PEDIATRICS

EEG Changes across Multiple Nights of Sleep Restriction and Recovery in Adolescents: The Need for Sleep Study

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Study Objectives: To investigate sleep EEG changes in adolescents across 7 nights of sleep restriction to 5 h time in bed [TIB]) and 3 recovery nights of 9 h TIB.

Methods: A parallel-group design, quasi-laboratory study was conducted in a boarding school. Fifty-five healthy adolescents (25 males, age = 15–19 y) who reported habitual TIBs of approximately 6 h on week nights (group average) but extended their sleep on weekends were randomly assigned to Sleep Restriction (SR) or Control groups. Participants underwent a 2-week protocol comprising 3 baseline nights (TIB = 9 h), 7 nights of sleep opportunity manipulation (TIB = 5 h for the SR and 9 h for the Control group), and 3 nights of recovery sleep (TIB = 9 h). Polysomnography was obtained on two baseline, three manipulation, and two recovery nights.

Results: Across the sleep restriction nights, total SWS duration was preserved relative to the 9 h baseline sleep opportunity, while other sleep stages were reduced. Considering only the first 5 h of sleep opportunity, SR participants had reduced N1 duration and wake after sleep onset (WASO), and increased total sleep time (TST), rapid eye movement (REM) sleep, and slow wave sleep (SWS) relative to baseline. Total REM sleep, N2, and TST duration remained above baseline levels by the third recovery sleep episode.

Conclusions: In spite of preservation of SWS duration over multiple nights of sleep restriction, adolescents accustomed to curtailing nocturnal sleep on school day nights evidence residual effects on sleep macro-structure, even after three nights of recovery sleep. Older teenagers may not be as resilient to successive nights of sleep restriction as is commonly believed.

Keywords: adolescents, sleep restriction, recovery sleep, sleep architecture, sleep homeostasis, slow wave activity

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Significance

Adolescents commonly sacrifice sleep for grades and social demands, believing that their youth confers superior resilience to allow them to recover from the effects of sleep restriction by sleeping in on weekends. We found that adolescents who are not habitual short sleepers respond to multiple nights of sleep restriction in a similar fashion as healthy young adults. Significantly, recovery to baseline sleep macrostructure did not appear complete even by the third night of recovery sleep. Given the pervasiveness of sleep restriction in adolescents across the globe, these findings should prompt students, educators and policy makers to re-evaluate their ideas about teenager's resilience to, and recovery from multiple nights of sleep restriction.

INTRODUCTION

Adolescence has been characterized as a period of heightened "storm and stress," accompanied by multiple physical, but also social and psychological transitions.[1–3] Sleep-wake patterns undergo changes due to both intrinsic and extrinsic factors.[4] Older adolescents sleep later due to a shift in their biological clock[4] as well as greater resistance to sleep pressure.[5] The increased use of electronic devices,[6,7] social obligations,[8] reduced parental control,[9] and greater academic demands[10] further collude to exacerbate delays in bedtime.

Despite the trend towards later sleep times in adolescence, wake times have not changed, resulting in insufficient sleep during school days, and an accumulation of sleep debt across the school week. These points have been observed in adolescent sleep surveys worldwide.[11] As many as 75% of adolescents in the USA and more than 90% in Korea and Japan sleep less than the recommended 8–10 h night.[12–15] To compensate for sleep loss during the school week adolescents engage in pronounced sleep extension on weekends.[11] In the current work, we explore the impact of multiple nights of sleep restriction on adolescents' sleep architecture and how these changes compare with prior findings in healthy young adults.

In healthy adults, 7 nights of sleep restriction to 4h TIB per night resulted in shorter sleep latency, reduced durations of N2 and rapid eye movement (REM) sleep with preserved slow wave sleep (SWS) duration.[16–19] Slow wave activity (SWA; EEG spectral power in the 0.5–4 Hz range), a marker of sleep homeostasis,[20,21] increased slightly after the first few nights of sleep restriction and stabilize over subsequent nights.[16,18] The time taken for complete recovery from sleep restriction differs across studies, but sleep architecture appears to return to baseline levels by the third recovery night. In one study involving 7 nights of sleep restriction to 3 h TIB per night,[17] sleep macrostructure returned to baseline levels by the first 8 h recovery night. In another study involving 4 nights of sleep restriction to 4 h TIB per night,[16] sleep macrostructure and SWA returned to baseline levels on the third 8 h recovery night.

