NEW RESEARCH

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Shortened Sleep Duration Causes Sleepiness, Inattention, and Oppositionality in Adolescents With Attention-Deficit/Hyperactivity Disorder: Findings From a Crossover Sleep Restriction/Extension Study

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Objective: Although poor sleep is often reported in adolescents with attention-deficit/hyperactivity disorder (ADHD), prior studies have been correlational. This study investigated whether sleep duration is causally linked to sleepiness, inattention, and behavioral functioning in adolescents with ADHD.

Method: A total of 72 adolescents (aged 14−17 years) entered a 3-week sleep protocol using an experimental crossover design. The protocol included a phase stabilization week, followed in randomized counterbalanced order by 1 week of sleep restriction (6.5 hours) and 1 week of sleep extension (9.5 hours). Sleep was monitored with actigraphy and daily sleep diaries, with laboratory visits at the end of each week. Analyses included 48 adolescents who had complete actigraphy data and successfully completed the sleep protocol (defined a priori as obtaining ≥1 hour actigraphy-measured sleep duration during extension compared to restriction). Parent and adolescent ratings of daytime sleepiness, ADHD symptoms, sluggish cognitive tempo (SCT), and oppositional behaviors were the primary measures. The A-X Continuous Performance Test (CPT) was a secondary measure.

Results: Compared to the extended sleep week, parents reported more inattentive and oppositional symptoms during the restricted sleep week. Both parents and adolescents reported more SCT symptoms and greater daytime sleepiness during restriction compared to extension. Adolescents reported less hyperactivity-impulsivity during sleep restriction than extension. No effects were found for parent-reported hyperactivity-impulsivity, adolescent-reported ADHD inattention, or CPT performance.

Conclusion: This study provides the first evidence that sleep duration is a causal contributor to daytime behaviors in adolescents with ADHD. Sleep may be an important target for intervention in adolescents with ADHD.

Clinical trial registration information: Cognitive and Behavioral Effects of Sleep Restriction in Adolescents With ADHD; https://clinicaltrials.gov/; NCT02732756.

Key words: adolescence, attention-deficit/hyperactivity disorder, comorbidity, sleep deprivation, sluggish cognitive tempo

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Adolescents with attention-deficit/hyperactivity disorder (ADHD) or ADHD symptoms experience more sleep problems, including shorter sleep, than their typically developing peers.[1-3] It is estimated that up to 75% of youth with ADHD have sleep problems.[4,5] Sleep problems are associated with more inattentive symptoms[1] and oppositional behaviors[6] in adolescents with ADHD. Furthermore, daytime sleepiness is common in adolescents with ADHD[5] and is associated with poorer academic performance.[7] These studies are important for identifying associations between sleep problems and functional impairment in adolescents with ADHD but cannot determine whether sleep is causally linked to impairment in adolescents diagnosed with ADHD.

The need for experimental research examining the impact of sleep in youth with ADHD has recently been identified as a research priority.[8] Only one, small-sample (n = 11) study[9] has manipulated sleep in children with ADHD to examine causal associations between sleep and ADHD behavior. Gruber *et al.* found that sleep restriction worsened attentional functioning compared to typical sleep in children with ADHD.[9] A comparable study has not been performed in adolescents with ADHD, nor has research been conducted examining the impact of sleep extension on functioning. Adolescents are especially prone to shortened sleep, due to a range of biological (eg, circadian clock shifts later associated with the progression of puberty) and environmental (eg, school start time, nighttime technology use)

factors.[10-12] There is also some indication that ADHD is associated with more eveningness/later chronotype,[13] which may exacerbate phase delay in adolescents with ADHD. It is thus essential to document the detrimental effects of inadequate sleep as well as the beneficial effects of longer sleep, if sleep is to be identified as a possible target for intervention.[3,14] The current study is the first to use an experimental protocol to determine whether shortened sleep is a causal contributor to daytime behaviors in adolescents with ADHD.

Studies with typically developing adolescents have found shortened sleep to be a causal contributor to increased daytime sleepiness, inattention, and oppositional defiant disorder (ODD) symptoms.[15,16] In contrast, these studies have not found associations between shortened sleep and hyperactive−impulsive symptoms.[15,16] Rather, because sleep restriction appears to contribute to greater hypoactivity rather than hyperactivity, Fallone *et al.* suggested more than a decade ago that "researchers investigating sleep and ADHD include symptoms of sluggish cognitive tempo."[17,p. 1565] Sluggish cognitive tempo (SCT) is a set of attentional symptoms characterized by daydreaming, mental confusion, and slowed behavior/thinking that are strongly related to both ADHD inattention and daytime sleepiness.[18] A recent study[19] with typically developing adolescents found that restricted sleep was causally related to increased SCT symptoms.

