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ORIGINAL ARTICLE

Differential and interacting effects of age and sleep restriction on daytime sleepiness and vigilance in adolescence: a longitudinal study

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Abstract

Study Objectives: There is contradictory evidence on whether sleep need decreases across adolescence. We investigated this question longitudinally with a dose-response design to test the effects of varied sleep durations on daytime sleepiness and on vigilance and to test whether these relations change with age across early and mid-adolescence.

Methods: Data from 76 participants who completed at least 2 years of the 3-year study are included in this report. Annually, participants ranging in age from 9.8 to 16.2 years completed three different time in bed (TIB) schedules each consisting of four consecutive nights of 7, 8.5, or 10 hours. Daytime sleepiness (multiple sleep latency test [MSLT]) and vigilance (psychomotor vigilance test [PVT]) were measured on the day following the fourth night of each TIB schedule.

Results: Electroencephalogram (EEG)-measured sleep durations changed linearly with TIB. MSLT-measured daytime sleepiness decreased with longer TIB and increased with age. The TIB and age effects interacted such that the TIB effect decreased with age. PVT performance improved with longer TIB and improved with age, but the benefit that increased TIB conferred on PVT performance did not change with age.

Conclusions: These results seem paradoxical because daytime sleepiness increased but vigilance improved with age. The significant age effect on the relation between TIB and sleepiness compared to the lack of an age effect on the relation between TIB and vigilance performance suggests different rates of maturation in underlying brain systems. We interpret these findings in relation to our model of adolescent brain development driven by synaptic elimination.

Statement of Significance

Current recommendations for sleep duration across childhood and adolescence are based primarily on correlational studies. Dose-response studies that measure effects of systematically varied sleep duration should provide stronger evidence-based recommendations. The different maturational effects on psychomotor vigilance test (PVT) performance and multiple sleep latency test (MSLT)-measured daytime sleepiness found here indicate the need for data from a spectrum of measures to determine the functional consequences of sleep changes across adolescence. Such data will ultimately permit more confident recommendations for adolescent sleep durations.

Key Words: sleep restriction; MSLT; PVT; brain maturation

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Introduction

Weeknight sleep duration declines steadily across adolescence. An Australian questionnaire study reported a 12 minutes/year decline in school night sleep duration across ages 9 to 18 years [1]. A longitudinal study by our group found a decline of 10 minutes/year in polysomnographically measured school night sleep duration over the same age range [2]. Throughout industrialized nations, time in bed (TIB) decreases across adolescence as bedtimes are delayed and rise times, largely determined by school schedules, remain relatively unchanged [3–5]. Our longitudinal study of children in California found an increase in bedtimes from 09:12 pm ± 0:05 (mean ± SE) at age 9 years to 10:58 pm ± 0:08 at age 18 years [2]. The later bed times have been attributed to such factors as electronic device use, scholastic demands, social commitments, extracurricular activities, reduced parental control, and altered circadian regulation. Reduced TIB does not appear to be the sole cause of the adolescent decline in sleep duration. TIB restriction reduces both non-rapid eye movement (NREM) and rapid eye movement (REM) sleep, whereas the adolescent decline in sleep duration is entirely a decrease in NREM sleep duration [6]. Thus, it remains unclear whether the adolescent decline in sleep duration is due to decreasing biological need for sleep or whether it occurs in spite of persisting or even increasing sleep need [7].

Based primarily on sleep propensity data, some investigators have concluded that sleep need is either constant or increases across adolescence [7, 8]. On weekends or vacations, when given the opportunity for greater TIB, adolescents sleep longer than preteens. Multiple sleep latency test (MSLT)-measured daytime sleepiness is greater in more mature adolescents even when prior sleep duration is held constant [9]. Carskadon *et al.* [10] found that when TIB was fixed at 10 hours on a 10:00 pm to 08:00 am schedule, younger children were more likely to wake spontaneously prior to 08:00 am, whereas more mature adolescents would have continued to sleep past 08:00 am if not woken by laboratory personnel.

