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Original Article

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Sleep restriction impairs item memory discrimination but not general recognition in young adolescents

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Abstract

Study Objectives: The impact of sleep loss on memory encoding is well described in adults, yet less understood in youth, despite the prevalence and educational relevance of adolescent sleep loss. Here, we implement at-home sleep restriction in youth ages 10–14 and a well-validated hippocampus-dependent learning task to elucidate how real-world levels of sleep loss affect distinct memory encoding processes at this young age.
Methods: A within-subject cross-over design involved five nights of at-home sleep restriction (7.5 h in bed) compared to sleep optimization (10 h in bed). Restriction was achieved by delaying bedtime and advancing risetime equally. All sleep was monitored with wrist actigraphy, sleep diaries, and daily calls to the laboratory. Testing involved the validated Mnemonic Similarity Task (MST), which can distinguish between two components of successful memory encoding: general memory recognition for old items and "lure discrimination," a hippocampus-dependent ability to distinguish similar yet distinct items.
Results: As estimated by actigraphy, our manipulation reduced sleep by 1.4 ± 0.48 h per night for five nights. This reduction resulted in a selective deficit in MST-indexed memory encoding; we observed a decrease in lure discrimination (i.e. the ability to distinguish highly similar items), but no impact on recognition of old items.
Conclusions: We present evidence that low levels of sleep loss for five nights (typical of a school week) are sufficient to alter memory encoding in youth. We interpret these data in the context of classroom-based learning and speculate that reduced lure discrimination may yield memory that is less capable of distinguishing closely related facts and concepts.

Key words: sleep restriction; learning; memory encoding; adolescents

Statement of Significance

Sleep loss impacts learning; however, most of this literature stems from adults. Despite the sleep loss experienced by youth as developmental pressures mix with early school-start-times, we know relatively little about how insufficient sleep at this age impacts learning. Here, we present a repeated-measures experiment in 10–14-year-old youth using an at-home sleep manipulation. We show five nights of sleep restriction (mirroring a typical school week by reducing time-in-bed from 10 to 7.5 h) alters hippocampus-dependent memory encoding with selectivity for discerning similar items from one another. We discuss these data in the context of a sleep and memory literature rooted in the career of Prof. Robert Stickgold and offer educational implications for this vulnerable time of life.

Introduction

Once hotly contested [1, 2], sleep is now widely recognized to contribute to human learning and memory [3–9]. Within the context of hippocampus-dependent memory, sleep supports the neural mechanisms underlying memory processing, whether through overnight [10–15] or nap sleep [16–19]. Likewise, not sleeping can impair the same neural mechanisms and diminish learning and memory outcomes [20–23]. Though most studies of sleep loss have focused on adult populations, recent experiments probing naps in young children have suggested that sleep loss has

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similar consequences for learning in early development [24–27]. The limited data investigating the impact of sleep loss on learning and memory in adolescents is striking, since teens are routinely exposed to insufficient sleep [28, 29]. While research has begun to confirm an adult-like role of sleep for memory consolidation in the second decade of life [30], fewer studies have probed the impact of sleep loss particularly for young pre- and mid-pubertal adolescents [31]. We present the current report in recognition of the many contributions of Prof. Robert Stickgold to this field. Specifically, we used a sensitive hippocampus-dependent memory task to investigate the influence of sleep restriction (SR) on learning and memory in young adolescents.

Cataloging the real-world impact of sleep-dependent memory, one can quickly point to adolescents as a group of individuals for whom sleep-dependent memory is immediately consequential. Early adolescence is a unique window for learning. Hippocampus anatomy and functional outcomes mature in late childhood [32] right as youth enter middle and secondary school education where flexible and robust learning is paramount to academic success. At the same time, puberty-gated changes in sleep and circadian rhythms begin to emerge [28, 29]. The perfect storm model [28, 29] of adolescent sleep outlines how bioregulatory shifts in sleep homeostasis and circadian rhythms collide with societal pressures such as early school-start-times [33] to curtail nighttime sleep. The resulting chronic SR can produce as much as 10 h of lost sleep opportunity each week [34]. The impact of insufficient and inadequate sleep on adolescent life is a major public health concern. Despite the clear links to education [33, 35–39], limited research has examined the impact of adolescent sleep loss on sleep-dependent learning and memory capacity. Yet, the evidence from adults is clear.

In adults, total [20, 23] and partial [21] sleep deprivation, as well as chronic SR [40, 41], diminish encoding and retrieval of fact-based episodic memories that rely on hippocampal function. Most sleep and memory studies focus on the overall presence or absence of a sleep-dependent effect. However, as Stickgold [3] and others [7, 42] have noted, not all memories are impacted similarly by sleep and sleep loss. For example, sleep-dependent consolidation may preference memories with high emotional content [13] or for which later recall is of greater relevance [42]. Additionally, the degree to which task success is sleep dependent can be gauged by the role of the hippocampus in the memory task. Familiarity judgments (e.g. a nonspecific sense of memory for an item) appear to depend on extrahippocampal regions such as the perirhinal and entorhinal cortices that lie along the parahippocampal gyrus [43]. More complex memory judgments that require discrimination between highly similar but unique stimuli rely on the hippocampus per se, and on subregions therein.