Few studies have investigated sleep restriction and recovery dynamics over multiple nights *in adolescents*. An early seminal study,[22] found that one night of 4 h sleep restriction in young adolescents aged 11–13 resulted in reduced stage 1–3 and REM sleep but preserved stage 4 sleep duration. In contrast to adult studies, TST, N1 and sleep latency did not return to baseline after 1 night of 10 h recovery sleep. Subsequent sleep restriction studies in children between the ages of 10–16 have yielded similar findings after one[23,24] or 4 nights of 5 h sleep opportunity.[25] Sleep restriction in these studies was associated with reduced sleep latency, decreased N1 and N2 sleep, increased percentage of slow wave sleep (SWS), and reduced percentage of REM sleep relative to sleep period time, as well as an increase in EEG spectral power within the low frequency range. However, changes in sleep macrostructure and sleep homeostasis across

multiple nights of sleep restriction and recovery sleep have either not been studied or only studied on the first recovery night.

The present work follows up on our previous report on the impact of sleep restriction on cognitive performance, sleepiness, and mood.[26] We evaluated the effect of seven nights of 5 h sleep restriction on the sleep physiology of older adolescents (15- to 19-year olds)—a period when maximum eveningness[27] and scholastic demands conspire to curtail sleep duration. It was conducted simultaneously on 56 participants in a school dormitory where participants' activities were constantly monitored for 2 weeks. Sleep macrostructure and SWA were evaluated using polysomnography (PSG) across 7 nights of sleep restriction and the subsequent 3 recovery sleep episodes.

We predicted that SWS and SWA would be preserved across multiple nights of 5 h sleep restriction at the expense of other sleep stages (N1, N2, and REM sleep), resembling young adults. However, given the greater need for sleep in adolescents compared to adults,[15] we expected that full restitution to baseline sleep structure might not be attained even by the third night of recovery sleep.

METHODS

Participants

Sixty adolescents took part in the Need for Sleep Study. They were selected from volunteers who reported that they: (1) were between 15 to 19 years of age, (2) had no history of chronic medical conditions, psychiatric illness or sleep disorders, (3) had a body mass index (BMI) ≤ 30, (4) were not habitual short sleepers (i.e., had an average actigraphically assessed time-in-bed (TIB) of < 6h and no sign of sleep extension for more than 1h on weekend compared to weekday nights), (5) consumed < 5 cups of caffeinated beverages a day and (6) did not travel across > 2 time zones one month prior to the experiment. Full details of the recruitment and screening criteria are detailed in Lo et al.[26] The study was approved by the Institutional Review Board of the National University of Singapore.

Participants were subsequently randomized into Sleep Restriction (SR) or Control groups. Three participants withdrew from the study for personal reasons—two prior to the study commencement, and one during the study itself. A fourth participant was further excluded for noncompliance with experimental protocols, while a fifth was excluded for excessive artifacts (refer to *Sleep Staging and EEG spectral analysis* for details). The final sample comprised 55 participants (25 males, 16.6 ± 1.0 years [mean ± SD]). The SR (n = 29) and Control (n = 26) groups did not differ in age, gender distribution, BMI, consumption of caffeinated beverages, or on tests of nonverbal intelligence, levels of anxiety and depression, morningness-eveningness preference, levels of daytime sleepiness, symptoms of chronic sleep reduction, subjective sleep quality, or self-reported and actigraphically assessed sleep habits. Habitual bed and wake times, along with TST and TIB of this sample are reported in Table S1 in the supplemental material.

One-Week Pre-Study Protocol

One week prior to the study, participants were required to adhere to a fixed 9 h sleep schedule (23:00–08:00) for circadian entrainment and to minimize the effects of any prior sleep restriction. This was carried out in participants' homes and compliance was verified using wrist-worn actigraphy (Actiwatch 2, Philips Respironics, Inc., Pittsburgh, PA) on the non-dominant hand. Data were recorded at 2-min intervals and scored using Actiware (version 6.0.2). TST was computed using the medium-sensitivity threshold (where activity counts ≥ 40 were scored as wake). Participants were informed that any deviation > 15 min from the required sleep schedule would lead to disqualification from the study. However, the actual exclusion criterion applied was deviation > 30 min from the required schedule for at least 2 days.

Actigraphically assessed TIB, TST, bed and wake times for this 1-week pre-study protocol are reported in Table S2 in the supplemental material. Compliance was satisfactory as bed and wake times were close to the required sleep schedule (23:00–08:00), while mean TIB (± SEM) was 8.75 ± 0.07 for the SR group and 8.84 ± 0.04 for the Control group (P = 0.49).

Two-Week Study Protocol

The experimental protocol during the 2-week period is shown in Figure 1. In the first 3 nights of the study (B1-B3), both SR and Control participants were given a 9-h nocturnal sleep opportunity (23:00–08:00). This was followed by a 7-night manipulation period (M1-M7), where the participants were given either 5 h (SR group; 01:00–06:00) or 9 h (Control group; 23:00–08:00) sleep opportunities. The protocol ended with 3 nights of 9-h sleep opportunity for both groups (R1-R3: 23:00–08:00). All participants were housed in a boarding school and slept in darkened, twin-share, and air-conditioned rooms.