Other measures of sleep need decline across adolescence, such as the optimum sleep duration for daytime mood [11]. The strongest evidence for an adolescent decrease in sleep need is the adolescent change in slow wave (delta) electroencephalogram (EEG) during NREM sleep. NREM delta (1–4 Hz) power declines by more than 60% between ages 12 and 16.5 years, after which the rate of decline slows markedly [12]. NREM delta is believed to be an indicator of a recuperative brain process that occurs during deep sleep [13, 14], and we have interpreted the adolescent delta decline as a decrease in the need for this recuperative process [12]. A slower build-up of homeostatic sleep pressure across the day [15] also suggests that sleep need is decreasing with age in adolescents. Furthermore, in younger teens the proportion of delta energy in the first NREM period is high, as is the level of standardized delta power at the start of the night, and these values decrease across adolescence [16]. Although the time-constant of the exponential decline of delta power across the night does not appear to change with age [15, 17], the age-related change in the initial level of delta power results in homeostatic pressure dissipating more slowly across the night in older adolescents [16], which is consistent with decreasing sleep need with age in adolescents.

While the apparent increase in daytime sleepiness is paradoxical given the apparent decrease in sleep need by sleep-based criteria, neither sleep propensity nor NREM delta activity directly measure sleep need. In a 3-year longitudinal study, we attempted to address this issue by systematically varying sleep duration and measuring effects on daytime sleepiness. The sleepiness findings from the first year of this study [18] replicated an earlier finding [9] that, compared to younger subjects, older adolescents who kept a 10-hour TIB schedule (and obtained sleep durations similar to those of younger participants) are more likely to fall asleep during an MSLT. The dose-response design of our study allowed us to show that TIB and age effects interact, such that the benefits of extending TIB decrease with age in early adolescence (10 to 14 years) [18].

In addition to objective daytime sleepiness measured with the MSLT, our study evaluated psychomotor vigilance performance, a standard measure of the effects of sleep loss on vigilant attention [19]. The number of lapses of attention increases with sleep restriction in both adults [20, 21] and teenagers [22, 23]. Furthermore, in adults, the pattern of degradation in psychomotor vigilance test (PVT) performance across multiple days of sleep restriction is strongly related to the pattern of decreasing MSLT sleep latency [24]. Sleep extension, conversely, improves PVT performance [20, 25], provides resilience against the effects of sleep loss [25], and increases the speed of recovery from sleep loss [25].

By evaluating age-related changes in the dose-response relations between sleep duration and daytime sleepiness and between sleep duration and daytime vigilance, we sought to evaluate how sleep need changes during adolescence. We present here dose-response data from 3 years of a longitudinal study covering ages 10 to 16 years, to address two questions. (1) Do the longitudinal data over a larger age range support the cross-sectional MSLT finding that the benefits of extending TIB decrease with age? (2) Does the improvement in daytime vigilance (PVT) produced by extended TIB also diminish with age?

Methods

Participants

Data are presented here for 76 subjects who participated in at least 2 years of this 3-year longitudinal study. A parent provided informed consent and all participants older than 12 years of age provided assent. The UC Davis Institutional Review Board approved all procedures. Details of the recruitment and screening process have been published elsewhere [18]. Sixty-seven subjects participated in year 3. The most common reason for withdrawing from the study was conflicts between school work or extracurricular activities and the TIB schedules required by the study.

Study design

Each year participants completed three different TIB schedules where they spent four consecutive nights with either 10, 8.5, or 7 hours TIB. For all three TIB schedules, three nights with 8.5 hours in bed preceded the four nights. Subjects kept their habitual rise time and altered their bedtimes to achieve the prescribed TIB. Allowing flexibility in scheduling (to accommodate schoolwork, etc.) prevented us from randomly assigning the order in which each participant completed the schedules. Instead, order

Table 1. Participant information for each time in bed condition for each year of the study

		Year 1	Year 2	Year 3
7 hours	*N*	75	72	62
TIB	Female: Male	35:40	32:40	28:34
	Age (mean, *SD*)	12.21, 1.18	13.39, 1.14	14.31, 1.15
8.5 hours	*N*	76	69	61
TIB	Female: Male	35:41	33:36	27:34
	Age (mean, *SD*)	12.23, 1.18	13.33, 1.19	14.27, 1.14
10 hours	*N*	75	65	61
TIB	Female: Male	35:40	27:38	26:35
	Age (mean, *SD*)	12.21, 1.18	13.24, 1.15	14.28, 1.16

is included as a covariate in statistical analyses. In years 2 and 3 some subjects failed to complete all three schedules. See Table 1 for the distribution.