Prior work [23] indicated that sleep loss impacts complex judgments on the Mnemonic Similarity Task (MST). The MST has advantages over simple old–new item recognition paradigms by including "lure" images that range in visual similarity to learned items. The "Lure Discrimination Index" (LDI) score from the MST [44, 45] serves as a behavioral marker of hippocampal pattern separation, the ability to keep these separate yet related items distinct in memory. This behavioral metric depends on specific hippocampus circuitry in the CA3/Dentate-Gyrus subfields. Saletin and colleagues demonstrated in adults that lure discrimination was diminished by total sleep deprivation; critically, general old–new recognition memory was spared. Parallel findings have also been identified on this task for memory consolidation after overnight sleep [46–48]. As one begins to investigate the influence of commonplace adolescent SR on cognitive function, it is critical to use tasks such as the MST that can dissociate anatomically distinct memory processes. In so doing, we may be better able to parse the specific hippocampus-dependent mechanisms that underscore real-world learning and short-term memory in the classroom, which relies on flexible and sensitive memory encoding and successful and precise integration and recall [49, 50].

Guided by this literature, we present the first results from an ongoing within-subject cross-over experimental SR study of young adolescents. We manipulate sleep schedules in the home to model the sleep loss that occurs in a typical school week—that is, a less drastic reduction than the experimentally induced sleep deprivation of many studies. Adding to this ecological validity, we include participants who, while present in the classroom are typically excluded from sleep and memory experiments: youth with varying symptoms of attention-deficit/hyperactivity disorder (ADHD). As ADHD affects 1 in 10 school-aged children, enrolling these youth in experimental studies is necessary to gain translational relevance to the classroom and enhance ecological validity [51]. The current analyses advance a first examination of the within-subject experimental effect of SR on learning in such a neurocognitively diverse set of adolescent participants. We have previously proposed that youth with ADHD symptoms may be particularly vulnerable to the effects of routine adolescent sleep loss [52]. While not the focus of the current analysis, our future work will extend these findings to investigate whether inter-individual differences in ADHD symptoms predict the behavioral and brain consequences of SR for youth.

With this lens in mind, the current analyses examine how restricting sleep (i.e. assigning 7.5 h in bed) for five nights impacts short-term hippocampus-dependent learning on the MST. We hypothesized that, (1) SR for five nights would impair MST learning performance and (2) that MST lure discrimination, a more sensitive marker of hippocampus function, would be more impaired than general memory recognition.

Materials and Methods

This study was approved by the local institutional review board. Written consent (and for children, assent) was acquired from all participants. Recruitment methods included community events, print media, community flyers, and online social media advertisements to parents. All participants were paid for their time and effort.

Participants

Youth aged 10–14 were enrolled into an ongoing experimental protocol involving within-participant cross-over manipulation of at-home sleep schedules. As of Summer 2024, 52 children had enrolled in the study. The current analysis includes 47 individuals (23F, 14 non-white, age: 11.7 ± 1.1 years); five children's data were excluded from analysis due to technical issues and attrition (e.g. failure to keep the schedule, withdrawal).

Exclusion criteria included a diagnosis of autism; major or bipolar depression, schizophrenia, or evidence of suicide attempts; a major medical, genetic, or neurological disorder; psychiatric and sleep medication (with exceptions noted below); a first-degree relative with bipolar disorder or psychosis; or an intellectual or physical disability prohibiting participants' ability to complete study procedures.

Table 1. Sample characteristics

	n	%
Sex		
Female	23	48.9
Male	24	51.1
Gender identity		
Female	22	46.8
Male	22	46.8
Non-binary/other	3	6.4
Race		
Asian	1	2.1
Black	2	4.3
White	33	70.2
Multiracial	8	17.0
Other non-white	3	6.4
Ethnicity		
Hispanic	6	12.8
Non-Hispanic	41	87.2
Child-rated pubertal status (PDS category)		
Prepubertal	8	17.0
Early pubertal	6	12.8
Mid-pubertal	19	40.4
Late pubertal	11	23.4
Postpubertal	0	0.0

	m	*SD*	min	max
Age	11.7	1.1	10	14
MacArthur Scale of Subjective Social Status	6.4	1.3	4	10
Morningness/Eveningness	29.7	6.2	22	38
WASI-II Two-test IQ	115.8	10.9	87	140
Conners 3 Parent ADHD Index	43.2	33	9	99

Pubertal status derived from child-reported pubertal development scale [54]. Three individuals did not provide pubertal ratings. The MacArthur Scale of Subjective Social Status [55] ranges from 1 to 10 with greater numbers indicating higher self-reported social status. Morningness/Eveningness [55] scores range from 14 to 42 with greater numbers indicating greater morningness.

Our larger study aims to examine how SR differentially influences cognitive and brain function in children with varying levels of ADHD symptoms. In this regard, our study takes an inclusion-oriented dimensional approach to community-based recruitment. We did not require a diagnosis of ADHD for participation in the study; however, children with ADHD (or common comorbid conditions of oppositional defiant disorder, conduct disorder, or anxiety) were not excluded from enrollment. Children taking stimulant medication (e.g. methylphenidate, but not guanfacine; $n = 3$) or melatonin ($n = 6$), which are both common in ADHD, were allowed to participate if they were able to withdraw from these during the study. Parsing ADHD traits is not a focus of this report; however, we report general ADHD symptom levels in Table 1 together with other demographic information. The Conners 3 ADHD Index scores for excluded youth (44.2 ± 39.2) were similar to those for the final analytic sample (43.2 ± 33; see Table 1).