The current study used an experimental sleep restriction/extension protocol to examine shortened sleep duration as a causal contributor to poorer daytime functioning in adolescents with ADHD. Based on previous research,[8,15,16,19] we hypothesized greater daytime sleepiness, inattention, SCT, and oppositional behaviors during a sleep restriction condition compared to a sleep extension condition, but did not expect sleep to have an impact on hyperactivity−impulsivity. As in previous research,[15] and given limitations surrounding adolescent self-report of ADHD symptoms,[20] we expected effects to be more robust when examining parent-reported as compared to adolescent-reported behavior.

METHOD

Participants

Participants were 72 adolescents (71% male and 29% female) aged 14 to 17 years (mean ± SD, 15.10 ± 1.06 years) diagnosed with ADHD. All participants had an IQ ≥70 (range, 79−132) based on the Kaufman Brief Intelligence Scale, Second Edition.[21] Sample characteristics, including comorbid diagnoses based on the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS)[22] interview conducted separately with the adolescent and parent, are provided in Table 1.

Full *DSM-5* criteria for ADHD Predominantly Inattentive or Combined Presentation on the K-SADS parent interview was required for eligibility. Exclusion criteria included the following: autism, bipolar disorder, obsessive-compulsive disorder, or psychosis; meeting screening criteria for the possible presence of sleep-disordered breathing or restless legs syndrome using the Pediatric Sleep Questionnaire (PSQ)[23,24]; history of epilepsy or head trauma resulting in loss of consciousness; IQ <70; regular high caffeine use (>1 coffee/energy drink per day or 3 caffeinated soft drinks per day); highly atypical sleep duration (routinely obtaining <6 hours or >9.5 hours on school nights); or obligations that required a bedtime later than 10 pm or waking earlier than 6 am. Participants taking stimulant medication were allowed if the family was willing to discontinue the medication for the 3-week sleep protocol during the summer. In the first year, all participants taking melatonin or a nonstimulant psychiatric medication were excluded; in the second year, these were not exclusionary but were allowable only if discontinued during the summer.

Procedures

All study procedures were approved by the Cincinnati Children's Hospital Medical Center Institutional Review Board. Signed informed consent and assent were obtained. Recruitment materials were distributed via local schools, in the community, and at Cincinnati Children's Hospital Medical Center where the study was conducted during the summers of 2016 and 2017. Materials described a study examining sleep in adolescents with ADHD but did not specifically mention (or target) adolescents with sleep problems.

Sleep Protocol. The sleep manipulation protocol used here has been used in other studies of typically developing adolescents.[15,16,25] As summarized in Figure 1, study participants were involved in a 3-week sleep manipulation protocol administered during the summer break from school to avoid affecting scholastic performance. All sleep occurred in the home environment and was monitored via sleep diaries and actigraphy. After a stabilization week in which participants were asked to wake at a time that would allow them to arrive at the research location by 8 am (see Supplement 1, available online, for additional details), participants were asked to systematically change their bedtimes to accommodate Sleep Extension (SE) and Sleep Restriction (SR) conditions. A within-subjects, crossover design was used, such that all adolescents participated in both the SR and SE conditions, with the order of conditions randomly counterbalanced across participants. During the SE condition, adolescents adjusted their bedtime to obtain 9 hours of nightly sleep (9.5 hours in bed, leaving up to half

TABLE 1 Sample Characteristics

	Full Sample (N = 72)	Adherent Participants (n = 48)
	Mean ± SD	**Mean ± SD**
Age, y	15.10 ± 1.06	15.21 ± 1.15
IQ	102.78 ± 11.67	102.90 ± 11.64
	n (%)	**n (%)**
Sex		
Male	51 (70.8%)	36 (75.0%)
Female	21 (29.2%)	12 (25.0%)
Race/ethnicity		
White	58 (80.6%)	37 (77.1%)
Black	6 (8.3%)	5 (10.4%)
Hispanic	1 (1.4%)	1 (2.1%)
Multiracial	7 (9.7%)	5 (10.4%)
Baseline Medication Status[a]		
Stimulant	53 (73.6%)	35 (72.9%)
Nonstimulant	5 (6.9%)	4 (8.3%)
Melatonin	1 (1.4%)	1 (2.1%)
Any medication	56 (77.8%)	37 (77.1%)
Family Income[b]		
Up to $40,000	9 (12.5%)	4 (8.3%)
$40,001—$60,000	10 (14.1%)	8 (17.0%)
$60,001—$80,000	7 (9.9%)	4 (8.5%)
Over $80,000	45 (63.4%)	31 (66.0%)
ADHD Presentation[c]		
Combined	15 (20.8%)	11 (22.9%)
Inattentive	57 (79.2%)	37 (77.1%)
Comorbid Diagnoses[c]		
Depression/dysthymia	1 (1.4%)	1 (2.1%)
GAD	6 (8.3%)	4 (8.3%)
PTSD	1 (1.4%)	0 (0%)
Mania	0 (0%)	0 (0%)
ODD	4 (5.6%)	2 (4.2%)
CD	0 (0%)	0 (0%)
Any comorbidity	10 (13.9%)	6 (12.5%)

