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**Original Article**

## Original Article

# Extending weeknight sleep duration in late-sleeping adolescents using morning bright light on weekends: a 3-week maintenance study

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## Abstract

**Study Objectives:** Our sleep extension intervention in adolescents showed that gradually shifting weekday bedtime earlier plus one weekend of morning bright light advanced circadian phase and increased weeknight sleep duration. Here, we examine at-home maintenance of these changes.

**Methods:** Fourteen adolescents (15.3–17.9 years; 7 female) completed a 7-week study. After usual sleep at home (2-week baseline), intervention participants ($n = 8$) gradually advanced weekday bedtime (1 hour earlier than baseline during week 3; 2 hours earlier in week 4) and received bright light (~6000 lux; 2.5 hours) on both mornings of the intervening weekend. During three maintenance weeks, intervention participants were instructed to maintain their school-day wake-up time on all days, keep their early week four bedtimes, except on weekends when they could go to bed up to 1 hour later, and get a 2.5-hour light box exposure within 5 minutes of waking on one morning (Saturday or Sunday) of both weekends at home. Control participants ($n = 6$) slept as usual at home and did not receive weekend bright light. Dim light melatonin onset (DLMO) was measured after the 2-week baseline, 2-week intervention, and 3-week maintenance in all participants. Actigraphic sleep–wake was collected throughout.

**Results:** After the 2-week intervention, DLMOs advanced more compared to control (37.0 ± 40.0 minutes vs. –14.7 ± 16.6 minutes), weekday sleep duration increased by 69.7 ± 27.8 minutes and sleep onset was 103.7 ± 14.2 minutes earlier compared to baseline. After three maintenance weeks, intervention participants showed negligible DLMO delays (–4.9 ± 22.9 minutes); weekday fall-asleep times and sleep durations also remained stable.

**Conclusions:** Early circadian phase and extended sleep can be maintained with at-home weekend bright light.

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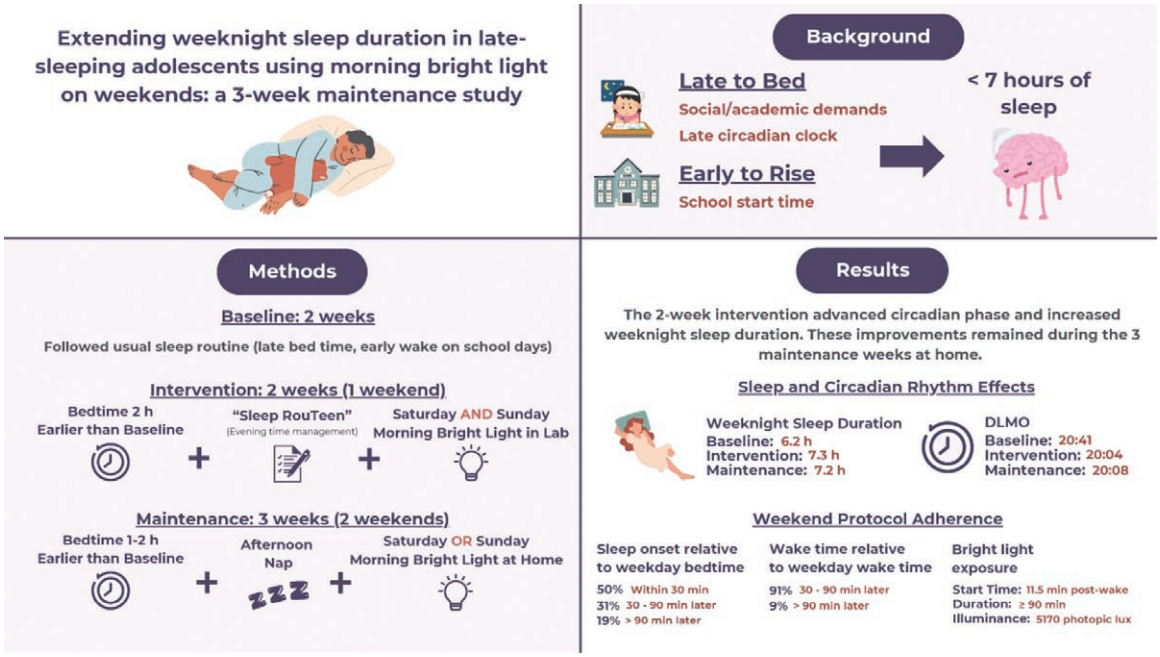
## Graphical Abstract




**Keywords:** delayed sleep onset; adolescence; bright light; DLMO; circadian rhythms; phase shifts; school start time; sleep extension

### Statement of Significance

Most adolescents are chronically sleep-deprived on school nights, getting far less sleep than the recommended 8–10 hours/night. This is partly due to developmental changes making their internal circadian (~24-hour) clocks and thus fall asleep times later and in conflict with high school start times that are too early. Although advocacy efforts to shift school start times later have made progress, not all regions have adopted this change, or these times are still too early for some adolescents. We present behavioral strategies for late-sleeping adolescents to reset their circadian clocks to an earlier clock time with bright morning light administered on the weekend to maintain these early times and increase school night sleep duration at home.

A majority of adolescents obtain less than the recommended [1] 8 to 10 hours of sleep on school nights [2–4] due to a combination of biological, psychosocial, and societal pressures that impinge on sleep opportunity [5, 6]. Late sleep onset times associated with mid-to-late adolescence are partly driven by a puberty-related slowing of homeostatic sleep pressure buildup across waking [7], a delay shift of the central circadian timing system [8, 9], and potentially a robust circadian-driven wake maintenance zone around the time of the dim light melatonin onset (DLMO) [10]. Late school-night sleep onset times with early rise times on school-day mornings make it difficult for adolescents to obtain sufficient sleep [11, 12]. Chronic partial sleep deprivation is especially common for adolescents who endorse an evening chronotype [13]. The conflict between biologically driven late sleep onset times and school-driven early wake times also results in circadian misalignment (mistimed sleep relative to the circadian system) on school nights [14] and social jetlag [15, 16]. Insufficient and mistimed sleep in adolescents is associated with a number of poor outcomes, including heightened anxiety [17] and depressed mood [18–20], poor emotion regulation [21], daytime sleepiness [14, 22–26], and poor academic performance [18, 27–32].

We recently reported on a sleep-extension intervention in high-school-aged adolescents who had late bedtimes (school night ≥23:00; non-school ≥ midnight) and short (≤7 hours) school-night sleep duration [33]. In that 4-week protocol, participants slept as usual at home during the school year for 2 baseline weeks. During the subsequent 2 weeks, participants in an intervention group gradually shifted their school-night bedtime earlier (advanced) from their own baseline; they were instructed to go to bed 1 hour earlier in week 3 and another hour earlier in week 4. An individualized time-management strategy ("SleepRouTeen") with behavioral goals and sleep health recommendations (e.g. avoid caffeine in the afternoon) was implemented to facilitate an earlier bedtime. Participants in the Intervention group also received bright light in the laboratory on both mornings of the intervening weekend (between weeks 3 and 4). In response to these interventions, circadian phase advanced by an average of 36 minutes, sleep onset advanced by an average of 90 minutes, and school-night sleep duration was more than 60 minutes greater than baseline, on average. A control group, which was not given any instruction about their sleep/wake times or time management, and did not receive weekend morning bright light, did not show a systematic change in their DLMO phase, sleep onset time, or sleep duration.