Throughout the entire study duration, participants also completed a computerized cognitive test battery comprising 7 tasks. Further details of these tasks and analyses are detailed in our prior report.[26]

Polysomnography (PSG)

Sleep was recorded using portable EEG recording devices (SOMNOtouch RESP, SOMNOmedics GmbH, Germany). Recordings were obtained on 7 nights: B1 (adaptation), B3 (baseline), M1, M4, M7, R1, and R3. B1 recordings, which were performed to allow participants to adapt to new sleeping conditions, were not used in the present analysis. EEG was recorded from 2 main channels (C3 and C4 in the international 10–20 system of electrode placement) referenced to the contralateral mastoids. The common ground and reference electrode were placed at Cz and Fpz. Electrooculography (EOG; right and left outer canthus) and submental electromyography (EMG) were also used for sleep stage classification. Signals were sampled at 256 Hz and band-pass filtered between 0.2 and 35 Hz (EEG and EOG) or 1–128 Hz (EMG).

Sleep Staging and EEG Spectral Analysis

Sleep scoring was performed in 30-s epochs using the FASST toolbox.[28] Scoring was performed by trained technicians following the criteria set by The AASM Manual for the Scoring of Sleep and Associated Events.[29] The following sleep macrostructure parameters were computed: total sleep time (TST), duration of individual sleep stages (N1, N2, SWS, and REM),

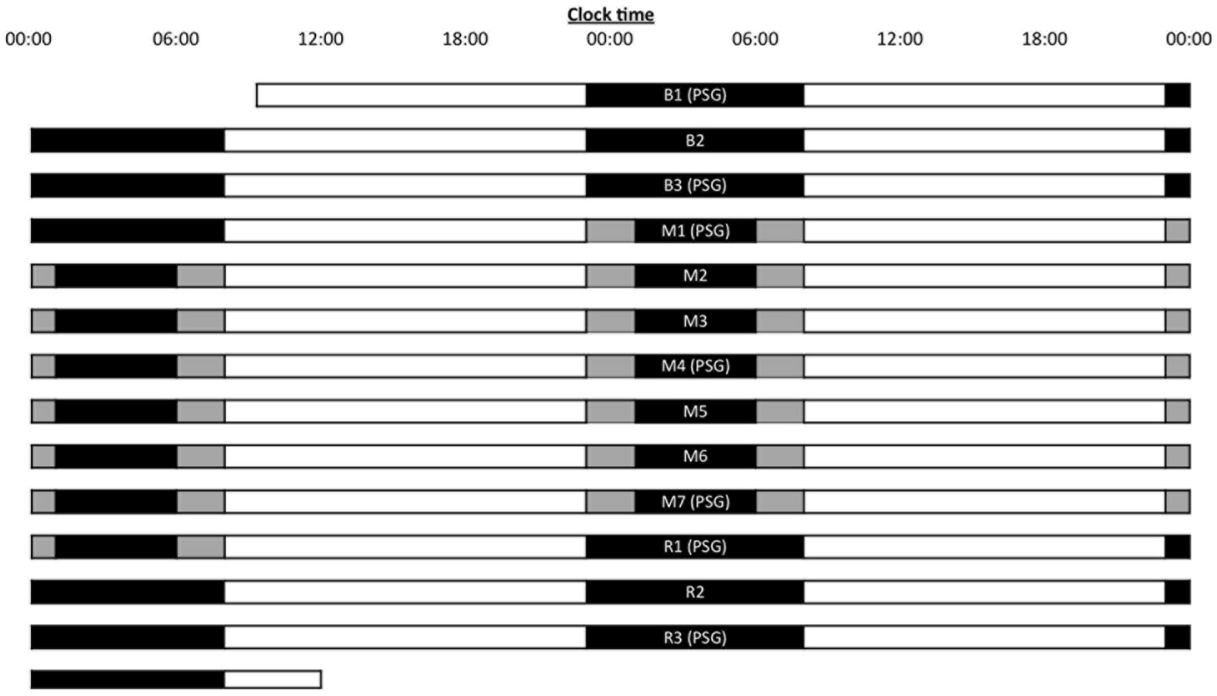

Figure 1—Two-week experimental protocol illustrated in a double raster plot. Both SR and Control groups underwent 3 baseline nights of 9 h sleep opportunity (B1 to B3), followed by 7 nights of either 5 h (SR; black bars) or 9 h (Control; gray bars) sleep opportunity (M1 to M7), and ending with 3 recovery nights of 9 h sleep opportunity (R1 to R3). Nights where polysomnography (PSG) was recorded are indicated on the plot.

sleep efficiency, wake after sleep onset (WASO), as well as sleep latencies for N2 (time from lights off to N2 sleep onset), SWS (time from sleep onset to the first N3 epoch), and REM sleep (time from sleep onset to the first REM epoch). These parameters were computed for (1) the entire night and (2) in the first 5 h of sleep in the SR and Control groups as previously described.[30] The latter procedure is more sensitive for detecting increases in sleep pressure as it considers a temporal window that is common across baseline, manipulation, and recovery nights.