Actigraphy recordings and all night polysomnography (see below) determined adherence to the prescribed schedule. If a participant's TIB deviated by more than 1 hour from the prescribed TIB, the condition was rescheduled, or, if rescheduling was not possible, the data were excluded. Deviations causing data exclusion occurred in less than 1% of conditions.

Nocturnal polysomnography

All night polysomnography was performed on the second and fourth night of the prescribed sleep schedule. Details have been previously published [6]. Records were visually scored for sleep stages using modified 2007 AASM criteria. The five-channel montage included C3-A2, C4-A1, O1-A2 or O2-A1, LOC or ROC, and chin electromyogram (EMG). Frontal electrodes were not used, nor was the 75 μV criterion for delta waves.

Daytime sleepiness and performance testing

Following four nights on the prescribed sleep schedule, participants reported to the lab for a full weekend day of performance and sleepiness testing. We studied up to four participants each day. Participants arose at their habitual rise time and arrived at the lab at 08:30 am. Lighting in the lab was kept dim (<100 lux at 6′), except during the MSLT when lights were turned off. Participants were not permitted to leave the building during the test day in order to avoid uncontrolled light exposure. The ambient temperature was kept between 72°F and 76°F.

Subjects completed four test batteries at 2-hour intervals starting at 09:00 am. All test batteries included a Karolinska Drowsiness Test/Alpha Attenuation Test (KDT/AAT), a 10-minute PVT [19], and a 20-minute MSLT [26]. Participants also completed the Karolinska Sleepiness Scale (KSS) to assess subjective sleepiness [27] and a positive and negative affect scale for children (PANAS-c) [28]. The 11:00 am and 03:00 pm test batteries included a modified Sternberg test of working memory [29]. For all tests, participants were alone in a bedroom furnished with a desk, chair, extra-long single bed, and night stand, and equipped with video cameras, speakers, and microphones. Breaks between test batteries were spent in a kitchen under supervision of lab personnel. KSS, PANAS, Sternberg, and KDT/AAT results will be presented elsewhere. This report focuses on the MSLT and PVT results.

Participants completed an MSLT session every 2 hours beginning at 09:30 am. Participants lay down in bed and assumed a comfortable position. The test began with instruction to try to fall asleep, and the lights were turned off. Left and right central EEG, occipital EEG, electrooculogram (EOG), and chin EMG were monitored on computer screens for each of the four bedrooms and body position was monitored with infrared video cameras. The MSLT concluded with the occurrence of five consecutive 20-second epochs of stage N1 sleep, or a single epoch of stage N2, N3, or REM sleep. Participants were awakened immediately after sleep was detected. The test was stopped after 20 minutes if the participant was unable to fall asleep.

Participants completed a laptop computer-based PVT (Pulsar Informatics, Seattle, WA) while seated comfortably at their desk. Subjects focused on a red rectangle in the middle of the laptop screen and pressed the space bar as quickly as possible in response to a yellow millisecond counter appearing inside the rectangle. Participants were instructed to respond as quickly as possible and to avoid false starts (pressing the space bar prior to the counter appearing). Response time in ms was displayed for 1 second following each trial. The inter-trial interval varied randomly from 2 to 10 seconds over the course of the 10-minute test.

As the primary measure of PVT performance, we extracted a signal-to-noise ratio (SNR) from the reaction times (RTs) of each test session. The log of the SNR (LSNR) is a measure of the fidelity of information processing and does not show metric ceiling or floor effects [30]. The LSNR is particularly suitable as an outcome measure in longitudinal studies where reference performance may be dynamically changing over time (age), as the interpretation of the difference between conditions is independent of the reference point (e.g. a –3dB change in LSNR always means a 50% reduction in the fidelity of information processing regardless of the reference point from which the change is measured). Details on LSNR calculation are provided in the Supplementary Material.

Additional PVT measures including lapses and average RT are presented in the Supplementary Material. The supplement also includes analyses of sex effects and pubertal maturation effects.

