration of our sample to what is considered typical for this age range, our sample would be considered to be sleeping less than expected, both during no medication conditions and when on medication. Typical sleep duration for children between 6 and 12 years of age is 10–11 h (Meltzer & Mindell, 2006). Children in our sample were in bed (not necessarily sleeping) for 9.5 h when not on medication and for 8.5 h when on medication. This is clearly below the recommended sleep duration for children in this age range.

Referring back to our previous study (Corkum et al., 2001), which did have a TD control group, we found that while the ADHD group slept longer than the TD group, the children with ADHD were not being put to bed significantly earlier than the children in the TD group. There were also no group differences in wake times and, in fact, the children with ADHD were no more likely to awake spontaneously than the TD group (which one would expect if these children required less sleep). In order to address an issue raised in the commentary, new analyses were conducted that examined the differences between groups in the variability (as defined by the mean SD) of bedtimes and wake times. We did not find that children with ADHD had more variable bedtimes \([F(1,47) = .31, \ p = .58]\) or wake times \([F(1,47) = .02, \ p = .88]\) when compared to the TD group. Taken together, these results would suggest that the sleep differences between children with ADHD and children who are TD are not the result of difficulties with parent limit setting around sleep or wake schedules.

To date, there is no research which examines whether children with ADHD may be by nature short sleepers who are being negatively affected by imposing normative sleep expectations. However, one study may address this to some degree. Lecendreux, Konofal, Bouvard, Falissard, and Mouren-Siméoni (2000) found that children with ADHD who did not have any clinical sleep problems were more likely than TD children to evidence daytime sleepiness based on the results of Multiple Sleep Latency Tests. One would assume that if children with ADHD need less sleep, they would not be sleepy during the day if in fact they had similar nocturnal sleep as the TD group. Also, it is important to note that a review of polysomnography studies (Sadeh, Pergamin, & Bar-Haim, 2006) did not find any differences in rapid eye movement sleep between children with ADHD and TD children.

If, as suggested by the author of the commentary, medication exerts its therapeutic effects through reducing sleep, a significant correlation between decreased sleep duration and improvement in ADHD symptoms should exist. In order to directly test the hypothesis proposed by the author of the commentary, we conducted further analyses based on our most recent data (Corkum et al., 2008). We correlated change in parent and teacher ratings of ADHD on the Conners’ Rating Scales (ADHD Index) with change in sleep duration (based on actigraphy ratings). Neither correlation was significant (teacher: \(r = -.09, \ p = .69\); parent: \(r = -.25, \ p = .28\)). However, an empirical test of the hypothesis put forth in the commentary would require depriving medication naïve children with ADHD of sleep and measuring the resulting changes in daytime ADHD symptoms.

Determining the mechanism for the therapeutic effects of stimulant medication is an important goal. We believe it is unlikely that the therapeutic effects of medication are, as suggested in the commentary, related to the reduction of sleep duration. In fact, the beneficial effects of stimulant medication can often be found immediately after the child’s first dose of medication, prior to any sleep (Hood, Baird, Rankin, & Isaacs, 2005). Currently, we are conducting a study using polysomnography to examine the changes in sleep which result from treatment with stimulant medication and the daytime consequences of shortened sleep duration in children treated with stimulant medication. The question that we are most interested in is whether reduced sleep duration has consequences for daytime functioning that are being masked by the administration of psychostimulants, which are known to reduce sleepiness.

Conflict of interest: None declared.
Received, revisions revised, and accepted September 23, 2008

References


