Sleep tendency during extended wakefulness: insights into adolescent sleep regulation and behavior

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SUMMARY   Sleep tendency (latency to sleep onset) was examined during extended waking in prepubertal and mature adolescents to determine whether sleep pressure is lower near bedtime in the latter group. Participants were nine prepubertal (pubertal stage Tanner 1, mean age 11.1 years, SD ± 1.3 years, five males) and 11 pubertally mature adolescents (Tanner 5, 13.9 ± 1.2 years, three males). They spent 10 nights at home on an identical fixed 10-h sleep schedule followed by a 36-h constant routine with sleep latency tests at 2-h intervals using standard polysomnography. Saliva was collected to assess dim-light melatonin onset (DLMO) phase. DLMO was earlier in the Tanner 1 (mean clock time = 20:33 hours, SD = 49 min) than Tanner 5 group (21:29 hours ± 42 min). Sleep latency compared at a 'critical period' spanning 12.5 (20:30 hours clock time) to 18.5 h (02:30 hours) after waking did not differ at 20:30 hours, but was shorter for the Tanner 1 group at 22:30 hours (Tanner 1 = 9.2 ± 6.3 min; Tanner 5 = 15.7 ± 5.8 min), 00:30 hours (Tanner 1 = 3.6 ± 1.7 min; Tanner 5 = 9.0 ± 6.4 min), and 02:30 hours (Tanner 1 = 2.0 ± 1.7 min; Tanner 5 = 4.3 ± 3.2 min; trend). These differences were apparent controlling for circadian phase by partial correlation. Sleep tendency after 14.5, 16.5, and 18.5 h awake was lower in mature versus prepubertal adolescents, supporting our hypothesis that a developmental change of intrinsic sleep–wake regulation may provide physiologically mediated 'permission' for later bedtimes in older adolescents.

KEYWORDS   circadian rhythms, puberty, sleep deprivation, sleep homeostasis

INTRODUCTION

As children mature into adolescence, they begin going to sleep later and will stay asleep later when permitted (Carskadon, 1990; Gau and Soong, 2003; Laberge *et al.*, 2001; Wolfson and Carskadon, 1998). Explanations for this delay of sleep timing are easy to find in the changing adolescent psychosocial milieu. Until recently, a common belief held that the habitual bedtime of teenagers is entirely under their behavioral control; for

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example, teens delaying bedtime by socializing in the evening with peers, having jobs, or enjoying late night activities (e.g. television or Internet). Yet, certain features of the sleep delay have led to an examination of possible biological processes contributing to the changes in sleep–wake timing during adolescence (for reviews see Carskadon and Acebo, 2002; Carskadon *et al.*, 2004).

The most well-known model describing sleep–wake regulatory mechanisms in adults was first expressed by Borbély as the two-process model of sleep regulation (Borbély, 1982). This model also serves as a conceptual framework for studying developmental changes of sleep–wake processes around puberty (Carskadon *et al.*, 2004; Jenni and Carskadon, 2004; Jenni *et al.*, 2005). The model includes a sleep–wake dependent

homeostatic process that increases during waking, decreases during sleep, and interacts with a sleep–wake independent clock-like circadian process (Borbély and Achermann, 2005).

Recent studies provide evidence that developmental changes may occur in these regulatory processes during adolescence (Carskadon *et al.*, 2004). For example, pubertal stage is correlated with circadian phase as marked by the timing of the melatonin secretion rhythm: more mature children show a later melatonin secretion offset phase, indicating a possible impetus for later adolescent sleep timing in the circadian timing system (Carskadon *et al.*, 1997). Furthermore, we have suggested that a heightened sensitivity to evening light, a decreased sensitivity to morning light, or a lengthening of circadian period across adolescent development might also contribute to the delay of sleep timing (Carskadon *et al.*, 1999, 2004).