EEG spectral analysis was performed on non-overlapping 5-s epochs using custom routines written in Matlab R2012a (The MathWorks, Inc. Natick, MA). Analysis was conducted primarily using C3/A2, unless data from C4/A1 was assessed as having fewer artifacts (10.7% of all records). For each epoch, power spectral density estimates were computed using Welch's modified periodogram method[31] (Hamming window; 0.2-Hz bin resolution) and spectral power was computed from 0.6 to 4 Hz using the trapezoidal rule for integral approximation. Following prior work,[18,30] total SWA summed across all NREM epochs was computed for (1) the entire night and (2) the first 5 h of sleep as a marker of sleep homeostasis and then expressed in percentage relative to baseline values. Mean SWA (total SWA divided by duration spent in NREM sleep) was also computed.

All records were visually inspected to identify artifact-free 5-s epochs. Recordings containing > 11% artifacts (mean ± SEM; 6.07% ± 0.14 %) from epochs scored as sleep were excluded from further analyses (35 of 336 total records from 56 participants who completed and complied with the protocol). Participants with > 2 unusable nights (macrostructure/spectral) or unusable B3 (spectral only) were removed from subsequent analyses. For sleep macrostructure, 29 SR and 26 Control entered the final analyses while for spectral data, 25 SR and 23 Control participants entered the final analyses.

Statistical Analysis

Statistical analyses were performed with SAS 9.3 (SAS Institute, Cary, NC). A general linear mixed model with PROC MIXED was used to investigate the effects of group, night and group × night interactions on macro-structure and SWA measures. Differences of least square means were used to determine significant differences between the 2 groups and across nights at $P < 0.05$.

RESULTS

Sleep Macrostructure

Between-Group Differences (SR vs. Control)

At baseline (B3), the 2 groups had comparable TST and sleep macrostructure (P's > 0.05) and differed only in the amount of WASO, which was on average 12 min longer in the SR group (mean ± SEM of SR: 29.38 ± 5.40 min vs. Control: 17.48 ± 3.87 min; $P = 0.005$). Throughout the course of the study, a group × night effect was observed across most sleep variables—TST, N1, N2, SWS, REM sleep, WASO, sleep efficiency, and N2 latency (Figure 2A–2H). There was no significant main or interaction effect for SWS latency (Figure 2I). REM sleep latency showed a main effect of night ($P < 0.05$), but the group