Note: ADHD = attention-deficit/hyperactivity disorder; CD = conduct disorder; GAD = generalized anxiety disorder; ODD = oppositional defiant disorder; PTSD = posttraumatic stress disorder.
[a]*All participants were taken off any medication before starting the 3-week sleep protocol.*
[b]*One parent declined to answer the family income question.*
[c]*ADHD and comorbid diagnoses established using the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS) conducted separately with the parent and adolescent (using an "or" rule), with ADHD diagnosis and presentation based on interview with the adolescent's parent.*

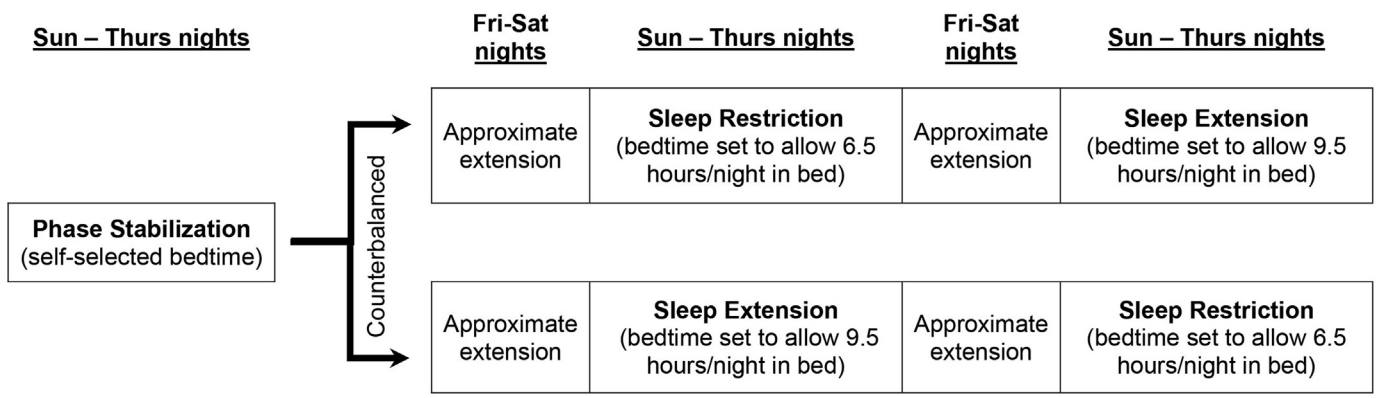
an hour to fall asleep). A 9-hour window was selected because (1) this is how long adolescents sleep during controlled trials of sleep satiation[26] and naturally on nonschool nights[27]; (2) 9 hours results in a well-rested state in adolescents[15]; and (3) this matches clinical recommendations for adolescents.[28] During the SR condition, adolescents adjusted their bedtime to allow 6.5 hours in bed, which, in previous studies using this protocol, resulted in an average of 6.1 to 6.3 hours of nightly sleep.[15] This SR condition reflects a realistic dose of sleep restriction (similar to school-night sleep in 15%−20% of healthy adolescents)[29] that is feasible and induces daytime sleepiness, inattention, and oppositionality in typically developing adolescents.[15,16] (See Supplement 1, available online, for details regarding weekend sleep and caffeine use/napping during the protocol.) Primary data collection occurred throughout the week via actigraphy and daily diaries, and at a laboratory visit each Friday at the end of each condition. Families were able to schedule their visit at any time during the day but were required to remain consistent across the three visits.

Measures

Actigraphy. Participants wore a wrist-mounted actigraph (Micro Motionlogger, Ambulatory Monitoring, Inc.) throughout the sleep protocol to gather an objective measure of sleep. At each Friday assessment, actigraph data were downloaded, and both the actigraphy data and sleep diaries (described next) were reviewed with the adolescent and their parent. In tandem with visually inspecting the sleep diaries, a validated algorithm[30] was used to obtain estimates of sleep onset, sleep offset, time in bed, sleep duration, sleep efficiency, and wake after sleep onset (WASO), which were then averaged into composite sleep variables for each week.

Daily Diaries. Both adolescents and parents completed a daily diary. The adolescent diary included bedtime, sleep latency (time it took to fall asleep), rise time, napping, and caffeine consumption. Sleep onset (bedtime + latency), sleep latency, rise time, and total nocturnal sleep minutes (onset to offset) were averaged for each participant for each week. In addition, both the adolescent and parent diaries included an item assessing the adolescent's difficulty to rise/wake up in the morning (1 = very easy, 2 = easy, 3 = neutral, 4 = difficult, 5 = very difficult) and three items assessing daytime sleepiness ("felt sleepy or tired," "had trouble staying awake," and "yawned or put head down during day") rated on a four-point scale (0 = not at all, 1 = just a little, 2 = pretty much, 3 = very much). Diary items were averaged into composite variables for each week (adolescent diary α = .78 and .89 across days for SE and SR, respectively; parent diary α = .89 and .96).