Our intervention to lengthen sleep duration was effective in the short term; however, it was unclear whether adolescents could maintain an early circadian phase and sleep onset time

after completing the 2-week intervention. Indeed, one weekend of sleeping later can delay rhythms by Monday morning [34] and quickly reverse a circadian phase advance obtained in response to the intervention. A feasible long-term maintenance strategy is needed to keep circadian phase early and facilitate an early sleep onset on school nights. The weekend morning bright light exposure that we tested in the intervention was intended to be a one-time bolus of bright light to quickly reset rhythms ("reset weekend"). The light duration was 2.5 hours because this was the most effective bright light duration to advance circadian rhythms in one weekend when we compared it to 1.5 hours of bright light and room light [35]. The bright light started 2 hours after the midpoint of baseline sleep on Saturday morning (started between 04:30 am and 09:00 am, depending on the individual) and then started one hour earlier on Sunday because these times target the most sensitive portion of the advance region of the adolescent phase response curve (PRC) to light [36]. Following the reset weekend, we predicted that an early bedtime, early wake-up time and presumably any outside morning light exposure during the following school week could help maintain the phase advance. This bright light treatment protocol with an early wake-up time every weekend morning; however, is not sustainable for most adolescents. We propose that one way to maintain an early circadian phase (and thus early sleep onset) is to keep the early bedtime and wake-time schedule on weekdays and then introduce some flexibility in the schedule on subsequent "booster" weekends.

In this follow-up study, we examined whether changes to sleep and circadian phase in response to our intervention are maintained in a 3-week maintenance protocol in which bright light is used on only one weekend morning (Saturday or Sunday) instead of two (Saturday and Sunday). In addition, weekend bedtimes were allowed to be 1 hour later. Because weekend wake-up times were kept the same as school days, we utilized afternoon naps to minimize sleep loss when bedtimes were late. Moreover, to assess feasibility of this maintenance protocol, all procedures were completed in the adolescent's home with adherence measures in place. Compared to a control group who continued to sleep as usual for 3 weeks without instruction, we hypothesized that circadian phase measured by the DLMO and sleep onset measured from wrist actigraphy would be maintained in the intervention group and remain earlier than the control group.

# Methods

## Participants

Healthy adolescents (14.0 to 17.9 years) enrolled in high school and who reported an average sleep duration of ≤7 hours on school nights, average school-night bedtime ≥23:00, average non-school night bedtime ≥ midnight, and average sleep duration ≥1 hour longer on non-school nights than school days were included in the larger intervention study [33] and then agreed to continue to participate in this 3-week follow-up maintenance study. Participants were invited to continue in the 3-week maintenance study during the fourth week of the larger intervention study. Inclusion and exclusion criteria were reported previously [33]. In brief, adolescent participants were healthy with no history of psychopathology, chronic illness, or sleep disorder. Elevated depressive symptoms (scores >16 on the Center for Epidemiological Studies-Depression [CES-D] scale) [37] were not exclusionary, but a history of suicidal ideation was. Participants did not work night shifts or travel beyond three time zones in the month before starting the study and were not color blind or deficient as measured by the Ishihara Color Blindness test [38]. Participants reported not taking any medication.

The study was approved by the Rush University Medical Center's Institutional Review Board, in adherence with the Declaration of Helsinki. A parent of the participant provided written consent for the child to participate in the study, and the adolescent cosigned the consent form to acknowledge assent. The study was registered on clinicaltrials.gov (NCT#04087603). Intervention participants were paid $400 and Control participants were paid $250 for completing the 3-week maintenance study. The payment was not tied to adherence.

## Study protocol

Participants completed the 51-day protocol illustrated in Figure 1 during the school year. Participants slept at home as usual for the first 14 nights ("baseline") and then lived in the laboratory for a weekend (days 15–16 in Figure 1), during which the baseline DLMO, the most reliable phase marker of the central circadian clock [39], was measured. Following the baseline lab weekend, the 2-week sleep extension intervention (days 17–28 highlighted in green in Figure 1) commenced for the intervention group. A control group slept at home as usual without instruction about their evening activities, sleep timing, or light exposure. Final DLMO was measured in the laboratory on day 30 in both groups to determine the immediate effects of the intervention on DLMO phase. See our previous report for more details of the sleep extension intervention [33].

Before leaving the laboratory after the final lab weekend, intervention participants were instructed to maintain their early bedtime on weekdays for the next 3 weeks (maintenance weeks 1, 2, and 3 highlighted in orange in Figure 1), which was scheduled to be 2 hours before their baseline weekday sleep onset. Scheduled weekday wake-up time remained the same so that adolescents could report to school on time. To facilitate maintenance of a bedtime that was 2 hours earlier than their baseline schedule on weekdays, participants were encouraged to follow their individualized Sleep RouTeen time management goals that they established during the 2-week sleep extension intervention. Naps were not allowed on weekdays. On two "booster" weekends (days 36–37 and 43–44 in Figure 1), intervention participants were allowed to go to bed up to 1 hour later than their scheduled bedtime on Friday and Saturday nights to allow for socializing or other activities on the weekend. If they chose to go to bed later, they were asked to take an afternoon nap on Saturday or Sunday during a 2-hour "nap zone" centered 12 hours after the midpoint of their nocturnal weekday sleep on days 24–28. The timing of this nap should have little effect on circadian phase [40] but provided an opportunity for the adolescent to sleep more on the weekend as morning sleep-ins were not allowed.

Intervention participants were given a bright light box (12" × 24" × 3½"; Bio-Light Ultra 10 000 LUX Light Therapy System, Enviro-Med, Vancouver WA) to use at home during the two "booster" weekends (on days 36 or 37 and on days 43 or 44 in Figure 1). Intervention participants were asked to choose one morning of each weekend (Saturday or Sunday) to sit in front of the light box within 5 minutes of waking for 2.5 hours. A light sensor (Actiwatch-L, Mini Mitter Co., Inc., Bend, Oregon) was hidden inside the light box to determine when the light box was on and off. Participants were allowed to take a break from sitting by the light box after the light had been on for at least 30 minutes and they were asked to try not to leave the light box for more than 10 minutes. Participants recorded clock times of when they