Pre-study procedures

Following informed consent, participants and their caregivers were characterized on several self-report indices. Key demographic and contextual variables in Table 1 include age, sex assigned at birth, child-reported gender identity, race and ethnicity, the MacArthur Scale of Subjective Social Status (a child-report proxy of SES) [55], and IQ measured on the Wechsler Abbreviated Scale for Intelligence (WASI) II two-test version [57]. We also included circadian phase preference via the Morningness-Eveningness Scale [55] and pubertal development with the Pubertal Development Scale [54,58], both measured on a Sleep Habits Survey [58]. Our ongoing recruitment aims to enrich along ADHD status (see the Discussion section); thus, we included the parent-rated Conners 3 ADHD Index [59].

Protocol overview

Participants entered a cross-over experimental protocol, during which they were instructed to refrain from napping or consuming caffeine during the study. All sleep occurred in the home (see sleep monitoring procedures below). The protocol (Figure 1) began with three to five nights of schedule stabilization (time-in-bed set to 10 h with risetime anchored to accommodate family schedules). Next, two five-night counterbalanced experimental conditions followed: sleep optimization (SO) on the same schedule of 10 h of assigned time-in-bed, and SR with assigned time-in-bed reduced by 25 percent to 7.5 h. In the SR condition, bedtime was delayed and risetime was advanced by equal 75-min intervals; this procedure was implemented to minimize circadian shifts induced by the manipulation [60]. The order of conditions was randomized, and each participant who was randomized to complete SR prior to SO had 4–7 nights of washout sleep on the stabilization schedule (10 h) prior to the five nights of SO commencing. Stabilization and SO schedules are identical with SO referring specifically to the five nights before testing, which match parallel nights of SR. In either randomization, SR always followed at least five nights of the stabilization/SO schedule.

A testing visit after each of the five-night windows of SO and SR was scheduled in the afternoon or early evening (i.e. typical

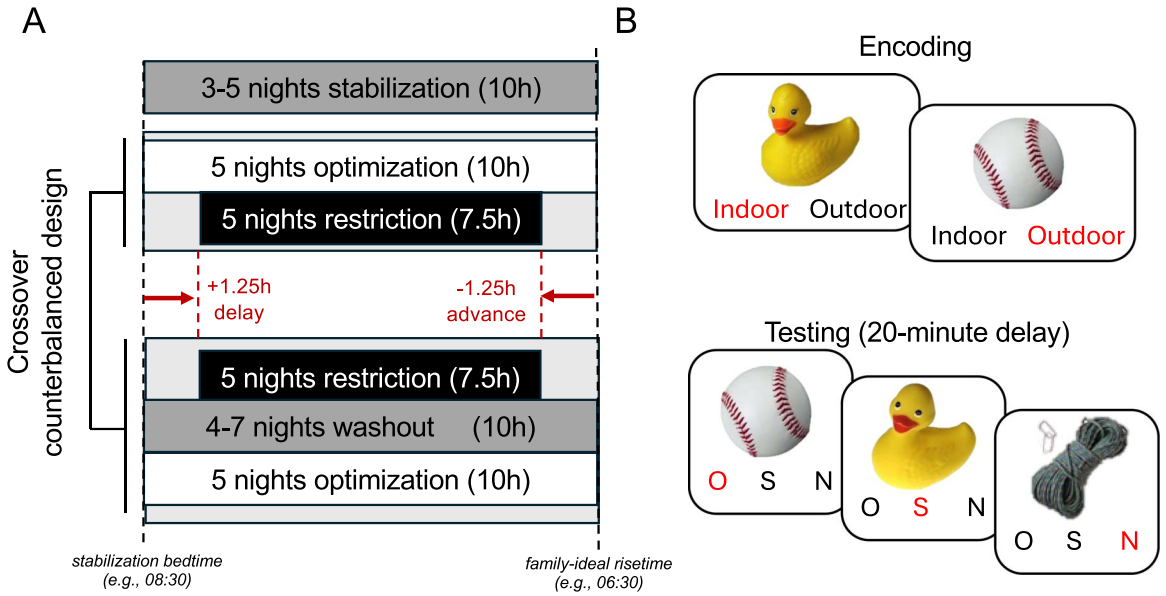

Figure 1. Study procedures. (A) Protocol schematic depicting our cross-over within-participants at-home SR protocol. At least five nights of stabilization to 10 h in bed (anchored to family-optimal risetime) was followed by five nights each on SO (10 h; same schedule as stabilization) and SR (7.5 h) schedules. Sleep restriction targeted 25 per cent reduction and was achieved by delaying bedtime and advancing risetime equally. Where applicable, a washout period of at least four nights followed restriction on the original 10 h stabilization schedule. (B) MST [44, 45] testing sessions occurred in-person after five nights on each schedule. An encoding consisted of viewing 40 namable objects and indicating whether the object was an "indoor" or "outdoor" object. A testing phase after a 20-min delay consisted of 20 repeated target images, 20 related but distinct lure images, and 20 novel foils; participants indicated memory judgments of "old" (correct for targets), "similar" (correct for lures), or "new" (correct for foils). The correct choice for each depicted example is indicated.

after-school homework hours). Assessments included a series of neurocognitive tasks, including declarative memory (i.e. the MST described in this report) as well as other tests probing working memory, response inhibition, cognitive throughput, and vigilant attention, which will be reported elsewhere.

Additional procedures outside the scope of the current analysis include an at-home dim-light melatonin assessment, wake EEG tests, and functional magnetic resonance imaging (fMRI; for eligible and willing participants).