FIGURE 1 Sleep Restriction/Extension Protocol Using a Within-Person Counterbalanced Design



Daily Attention and Behavior. The parent diary also included the 10-item IOWA Conners Rating Scale[31] that assesses inattention (2 items), hyperactivity–impulsivity (3 items), and oppositionality (5 items) on a four-point scale (0 = not at all, 3 = very much). Daily items were averaged into composite variables for each week (inattention $\alpha = .96$ and .93 for SE and SR, respectively; hyperactivity–impulsivity $\alpha = .87$ and .86; oppositionality $\alpha = .94$ and .91).

Daytime Sleepiness. At each laboratory visit, adolescents completed the 8-item Pediatric Daytime Sleepiness Scale (PDSS).[32] Two items assessing sleepiness/drowsiness during the school year (ie, during class and homework completion) were not used, resulting in six items rated on a five-point scale (0 = never, 4 = always). Mean scale scores were calculated ($\alpha = .65$ and .70 for SE and SR, respectively).

ADHD and ODD Symptoms. At each laboratory visit, parents completed the Vanderbilt ADHD Diagnostic Parent Rating Scale (VADPRS)[33] that includes the nine inattentive (ADHD-IN) symptoms, nine hyperactive–impulsive (ADHD-HI) symptoms, and eight ODD symptoms of the *DSM-5*. Each item is rated on a four-point scale (0 = never, 3 = very often). Mean scale scores were calculated (ADHD-IN $\alpha = .95$ and .93 for SE and SR, respectively; ADHD-HI $\alpha = .94$ and .91; ODD $\alpha = .91$ and .90).

Adolescent self-report of ADHD symptoms was assessed at each laboratory visit using the short version of the Conners, 3rd Edition (Conners-3),[34] which includes six ADHD-IN items and five ADHD-HI items rated on a four-point scale (0 = not true at all, 3 = very much true). Mean scale scores were calculated (ADHD-IN $\alpha = .89$ and .89 for SE and SR, respectively; ADHD-HI $\alpha = .80$ and .82).

SCT Symptoms. The Child and Adolescent Behavior Inventory (CABI)[35] and the Child Concentration Inventory, Second Edition (CCI-2)[36] were used at each laboratory visit to assess parent- and adolescent-reported SCT symptoms, respectively. The same 15 items (eg, daydreams, gets lost in own thoughts, slow behavior) are included on the CABI SCT module (0 = almost never, 5 = almost always) and CCI-2 (0 = never, 3 = always), are internally consistent, and demonstrate discriminant validity from ADHD symptoms.[35,36] Mean scale scores were calculated (CABI $\alpha = .95$ and .95 for SE and SR, respectively; CCI-2 $\alpha = .91$ and .95).