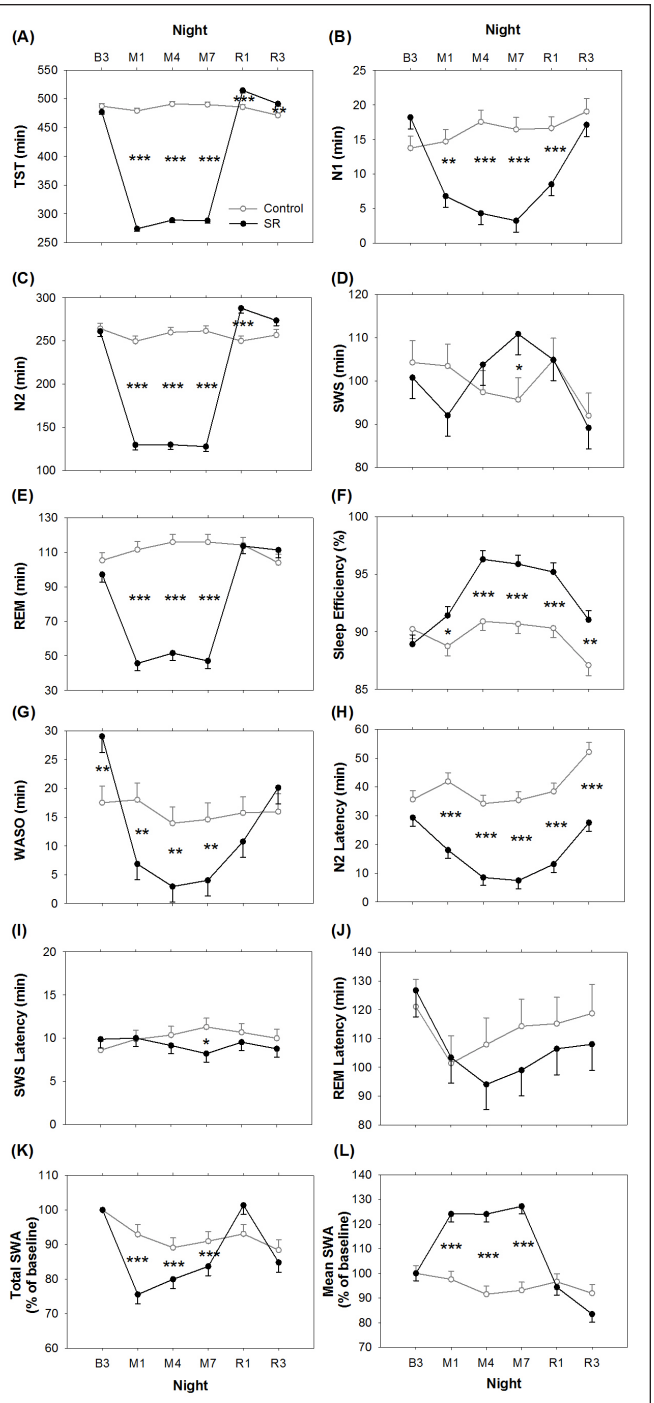

Figure 2—Effects of sleep restriction on sleep macrostructure and SWA power across experimental nights. Mean ± SEM of the SR (black filled circles) and the Control (gray open circles) groups were plotted for the last baseline night (B3), first, fourth, seventh sleep manipulation nights (M1, M4, and M7), and first and third recovery nights (R1 and R3). Asterisks indicate significant differences between groups for each night (*P < 0.05, **P < 0.01, ***P < 0.001).

and group × night interaction was not significant (P's > 0.43) (Figure 2J).

During the manipulation period, SR participants displayed shorter TST, N1, N2, REM sleep, WASO, and N2 sleep latency, as well as higher sleep efficiency, relative to the Control group. Although SWS duration was slightly elevated on M7 in the SR group (SR: 109.26 ± 3.33 min vs. Control: 97.17 ± 5.31 min; P = 0.03), mean SWS across manipulation nights did not differ between groups (SR: 101.10 ± 3.36 min vs. Control: 99.13 ± 5.42 min; P = 0.75).

On both recovery nights examined (R1 and R3), the SR group demonstrated longer TST, greater sleep efficiency and reduced N2 sleep latency than the Control group. The durations of N1, N2, SWS, REM sleep, and WASO were comparable between groups on R3.

Within-Group Differences (Changes from Baseline)

When sleep opportunity was reduced from 9 h to 5 h in SR participants, TST and durations of N1, N2, and REM sleep across the entire night decreased (Figure 3A–3C, 3E) relative to the last baseline night (B3). N2/REM sleep latency and WASO (Figure 3F–3G) were also reduced (P's < 0.05) reflecting increased sleep/REM sleep pressure. SWS latency remained at baseline levels throughout the experimental protocol (P's > 0.09). Duration of SWS (Figure 3D) dipped on the first night (P = 0.009) and then increased to above baseline levels (P = 0.004) on M7. However, the mean SWS duration across manipulation nights did not significantly differ from baseline (P = 0.76). As such, TST and all sleep macro-structure measures, except for SWS duration, were affected by our sleep restriction manipulation. Most parameters did not return to baseline levels on the first recovery night (Figure 3A–3G). N1 duration and N2 sleep latency only returned to baseline levels on R3, while TST, N2, and REM sleep duration remained elevated even on R3. TST was elevated by 14 min, N2 by 23 min, and REM sleep by 16 min on average compared to baseline levels (P's < 0.05). WASO was also reduced by 18 min on R1 and 9 min on R3 (P's < 0.05). SWS was at the baseline level on R1 but was 13 min less than baseline on R3 (P < 0.01), possibly to allow for REM rebound to occur.

When considering only the first 5 h of sleep in the SR group (Figure 3A–3G), we found that TST, SWS, and REM sleep durations significantly increased in the manipulation nights compared to baseline, while N1 duration was shortened. N2 duration remained unchanged. In the recovery period, TST, and durations of N1, N2, SWS, and REM sleep returned to baseline levels on R3.

Isolated changes from baseline also occurred in the Control group (Figure 4A–4G), but these were not systematic, and changes in the SR group were more prominent.

SWA Power

Between-Group Differences (SR vs. Control)

Throughout the course of the study, a group × night interaction effect was observed for both total and mean SWA (P < 0.001). In line with the reduction in sleep opportunity, *total* SWA in the SR group was *lower* across manipulation nights (P's < 0.001; Figure 2K) compared to the Control group. However, due to the predominance of SW power early in the night, and shorter NREM sleep (especially N1 and N2) during nights of 5h TIB, *mean* SWA was *higher* in the SR group compared to the Control (P's < 0.001; Figure 2L). Both total and mean SWA were comparable between groups during the recovery nights.