Sleep monitoring

At-home sleep duration was monitored with a combination of wrist actigraphy, morning and evening sleep diaries, and morning and evening calls to a lab voicemail. As in our prior work, participants were fitted with a Micro Motionlogger actigraph (Ambulatory Monitoring, Inc., Ardsley, NY) worn on the non-dominant wrist. Each child completed daily diaries in the evening before bed and the morning after waking to log bedtimes, risetimes, naps, actigraph removal, and other relevant information. Diary completion and schedule adherence were checked with evening and morning voicemails to the lab. If these voicemails indicated that either bedtime or risetime varied by ≥15 min from the assigned schedule, a research assistant called the family that morning to reinforce the study schedule.

We followed established procedures [61–63] for scoring all actigraphy data. An interview with the child (and parent where necessary) occurred at each testing session to compare actigraphy with provided diaries and lab call-ins. Interview questions focused on reconciling discrepancies and ambiguities in the activity record. Next, we scored nocturnal sleep periods in the actigraphy in 1-min epochs using the Sadeh algorithm [64]. The sleep period began with the first of three consecutive minutes of scored sleep and ended with the last five consecutive minutes [63]. Consensus meetings resolved uncertainties with convergent information from daily diaries, lab voicemails, and in-person interviewing.

Mnemonic Similarity Task

The MST indexes the sensitivity of hippocampus-dependent memory encoding for namable objects by manipulating the similarity of images presented. A schematic of the MST task is depicted in Figure 1. In brief (see [44, 45] for an extensive review), the task comprises two phases: (1) an initial encoding phase and (2) an object recognition test phase, separated by 20 min. A pre-study orientation visit included instruction and practice on all study procedures, including a full practice of the MST (encoding and test phases) using distinct stimuli. All task instructions were represented at the start of the SO and SR sessions.

Encoding. Individuals were shown 40 namable objects on a laptop computer screen for 3 s each with a 1-s inter-stimulus interval. For each object, participants indicated on the keyboard whether the object belonged "indoors" or "outdoors."

Test. The test phase followed a 20-min delay consisting of tasks of vigilant attention and cognitive throughput. Sixty images were shown as before, and participants were asked to make a keyboard-registered memory judgment from three options: "old," "similar," and "new." Twenty images were repeated from encoding as target images; the correct response for these images was "old." Another 20 images were lure images visually related yet distinct from the originals; the correct answer was "similar." Finally, 20 foils were included that were not part of the encoding session; the correct answer was "new." Independent stimuli sets were used for SO and SR study sessions, counterbalanced across condition and order.

Quantifying memory. The MST expands on traditional two-alternative forced-choice memory recognition paradigms by including similar lures in addition to repeated targets. Thus, it yields two distinct memory metrics with distinct neurobiological correlates. An LDI is highly hippocampus-dependent and serves as a behavioral approximation of the success of pattern separation—i.e. the ability for memory to distinguish between highly similar objects. As in prior studies by us [23] and others [45], we computed the LDI as a difference score: the probability of correctly endorsing a lure image as "similar" minus the probability of incorrectly endorsing a novel foil as "similar."

We also computed a "Recognition" accuracy score using traditional signal detection theory conventions [65]: the probability of correctly identifying a repeated target as "old" (i.e. hit rate) minus the probability of incorrectly labeling a new foil as "old." (i.e. false alarm rate).

Statistical analysis

All analyses occurred in R using the lmerTest and effectsize packages. We specified separate restricted maximum likelihood linear mixed-effects models for LDI and Recognition scores, respectively. Each model was parameterized with a fixed effect of condition (SO vs. SR) and a random individual intercept. Type-III analysis of variance (ANOVA) effects and Satterthwaite degrees of freedom are reported together with partial-eta-squared estimates of effect-size. Figure 2 includes both individual-level data (i.e. spaghetti plots) and aggregate means, as well as 95% confidence intervals derived from within-subject standard errors. We used similar models to evaluate the effectiveness of our experimental protocol on reducing actigraph-estimated sleep.

To examine the robustness of our design and preliminary sample of paired within-subject data to detect meaningful effects, we computed a sensitivity power analysis to assess differences between two-dependent means (presuming 0.8 power and $\alpha = .05$ and our sample of $n = 47$). This analysis, conducted in G*Power3.1.9.6 indicated our ability to detect effects as small as Cohen's $d = 0.42$ (in the small-effect range of .2–.5).

Results

Success of sleep restriction

Actigraphy during sleep stabilization prior to randomization revealed an average sleep period time (sleep onset to offset) of 9.21 ± 0.33 h and sleep duration of 8.11 ± 0.64 h. After randomization, our experimental protocol was reasonably successful at reducing sleep over five nights in the home. During SO, average sleep period time was estimated as 9.07 ± 0.42 with an average sleep duration of 7.9 ± 0.69 h. During SR, sleep period time averaged 7.25 ± 0.44 h with sleep duration averaging 6.6 ± 0.65 h. Using sleep duration as the ultimate measure of restriction, our data indicated an average reduction of 1.4 ± 0.48 h ($F(1,46) = 372.44$, $p < .01$, partial-$\eta^2 = .89$ [large effect]). Examining individual children, all participants lost sleep during restriction; however, we found high variability in the experimental success of the protocol. The maximal five-night average reductions ranged from 0.31 to 2.11 h; we consider this variability in the Discussion section. Individual-wise sleep estimates are included in Supplementary Table S1.