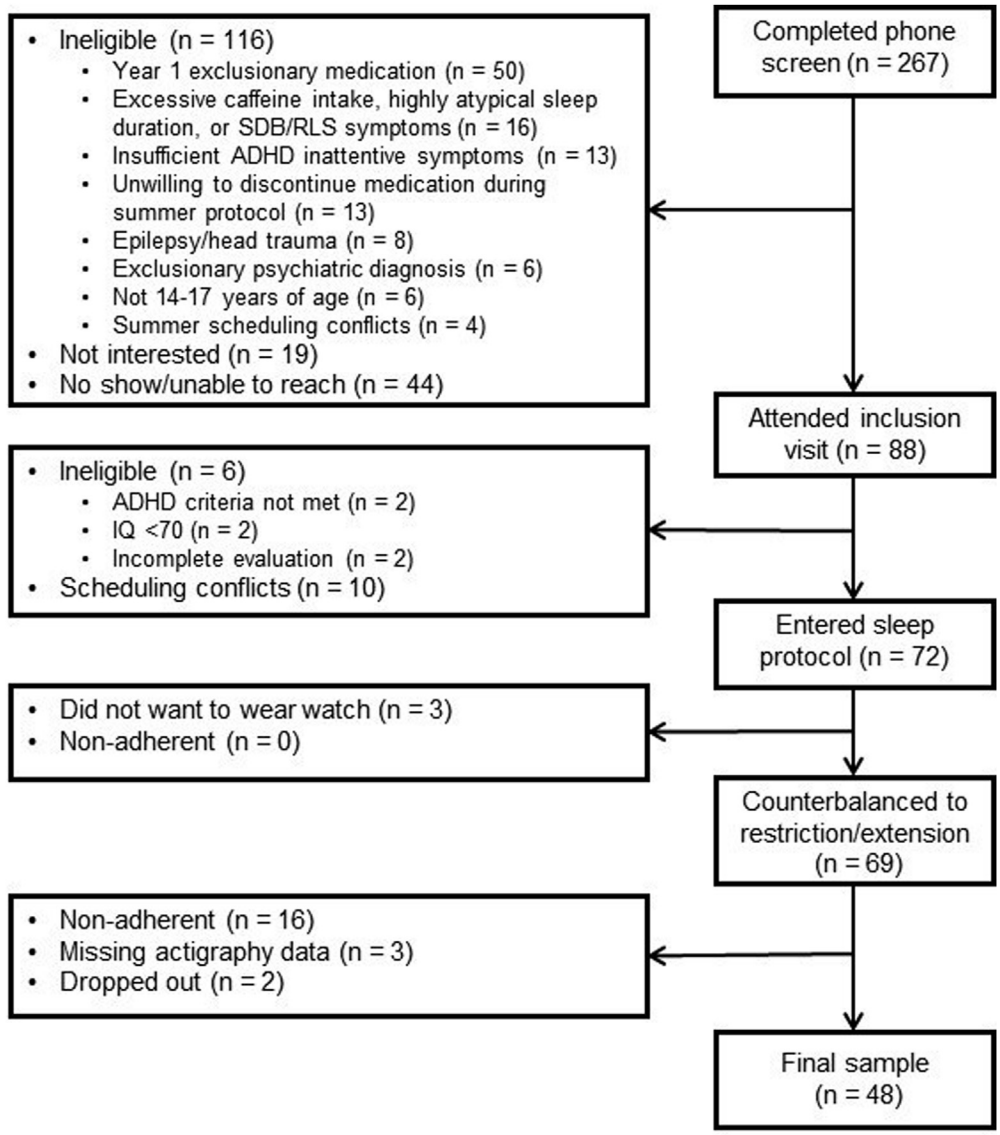
Continuous Performance Test

The A-X Continuous Performance Test (CPT)[37,38] consists of 400 letters that appear individually for 200 milliseconds, with a 1.5-second interstimulus interval and 10% target frequency. Participants were instructed to press the spacebar when they saw an "A" followed by an "X". Accuracy, mean reaction time, number of omission errors (an index of inattention), and number of commission errors (an index of impulsivity) were calculated.

Data Analyses

Given the aim of this study to test whether sleep restriction and extension affected daytime functioning in adolescents with ADHD, only adolescents adherent to the sleep protocol (defined a priori as obtaining ≥1 hour sleep duration during SE compared to SR) were included in analyses. We first confirmed that the sleep manipulation affected sleep as designed. Next, our primary analyses examined the extent to which SR was associated with daytime sleepiness, attention, and behavior. A paired-samples t test for each outcome variable was conducted comparing the SR and SE conditions, with an alpha threshold of .05. Cohen's d, corrected for dependence in within-subjects data, was computed as a measure of effect size. McNemar tests were used to examine whether participants were more likely to nap or to consume caffeine during SR compared to SE.

FIGURE 2 Flow Diagram of Participant Recruitment and Completion of Sleep Protocol



Note: *ADHD = attention-deficit/hyperactivity disorder; RLS = restless legs syndrome; SDB = sleep-disordered breathing.*

RESULTS

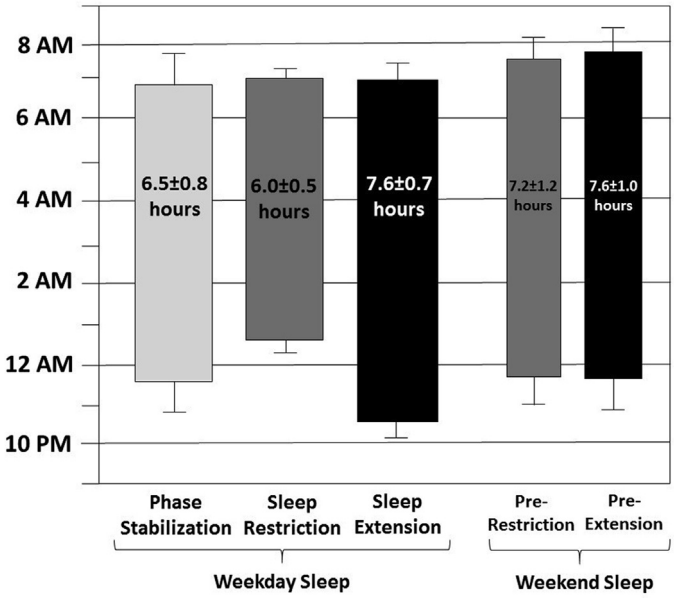
Participant Recruitment, Retention, and Adherence

Figure 2 provides a flow diagram of study recruitment and retention. A total of 64 participants completed the sleep protocol and had complete actigraph data to determine adherence. As shown in Figure 2 and as detailed in Figure S1, available online, 48 of these 64 adolescents (75%) were adherent to the sleep protocol and were included in primary analyses. Additional details regarding adherence can be found in the Supplemental Materials, available online, including analyses indicating no differences between adherent and non-adherent participants in demographic characteristics, symptom severity/comorbidity, and sleep/sleepiness (Table S1, available online) and strategies used by participants to foster adherence (Table S2, available online).