Statistics

Effects of age, TIB, and their interaction were analyzed with mixed-effects regression analyses. Mixed-effects regression is appropriate for longitudinal studies because it accounts for the inherent correlation of multiple observations from the same subject [31]. Time of day effects were accounted for by including a categorical variable for time of day in all analyses.

For the MSLT, latency to fall asleep cannot be analyzed directly as the outcome variable because of the 20-minute cutoff. Instead, MSLT results were analyzed with a nonlinear mixed-effects survival analysis that determined the probability of falling asleep during each minute of the MSLT. The analyses determined how the sleep probability was affected by TIB, age, and time of day, as well as the interaction of TIB by age. Likely due to participants becoming more comfortable with the lab environment, sleep latency decreased with repeated visits to the lab. This effect was accounted for by including a covariate

for order in the analyses. Age was treated as a continuous factor and TIB was treated as a categorical variable.

An order effect was also apparent in the PVT results. Instead of showing a learning effect, PVT performance decreased with repeated visits to the lab. As has been observed in adults [32, 33], the performance decline depended on the preceding conditions; the decline was greatest following the 7-hour TIB condition. This confound was overcome by including in the TIB model the order of each TIB condition as a covariate. For example, the 7-hour TIB condition could be completed in five different orders: 7 hours first, 7 hours second preceded by 8.5 hours, 7 hours second preceded by 10 hours, 7 hours third preceded by 8.5 hours then 10 hours, and finally 7 hours third preceded by 10 hours then 8.5 hours [33]. Age was treated as a continuous factor and TIB was treated as a categorical variable.

Results

TIB effects on sleep duration

TIB restriction effectively reduced sleep duration and did so in a nearly linear manner. The 3 years average (±SE) of night 4 total sleep time (TST) for all subjects was 530 ± 2 minutes, 471 ± 2, and 405 ± 1 for 10, 8.5, and 7 hours in bed, respectively. Mixed-effect analysis showed that this TIB effect represented a significant ($F_{1,471}$ = 2777, p < 0.0001) increase of 41.5 ± 0.8 minutes of TST for each additional hour of TIB. The analysis also showed a 1.9 minutes/year decrease with increasing participant age ($F_{1,471}$ = 5.71, p = 0.017). There was a trend ($F_{1,471}$ = 3.35, p = 0.068) for an interaction, such that the age-related decrease was larger for longer TIB conditions (Figure 1).

Multiple sleep latency test

The survival analysis estimated the log-likelihood of falling asleep in each minute of the MSLT given that the subject was still awake prior to that minute. It also evaluated how TIB and age affected this likelihood. Reducing TIB from 10 to 8.5 hours increased the log-likelihood by 0.86 ± 0.07 (t_{75} = 11.6, p < 0.0001), or a 2.4-fold ($e^{0.86}$) increase in the odds of falling asleep in each minute. Reducing TIB from 10 to 7 hours increased the log-likelihood by 1.90 ± 0.08 (t_{75} = 24.8, p < 0.0001), or a 6.7-fold increase in the odds of falling asleep in each minute. For each additional year of age, the log-likelihood of falling asleep increased by 0.46 ± 0.04 (t_{75} = 10.4, p < 0.0001), or a 1.6-fold increase in the odds. The age and TIB effects interacted such that the effect of TIB on sleepiness decreased with age (Figure 2). For the 7 hours versus 10 hours TIB comparison, the increased log-likelihood of falling asleep declined by 0.14 ± 0.05, or a factor of 0.87, with each additional year of age (t_{75} = –2.83, p = 0.0060); for the 8.5 hours versus 10 hours comparison, the log-likelihood decline, 0.096 ± 0.049, did not reach significance (t_{75} = –1.95, p = 0.055).

Evaluating MSLT results against night 4 TST rather than TIB conditions produced similar results. With TST as a continuous measure, MSLT sleep likelihood decreased significantly with longer TST (t_{75} = –22.7, p < 0.0001). As with TIB, this TST effect decreased significantly with age (t_{75} = –2.51, p = 0.014) by a factor of 0.93 (log-likelihood estimate = 0.071 ± 0.028).

In summary, objective daytime sleepiness measured with the MSLT decreased with extended TIB (and increased sleep duration) and increased across ages 10 to 16 years, but the sleepiness reduction benefit of extending TIB decreased across this age range.