Our group has begun to examine homeostatic aspects of sleep regulation in teenagers using EEG spectral analysis (Jenni and Carskadon, 2004; Jenni *et al.*, 2004, 2005). Developmental changes in this aspect of sleep regulation might also influence sleep timing. We found no differences in the dynamics of the homeostatic process across the night in adolescents; however, indicating that the homeostatic recuperative drive during sleep – and possibly sleep need – remains stable across pubertal development (Jenni and Carskadon, 2004; Jenni *et al.*, 2005). In contrast, analysis of slow-wave activity (SWA; EEG spectral power in the 0.6–4.6 Hz frequency range) during recovery sleep after 36-h of wakefulness indicated that the homeostatic process during waking – essentially reflecting the rise in sleep pressure across the day – may increase at a slower rate in mature adolescents compared with prepubertal children (Jenni *et al.*, 2004). A consistent, though tentative conclusion from these studies of sleep–wake regulation in teenagers is that the maturation of these processes across puberty is permissive of later bedtimes in older adolescents (Carskadon *et al.*, 2004).

Another approach to examine the accumulation of sleep pressure during waking is to assess sleep tendency (measured as the latency to initiate sleep) after prolonged waking in prepubertal and postpubertal adolescents. In this analysis, we make the presumption that speed of falling asleep provides a marker of the process underlying the accumulation of sleep pressure (Carskadon and Acebo, 2002). With this in mind, we predict that sleep tendency during a 'critical window' that falls after scheduled bedtime is sustained at lower levels in older compared with younger adolescents. The chief goal of the present study is to examine this hypothesis using data from young humans in whom multiple sleep latency assessments occurred at 2-h intervals across a 36-h waking vigil.

METHODS

Participants

A sample of 20 children was selected from those who participated in studies of circadian timing that included an episode of extended waking. Sample selection was based upon pubertal stage at the time of study, such that participants were either prepubertal or fully mature. The sample included nine prepubertal (pubertal stage Tanner 1, mean age 11.1 years, SD ± 1.3 years, five males) and 11 mature adolescents (Tanner 5, 13.9 ± 1.2 years, three males). The racial breakdown was as follows: White people ($n = 17$), Native American ($n = 1$), Hispanic ($n = 1$), multi-racial ($n = 1$).

Pubertal status had been determined at a brief physical examination using standardized assessment criteria (Tanner, 1962), in which a rating of stage 1 indicates failure to manifest pubertal changes in secondary sexual characteristics and stage 5 indicates maturity based upon these characteristics. Tanner staging yields two measures for each child: (i) genital development for boys and breast development for girls and (ii) pubic hair growth and distribution for boys and girls. When these ratings differed for a child, pubic hair staging was used because of its higher measurement reliability (Tanner, 1962). Ratings were achieved by consensus from staging by two or three independent research clinicians.

Procedures

Participants were recruited through pamphlets distributed at schools, newspaper advertisements, and solicitations of participants from previous studies. Volunteers and their parents were screened by telephone interview and questionnaires to exclude those reporting a personal history of psychopathology, sleep disorders, current erratic sleep schedules (reported bedtime, rise time, or total sleep time varying more than 3 h across week), more than one nap per week, chronic major illness, current illness, current use of psychoactive agents or other compounds known to affect sleep–wake patterns, history of head trauma, evidence of a learning disability, or a physical handicap that would interfere with testing. Report of psychopathology or major sleep disorder in a first-degree relative was also exclusionary. Volunteers were invited to tour the laboratory facilities with their families, and procedures were explained and demonstrated. Informed consent was obtained from the parents, and participants gave their assent. Participants with no prior experience in the laboratory were asked to take part in a sleepover before the study to ensure they were fully aware of the study conditions.