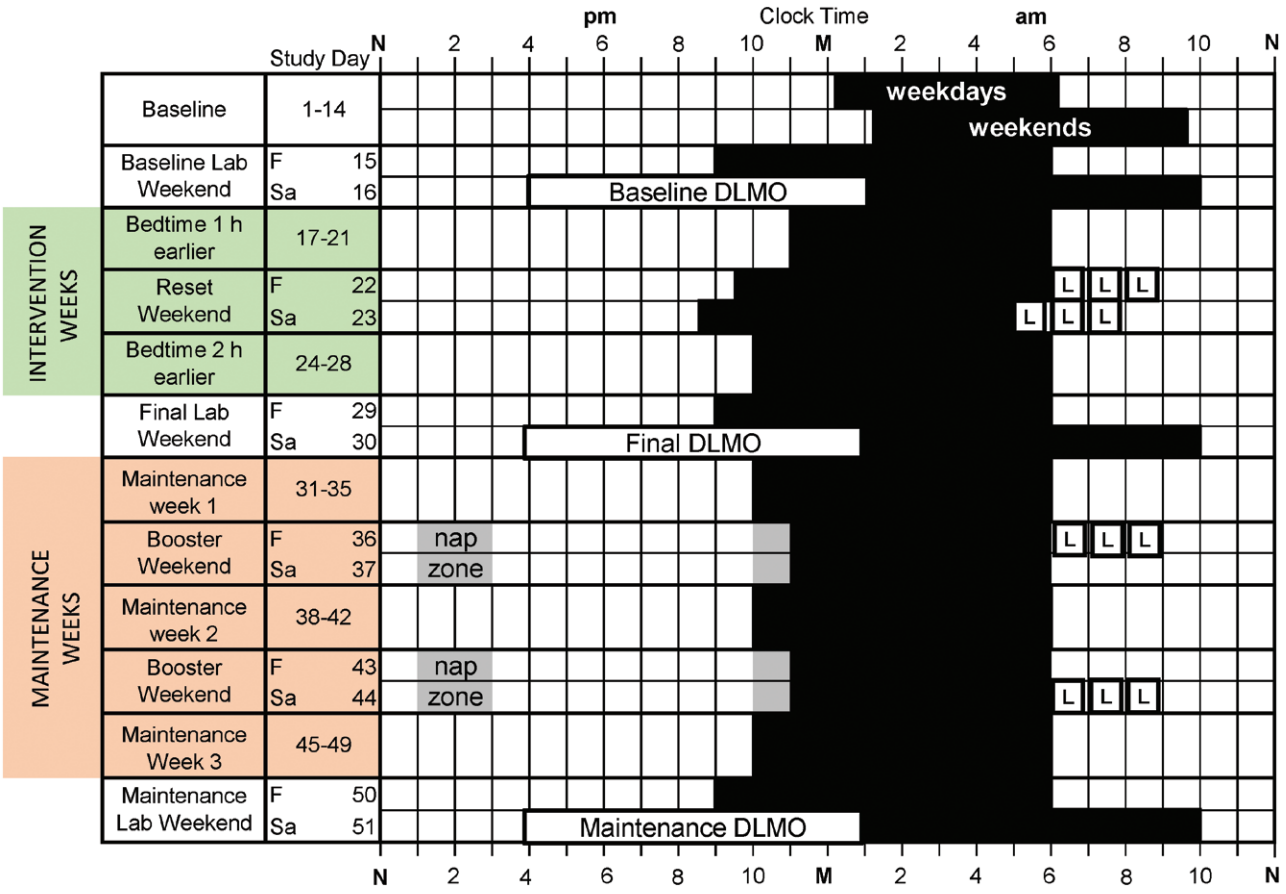



**Figure 1.** Example protocol for intervention participants. M, midnight; N, noon. Black rectangles illustrate dark/sleep. Participants had two baseline weeks of usual sleep at home (days 1–14). Average baseline sleep onset and wake-up times on weekday and weekend nights are shown. Days of the 2-week intervention [33] are shown as days 17–28. Participants in the intervention group were instructed to go to bed and try to fall asleep 1 hour earlier than their average baseline school-night sleep onset on days 17–21 (intervention week 1) and 2 hours earlier than baseline on days 24–28 (intervention week 2). For example, for an individual with a baseline weeknight sleep onset of midnight, they were instructed to go to bed at 23:00 on days 17–21 and at 22:00 on days 24–28. Individualized behavioral goals ("Sleep RouTeen") were identified for each participant to help them manage their time in the evening and facilitate earlier bedtimes during these weeks. Intervention participants lived in the laboratory during the intervening weekend (Reset Weekend, days 22–23) to receive 2.5 hours intermittent bright light (three 50-minute exposures with 10-minute breaks between) from light boxes (~6000 lux) on both mornings to advance circadian rhythms. Maintenance days are on days 31–49. Intervention participants were instructed to keep their bedtime 2 hours earlier than their baseline and wake up at their average school day wake time for 3 weeks. On two booster weekends (days 36–37 and days 43–44), participants were allowed to go to bed up to 1 hour later, but wake-up time was stable and set to their average school-day wake time. If they went to bed late, they were instructed to take a nap during a 2-hour nap zone centered 12 hour after the midpoint of their nocturnal sleep. On one morning of each booster weekend (day 36 or 37 and days 43 or 44), intervention participants were instructed to sit in front of a loaned light box within 5 minutes of waking at home for 2.5 hours. Control participants slept as usual at home during the intervention and maintenance weeks, did not receive the "Sleep RouTeen" behavioral goals, and did not receive bright light from a light box. All participants lived in the laboratory for 3 weekends (Baseline, Final, and Maintenance Lab Weekends). On Friday (days 15, 29, and 50), a 9-hour sleep opportunity was timed to end at the individual's average school-day wake-up time. Dim light melatonin onset (DLMO) was measured on Saturday (day 16, 30, and 51) via serial saliva sampling every 30 minutes starting at 1600 and ending at the individual's average weekend bedtime (±30 minutes). Participants were given a 9-hour sleep opportunity in the laboratory after the DLMO phase assessment.

turned the light box on, when were away from the light box, and when the light box was turned off on a paper log (Supplementary Materials). The light box was equipped with a 53-cm string anchored to the top and a bead at the opposite end of the string. To ensure that participants were sitting close enough to the light box, they were instructed to sit comfortably in front of the light box and hold the bead at the end of the string next to their eye so that the string was taut. They were allowed to complete homework, read, use a phone or tablet, or watch television while sitting in front of the light box; however, they were instructed not to block the light box with these activities (i.e. with a book, tablet or laptop). Participants were allowed to offset the light box to the left or right, but we asked them to ensure that the light box was angled toward their faces and eyes. Participants wore a light sensor on a string around their necks to measure illuminance levels and were instructed to make sure that the sensor was facing out when the light box was turned on.

The control group was not given any instruction about the sleep schedules or time management just like the main study, and they were not given a bright light box to take home. After the final lab weekend, they were instructed to continue wearing their actigraph and complete their daily sleep logs on days 31–49. Both groups completed weekly appointments to review their data and the study team provided feedback and encouragement. Participants were not dropped during the maintenance weeks for not adhering to the sleep schedule or bright light instructions, but rather adherence was measured to understand feasibility of this approach.

All participants lived in the laboratory during the Maintenance Lab Weekend (days 50–51), during which the maintenance DLMO

was measured to assess the stability of circadian phase after the 3 maintenance weeks.

## DLMO phase assessments

During circadian phase assessments (days 16, 30, and 51), salivary melatonin concentration was measured from approximately 2 mL of saliva collected every 30 minutes using Salivettes (Sarstedt, Nümbrecht, Germany). Participants remained awake in dim light (<5 lux) sitting in recliners, except when they needed to use the attached washroom (also <5 lux). They were not allowed to eat or drink in the 10 minutes before each sample and washroom trips were not allowed during this time. Saliva samples were centrifuged immediately after collection and frozen. These samples were later radioimmunoassayed (RIA) for melatonin concentration using commercially available kits (Bühlmann Laboratories AG, Schönenbuch, Switzerland) by SolidPhase, Inc (Portland, ME). The manufacturer reports that the analytic sensitivity (limit of detection) of the assay is 0.9 pg/mL. Intra-assay coefficients of variation for low (daytime), medium (evening), and high (nighttime) levels of salivary melatonin are 20.1%, 4.1%, and 4.8%, respectively. The inter-assay coefficients of variation for low, medium, and high levels of salivary melatonin are 16.7%, 6.6%, and 8.4%, respectively. DLMO phase, expressed in 24-hour clock time, was determined by linear interpolation across the time points before and after the melatonin concentration increased to and stayed above 4 pg/mL [8, 41].

DLMO phase shifts were computed to determine (1) the acute phase-shifting effects of the sleep extension intervention (Baseline DLMO on day 16—Final DLMO on day 30); and (2) whether the circadian phase changed after the 3-week maintenance protocol (Final DLMO on day 30—Maintenance DLMO on day 51). Positive numbers indicate a phase advance.

## Actigraphic sleep

Throughout the 51-day study, participants in both groups wore an actigraph (Actiwatch Spectrum, Philips Respironics, Inc., Bend Oregon, USA) on their non-dominant wrist and completed daily sleep logs to record the time they got into bed, the time they tried to fall asleep, wake time, and sleep disturbances. They also telephoned daily to a time-stamped voicemail messaging system at bedtime and wake time. Participants visited the lab each week so that we could download the actigraphy data and review sleep logs with them; participants were questioned about any inconsistencies between the actogram and sleep logs.