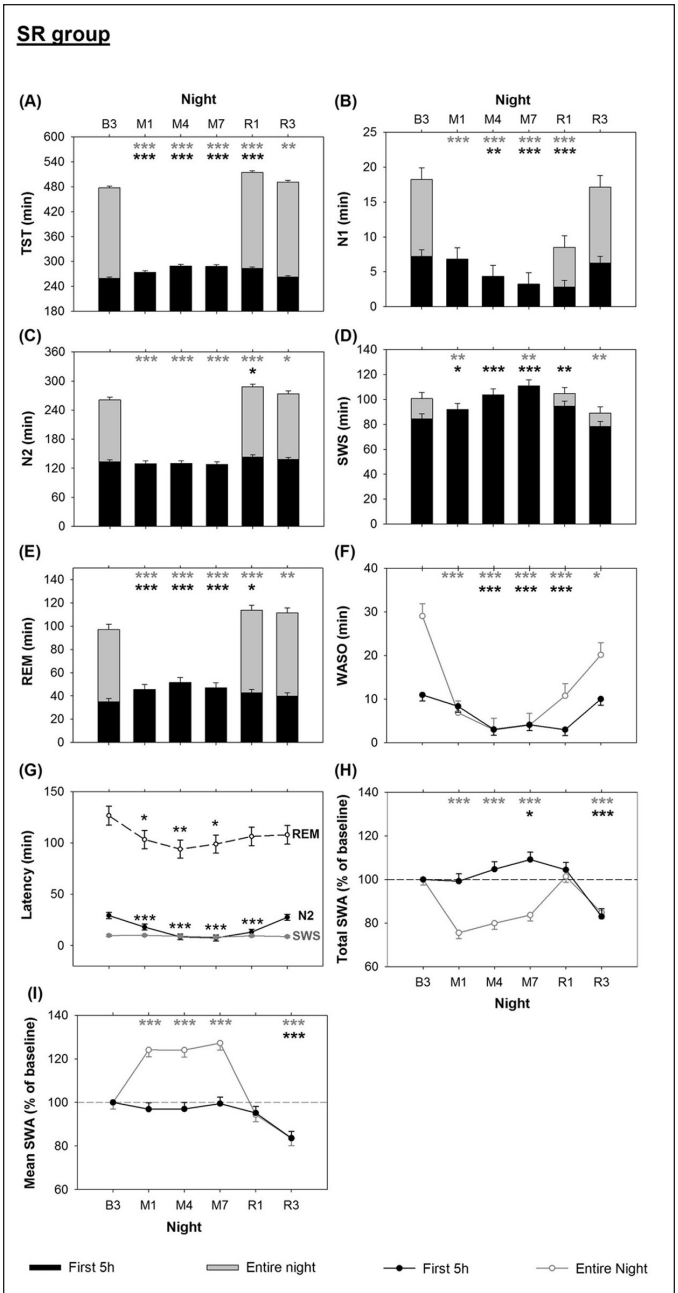

Figure 3—Sleep macrostructure and SWA power of the SR group. Black bars indicate mean ± SEM of sleep variables in the first 5 h of sleep for all nights, while gray bars indicate mean ± SEM in the remainder of full sleep (9 h for B3, R1, and R3). Black asterisks indicate significant differences from B3 considering only the first 5 h of sleep in all nights while gray asterisks indicate differences from B3 considering the full sleep opportunity. (*P < 0.05, **P < 0.01, ***P < 0.001).