All assessments occurred between June 21 and August 25, i.e. north American summertime. Experimental protocols were designed to stabilize and 'optimize' prestudy sleep schedules so that participants would not carry a significant sleep deficit and would have a similar prestudy light/dark exposure. Therefore, participants slept at home on a fixed schedule (lights off = 22:00 hours, lights on = 08:00 hours) for at least 10 nights. Previous studies have indicated that the need for sleep is stable at about 9.25 h across this age span and that a 10-h time in bed provides an adequate opportunity to attain sufficient sleep span (Carskadon, 1979, 1982). Prescreening information from these participants indicates that their sleep needs were likely met by this schedule. For example, the pre-pubertal group

reported total sleep time of 9.375 h (SD = 1.06 h) on school nights and 9.5 h (1.41 h) on weekends, and the mature group reported an average total sleep time of 8.45 h (1.13 h) on school nights and 9.91 h (1.51 h) on weekends. At-home restrictions during scheduled sleep included no visitors, lights off, no TV or radio on, sleeping alone in a separate room, and wearing eyeshades provided by the laboratory. Participants called up the lab each morning and evening to report bed and rise times, completed daily sleep diaries, and wore actigraphs to ensure schedule compliance. They were also asked to remain free of caffeine, medications, and such substances as alcohol or illicit drugs throughout the study.

The in-lab portion of the study began immediately after the home monitored nights. Participants were continuously monitored with polysomnography during in-lab scheduled sleep episodes, sleep-latency test (SLT), and performance tests. During the first in-lab night, participants slept on the same at-home schedule and were monitored with polysomnograpy as described in the SLT section below, with the addition of ECG, thermocouples, and sometimes leg EMGs to screen for occult sleep disorders. None was detected.

Extended waking was achieved using a constant routine protocol which was part of the larger study and designed to control for factors that may affect (i.e. mask) circadian rhythms (Carskadon *et al.*, 1997; Czeisler *et al.*, 1985). Thus, participants stayed in individual rooms sitting in bed in a semi-recumbent (45°) posture in dim lighting (15–20 lx) for 36 h (08:00 hours day 1 to 20:00 hours day 2). The sleep latency tests were performed in the seated position, as mandated by the postural control feature of the constant routine. We have collected SLT data in constant routines in the past to test other hypotheses (Carskadon and Dement, 1992) and encountered no unexpected results that could be attributed to posture. The SLTs occurred at 2-h intervals. A performance battery was also given every 2 h. Meals were provided every 2 h, and meal sizes were based on 24-h caloric need determined by height, weight, age, and sex. Saliva (2 mL) was collected half hourly from 18:00 to 12:00 hours. A researcher stayed with each participant at all times, except during sleep testing and restroom activities.

Dim-light melatonin onset

Dim-light melatonin onset (DLMO) was determined as a marker of circadian phase. Saliva samples were frozen (−20 °F) and melatonin levels were subsequently determined with radioimmunoassay (Alpco, Windham, NH, USA). Details of the RIA are available in Carskadon *et al.* (1999). DLMO was determined using linear interpolation from samples bracketing a 4 pg mL^{-1} threshold.

Sleep Latency Test

The SLT is a measure of sleep tendency (speed of falling asleep) obtained using polysomnography under standard conditions (Carskadon and Dement, 1982; Carskadon *et al.*,

1986). Electroencephalogram (EEG; international 10–20 system, C3/A2 or C4/A1, and O2/A1 or O1/A2), electro-oculogram (EOG), and chin electromyogram (EMG) were recorded at 10 mm s^{-1} on Grass Model 8 or Model 78 polygraphs. For each test, participants sat in bed at a 45° angle in dim light (about 0.1 lx) and were instructed to 'sit quietly, keep your eyes closed, and try to fall asleep.' SLTs were ended after unequivocal sleep onset or at 20 min if sleep onset did not occur (Carskadon *et al.*, 1986). Sleep latency is the interval from the start of the test to the first 30-s epoch scored as any sleep stage.

Given the nature of our hypothesis, the ceiling effect on sleep latency after a good night's sleep and the floor effect on sleep latency after a night of sleep loss, we were most interested in SLT scores from a 'critical period' near the participants' scheduled bedtime.