Actigraphy data were collected in 1-minute epochs. The low wake threshold and the sleep epochs sleep interval detection algorithm in Actiware 6 (version 6.0.9, Philips Respironics, Inc., Bend Oregon, USA) were used to determine sleep and wake. In a validation study against PSG, Meltzer and colleagues [42] showed that low sensitivity is the best threshold for healthy adolescents aged 13–18 years. Each sleep episode was manually inspected within a rest interval beginning 15 minutes before the participants' reported try to fall asleep time and ending 15 minutes after their reported wake-up time on their daily sleep log. The first of 3 consecutive 1-minute epochs of sleep defined sleep onset and the first epoch after the last 5 consecutive 1-minute epochs of sleep defined wake-up time [43]. The following variables were derived separately for weekdays and weekends: sleep onset time, wake-up time, total sleep time (total hours of sleep scored by the program between sleep onset and wake-up time), and duration (total hours between sleep onset and wake-up time). Naps on booster weekends were scored using the same method as nocturnal sleep.

Deviations from scheduled bedtime and scheduled wake-up time were computed for maintenance weeks (weekdays) and booster weekends.

## Booster weekend morning bright light exposure

A second actigraph with a light sensor (Actiwatch Spectrum, Philips Respironics, Inc.) and without the wristband was mounted on a lanyard and strung around participants' necks like a medallion to measure ambient white light (in photopic lux). The sensor was positioned at the center of the chest. All participants wore light sensors, but here we examine the ambient light data for the intervention group only to ensure fidelity of the booster weekend morning bright light exposure intervention.

Booster weekend morning bright light box exposure was measured using three methods: (1) the self-report weekend light box logs (Supplementary Materials); (2) the light sensor around their neck ("neck light sensor"); and (3) the light sensor hidden inside their light box ("light box sensor"). For the weekend light box log, total minutes were summed between self-reported lights-on clock time to the minute before their self-reported lights-off clock time. If the participant reported leaving the light box, those minutes were excluded from the sum. For both light sensors, the total number of minutes that the bright light box was on (defined as illuminance readings ≥500 photopic lux) was examined from the scheduled wake-up time to 2 hours 35 minutes after the scheduled wake-up time because they were instructed to turn the bright light box on within 5 minutes of waking and sit in front of the light box for 2 hours 30 minutes. The total number of minutes in which illuminance readings ≥500 lux were summed separately for the neck light sensor and the light box sensor. Average photopic illuminance (in lux) was computed from wake-up time until 2 hours and 35 minutes after wake from the neck light sensor only.

Participants were instructed to start the bright light box within 5 minutes of waking. Therefore, we also examined when booster weekend bright light started relative to their scheduled wake-up time using self-reported lights-on time (weekend light box logs), the clock time of the first epoch when illuminance ≥500 lux after scheduled wake-up time on the neck light sensor, and the clock time of the first epoch when illuminance ≥500 lux after scheduled wake-up time on the light box sensor. Finally, because intervention participants were instructed not to leave their light box within the first 30 minutes of turning it on, we computed the number of minutes illuminance ≥500 lux and average illuminance from the neck light sensor only in the first 35 minutes after the scheduled wake.

## Statistical analyses

One female in the Control group completed the maintenance protocol during the transition from standard time to daylight savings time (DST). All DLMOs were adjusted to be in DST to compute phase shifts. For this participant, the week of actigraphic sleep data following the change to DST (maintenance week 2) was omitted from the analysis, and the remaining actigraphic sleep data measured during baseline, the 2-week sleep extension intervention, and the first week of the maintenance protocol were adjusted to DST. One male in the Control group had missing actigraphy data during the first maintenance week 1 due to the failure of the actigraph.

We employed linear mixed models (LMMs) to analyze changes in actigraphic sleep outcomes (sleep onset time and sleep duration) and circadian phase (DLMO) across Baseline, Intervention,

and Maintenance time points. For each outcome, we compared a baseline model to a random intercepts model, with the latter consistently providing a better fit across all measures. This was indicated by lower AIC and BIC values and significant likelihood ratio tests (all $p < .001$). Therefore, we report results from the random intercept models, which include group, time, and their interaction as fixed effects, with a random intercept for each participant. The interactions between group and time are central to our analysis. These interactions reveal whether the Intervention and Control groups show different patterns of change over the study period. Significant interactions during the intervention weeks (compared to baseline) indicate that the intervention had an effect. Similarly, significant interactions during the maintenance weeks suggest that the effect of the intervention persisted over time. Fixed effects for time were relative to baseline, and we conducted pairwise comparisons using estimated marginal means to compare each time point. Group means and standard errors at each time point are illustrated in figures and tables for clarity and simplicity. Spaghetti plots of the individual data are also included in Supplementary Figures S1-S3). Post hoc *t*-tests and effect sizes (Cohen's *d* = group mean difference/pooled SD) to illustrate group differences in sleep and circadian outcomes at each time point are also noted in figures and tables.

# Results

## Participants

Forty-six adolescents completed the first 4 weeks of the study [33]. Of those adolescents, 34 were invited to complete the 3-week maintenance study and 12 were not invited because the maintenance study coincided with the beginning of summer vacation when adolescents were no longer attending school. Of the 34 adolescents invited, 18 declined to participate (seven for schedule conflicts with the maintenance lab weekend and 11 did not provide a reason). Therefore, 16 participants (eight biological females; eight biological males) enrolled in the 3-week maintenance study immediately after they completed the 4-week study. Participants self-identified their race as African American ($n = 7$), white ($n = 7$), or multiracial ($n = 2$). Nine participants identified as non-Hispanic and seven identified as Hispanic/Latinx. Race and ethnicity were confirmed by a parent or guardian. The group of adolescents who did not complete the maintenance study ($N = 30$) showed a similar demographic distribution for sex assigned at birth ($n = 14$ males, $n = 16$ females; $\mathbf{X}^2$ (1, $N = 46$) = 0.05, $p = .83$), self-identified race ($n = 11$ African American, $n = 13$ white, $n = 3$ multiracial, $n = 3$ another race; $\mathbf{X}^2$ (3, $N = 46$) = 1.79, $p = .62$), and ethnicity ($n = 20$ non-Hispanic; $n = 10$ Hispanic/Latinx; $\mathbf{X}^2$ (1, $N = 46$) = 0.49, $p = 0.49$). Compared to the participants that did not continue in the maintenance study ($N = 30$), the maintenance sample also did not differ in age ($t(44) = 0.82$, $p = .42$), morningness score ($t(43) = -0.91$, $p = .37$), Midsleep time on Free Days (MSFsc; $t(37) = -0.59$, $p = .56$), social jetlag ($t(37) = -0.27$, $p = .80$), depressive symptoms ($t(44) = 0.11$, $p = .91$), or school start time ($t(44) = -1.14$, $p = .26$). School start time for the maintenance sample averaged 07:42 ± 00:26, with the earliest reported school start time at 06:30 and the latest at 08:25. Two participants in the Control group (1 biological male and 1 biological female) discontinued participation due to a family death and a schedule conflict with the 3-week maintenance study protocol, respectively.