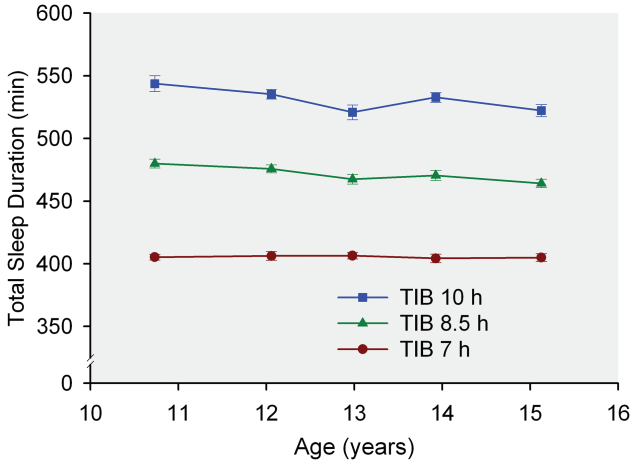

Figure 1. Average (±SE) total sleep duration (TST) plotted against participant age for the three TIB conditions (10, 8.5, and 7 hours). Data are averaged for five age groups: <11.5 (n = 27), 11.5–12.5 (n = 43), 12.5–13.5 (n = 46), 13.5–14.5 (n = 53), and >14.5 years (n = 48).

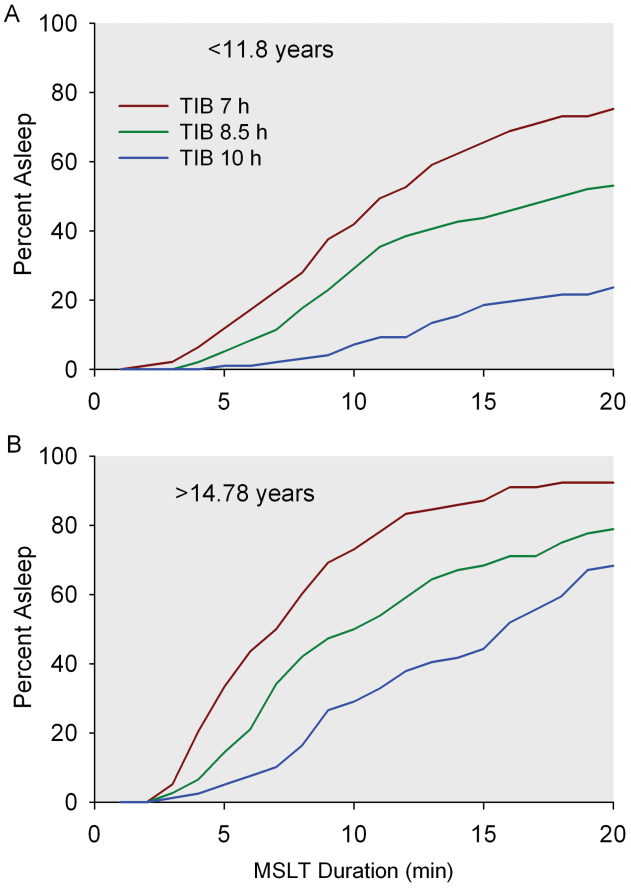

Figure 2. MSLT sleep propensity for the three TIB conditions for the youngest third of participants (A) in the first year of the study (n = 25) and the oldest third of participants (B) in the third year of the study (n = 21). The percentage of subjects asleep is plotted against the 20 minutes of the MSLT. Data for the four daily MSLTs are pooled.

PVT

PVT performance measured as LSNR improved with both increasing TIB and age. PVT performance decreased across the day ($F_{1,2318} = 106, p < 0.0001$). With this time of day effect accounted for, LSNR improved ($F_{2,2318} = 32.1$, $p < 0.0001$) with increasing TIB (Figure 3A). As with the MSLT, the TIB effect was approximately linear. With age centered at 13.2 years and time of day centered at 09:00 am, model estimates of LSNR for 7, 8.5, and 10 hours TIB were 12.15, 12.55, and 12.88 dB, respectively, constituting an 18.3% ($10^{[(12.88-12.15)/10]}-1$) improvement in the fidelity of information processing from 7 to 10 hours TIB. For comparison, the 10-hour TIB value of 12.88 dB is about 2 dB lower than well-rested baseline performance observed in adults, and performance in adults drops by approximately –3 dB on the LSNR scale (i.e. 50%) during the early morning trough of performance after being kept awake all night [30]. LSNR improved by 0.16 points (a 3.8% improvement) for each additional year of age ($F_{1,2318} = 14.6$, $p = 0.0001$). As shown in Figure 3B, the age effect on PVT performance was not linear. Instead, the magnitude of the age effect decreased across the 10- to 16-year age range studied.