The study was approved by the Lifespan Institutional Review Board for the Protection of Human Subjects and performed according to the Declaration of Helsinki. Participants received monetary compensation and small gifts for their efforts.

RESULTS

As mentioned above, sleep times were strictly controlled for a minimum of 10 nights before the in-lab study. Nevertheless, the mean time of DLMO in the Tanner 1 group was significantly earlier (mean clock time = 20:33 hours; SD = 49 min) than in the Tanner 5 group (21:29 hours; 42 min) as determined by a two-tailed *t*-test ($t = -2.78$; df = 18; $P = 0.01$).

Fig. 1 illustrates the pattern of SLT scores across the waking vigil for each group. Repeated measures analysis of variance with 'Tanner stage group' as the between-group factor and test time as the repeated measure, revealed no main group effect for the 17 SLTs. A main effect of test time was statistically significant ($F_{5.8,288} = 137.6$, $P < 0.001$, Greenhouse–Geisser correction). *Post-hoc* comparisons (with α set at 0.001) identified significant differences for each of the first six tests (10:30–20:30 hours) compared with every test later in the sequence. Furthermore, tests 6–9 (20:30–02:30 hours) were significantly different from each other, and values from the tests at 02:30 and 04:30 hours were higher than at 06:30 hours.

The interaction of group-by-time was also statistically significant ($F_{5.8,288} = 3.3$, $P = 0.006$, Greenhouse–Geisser correction). Given our chief hypothesis – that sleep pressure builds more slowly in more mature adolescents – we were most interested in SLTs that occurred late in the day and beyond usual bedtime. Thus, we performed *post-hoc* testing of the interaction (independent samples *t*-tests) comparing groups on SLT scores for one session before and three sessions after the participants' normally scheduled 22:00 hours bedtime (i.e. SLTs performed at 20:30, 22:30, 00:30, 02:30 hours). Table 1 provides SLT data from these four test times and indicates that prepubertal children had significantly lower SLT scores than the mature adolescents at 22:30 and 00:30 hours, with a trend
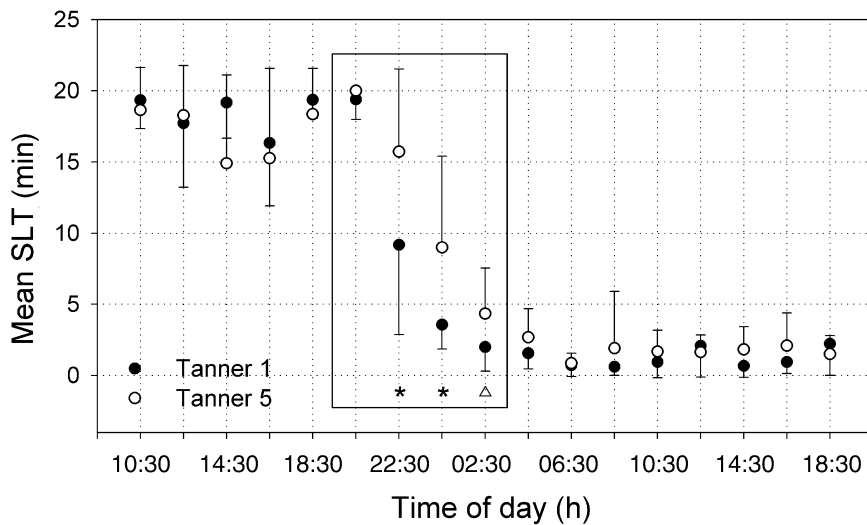
Figure 1. Mean and SD of SLT scores for groups: Tanner 1 shown in filled circles ($n = 9$); Tanner 5 shown in open circles ($n = 11$). Participants woke at 08:00 hours, SLTs were administered at 2-h intervals beginning at 10:30 h. The box isolates the 'critical window' of four tests subjected to *post-hoc* analysis. Asterisks indicate statistically significant differences. The triangle indicates a trend.