Mnemonic Similarity Task

Figure 2 and Table 2 capture our core findings for individual data and aggregate estimates. Sleep restriction had a selective impact on MST-indexed memory. On the LDI, we identified an effect of SR ($F(1,46) = 4.74$, $p = .035$, partial-$\eta^2 = .09$ [medium effect]). That is, LDI was lower (−5.9 ± 18.5 [$m \pm SD$]) in SR (60.9 ± 18.6) compared to SO (66.8 ± 17.2). In contrast, no such impact of SR was found for Recognition memory scores ($F(1,46) = 0.80$, $p = .38$, partial-$\eta^2 = .02$ [small effect]). Recognition scores did not statistically differ between SR (79.6 ± 14.5) and SO (81.6 ± 13.0). Thus, SR reduced LDI scores by 8.8 per cent, and Recognition memory was relatively spared.

Table 2 reports full performance metrics from the MST, including overall item-accuracy as well as component scores for *target*, *lure*, and *foil* items with mean rates of endorsing *old*, *similar*, and *new* memory judgments for each. Similar analytic models compared the effects of SR in these scores. Like LDI, SR was associated with a reduction in overall accuracy (per cent of right responses), with SO performance (82.7 ± 8.11 per cent) reduced to (80.0 ± 8.89 per cent) in SR ($F(1,46) = 4.65$, $p = .036$, partial-$\eta^2 = .09$ [medium effect]). The only raw item-score significantly different between SR And SO was the rate of new *foil* images correctly identified as new. Performance dropped from SO (91.6 ± 13.8) to SR (88.8 ± 13.4; $F(1,46) = 4.24$, $p = .045$, partial-$\eta^2 = .08$ [medium effect]). No other effects were identified.

Sensitivity analyses: individual differences

We performed a series of sensitivity analyses to examine whether inter-individual variability in puberty status or ADHD status (Table 1) was informative of MST performance in either condition (SO or SR) or in the impact of SR (SR–SO). With respect to ADHD symptoms, no correlations emerged as significant for LDI (r's: −.16 to .16; p's ≥ .28) or Recognition (r's: −.11 to .16; p's > .28). Similarly, correlations with puberty status were not statistically significant (LDI: r's: .06–.17; p's > .15; Recognition (r's: −.037 to .28, p's > .064). A trending positive relationship between the change in Recognition after SR and puberty status ($r = .28$, $p = .064$) may be driven by a similar non-significant positive association between puberty status and SR Recognition performance ($r = .22$, $p = .15$).

We likewise examined whether individual differences in the change in actigraph-estimated sleep period time or sleep duration, as described above, account for within-subject differences in MST outcomes. No associations were identified for either LDI (sleep period time: $r = -0.078$, $p = .61$; sleep duration: $r = -.13$, $p = .38$) or Recognition performance (sleep period time: $r = .042$, $p = .78$; $r = .0097$, $p = .95$). Finally, we probed whether sex-differences may be present in the impact of sleep loss. We compared the difference of SR and SO performance between female and male participants and identified no significant sex-differences in the impact of SR on either LDI ($t(45) = 0.070$, $p = .94$, $d = 0.02$ [small effect]) or Recognition ($t(45) = -1.40$, $p = .17$, $d = -.41$ [small effect]) performance.

Sensitivity analyses: sleepiness

A Stanford Sleepiness Scale [66] to assess self-reported sleepiness was administered before and after cognitive testing in the experimental protocol. To examine how sleepiness and alertness may be influencing our results we conducted a sensitivity analysis of these data using similar modeling as above. Minor missingness in this data diminishes the sample size slightly (two participants each with missing data for the pre-test sleepiness scale in BSL and SR conditions, four with missing data for the post-test sleepiness scale in SR). Nevertheless, we observed a statistically significant and expected increase in self-reported sleepiness following SR for the pre-test (SO: 3.3 ± 1.2; SR: 4.2 ± 1.4; $F(1,44.47) = 26.6$, $p < .001$, partial-$\eta^2 = .37$ [large effect]) scale. A similar but not significant