The TIB effect did not change significantly ($F_{2,2318} = 2.10$, $p = 0.12$) with age (Figure 3C). However, for the older participants 10 hours TIB did not produce better performance than 8.5 hours TIB. Post hoc analyses for the 10 versus 8.5 hours TIB conditions showed a significant LSNR improvement for the youngest quartile ($F_{1,562} = 6.47$, $p = 0.011$) but not for the oldest quartile ($F_{1,544} = 1.17$, $p = 0.28$). However, analyzing all data together showed no significant age interaction with the 10 versus 8.5 hours TIB effect on LSNR ($F_{2,2318} = 1.01$, $p = 0.32$).

Evaluating PVT results against night 4 TST rather than TIB conditions produced similar results. With TST as a continuous measure, PVT LSNR increased by 0.29 ± 0.04 dB (mean ± SE) for each additional hour of TST ($F_{1,2067} = 45.2$, $p < 0.0001$). TST effects did not interact significantly with age ($F_{1,2067} = 2.19$, $p = 0.14$).

In summary, PVT measured daytime vigilance increased with extended TIB (and with increased sleep duration) and increased across ages 10 to 16 years, but the performance improvement benefit of extending TIB did not change significantly with age.

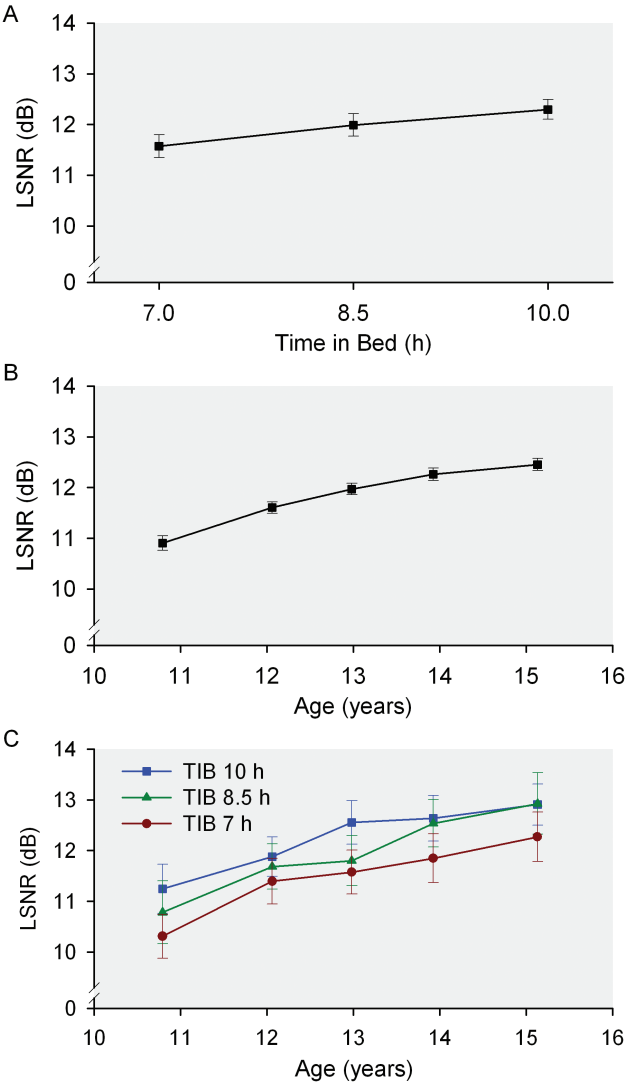

Figure 3. Mean (±*SE*) log signal-to-noise ratio (LSNR), a measure of PVT performance, plotted (A) versus TIB, (B) versus age, and (C) versus age for the three TIB conditions.