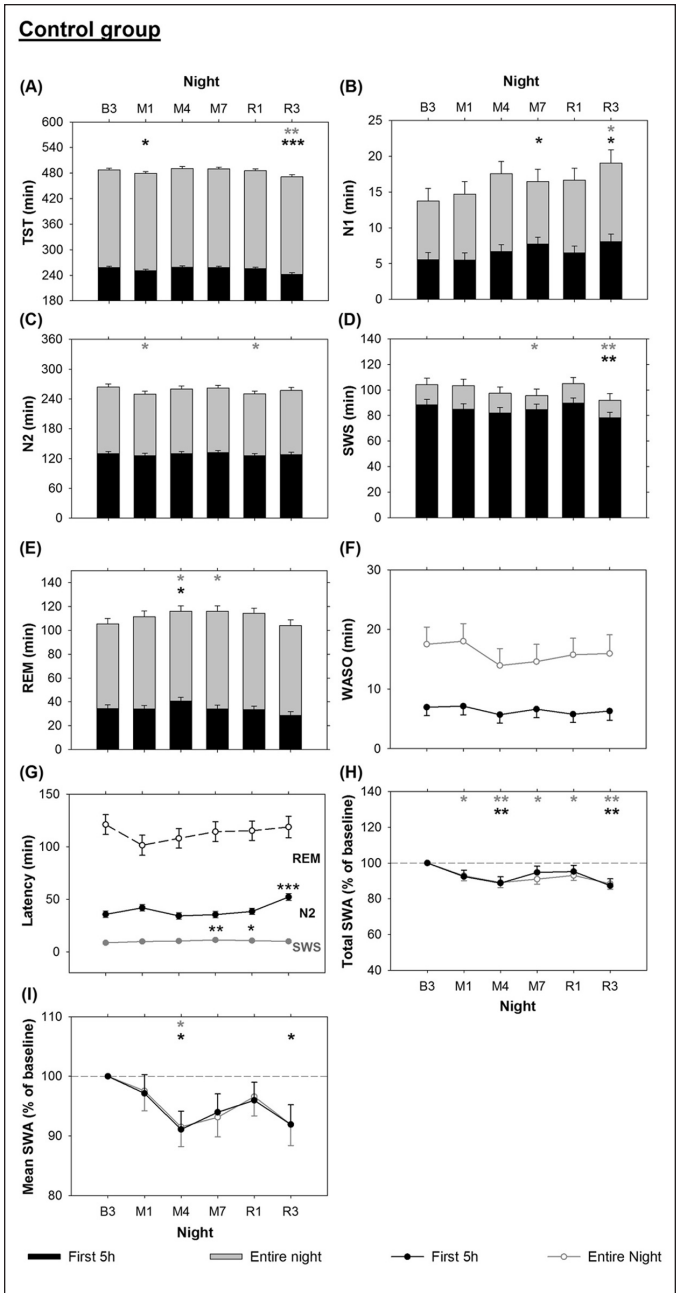

Figure 4—Sleep macrostructure and SWA power of the Control group. Black bars indicate mean ± SEM of sleep variables in the first 5 h of sleep for all nights, while gray bars indicate mean ± SEM in the remainder of full sleep (9 h for B3, R1, and R3). Black asterisks indicate significant differences from B3 considering only the first 5 h of sleep while gray asterisks indicate differences from B3 considering the full sleep opportunity. (*P < 0.05, **P < 0.01, ***P < 0.001).

Within-Group Differences (Changes from Baseline)

In the SR group (Figure 3H), *total* SWA power over the entire night (9 h on B3 vs. 5 h on M1-M7) was reduced in the manipulation nights compared to the baseline night, in line with the reduction in sleep opportunity. When considering only the first 5 h of sleep, and comparing this with the equivalent period in the baseline and recovery nights, total SWA increased on M7 compared to baseline (P = 0.03), returned to baseline on R1 and reduced below baseline levels on R3 (P < 0.001). In the Control group (Figure 4H), total SWA power across the entire 9 h period on manipulation and recovery nights was reduced from baseline levels (P's < 0.05) although as mentioned previously, the average total SWA power in the manipulation nights was still higher than that observed in the SR group (Control: 90.45% ± 2.0% vs. SR: 77.44% ± 2.8%, P < 0.001; Figure 2K). This appears to be a result of the additional 4-h sleep opportunity available to Control participants. When considering only the first 5 h of

sleep, total SWA was either reduced or did not differ from baseline levels.

In the SR group (Figure 3I), *mean* SWA power over the entire night (9 h on B3 vs. 5 h on M1-M7) *increased* on manipulation nights compared to the baseline night because as mentioned previously, SW power was higher in the first half of the night, and during sleep restriction, NREM sleep (particularly N1 and N2) duration was shortened. If SWA in the first 5 h of sleep was compared with that of an equivalent period in the baseline condition, mean SWA was found to be maintained throughout the manipulation period and on R1, and only reduced below baseline on R3 ($P < 0.001$). Isolated nights of reduced mean SWA power were observed in the Control group (Figure 4I).

DISCUSSION

In the largest PSG-verified, multiple-night sleep restriction and recovery sleep study on older adolescents to date, nocturnal sleep architecture was found to be altered similarly to that previously reported for healthy young adults. Across 7 nights of 5 h TIB, N2 sleep latency, REM sleep latency, and WASO were reduced alongside relative increases in TST and durations of REM and SWS in the first 5 h of sleep. REM, N2, and TST showed a delayed and prolonged rebound and did not return to baseline levels even by the third recovery night. In addition, REM sleep pressure appeared to build quickly evidenced by reduced REM sleep latency across sleep restriction nights and prolonged REM sleep rebound on recovery nights.

Adolescents Display Preserved Homeostatic Sleep Drive following Sleep Restriction

SWS and SWA demonstrate homeostatic regulation[32] following total[33,34] or partial sleep deprivation.[16,35] In adults, the duration of SWS/SWA was maintained against a backdrop of curtailed N2 and REM sleep across 14 nights of restriction to 4 h TIB.[18] Similar selective preservation of SWS occurred in the recovery nights that followed 7–8 nights of 3 h sleep opportunity.[17,36] As the adolescent brain is often thought of as highly plastic and susceptible to environmental influences,[37] it might be predicted that recurrent sleep restriction would alter sleep homeostasis, resulting in better tolerance of sleep loss over time.[38]

Instead, we found that even in our sample of adolescents who had a habitual TIB of approximately 6 h on school weekday nights (Table S1) SWS duration and SWA were robustly maintained. This effect was particularly evident when considering the first 5 h of sleep, when similar to young adults,[30] adolescents showed *increases* in SWS and total SWA across nights of sleep restriction compared to baseline levels. This increase in total SWA appears to be related to an increase in SWS duration in the first 5 h of sleep, as mean SWA across same period was maintained across nights of sleep restriction.