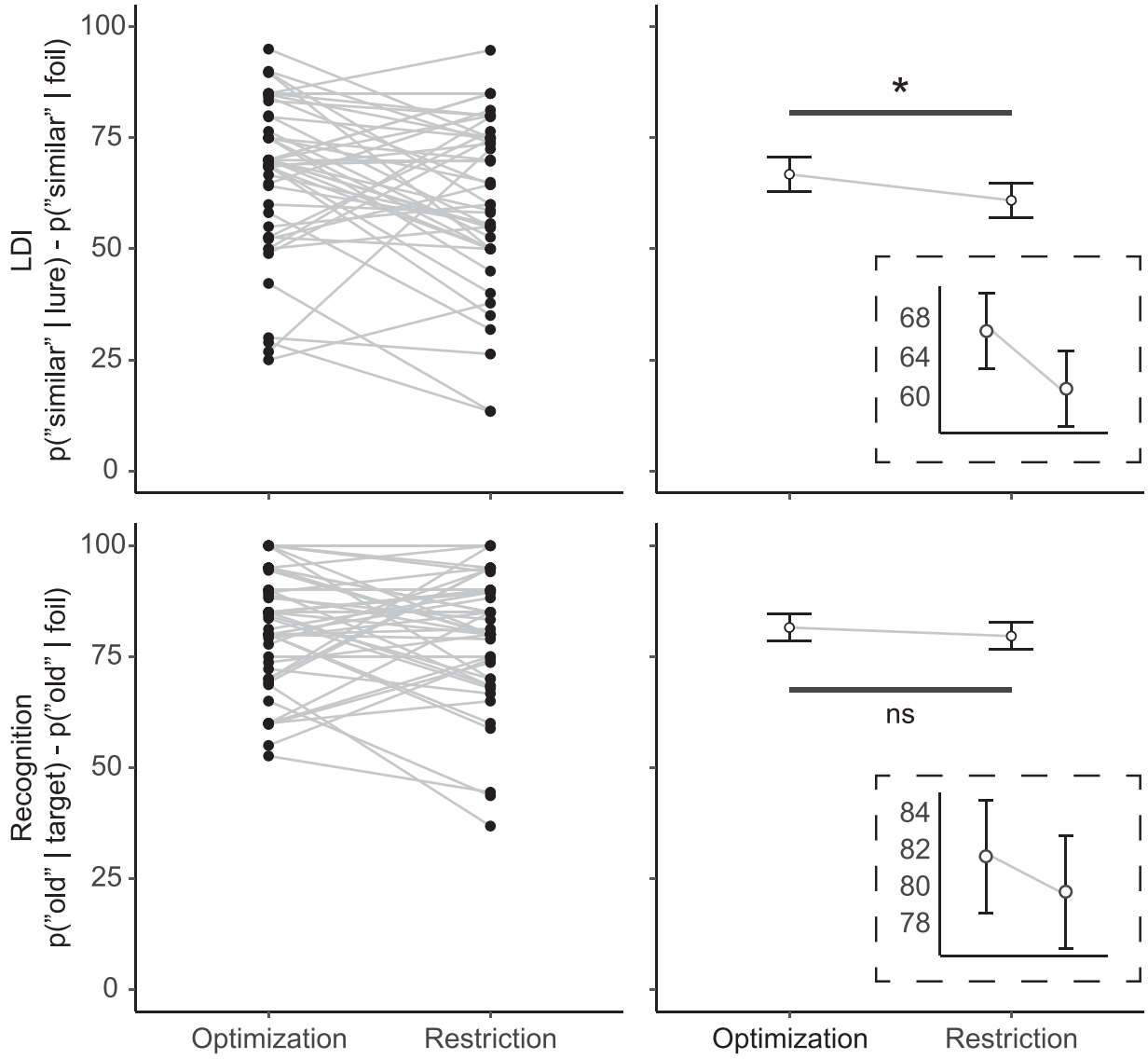

Figure 2. MST performance. Performance is plotted for the (top row) LDI—probability of endorsing a lure image as "similar" minus the probability of endorsing a novel foil as "similar"—and the (bottom row) Recognition—probability of endorsing an old image as "old" minus the probability of endorsing a novel foil as "old"—scores, respectively. For each, individual data points are plotted (left column) for Optimization and Restriction conditions with gray lines illustrating individual-trajectories of performance in the protocol. Finally (right column) mean performance across individuals in each condition is illustrated with error-bars depicting 95% confidence intervals. Dashed box inlets zoom within range of effect.

effect was identified at the post-test assessment (SO: 3.1 ± 1.4; SR: 3.4 ± 1.3; $F(1,41.65) = 3.95$, $p = .053$, partial-$\eta^2 = .09$ [medium effect]). Given the expected rise in sleepiness, we next examined associations between within-condition (SO, SR) sleepiness or the change in sleepiness (SR–SO)—at either pre-test or post-test assessments—and within-condition, or between-condition change in, MST performance on LDI and Recognition scores. To that end, no significant associations were found for any correlation (r's: −.21 to .17, p's ≥ .18).

Discussion

These data—the first from an ongoing study—indicate the effect of at-home SR on hippocampus-dependent memory encoding in children. We find effects of modest SR (on average 1.4 h/night for 5 days) on new learning. Despite subtle changes in sleep, we observed measurable and specific effects. Our study leveraged the MST, a highly replicated experimental paradigm [45] capable of dissociating two indices of memory encoding: first, the LDI highly dependent on hippocampus subfield circuitry; and second, a traditional nonspecific memory recognition score. Only the LDI, a marker of behavioral pattern separation, was significantly impaired after five nights of experimental SR. Below, we discuss the implications of these data across the literatures of sleep and memory broadly, as well as the impact of sleep loss during adolescence. We note how these findings further support the importance of adequate sleep during the "perfect storm" of adolescence [28, 29], in which even small changes to sleep during the school week may result in demonstrable changes in learning.

The impact of sleep loss on hippocampus-dependent learning and memory is well described in adults (see [8] for extensive discussion). Moving beyond traditional item memory paradigms, several studies have used the same MST used here to probe these effects more mechanistically. A great advantage of the MST is its construct validity to parse distinct components of learning: namely, lure discrimination reliant on pattern

Table 2. MST performance

	SO		SR		P	Partial-η^2
	m	*SD*	*m*	*SD*		
Summary statistics						
Overall accuracy (%)	82.7	8.11	80.0	8.89	.036	.09
LDI	66.8	17.2	60.9	18.6	.035	.09
Recognition	81.6	13.0	79.6	14.5	.38	.02
Response rates by item type (%)						
Target						
Old	83.3	11.8	82.9	12.5	.47	.01
Similar	13.8	9.3	14.8	11.1	.58	<.01
New	2.9	5.6	3.3	5.2	.69	<.01
Foil						
Old	2.0	3.7	2.7	5.5	.39	.02
Similar	6.36	8.5	8.47	12.0	.097	.06
New	91.6	13.8	88.8	13.4	.045	.08
Lure						
Old	21.0	11.1	24.1	11.7	.11	.05
Similar	73.2	13.8	69.4	13.4	.14	.05
New	5.87	8.6	6.47	7.5	.69	<.01