Confirmation That the Sleep Protocol Affected Sleep

Actigraphy-measured sleep onset, offset, and duration throughout the sleep protocol for the 48 adherent participants is depicted in Figure 3. Participants averaged 1.6 hours more sleep per night during SE than during SR. Table S3, available online, details effects of the sleep manipulation on actigraphy- and diary-measured sleep. Very large effects were found for both sleep duration and sleep onset time per actigraphy (d = 2.76 and 4.08) and daily diary (d = 3.76 and 4.10). Participants experienced

FIGURE 3 Sleep Patterns Based on Actigraphy for Each Phase of the Sleep Protocol



Note: Mean sleep onset is represented by the bottom of each bar, and mean wake time by the top of each bar (error bars representing the standard deviation of each), with average sleep duration printed within each bar.

greater actigraphy-measured sleep efficiency ($d = 0.63$) and less actigraphy-measured waking after sleep onset ($d = 1.00$), as well as shorter diary-reported sleep onset latency ($d = 0.78$) and greater diary-reported difficulty waking ($d = 0.75$ and 0.91 for adolescent and parent diaries, respectively) during SR than SE.

Impact on Daytime Sleepiness, Attention, and Behavior

Table 2 summarizes findings for the outcome variables (Table S4, available online, provides intercorrelations among the outcome variables during SR and SE). Greater daytime sleepiness was reported during SR compared to SE, with large effect sizes across parent- and adolescent-completed daily ratings of daytime sleepiness ($d = 1.51$ and 1.00, respectively) and the adolescent-completed PDSS ($d = 1.05$). Participants were significantly more likely ($p = .003$) to report napping during SR (n = 14) compared to SE (n = 3). There was no difference ($p = 1.00$) in reports of caffeine consumption during SR (n = 14) or SE (n = 13).

Across both daily and weekly measures, parents reported significantly greater inattention and oppositional behaviors during SR compared to SE, with medium-sized effects for inattention ($d = 0.53$ and 0.60) and small effects for oppositionality ($d = 0.43$ and 0.33). Significant differences were not found for measures of parent-reported hyperactivity–impulsivity or for adolescent-reported inattention. However, adolescents reported significantly less hyperactivity–impulsivity during SR compared to SE ($d = 0.41$). Both parents and adolescents reported significantly greater SCT symptoms during SR compared to SE, with a small-to-medium effect for adolescent self-reported SCT ($d = 0.44$) and a large effect for parent-reported SCT ($d = 1.00$). No significant effects were found when comparing CPT performance indices during SR and SE (a marginal effect was found for increased errors of commission during SR compared to SE).

DISCUSSION

This study provides the first evidence that insufficient sleep causes impairments in self- and parent-reported daytime functioning in adolescents with ADHD. Restricted sleep worsens attentional functioning and increases oppositional behaviors and daytime sleepiness in adolescents with ADHD.

The impact of restricted sleep on attention was found for parent-reported inattentive symptoms and for both parent- and adolescent-reported SCT symptoms. This is robust evidence for restricted sleep worsening attentional functioning in adolescents with ADHD, although it should be noted that effects were not found for adolescent-reported inattention. There is some evidence from the broader sleep restriction literature that individuals themselves may be less aware of worsened cognitive functioning associated with restricted sleep.[39] However, in typically developing adolescents, sleep restriction worsened ADHD inattentive symptoms across both parent and adolescent ratings, although effects were larger for parent ratings.[15] Adolescents with ADHD may not be able to accurately report on their own ADHD symptomatology,[20] and so this may have contributed to our lack of findings for adolescent-reported inattention, more so than a lack of awareness of worsened cognitive functioning. Furthermore, in contrast to ADHD inattention, there is emerging evidence that youth can provide valid self-reports of their own SCT symptoms[36,40] and we found sleep restriction to worsen SCT across both parent and adolescent ratings. These multi-informant effects are important, as it is estimated that 25% to 40% of youth with ADHD also have elevated SCT symptoms,[41,42] and SCT symptoms are themselves linked to a range of functional impairments such as social withdrawal and internalizing psychopathology.[18]

Consistent with other studies,[8,15,16] parents did not report greater hyperactivity–impulsivity during sleep restriction compared to sleep extension; rather, adolescents in our study reported less hyperactivity–impulsivity during restriction compared to extension. These findings are consistent with the hypothesis that sleep restriction

TABLE 2 Differences in Daytime Sleepiness, Attention, and Behavior Ratings and Continuous Performance Test (CPT) Performance During Sleep Restriction (SR) and Sleep Extension (SE)