Discussion

The 7, 8.5, and 10 hours TIB schedules produced a nearly linear change in total sleep duration and significantly affected MLST-measured daytime sleepiness and PVT-measured daytime vigilance. Therefore, the data presented here can answer the two questions posed in the Introduction. First, the longitudinal MSLT data covering ages 10–16 years show that the benefit, that is, reduced MSLT-measured sleepiness, of longer TIB decreases with age. Second, the improvement in daytime vigilance with longer TIB does not diminish with age. The findings raise the following paradoxes: over early/mid-adolescence (10–16 years), MSLT-measured daytime sleepiness increases but PVT-measured daytime vigilance also increases; over early/mid-adolescence there is an age-related change in the sleep extension effect on daytime sleepiness but not in the sleep extension effect on daytime vigilance. These different age effects and different interactions of age and sleep extension may represent the effects of brain maturation on two different brain systems, those controlling global arousal level and those concerned with cognitive processing.

We interpret both the daytime sleepiness and daytime vigilance effects in relation to the model of adolescent brain reorganization driven by synaptic elimination [34]. In this synaptic elimination model, the high levels of synaptic density at the onset of adolescence are associated with high levels of cerebral metabolism during waking. This elevated brain activity produces an intense need for the sleep-dependent processes by which the brain reverses (or recovers from) the changes induced in plastic brain systems during waking. We have previously argued that this recovery takes place during NREM sleep and that the intensity of the recovery process is proportional to delta power [14]. As synaptic elimination proceeds during adolescence [35], cerebral metabolic rate declines [36], reducing the amount of recovery needed during NREM sleep. As a result, synaptic density, brain metabolic rate and NREM delta power decline, roughly in parallel, across adolescence [37]. Furthermore, the maturational decline in NREM delta power is associated with a decline in cortical gray matter volume [38, 39]. Although we interpret

the age-related changes in both daytime sleepiness and daytime vigilance as products of synaptic pruning, the consequences of pruning are not the same for the two measures.

MSLT age-related changes and age × TIB interaction

An increase in daytime sleepiness during adolescence is well-documented [9, 40–44]. One interpretation of the greater likelihood of MSLT sleep in older participants is that it indicates a greater sleep need. We propose instead, as we have previously [18, 40], that the increased sleepiness reflects increased sleep propensity resulting from the effects of synaptic elimination on waking brain activity. In the years immediately preceding adolescence daytime sleepiness is rare, and preteens are typically unable to nap unless ill or sleep deprived. We have proposed that a high level of brain activity produces a state of arousal that precludes daytime sleepiness [18, 40]. This high level of brain activity is reflected in the elevated waking brain metabolic rates in preteens [36]. As synaptic pruning proceeds, the intensity of waking brain activity declines, allowing sleepiness to emerge.

The adolescent decline in delta power is associated with the adolescent increase in daytime sleepiness [40]. Prior to adolescence, the higher levels of waking brain activity produce a more rapid accumulation of sleep need [15], and delta power during night-time sleep is elevated. In early adolescence, the intensity of waking brain activity is still sufficiently high to create a relatively high need for sleep-dependent recuperation, as in the younger participants of the current study. Interfering with this recuperation by restricting TIB, therefore, produces a greater increase in daytime sleepiness in younger participants. Conversely, extending sleep benefits the younger participants more (a greater decrease in daytime sleepiness).

PVT age-related changes and age × TIB interaction

PVT response times in our study were considerably slower than those normally observed in adults; as would be expected, increasing age was associated with progressively faster mean RTs and faster maximum response speed (Supplementary Material). The effect of age on PVT performance would be an anticipated consequence of adolescent brain maturation. Rapid responses on vigilant attention tasks such as the PVT involve thalamocortical networks and the reticular activating system [19]. Adolescent development of these systems should increase response speed and increase the ability to sustain attention.