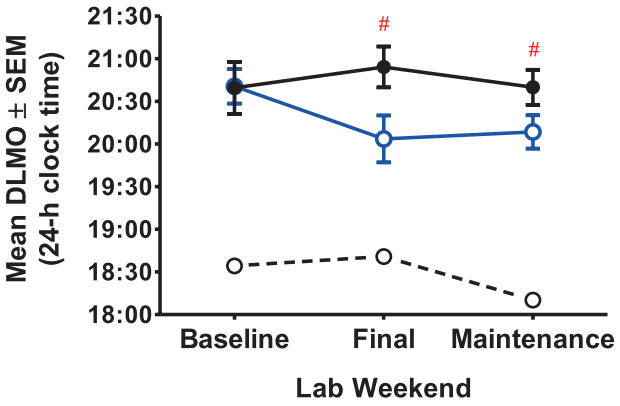



**Figure 2.** Circadian phase (DLMO) averaged for the intervention group (blue open circle; $n = 8$) and control group (black closed circle; $n = 5$). An extreme outlier in the control group with a baseline DLMO more than 2 SDs earlier than average is plotted separately and not included in the analysis (open circles and dotted line; see text). #$p < .10$ and effect size ($d$) > 0.9.

## Circadian phase and phase shifts

Figure 2 illustrates DLMO phases at baseline (day 16), after the 2-week sleep extension intervention (day 30), and after the 3-week maintenance protocol (day 51) for both groups. One male participant in the Control group was an extreme outlier with a baseline DLMO (18:34) of more than 2 standard deviations earlier than the mean of all participants (20:45 ± 00:44). For this outlier, DLMO phases remained stable across assessments, shifting less than 24 minutes from his baseline (18:41 on day 30; 18:10 on day 51). DLMO data for the outlier is plotted separately in Figure 2 (hashed line) but omitted from the analysis to avoid misrepresentation of the control group means.

DLMO for the Intervention group advanced 37.0 ± 40.0 minutes in response to the 2-week sleep extension intervention and then remained stable during the 3-week maintenance protocol (DLMO phase shift from day 30 to day 51 averaged −4.9 ± 22.9 minutes). This is compared to the Control group with average phase shifts of less than 15 minutes across the study (Figure 2 and Table 1). Group-by-time interactions were detected for DLMO for both the 2-week intervention ($\beta = 0.86$, $SE = 0.26$, $p = .003$) and 3-week maintenance ($\beta = 0.54$, $SE = 0.26$, $p = .049$) relative to Baseline. The ICC was 0.67. Pairwise comparisons revealed that the Intervention group had a significantly greater phase advance after the 2-week intervention compared to the Control group (estimated difference = −0.84 hours, $SE = 0.35$, $p = .02$). The EMM contrast for the Intervention group from baseline (day 16) to maintenance (day 51) lab weekends was significant (estimated change = −0.53 hours, $SE = 0.18$, $p = .005$), but the EMM contrast from the final lab weekend (day 30) to the maintenance weekend (day 51) was not (estimated change = 0.08 hours, $SE = 0.18$, $p = .30$) Figure 3 and Table 2.

## Weekday sleep

Figure 3 illustrates weekday (Sunday–Thursday) sleep onset time and sleep duration for both groups at baseline, during the two sleep extension intervention weeks, and during the three maintenance weeks. Sleep onset time for the intervention group advanced 1.73 ± 0.24 hours from their baseline by intervention week 2, and weekday sleep onset averages remained relatively stable at about 23:00 across the three maintenance

**Table 1.** DLMO and DLMO Phase Shift (Mean ± SD)

| | Control | Intervention | Effect Size (d) |
|---|---|---|---|
| N | 5[a] | 8 | |
| Baseline DLMO (day 16) | 20:39 ± 00:41 | 20:41 ± 00:34 | 0.03 |
| Final DLMO (day 30)[#] | 20:54 ± 0:32 | 20:04 ± 00:47 | 1.27 |
| Maintenance DLMO (day 51)[#] | 20:40 ± 00:28 | 20:08 ± 00:33 | 1.03 |
| DLMO phase shifts (minutes)[b]: | | | |
| Intervention: days 16–30* | –14.7 ± 16.6 | 37.0 ± 40.0 | 1.69 |
| Maintenance: days 30–51 | 14.2 ± 27.4 | –4.9 ± 22.9 | 0.76 |

[a]excludes outlier in Control group (see text).
[b]Negative number = phase delay; positive number = phase advance.
Differences between Control and Intervention: [#]$p \leq .10$; *$p < .05$; large group effects sizes are in italics.

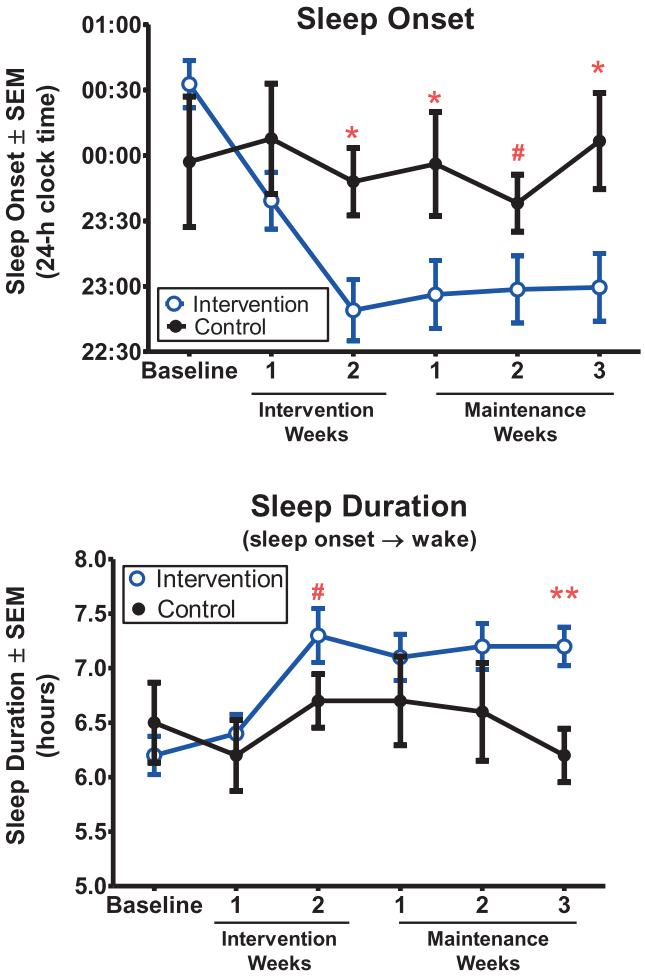



**Figure 3.** Weekday sleep measured from wrist actigraphy for the Intervention group (blue open circles; $n = 8$) and the control group (black closed circles; $n = 6$) during Baseline, Intervention Weeks, and Maintenance Weeks. *$p < .05$; **$p < .01$; #$p < .10$ and effect size ($d$) > 0.9.

weeks (Figure 3 and Table 2). The control group did not show a systematic change from their baseline across the intervention weeks or during the maintenance weeks with sleep onset averaging around midnight (Figure 3 and Table 2). Therefore, the intervention group was falling asleep 40 to 67 minutes earlier on average compared to the control group during the 3 maintenance weeks. Deviations from scheduled bedtime during the 3 maintenance weeks, however, did occur in the Intervention group (Supplementary Figure S4). LMMs detected significant group-by-time interactions for both the Intervention (β = 1.37, SE = 0.30, $p < .001$) and Maintenance weeks (β = 1.65, SE = 0.29, $p < .001$) relative to Baseline. The ICC was 0.66. Pairwise comparisons revealed that the Intervention group had significantly earlier sleep onset times during the Maintenance weeks compared to the control group (estimated difference = –0.94 hours, SE = 0.40, $p = .03$). The EMM contrast for the intervention group from Baseline to Maintenance weeks was significant (estimated change = –1.16 hour, SE = 0.23, $p < .0001$), but the EMM contrast from the intervention to the maintenance weeks was not (estimated change = 0.10 hours, SE = 0.25, $p = .68$).