MST performance as defined in the Methods section, with performance rates using omission-adjusted denominators. p-values and partial-η^2 statistics reflect within-subject mixed-effects modeling as described in the text.

separation in CA3/Dentate-Gyrus subfields [45]. Using the MST has refined our understanding of how sleep and sleep loss impact aspects of hippocampus-dependent learning. In a prior study of college-aged adults [23], Saletin and colleagues demonstrated that sleep deprivation impaired lure discrimination more than general item recognition through familiarity processes served by extrahippocampal cortices. From this initial demonstration of selectivity within sleep-loss's impact on lure discrimination, the MST has been used in several nap and overnight sleep consolidation studies to show that sleep can affect memory selectively. Compared to a day awake, overnight sleep may protect mnemonic discrimination [46–48] whereas naps may or may not bestow a similar benefit [67, 68]. While our study investigated new encoding, and not overnight or over-nap memory consolidation it joins this broader literature in documenting the selective influence of sleep—or in this case sleep loss—on hippocampus-dependent learning systems. Future studies could investigate sleep loss's long-term consequences to memory relying on consolidation and retrieval processes.

The current project investigated MST performance in isolation of other cognitive domains such as working memory, which are also routinely impacted by sleep loss [69]. While other cognitive domains, including fMRI-monitored working memory, were assessed as part of our study's larger protocol, they are not considered here. Parsing the independent contributions of cognitive control and attention systems on learning in MST will require a larger analytic sample. Examining task interactions in this way is a strong future direction for later consideration. Likewise, we examined whether sleepiness—here measured using a self-reported scale—may be contributing to diminished learning after SR. While sleepiness did increase in our experiment, we identified no significant associations between self-reported sleepiness and MST performance, either within or across conditions. These correlations are interpreted with caution given the sample size required to detect small inter-individual associations; we discuss this point in greater detail below.

The current study of SR used an ecologically valid sleep manipulation. Five nights of home-based, experimentally monitored SR decreased lure discrimination by 9 per cent from rested levels; by contrast, recognition memory was spared. This pattern of results is consistent with those from total sleep deprivation in adults [23], despite the striking difference between studies in terms of severity of sleep deprivation. A far cry from total sleep loss, our protocol resulted in 1.4 h less sleep per night on average. While 1.4 h may be minor on a night-to-night basis, five nights at the same rate would accumulate to 7 h or more of loss sleep, nearly a whole night in sum. We know that for outcomes like vigilant attention, clear dose–response functions relate the impact of total sleep deprivation to the length and dose of SR [70, 71]. We did not probe learning each day of the protocol, so we cannot deduce when the impairment seen here emerged nor when or whether it would equate to total sleep deprivation over time. Studies such as ours indicate a need for similar titrations of sleep loss to investigate how hippocampus-dependent learning responds dynamically to the severity and length of sleep loss. Correlations between individual-level dose of SR and MST outcomes were small and not significant in the current study, and future work with larger samples should investigate whether and how dose–response effects exist.

The average age of participants in our study was 11.7 years of age. While a number of studies (see [30] for review) have begun to examine sleep-dependent memory consolidation in younger adolescents (e.g. [72],) and others have used SR to probe memory encoding in older adolescents [22, 73], our study is the first to our knowledge to examine SR and hippocampus-dependent memory encoding at this young age. In this context, the levels of sleep loss exhibited here are in line with those experienced by school-age children and adolescents daily during the school week [34]. Nevertheless, even that shallow sleep loss altered learning for these participants.

While we know sleep loss during adolescence has been linked to reduced academic performance [38], translating tests such as the MST to real-world learning can be difficult (see [33] for hurdles in this regard). Nonetheless, if we speculate on the real-world implications, diminished lure discrimination in the presence of preserved general item recognition may result in more generalized learning, with less ability to recall and distinguish

closely related yet dissociable facts and concepts. Prior work compliments this finding by indicating that SR alters creativity in adolescents [74]. In addition, the inclusion of academic-oriented learning tasks (concept learning, mathematics, critical thinking and inference) together with hippocampus-dependent laboratory tasks such as the MST, may afford future mechanistic studies a true translational link between the learning changes from SR demonstrated here and processes and outcomes most relevant to classroom success for youth. Such evidence would begin to elucidate the mechanisms through which academic achievement is impaired when children receive inadequate sleep night after night. Building from this literature, one may propose to reverse our experimental protocol to extend sleep by equally small amounts (e.g. 30 min to 1 h) given that typical school-start-time interventions target increasing sleep opportunity by similar levels [33]. The current data present early rationale for such opportunities.

Our study comes with some clear limitations. While a strength of our design is its at-home protocol (as discussed below), we must acknowledge that it limits a strict control on sleep or an assessment of sleep neurophysiology, which may be relevant to understand these and other associations. While we aimed for 2.5 h of SR per night, our study yielded only 1.4 h less sleep each night on average. In our hands—as in others'—the core difficulty appears to be saturating sleep on the longer optimization schedule. We used a one-size-fits-all protocol wherein all children received the same schedule prescription, albeit adapted for household risetime. Developmental forces pushing ideal bedtimes later may make this schedule particularly difficult for older teens to implement [28], thus reducing in part the generalizability of our protocol. Finally, we note that our MST paradigm was brief compared to some other studies. Including more items would allow for a granular investigation of memory bias across dimensions of item similarity, for a more nuanced view of sleep loss's mechanistic impairment.