Table 1 Sleep latency in prepubertal children (Tanner 1) and mature adolescents (Tanner 5)

Time (hours)	Tanner 1 (n = 9)	Tanner 5 (n = 11)	t	P
20:30	19.4 (1.4)	20.0 (0.0)	−1.34	0.22
22:30	9.2 (6.3)	15.7 (5.8)	−2.42	0.026
00:30	3.6 (1.7)	9.0 (6.4)	−2.71	0.019
02:30	2.0 (1.7)	4.3 (3.2)	−2.12	0.051

Values are expressed in mean (SD). Maximum sleep latency score = 20, minimum = 0.

($P = 0.051$) at 02:30 hours. In contrast, SLTs for the groups were indistinguishable at 20:30 hours, which was earlier than their scheduled bedtimes (22:00 hours). After 18.5 hours awake, an apparent floor effect of the SLT limited distinctions between groups.

Because of significant group differences in circadian (DLMO) phase, we were concerned that the influence of the circadian timing system on sleep tendency might confound the analysis and invalidate conclusions based upon length of time awake. To control for effects of DLMO, therefore, we chose to perform partial correlations of Tanner stage, SLT, and DLMO for the four critical times evaluated above. The partial correlation (Tanner versus SLT, partialling DLMO, i.e. $r_{TannerSLT.DLMO}$) was not statistically significant for the test at 20:30 hours ($r_{TannerSLT.DLMO} = 0.25$; $t = 1.19$; $P > 0.1$), the correlations were significant for 22:30 hours ($r_{TannerSLT.DLMO} = 0.73$; $t = 5.81$; $P < 0.001$), 00:30 hours ($r_{TannerSLT.DLMO} = 0.70$; $t = 5.25$; $P < 0.001$), and 02:30 hours ($r_{TannerSLT.DLMO} = 0.58$; $t = 3.65$; $P < 0.01$), indicating an association of greater maturation level with longer SLTs at the latter times.

DISCUSSION

The major finding of this study was that less mature adolescents manifested greater sleep pressure than more mature adolescents for approximately 4 hours following

scheduled bedtime by falling asleep significantly faster under controlled conditions. Thus, after 14.5, 16.5, and 18.5 h awake [i.e. at 22:30, 00:30, and 02:30 hours (trend)], SLT scores were significantly lower in the prepubertal than postpubertal group. This divergence of sleep tendency in pre- versus postpubertal adolescents may indicate a change in the homeostatic sleep regulatory process as youngsters mature, apparently independent of small differences in circadian phase position.

We are confident in ascribing the sleep tendency difference to an intrinsic process tied to the regulation of sleep rather than external circumstances, because the experimental protocol controlled for many behavioral and environmental factors known to influence sleep timing. For example, we placed participants on a fixed sleep–wake schedule after evaluating their preexisting sleep patterns through the screening questionnaire. As expected, older participants reported sleeping less on school nights, which is a typical behavioral response during adolescent development, even though data from longitudinal studies that control sleep schedules indicate no change in sleep need across this span (cf. Carskadon, 1979, 1982). Indeed, the recently reported finding (Jenni and Carskadon, 2004) that SWA in NREM sleep decays at a similar rate for pre- and postpubertal adolescents on schedules similar to those in the present study also supports a lack of change in sleep need across this developmental span. We note as well that the participants in the Tanner stage 1 group tended to report equivalent sleep on school nights versus weekends, in contrast to the Tanner 5 participants who reported significantly extended sleep on weekends. We interpret this behavioral pattern to indicate that the older children are not acquiring the sleep they 'need' on their school night schedules and must extend sleep on weekends in a compensatory response. Indeed, a major reason that our experimental protocols begin with a substantial lead-in period (in this case a minimum of 10 nights) with a fixed sleep schedule of 10 h a night is to provide the opportunity for participants of this age range to become 'sleep satiated.' The SLT scores above 15 m for the first six tests, indicate that this

goal was achieved. We, therefore, propose that underlying differences in sleep need do not account for the group differences in the later SLTs.