Weekday sleep duration increased by 1.2 ± 0.5 hours from baseline by intervention week 2 in the intervention group, and average weekday sleep duration remained stable during the three maintenance weeks (Figure 3 and Table 2). The control group did not show a systematic change from baseline across the study. The average sleep duration for the intervention group was 1.0 hours greater, on average compared to the control group in maintenance week 3 [$t(12) = 3.6$, $p = .004$] and the effect size was large (see Table 2). LMMs detected significant group-by-phase interactions for the Intervention (β = –35.24, SE = 20.14, $p = .04$) and Maintenance (β = –42.13, SE = 16.09, $p = .01$) weeks relative to the baseline. The ICC was 0.48. Pairwise comparisons revealed a trend towards longer sleep duration in the intervention group during the Maintenance weeks (estimated difference = 37.6 minutes, SE = 20.0, $p = .07$) compared to the control group. The EMM contrast for the intervention group from baseline to Maintenance was significant (estimated change = 32.13 minutes, SE = 15.2, $p = .04$), but the EMM contrast from intervention to maintenance weeks was not (estimated change = –1.58 minutes, SE = 16.4, $p = .93$).

## Weekend sleep

Table 3 shows weekend sleep at home derived from actigraphy during baseline and during the two booster weekends. On booster weekends (days 36–37 and days 43–44), the Intervention group was allowed to go to bed up to 1 hour later than their maintenance weekday schedule and we instructed them to wake up at their maintenance weekday wake-up time. On average, weekend sleep onset during the two booster weekends was about 30 minutes later than weekday sleep onset (see Table 2). Booster weekend sleep onset and wake-up times for each intervention participant expressed as deviations from their scheduled times for each night are shown in Supplementary Figure S5. All participants fell asleep more than 30 minutes later than their scheduled weeknight bedtime on at least 1 of the 4 weekend nights

**Table 2.** Weekday (Sundays—Thursdays) Sleep From Wrist Actigraphy (Mean ± SD)

| | Control | Intervention | Effect size (*d*) |
|---|---|---|---|
| N | 6 | 8 | |
| Baseline (days 3–7 and 10–14) | | | |
| Sleep onset time | 23:57 ± 01:13 | 00:33 ± 00:31 | –0.63 |
| Wake-up time | 6:25 ± 00:25 | 6:43 ± 00:24 | –0.70 |
| Total sleep time (h) | 5.5 ± 0.8 | 5.3 ± 0.6 | 0.30 |
| Duration (h) [a] | 6.5 ± 0.9 | 6.2 ± 0.5 | 0.40 |
| Intervention week 1 (days 17–21) | | | |
| Sleep onset time | 00:07 ± 01:02 | 23:39 ± 00:37 | 0.56 |
| Wake-up time | 6:21 ± 00:44 | 6:06 ± 00:36 | 0.38 |
| Total sleep time (h) | 5.3 ± 0.5 | 5.5 ± 0.5 | 0.48 |
| Duration (h) [a] | 6.2 ± 0.8 | 6.4 ± 0.5 | 0.34 |
| Intervention week 2 (days 24–28) | | | |
| Sleep onset time | 23:47 ± 00:38 | 22:49 ± 00:40* | 1.52 |
| Wake-up time | 6:30 ± 00:18 | 6:09 ± 00:48 | 0.58 |
| Total sleep time (h) | 5.6 ± 0.7 | 6.2 ± 0.7 | 0.80 |
| Duration (h) [a] | 6.7 ± 0.6 | 7.3 ± 0.7# | 0.96 |
| Maintenance week 1 (days 31–35) | | | |
| Sleep onset time | 23:56 ± 00:53 | 22:56 ± 00:44* | 1.22 |
| Wake-up time | 6:38 ± 00:36 | 6:04 ± 00:36 | 0.96 |
| Total sleep time (h) | 5.6 ± 0.6 | 5.9 ± 0.6 | 0.44 |
| Duration (h) [a] | 6.7 ± 1.0 | 7.1 ± 0.6 | 0.52 |
| Maintenance week 2 (days 38–42) | | | |
| Sleep onset time | 23:38 ± 00:29 | 22:59 ± 00:44# | 1.07 |
| Wake-up time | 6:15 ± 00:46 | 6:09 ± 00:34 | 0.15 |
| Total sleep time (h) | 5.5 ± 0.6 | 6.0 ± 0.8 | 0.86 |
| Duration (h) [a] | 6.6 ± 1.0 | 7.2 ± 0.6 | 0.72 |
| Maintenance week 3 (days 45–49) | | | |
| Sleep onset time | 0:07 ± 00:54 | 23:00 ± 00:44* | 1.36 |
| Wake-up time | 6:17 ± 00:27 | 6:11 ± 00:33 | 0.18 |
| Total sleep time (h) | 5.4 ± 0.7 | 5.9 ± 0.9 | 0.76 |
| Duration (h) [a] | 6.2 ± 0.6 | 7.2 ± 0.5** | 1.92 |

Differences between Control and Intervention: #$p \leq .10$; *$p < .05$; **$p < .01$; large effects sizes are in italics.
[a]interval from sleep onset to final wake.

(Supplementary Figure S5, top). Of all the booster weekend nights (8 participants × 4 booster weekend nights = 32 nights), 69% of the nights had sleep onsets 30 minutes or later than scheduled weekday bedtime and 31% had sleep onsets within 30 minutes of scheduled weekday bedtime. There were 6 booster weekend nights in which sleep onset was more than 1.5 hours after scheduled bedtime, and 4 of those nights were from the same individual (Supplementary Figure S5).

Intervention participants were asked to wake up at their average school-day wake-up time on both mornings of the two booster weekends. Of the 32 booster weekend mornings, there were 3 (9%) mornings in which wake-up time was more than 1 later than prescribed and these late wake-up times occurred once for 3 separate intervention participants and occurred during the second booster weekend only (Supplementary Figure S5). For the remaining mornings (91%), wake-up times were within 32 minutes of prescribed wake-up time.

Intervention participants were instructed to take a nap on Saturday or Sunday of the booster weekends if they chose to go to sleep later on Friday or Saturday nights, respectively. Of the 22 nights in which sleep onset was 30 minutes or more later than weekday sleep onset, naps occurred the next day 50% of the time (11 out of 22). All but one intervention participant took at least 1 nap during the booster weekends. On days when naps were taken, the 24-hour (nighttime + daytime) sleep duration averaged 490 ± 24 minutes and total sleep time averaged 406 ± 78 minutes.