	SR	SE	Paired-Samples *t* Tests Comparing SR and SE		
	Mean ± SD	Mean ± SD	*t*	*p*	*d*
Daytime sleepiness					
Sleep diary (parent)[a]	1.24 ± 0.75	0.33 ± 0.36	9.19	<.001	1.51
Sleep diary (adolescent)[a]	0.93 ± 0.68	0.41 ± 0.47	6.49	<.001	1.00
PDSS (adolescent)	1.85 ± 0.78	1.08 ± 0.72	7.25	<.001	1.05
Attention					
IOWA-10 IN (parent)[a]	1.42 ± 0.81	1.13 ± 0.80	3.66	.001	0.53
VADPRS ADHD-IN (parent)	1.57 ± 0.72	1.25 ± 0.77	4.10	<.001	0.60
Conners-3 ADHD-IN (adolescent)	7.48 ± 4.22	7.38 ± 4.67	0.25	.802	0.04
SCT (parent)	1.50 ± 0.95	0.73 ± 0.74	6.75	<.001	1.00
SCT (adolescent)	0.83 ± 0.63	0.64 ± 0.49	2.90	.006	0.44
Behavior					
IOWA-10 HI (parent)[a]	0.66 ± 0.70	0.69 ± 0.69	0.57	.569	0.08
VADPRS ADHD-HI (parent)	0.58 ± 0.63	0.58 ± 0.70	0.05	.963	0.01
IOWA-10 ODD (parent)[a]	0.58 ± 0.56	0.38 ± 0.54	2.99	.004	0.43
VADPRS ODD (parent)	0.65 ± 0.59	0.49 ± 0.58	2.24	.030	0.33
Conners-3 ADHD-HI (adolescent)	4.44 ± 3.40	5.13 ± 3.55	2.82	.007	0.41
CPT					
Accuracy (%)	98.68 ± 2.22	98.77 ± 2.05	1.05	.300	0.17
Mean reaction time (ms)	576.93 ± 118.20	567.04 ± 139.06	0.96	.344	0.15
Errors of omission	3.35 ± 5.91	3.30 ± 5.94	0.26	.793	0.04
Errors of commission	1.94 ± 4.47	1.61 ± 4.48	1.80	.079	0.27

Note: N = 48. ADHD = attention-deficit/hyperactivity disorder; CPT = Continuous Performance Test; HI = hyperactivity–impulsivity; IOWA = IOWA 10-item Conners Rating Scale; IN = inattention; ODD = oppositional defiant disorder; PDSS = Pediatric Daytime Sleepiness Scale; SCT = sluggish cognitive tempo; VADPRS = Vanderbilt ADHD Diagnostic Parent Rating Scale.
[a]These measures were completed in a daily diary throughout SR and SE; all other measures were completed at a laboratory visit after SR and SE.

induces hypoactivity rather than hyperactivity.[8,17] Shortened sleep duration and sleep deprivation contribute to allostatic load throughout the human body,[43] leading to a host of metabolic, hormonal, and neural responses, including increased secretion of proinflammatory cytokines such as interleukin-1 (IL-6) and tumor necrosis factor–α (TNF-α).[44,45] These cytokines are associated with daytime sleepiness and fatigue,[45-47] as well as somatic symptoms of depression such as loss of energy and psychomotor retardation.[48] It would valuable for future research to directly measure cytokines and other indices of hypoactivity to further test this mechanism in adolescents following acute sleep restriction.

We did not find effects on our laboratory-based measure of attention. The literature has produced mixed findings for neurocognitive tasks in studies examining acute sleep restriction (as opposed to chronic sleep loss or sleep deprivation),[49,50] and the impact of sleep restriction on neurocognition is larger in adults than in children/adolescents.[51] Both of these factors may have contributed to the lack of CPT findings in our study. In addition, it would be beneficial for future studies to include other measures of neurocognition, including tasks and indicators that have been shown to be sensitive at detecting ADHD-related cognitive deficits (eg, reaction time variability).

A previous observational study found that sleep problems longitudinally predicted increases in ODD symptoms over 1 year in young adolescents with ADHD.[6] Our findings provide support for a causal link in this association. Establishing this link is theoretically and clinically important, because ODD symptoms commonly co-occur with ADHD, contribute to functional impairment, and may place youth with ADHD on a trajectory for more severe conduct and substance use problems in adolescence.[52] Insufficient sleep may contribute to, or exacerbate, oppositional-defiant behaviors in adolescents with ADHD, and may be important to include in theoretical models of

ADHD comorbidity and treatments targeting externalizing behavior problems.

Also, consistent with previous research with typically developing adolescents[15] and hypothesized based on physiological homeostatic mechanisms that maximize the quantity and quality of sleep in conditions of insufficient sleep,[53,54] adolescents had shorter sleep onset latency, less WASO, and greater sleep efficiency during the week of sleep restriction compared to sleep extension. Both parents and adolescents also reported that it was more difficult for the adolescent to wake in the morning during restriction than during extension. We also found that restricted sleep increased daytime sleepiness in adolescents with ADHD, with effects that were consistent across daily and end-of week measures and across adolescent and parent reports. Children and adolescents with ADHD have been reported to experience more daytime sleepiness than their peers,[5,55] and daytime sleepiness is specifically associated with poorer academic performance in adolescents with ADHD.[7,56] Our findings indicate that shortened sleep duration is likely one mechanism contributing to elevated sleepiness in adolescents with ADHD.