The somewhat unexpected small decrease (instead of increase) in total SWA/SWS observed from M7 to R1 (Figure 2D) might reflect the current experimental design rather than an anomaly of sleep homeostasis. In order to minimize shift in circadian phase, sleep restriction was implemented by aligning the midpoints of the 5-h and 9-h sleep periods, such that bedtimes were delayed and wake times advanced by 2 h, respectively. This resulted in a 2 h longer duration of preceding wakefulness in the SR group (19 h) during the manipulation period (including M7) compared to the recovery (17 h; M7-R1) period (Figure 1).

Ample Sleep Opportunity on 3 Recovery Nights may be Insufficient for Adolescents

In prior work investigating sleep restriction in adults, recovery in terms of restitution of specific baseline sleep stage durations, was complete by the first[17] or third[16] recovery night. In the present work, the amounts of TST, N2, and REM sleep remained elevated on R3 in the SR group (Figure 3A, 3C, 3E), indicating that 3 nights of 9 h recovery sleep might have been insufficient to fully reverse the effects of cumulated sleep loss. This statement is based on a within-group comparison in the SR group contrasting R3 and B3 (Figure 3), which would be less confounded by inter-individual differences than a between-group comparison (which might be inferred from Figure 2).

One solution to this incomplete recovery could perhaps be to ask adolescents to simply sleep in longer (e.g., on weekends) to increase recovery time.[39] Unfortunately, adolescents do not readily adjust to advances in sleep schedule.[40] Delaying weekend bed and wake times as is commonly practiced has been shown to lead to circadian phase delay in the ensuing week,[41] potentially exacerbating sleep loss. Supporting this point, postponing bedtime by 1.5 h and wake time by 3 h on Friday and Saturday in 10th and 11th graders resulted in delayed melatonin onset the following Sunday.[42,43]

Cumulative REM Sleep Pressure in Adolescents across Sleep Restriction Nights Exhibits Prolonged Recovery

The reduction in REM sleep latency across sleep restriction nights suggests a buildup of REM sleep pressure. Relative to the constancy of SWS duration during sleep restriction, a substantial REM sleep deficit accumulated across manipulation nights (Figure 3E). Perhaps as a trade-off for SWS preservation, there was reduced REM sleep opportunity until R1, when REM sleep duration showed a rebound over baseline levels in the SR group.

NREM sleep compensation has been shown to take priority over REM sleep,[16,36] suggesting that REM sleep restitution might be impeded under conditions of elevated slow wave pressure. For example, REM sleep has been shown to occur at a time when slow wave pressure was either low at the end of sleep[44] or much less increased than REM sleep pressure.[35]

REM sleep rebound on recovery nights could be beneficial to the maturing brains of adolescents. The proportion of sleep occupied by REM sleep increases from childhood to adolescence before decreasing in older persons.[45] REM sleep is important for consolidation of procedural memory[46,47] and modulating affective memories,[48] both of which are relevant to students.

Limitations and Future Directions

The relatively long N2 sleep latency of ~30 min during baseline nights merits comment. Given that adolescents in this sample reported habitual bed times close to midnight on average (Table S1), it is possible that the prolonged sleep latency

observed is a consequence of the relatively early lights-out of 23:00 taking place during a (delayed) wake maintenance zone. Perhaps sleep latency could have been shorter (and sleep duration longer) if a had a later bedtime been scheduled.

As the experimental protocol was designed to minimize the effects of circadian phase shifting by aligning the mid-points of the baseline, manipulation, and recovery nights, analysis of sleep macrostructure could be subject to circadian influences, as participants went to bed 2 h later on manipulation nights. This meant that bedtimes of the SR and Control group were out of and in the wake maintenance zone respectively, possibly contributing to increased sleep latency in the latter group. In addition, an increase in REM sleep priority in association with late nights has been shown[49] and could result in increased time spent in REM sleep, interfering with SWS maintenance and as a result, reduce SWS duration.[30,50] This could explain the slight dip in SWS duration from B3 to M1 and increase in REM duration in the first 5h of sleep on M1 compared to B3 in the SR group.

A parallel-group design was employed here to keep the duration of the experiment acceptable to parents and students, but a crossover design, where each participant serves as his or her own control, would have been ideal to examine inter-individual differences in adolescents' vulnerability to sleep restriction.

Our finding that some sleep parameters did not return to baseline levels even by the third recovery night highlight the need for further investigations into the recovery process of sleep-restricted adolescents.

CONCLUSION

In spite of preservation of SWS duration, adolescents demonstrate residual effects on sleep architecture following multiple nights of sleep restriction. Recovery to baseline sleep architecture was not achieved by the third night. As adolescents form habits that could persist throughout their adult lives, parents, educators, and policy makers would do well to consider the perils of chronic voluntary sleep loss and their remedy.

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