## Booster weekend morning bright light

Participants were instructed to sit by the light box for 150 minutes on either Saturday or Sunday morning. Bright light start time and duration were similar among the self-report measure, and both light sensors (light box and neck). Therefore, we report on the neck light sensor only. All eight intervention participants were exposed to bright light on one booster weekend morning

**Table 3.** Weekend (Fridays and Saturdays) Sleep From Wrist Actigraphy (Mean ± SD)

| | Control | | Intervention | |
|---|---|---|---|---|
| | Nighttime | Daytime[b] | Nighttime | Daytime[c] |
| Baseline (days 1–2 and 8–9) | | | | |
| Sleep onset time | 00:19 ± 01:21 | 14:15 ± 04:00 | 01:22 ± 00:55 | 13:50 ± 02:51 |
| Wake-up time | 08:56 ± 01:36 | 16:51 ± 01:34 | 10:10 ± 01:02 | 15:35 ± 02:31 |
| Total sleep time (h) | 7.1 ± 1.0 | 2.3 ± 2.1 | 7.4 ± 0.8 | 1.4 ± 1.4 |
| Duration (h) [a] | 8.6 ± 0.9 | 2.6 ± 2.4 | 8.8 ± 1.0 | 1.8 ± 1.7 |
| Booster weekend 1 (days 36–37) | | | | |
| Sleep onset time | 01:02 ± 1:20* | — | 23:25 ± 00:49 | 12:22 ± 01:56 |
| Wake-up time | 08:39 ± 00:41** | | 06:04 ± 00:38 | 13:35 ± 01:57 |
| Total sleep time (h) | 6.0 ± 1.1 | | 5.6 ± 0.7 | 0.9 ± 0.5 |
| Duration (h) [a] | 7.6 ± 1.7 | | 6.7 ± 0.7 | 1.2 ± 0.6 |
| Booster weekend 2 (days 43–44) | | | | |
| Sleep onset time | 23:54 ± 00:48 | — | 23:32 ± 1:10 | 12:55 ± 3:05 |
| Wake-up time | 08:07 ± 01:13* | | 06:38 ± 01:04 | 14:47 ± 03:17 |
| Total sleep time (h) | 6.9 ± 1.0* | | 5.8 ± 0.6 | 1.5 ± 0.6 |
| Duration (h) [a] | 8.2 ± 1.1* | | 7.1 ± 0.5 | 1.9 ± 0.6 |

[a]interval from sleep onset to final wake.
[b]2 control participants contributed to nap averages during baseline weekends; no control participants reported naps during booster weekends 1 or 2.
[c]6 intervention participants contributed to nap averages during baseline weekends; 4 intervention participants contributed to nap averages during booster weekend 1; 4 participants contributed to nap averages during booster weekend 2.
Nighttime differences between Control and Intervention: *$p < .05$; **$p < .01$.

for at least 90 minutes of the prescribed time. On those mornings, bright light started 11.5 ± 11.2 minutes after the scheduled wake and the duration of bright (≥500 lux) light exposure was 125.4 ± 18.2 minutes. (For individual start times and bright light exposure durations, see Supplementary Figure S6.) On bright light box mornings, the average photopic illuminance from scheduled wake-up time to 2 hours 35 minutes after scheduled wake was 5170 ± 4094 photopic lux. During the first 35 minutes after scheduled wake on bright light box mornings, intervention participants received 23.5 ± 10.1 minutes of bright light and illuminance averaged 4440 ± 3172 photopic lux.

## Discussion

In our previous 4-week study with a 2-week sleep extension intervention, we showed that a gradual advance of bedtime, an individualized time-management strategy ("SleepRouTeen") with behavioral goals to facilitate an earlier bedtime, and bright light box exposure on both mornings of one weekend in the laboratory ("reset weekend") advanced circadian phase and sleep onset, and increased weeknight sleep duration by more than 1 hour [33]. In the current 3-week follow-up, we showed that these favorable changes to circadian phase and sleep can be maintained at home when wake times remain stable, weekday bedtimes remain early but some flexibility is introduced on weekends, weekend naps are in place to compensate for sleep loss, and morning bright light exposure is used on one morning of each weekend.

Previous studies of late-sleeping adolescents have tested daily morning light exposure over 3–6 weeks using a combination of natural sunlight and a light box [44], light glasses [45], or a passive approach of prewake light flashes [46] to facilitate earlier sleep onset. Instead of daily morning bright light exposure like in these previous studies [44–46], we chose to use morning bright light exposure on the weekend only to advance the circadian phase, maintain an early phase, and facilitate an early sleep onset because this approach is likely more feasible for this age group. Adolescents usually have more time on weekend mornings to complete a bright light exposure protocol compared to school mornings. Asking an adolescent—especially a youngster who has difficulty waking in the morning—to wake up even earlier than usual to fit bright light treatment into their school morning routine is difficult. Adherence to morning bright light treatment, especially in younger groups, is described as poor [47]. In one of the few studies that reported adherence in this age group, Micic et al. [48] reported mediocre treatment adherence to 30 mins of daily morning bright light from LED glasses for 1 to 4 weeks in adolescents with Delayed Sleep–Wake Phase Disorder (DSWPD), providing further evidence that consistent daily morning bright light treatment is difficult for most adolescents. The only other study that tested morning bright light on the weekend in late-sleeping adolescents did so as an adjunct treatment to a school-based sleep education program in which students were asked to advance their sleep 30 minutes per day and use light glasses on both mornings of one weekend for 30–60 minutes at wake-up time [49]. Adding bright light to weekend mornings did not confer an added benefit to improvements in mood or sleep (circadian phase was not measured), and this may be related to protocol adherence or light exposure time being too short. In the current study, we asked participants to sit in front of the bright light box on one weekend morning and not both to keep the advanced circadian phase stable, recognizing that requiring both days may be difficult for many families who have other commitments on the weekend. The duration of bright light exposure was 2.5 hours (three 50-minute exposures) based on results from our previous study [35]; however, data from the current study suggests that at least 90 minutes of bright light may be sufficient on booster weekends.

The timing of weekend sleep (dark) schedules must also be considered to optimize circadian phase shifts. Previous studies of young adults [24, 26] and adolescents [34] show that sleeping in later on weekends shifts DLMO by about 30–45 minutes, on average, by Sunday evening. Taylor and colleagues [26] asked young adults to maintain a fixed bedtime and then allowed ad-lib wake-up on Saturday and Sunday mornings. DLMO was delayed by 29.6 minutes, on average. Yang et al. [24] delayed weekend bedtime and wake-up time by 2 hours on 1 weekend and DLMO delayed by 31.6 minutes, on average. Crowley et al. [34] identified that when high-school-aged adolescents without sleep complaints were prescribed a typical weekend "recovery" sleep schedule (bedtimes were 1.5 hours later than school nights and wake-up was 3 hours later than school mornings), the circadian phase measured by the DLMO shifted later by about 45 minutes on average by Sunday night. Asking adolescents to wake only 1 hour later on Saturday and Sunday morning instead of 3 hours later or to sit in front of a small light box emitting short wavelength light (454–484 nm) for 1 hour at home when they woke up on both mornings of the weekend did not stabilized phase; DLMOs delayed about 40 minutes in both conditions by Sunday night. Taken together, these studies demonstrate that sleeping late on the weekend competes with the desired outcome of stabilizing or advancing the circadian clock of adolescents. Therefore, to improve the effectiveness of weekend morning bright light, we reasoned that weekend sleep (dark) needs to gradually shift earlier to help facilitate a circadian phase advance [33, 35] or remain as similar to school days as possible to stabilize the circadian phase. In the "booster" weekends of the current study, adolescents were asked to maintain their school-day wake time so that morning light exposure would coincide with the most sensitive portion of the PRC to light and thus help keep their circadian clock early. Flexibility in the sleep schedule was therefore introduced at bedtime to increase the likelihood of adolescents being able to follow the schedule longer term at home. Later bedtimes on Friday and Saturday nights provide more time to engage in their usual weekend activities, like socializing with friends or part-time work.

In the current study, we did not instruct adolescents to limit evening light or screen time or wear eyeglasses that block or significantly reduce blue light on the weekend when they were allowed to stay awake 1 hour later because we were trying to simulate what they would naturally do at home. Data from adolescents [50] and adults [51] show that evening light exposure can attenuate phase advances in response to morning bright light given over 3 days. Ambient light exposure [52], including light emitted from device screens [53–55], in the evening before habitual bedtime can suppress melatonin secretion, though interindividual variation in this response is noted [56]. As we develop this intervention work, testing the added benefit of controlling illuminance and the spectral composition of evening light on booster weekends may be warranted.