Some might suggest that the results were contaminated by undertaking the SLT with participants in a seated posture. By contrast, we feel that posture did not affect the SLT measures; indeed, the well-controlled conditions of posture, lighting, feeding, and social interaction that are part of the constant routine protocol reduce the likelihood that the maturational differences represent an experimental artifact.

The chief goal of this study was to examine how length of wakefulness affected sleep tendency across this developmental phase. Sleep tendency is also influenced by the circadian timing system, and the current protocol design did not allow an independent analysis for effects of sleep homeostasis and circadian rhythms on sleep tendency. We had hoped that by holding the sleep/wake (and light/dark) schedule consistent during the lead-in period that the circadian phases might have aligned more closely. Confirming previous reports (Carskadon et al., 1993, 1997), however, physical maturation was associated with circadian phase in spite of the consistent sleep–wake schedule. Partial correlation analysis (Tanner versus SLT partialling DLMO), showing a significant partial association of Tanner and SLT at the three tests that occurred after 22:00 hours indicates that the group differences on these tests were associated with maturational stage and not driven by the circadian phase.

This study may contribute to understanding possible bioregulatory processes underlying adolescent sleep–wake behavior changes, i.e. the prominent temporal delay of sleep during adolescent development (Carskadon, 1990; Gau and Soong, 2003; Laberge et al., 2001; Wolfson and Carskadon, 1998). We interpret the difference in sleep tendency after 14.5–18.5 h awake as 'permissive' or 'enabling' for later bedtimes in more mature versus less mature adolescents. This physiological 'permission' may then allow youngsters to respond to psychosocial pressures that also drive later sleep onset. Our findings based on SLT are also in line with other data pointing to a slower rate of sleep pressure accumulation in older compared with younger adolescents as indicated from simulations of SWA according to the two-process model of sleep regulation (Jenni et al., 2004).

A limitation of this study is the cross-sectional nature of the sample, which raises concerns regarding conclusions about developmental or maturational implications of the finding (for discussion of such concerns, see Kraemer et al., 2000). Another caveat is the implicit assumption that maturational stage rather than chronological age is more relevant. We cannot separate the two factors in this study, as the Tanner 5 group was also significantly older than the Tanner 1 group.

On the basis of our findings, we suggest that a sleep phase delay driven by intrinsic alterations of circadian and sleep–wake regulation is a normative developmental process of adolescence. This notion is also supported by the observation that a sleep delay during the teen years occurs in numerous societies around the world despite substantial cross-cultural variations in adolescent lifestyles (see Jenni and O'Connor, 2005 and also Andrade et al., 1993; Carskadon et al., 1998; Gau and Soong, 1995; Gianotti and Cortesi, 2002; Iglowstein et al., 2003; Laberge et al., 2001; Reid et al., 2002; Roenneberg et al., 2004; Strauch and Meier, 1988). As a consequence of the adolescent sleep delay, specific societal demands – such as early school times – can result in curtailment of sleep duration that may influence cognitive and emotional functioning, and therefore school performance and mood (Wolfson and Carskadon, 1998, 2003).

The teenage years also likely represent a vulnerable period with respect to sleep–wake disorders (Wyatt, 2004). Accordingly, the prevalence of delayed sleep phase syndrome (DSPS), a specific clinical entity with inability to fall asleep at a socially acceptable sleep time, peaks during adolescence (7–16%; Garcia et al., 2001; Mercer et al., 1998; Regestein and Monk, 1995), and is relatively rare in adults (0.13–0.17%; Schrader et al., 1993). Future studies in adolescent DSPS patients examining homeostatic and circadian aspects of sleep–wake regulation may further contribute to understanding the pathophysiology of this disorder.

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