Although the protocol affected sleep as expected, the average sleep duration difference per night between the extension and restriction conditions was 1.6 hours. In contrast, studies using the same protocol with typically developing adolescents found participants to obtain an average of 2.5 hours of sleep per night during the extension condition compared to the restriction condition.[15,16] In considering possibilities that may explain the larger sleep duration effect in studies of typically developing adolescents compared to our sample of adolescents with ADHD, it is important to observe that adolescents with ADHD successfully obtained the instructed sleep duration during sleep restriction but fell short of the instructed duration during sleep extension (Figure 3). Furthermore, as designed, the longer sleep duration during extension compared to restriction was due almost entirely to a shift in the adolescents' bedtime while maintaining a constant wake time. Adolescents with ADHD are more likely than their peers to have a delayed sleep onset latency,[1,55] time-management and organization difficulties,[57] conflict with parents,[58] and reduced motivation,[59] as well as a potentially stronger preference for eveningness.[13] Any or all of these factors may have contributed to adolescents with ADHD being less successful than typically developing adolescents at going to bed earlier and/or falling asleep during the extension condition specifically. It is possible that the 1.6-hour difference in sleep duration was sufficient to produce strong effects on attention and behavior but was insufficient to produce effects on our neurocognitive outcome (ie, CPT).

Our findings have potentially important implications for the treatment of adolescents with ADHD. If shortened sleep duration worsens daytime functioning, then the flip side of the coin is that extended sleep duration improves daytime functioning. Sleep should be assessed when treating adolescents with ADHD, and it may be important to directly target sleep in ADHD interventions. Cognitive–behavioral sleep interventions have been shown to be effective in multiple non-ADHD populations. A recent meta-analysis determined that these interventions are feasible with adolescents, and result in improved sleep (eg, overall sleep quality, total sleep time, sleep onset, sleep efficiency) and functional outcomes (eg, daytime sleepiness, internalizing symptoms).[60] However, none have been tested in adolescents with ADHD specifically, making this an important direction for future study. If a cognitive–behavioral sleep intervention can demonstrate effects similar to what we found in this brief experimental protocol, which were generally medium to large and similar, if not larger, in magnitude to more intensive behavioral interventions,[61-63] sleep treatment would have an important place in the armamentarium of behavioral interventions for adolescents with ADHD. However, modifications to existing cognitive–behavioral sleep interventions may be needed to optimize effectiveness in adolescents with ADHD.[3] In addition, although not the focus of the current study, a public health approach to improving sleep in adolescents more broadly may also improve the daytime functioning of adolescents with ADHD. There is substantial interest in school start time for improving sleep and functioning in adolescents, with recent position statements calling for high schools to start no earlier than 8:30 AM.[64,65] Although the effects of a later school start time are far from settled,[66] the point here is that it is important to consider both individual (eg, pharmacological and/or cognitive–behavioral treatment) and public health (eg, school start time) approaches to improving the sleep and daytime functioning of adolescents with ADHD.

Several limitations are important to acknowledge. First, our significant findings were limited to clinical rating scales, which lack objectivity and blinding to sleep conditions. Still, the fact that we found effects on hypothesized domains (eg, inattention, oppositionality, sleepiness) and not on other hypothesized domains (eg, hyperactivity–impulsivity) lends confidence that findings are not simply due to response bias. Second, we did not include a comparison sample of typically developing adolescents, and thus were unable to test whether the magnitude of effects differs for adolescents with and without ADHD; it appears that effects may be smaller in our sample of adolescents with ADHD compared to typically developing samples.[15,16] Our sample

size was also modest. In addition, the level of ADHD severity in the sample was relatively mild, and rates of comorbidity were relatively low. It is unknown whether our findings generalize to more diverse or more clinically impaired adolescents with ADHD. Finally, the restriction/extension protocol was conducted during the summer and was limited to 5 nights of each condition. Although 5 nights corresponds to the typical school week in U.S. schools, it would be informative for future studies to extend restriction beyond 5 nights to evaluate the cumulative impact of restricted sleep and also to examine the beneficial effects of extended sleep on daily life functioning during the school year among adolescents with ADHD. Administering a sleep protocol during the school year would also allow for collecting ratings from teachers, who are important informants of impairment and could be kept blinded to the sleep conditions.

In conclusion, the current study makes a major contribution to the study of sleep and ADHD. This is the first study to demonstrate that shortened sleep duration is a causal contributor to sleepiness, inattention, SCT, and oppositionality in youth with ADHD. Conversely, extending sleep improves attentional and behavioral functioning in adolescents with ADHD, suggesting that sleep may be an important target for intervention in this population. The current study is a critical first step in showing sleep to be an important component in reducing impairment and improving the lives of adolescents with ADHD.

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