However, the absence of an age-related change in the effect of TIB duration on PVT performance was unexpected and might seem inconsistent with the age-related decrease in vulnerability on the MSLT. The synaptic elimination model [34] hypothesized that the enormous advances in cognitive power during adolescent are due more to brain reorganization than learning; synaptic pruning reduces the redundancy of neuronal pathways and makes cognitive processing faster and more efficient with increasing age. Although this decrease in redundancy may decrease processing speed, it may also elevate the vulnerability to sleep loss. It has been hypothesized that sleep loss can reduce the number of redundant functional circuits below the level required to for optimal performance of a task [45]. The level of redundancy may also vary across individuals, making some more vulnerable to the effects of sleep loss [46]. During adolescent development, synaptic pruning could reduce the redundancy in functional circuits, producing a vulnerability to sleep loss that offsets a maturational (age-related) improvement in PVT performance.

Limitations and future directions

Circadian phase may have affected participants' daytime sleepiness and performance, but this study included no measure of circadian phase; measuring circadian effects would have required a design that would have made the study less practical for our subjects. It remains possible that shortening TIB by delaying bedtime produced a circadian phase delay and that this delay differed by age.

In order to standardize sleep history immediately prior to the four nights of prescribed TIB, each TIB schedule was preceded by three nights of 8.5 hours TIB. This 8.5 hours TIB duration was based on average sleep duration of children in this age range from our previous longitudinal study [2] and was a TIB duration that did not interfere with participants' school schedule and extracurricular activities. The 8.5 hours TIB duration is at the lower end of sleep duration recommended for teenagers [47] and may have produced a sleep debt preceding the four-night TIB schedule. However, this debt would be the same for the three different TIB schedules and should therefore not have differentially impacted the effects of the three TIB doses of the study.

Another limitation is that the longitudinal study only covered early and middle adolescence. It leaves undetermined the trajectory of the MSLT and PVT trends across late adolescence and into adulthood. It is important to determine the age at which MSLT sleep likelihood is greatest and how changes in sleep duration affect MSLT-measured sleepiness through late adolescence and into young adulthood. Similarly, the PVT-sleep duration trends from adolescence into adulthood remain unknown. In adults, average RTs are approximately 100 ms faster than the 350 ms mean RTs for the oldest of our study participants on the 8.5 and 10 hours TIB schedule. The age-related improvement in PVT performance shown in Figure 3A likely continues to decelerate until reaching its adult level. Obtaining TIB dose-response data from late adolescence and into adulthood would allow us to test whether these improvements in PVT performance are accompanied by the maintained (or even increased) vulnerability to sleep loss that would be predicted by the loss of redundancy hypothesis expressed above.

Conclusion

Current recommendations for optimal sleep duration in children and adolescents [47] are based primarily on correlational data from studies relating self-reported habitual sleep duration to outcome measures of health, mood, or scholastic performance. A recent review of sleep duration recommendations noted the need for dose-response studies of the type presented here. Short *et al.*'s recent PVT dose-response study in late adolescence used modeling to arrive at a daily sleep requirement of 9.35 hours (out of 10 hours TIB) [23]. The 530 minutes sleep duration that we report for the 10 hours TIB condition is very similar to the 533 minutes mean for 15- to 17-year-old participants on a

10 hours TIB schedule in Short *et al.*'s study and is similar to the values reported by Carskadon *et al.* in 1983 [10]. Both Short *et al.* and Carskadon *et al.* viewed the sleep duration obtained with 10 hours TIB as an indicator of the required sleep duration for this age group. Our PVT data begin to suggest that this much sleep provides little improvement over 8.5 hours TIB in mid-adolescence, but the evidence is not yet firm.

Our data indicate that there are at least two valid but competing perspectives on the issue of whether sleep need changes across adolescence [48]. The MSLT data demonstrate a decrease across adolescence in the benefit provided by extending TIB duration, suggesting diminishing sleep need with age. By contrast, the PVT data do not show an age-related change in the benefit provided by extending TIB duration, raising the possibility that sleep need does not actually diminish across adolescence. It is, of course, possible or even likely that the brain systems controlling global sleepiness simply mature at a different rate from those controlling information-processing in discrete circuits. Whatever the ultimate explanation(s) it seems clear that further evidence on the relation of the sleep changes in adolescence to waking alertness and cognitive function will advance our understanding of sleep and late brain maturation. It also appears that we require additional data across a spectrum of functional outcome measures and over a wider age range in order to translate our findings to evidence-based sleep duration recommendations.

Supplementary material

Supplementary material is available at *SLEEP* online.

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Conflict of interest statement. None declared.

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