Our study aimed to understand how ADHD relates to the response to sleep loss. This question is rooted in the well-known association of ADHD report routine sleep disruption (including difficulty falling and remaining asleep and altered sleep timing [51]), which may accompany altered sleep neurophysiology [75, 76]. At a practical level, our recruitment required neither the presence nor absence of an ADHD diagnosis for inclusion. Instead, our dimensional approach sampled youth across typical and atypically developing levels of ADHD symptoms. We view this aspect of our study as a positive from the lens of ecological validity—as it acknowledges a real-world classroom setting in which all kids, regardless of neurocognitive profile, are susceptible to sleep loss's effects on learning. Nevertheless, we acknowledge this sample introduces multiple sources of variance not common in studies enrolling only typically developing youth. Beyond the presence of varying ADHD traits, some of young participants come to the study with prior history of commonly indicated ADHD medications and melatonin. While all participants abstained from relevant ADHD medications and melatonin, it remains possible that prior use of these medications may have altered the brain's response to sleep loss.

Understanding the influence of both ADHD traits and accompanying complexity will be the goal of subsequent papers using the final recruited sample size to test a broader hypothesis that ADHD traits—and the typical disruptions that accompany such presentations—expose a heighted sensitivity to sleep loss at brain and behavioral levels [52].

Taken together, this report is the first of a larger effort to examine our hypothesis of differential and greater vulnerability and resilience to sleep loss with respect to ADHD (see [52] for broader discussion). While we did not draw conclusions on the moderating effects of ADHD traits in this first report, our sensitivity analyses indicated that such effects may be modest and small. Our current data are only powered (.8 power at $\alpha = .05$) to detect associations greater than $r = .38$; thus, we will return to these questions of inter-individual differences in future work with larger samples. Other moderators such as sex-differences, while not present in the current sample, are worthy of future investigation.

Despite these limitations, our study has multiple strengths deriving from our experimental design. Our principal strength is that each child served as their own control in our cross-over repeated-measures design. This was particularly critical given the variability in sleep loss experienced in the sample. While all children lost sleep on average, the magnitude of this effect was variable. Currently our findings examine the aggregate results of our protocol; however, we look forward to the opportunity to examine, as our sample accrues, how these effects differ as a function of sleep loss magnitude. A second strength is the use of a sensitive learning task, i.e. the MST, which provides mechanistic insights into dissociable learning processes. The inclusion of the LDI revealed an effect not detectable in traditional memory recognition tasks. Using similarly, or more, complex tasks may be required to reveal the impact of SR and reconcile studies in which adolescents appeared to demonstrate resilience to sleep loss [77]. An additional strength of our design is that by simultaneously delaying bedtime and advancing bedtime, the experimental protocol proved amenable and attractive to prospective families. Moreover, we avoided introducing explicit circadian misalignment by keeping the midpoint of time-in-bed constant. We note that the use of an at-home manipulation makes our protocol not only increasingly accessible to families, but more ecologically valid in the context of real-world sleep loss experienced by youth. Finally, our study's success in reducing sleep by an average by 1.4 h a night (similar to other recent studies in older adolescents investigating functions beyond memory [60, 78–81]) was bolstered by monitoring procedures involving not only actigraphy and diaries but also daily calls to lab staff who were to respond quickly to family needs and schedule deviations.

Conclusion

We reiterate our debt to the pioneering work of Prof. Stickgold and hope to honor him and his efforts by extending these themes to the younger sample presented here. Taken as a whole, these data add to the basic science of sleep-dependent memory by pairing SR with a still understudied age range. This work joins a quickly expanding literature on the impact of insufficient sleep on cognition and behavior [31] in youth. Set against the backdrop of a developmental maelstrom that conspires with early school-start-times to erode adolescent sleep, studies such as these highlight the cost of such sleep loss while adding to the chorus of calls to improve sleep health for all youth.

Supplementary material

Supplementary material is available at *SLEEP Advances* online.

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Author contributions

Jared M. Saletin (Conceptualization [lead], Data curation [lead], Formal analysis [lead], Funding acquisition [lead], Investigation [lead], Methodology [lead], Project administration [lead], Resources [lead], Software [lead], Supervision [lead], Validation [lead], Visualization [lead], Writing—original draft [lead], Writing—review & editing [lead]), Sinéad M. Moyles (Data curation [supporting], Formal analysis [supporting], Investigation [supporting], Writing—review & editing [supporting]), Victoria O. Dionisos (Data curation [supporting], Investigation [supporting], Writing—review & editing [supporting]), Taylor G. Christiansen (Data curation [supporting], Investigation [supporting], Writing—review & editing [supporting]), Claire Mayew Sherman (Data curation [supporting], Writing—review & editing [supporting]), Gina M. Mason (Investigation [supporting], Writing—review & editing [supporting]), Silvia A. Bunge (Funding acquisition [supporting], Writing—review & editing [supporting]), Francisco Xavier Castellanos (Funding acquisition [supporting], Writing—review & editing [supporting]), Judith Owens (Funding acquisition [supporting], Writing—review & editing [supporting]), Daniel P. Dickstein (Conceptualization [supporting], Funding acquisition [supporting], Methodology [supporting], Writing—review & editing [supporting]), and Mary A. Carskadon (Conceptualization [supporting], Funding acquisition [supporting], Investigation [supporting], Methodology [supporting], Writing—review & editing [supporting])

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Data availability

The data underlying this article will be shared on reasonable request to the corresponding author and in adherence with Bradley Hospital data sharing policies.

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