In the intervention group, weekday sleep duration increased from an average of 6.2 hours to an average of 7.3 hours. Average weekday sleep duration remained stable at 7.1 to 7.2 hours during the 3 maintenance weeks. This 1-hour change in sleep duration for a group who reported chronic insufficient sleep is significant; however, it is still less than the minimum recommendation of 8 hours per night [1, 57]. In our previous report [33], 16 out of 23 participants in the intervention group (70%) were averaging less than 8 hours of sleep on weekdays. In the current group, the average weekday sleep duration was greater than 8 hours for 1 participant in maintenance week 1, and no one averaged more than 8 hours in maintenance weeks 2 and 3. Therefore, advancing bedtime an additional 30–60 minutes in a third intervention week may be needed for most adolescents with a late-sleeping behavioral phenotype. Time management strategies provided by their individualized Sleep RouTeens to support earlier bedtimes may be enough for most adolescents. However, falling asleep even 30 minutes earlier may still pose challenges for some who continue to have delayed circadian phases as bedtimes could coincide with the Forbidden Zone for Sleep [10]. For these delayed adolescents, additional morning light may be needed on the first booster weekend at wake-up time.

Sleep duration on baseline weekends for both the Control and Intervention groups averaged 8.6 to 8.8 hours, which is more than 2 hours longer than on their baseline weekdays. This weekend oversleeping is greater than the approximate 1-hour difference reported in previous studies [3, 58]. Some adolescents also took naps on baseline weekends, with durations averaging between 1.8 and 2.5 hours, further suggesting a level of weekday sleep restriction that required compensatory sleep on weekends. The booster weekends tested in the current study required that adolescents wake up at their school day wake time, which prevented the typical compensatory sleep on the weekend. Moreover, we allowed bedtimes to be up to 1 hour later on Friday and Saturday nights, which would theoretically reduce sleep duration by up to 1 hour too. Compared to the control group, the nocturnal sleep duration of the intervention group was shorter on weekend nights, particularly during booster weekend 2 (Table 3). In an attempt to provide some compensatory sleep on the weekend, we asked participants to take a nap in the afternoon if they went to sleep late on Friday or Saturday nights. For those adolescents who took a nap, their daily (nighttime + daytime) sleep duration averaged more than 8 hours, suggesting that napping on the weekend could provide additional sleep on the weekend. Although sleep duration on the booster weekends was descriptively shorter than during the baseline weekends in the intervention group, it must also be noted that weekday sleep duration was also about 1 hour longer per night during the maintenance weeks, and therefore, the need for weekend compensatory sleep may be less. Nevertheless, morning sleepiness or dysfunction before the afternoon nap may continue to be problematic and needs to be further assessed.

Although the tested intervention and 3-week maintenance protocol show promise to increase and maintain sleep duration in short and late-sleeping adolescents, limitations of the current study should be noted. First, the sample size was small in this 3-week follow-up study. Also, the adolescents who were in the intervention group and agreed to be in the follow-up study may have positively biased protocol adherence outcomes because they were more willing to participate than those who decided not to participate in the follow-up study. Finally, in this initial test of effectiveness, participants were paid to complete the intervention and follow-up study. It is unclear whether adolescents would show the same level of adherence without compensation, and whether additional motivators (e.g. parental involvement, peer support) may be needed.

## Conclusions

The biological propensity for alertness later in the evening permitted by maturing circadian and sleep–wake homeostatic systems, as well as social and academic pressures displace sleep to a later time. These biological and psychosocial factors compete with early school start times that force adolescents out of

bed too early on school days. Our previous study [33] showed that by shifting the circadian timing system earlier, prescribing a gradual advance of bedtime, and modifying after-school and evening time-use, adolescents were able to fall asleep early and increase their sleep duration during the school week. The current follow-up study shows that these gains can be maintained with only one morning of bright light each weekend, stable sleep times on weekdays, and some flexibility in weekend bedtimes. Adaptation and expansion of this approach for patients with DSWPD or other conditions with cooccurring delayed sleep may be warranted.

## Supplementary Material

Supplementary material is available at *SLEEP Advances* online.

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## Author Contributions

Stephanie Crowley (Conceptualization [lead], Data curation [equal], Formal analysis [equal], Funding acquisition [lead], Investigation [lead], Methodology [lead], Project administration [lead], Supervision [lead], Visualization [lead], Writing—original draft [lead]), Elaine Poole (Data curation [supporting], Formal analysis [supporting], Visualization [supporting], Writing—review & editing [supporting]), John Adams (Formal analysis [lead], Writing—review & editing [supporting]), and Charmane Eastman (Conceptualization [supporting], Funding acquisition [supporting], Methodology [supporting], Writing—review & editing [equal]).

## Data Availability

The data underlying this article will be shared on reasonable request to the corresponding author.

## References

1. Paruthi S, Brooks LJ, D'Ambrosio C, *et al*. Consensus statement of the American Academy of Sleep Medicine on the recommended amount of sleep for healthy children: methodology and discussion. *J Clin Sleep Med*. 2016;**12**(11):1549–1561. doi: 10.5664/jcsm.6288
2. Wheaton AGP, Jones S, Cooper ACM, Croft JBP. Short sleep duration among middle school and high school students - United States. *Centers Dis Control Prev*. 2018;**67**(3):6.
3. Galland BC, Short MA, Terrill P, *et al*. Establishing normal values for pediatric nighttime sleep measured by actigraphy: a systematic review and meta-analysis. *Sleep*. 2018;**41**(4). doi: 10.1093/sleep/zsy017
4. Baiden P, Tadeo SK, Peters KE. The association between excessive screen-time behaviors and insufficient sleep among adolescents: findings from the 2017 youth risk behavior surveillance system. *Psychiatry Res*. 2019;**281**:112586. doi: 10.1016/j.psychres.2019.112586
5. Carskadon MA. Sleep in adolescents: the perfect storm. *Pediatr Clin North Am*. 2011;**58**(3):637–647. doi: 10.1016/j.pcl.2011.03.003
6. Crowley SJ, Wolfson AR, Tarokh L, Carskadon MA. An update on adolescent sleep: new evidence informing the perfect storm model. *J Adolesc*. 2018;**67**:55–65. doi: 10.1016/j.adolescence.2018.06.001
7. Jenni OG, Achermann P, Carskadon MA. Homeostatic sleep regulation in adolescents. *Sleep*. 2005;**28**(11):1446–1454. doi: 10.1093/sleep/28.11.1446
8. Carskadon MA, Acebo C, Richardson GS, Tate BA, Seifer R. An approach to studying circadian rhythms of adolescent humans. *J Biol Rhythms*. 1997;**12**(3):278–289. doi: 10.1177/074873049701200309
9. Carskadon MA, Acebo C, Jenni OG. Regulation of adolescent sleep: implications for behavior. *Ann N Y Acad Sci*. 2004;**1021**:276–291. doi: 10.1196/annals.1308.032
10. Monterastelli AJ, Adams J, Eastman CI, Crowley SJ. The forbidden zone for sleep is more robust in adolescents compared to adults. *Front Sleep*. 2024;**2**:1304647. doi: 10.3389/frsle.2023.1304647
11. Meltzer LJ, Wahlstrom KL, Plog AE, Strand MJ. Changing school start times: impact on sleep in primary and secondary school students. *Sleep*. 2021;**44**(7). doi: 10.1093/sleep/zsab048
12. Meltzer LJ, Saletin JM, Honaker SM, *et al*. COVID-19 instructional approaches (in-person, online, hybrid), school start times, and sleep in over 5,000 U.S. adolescents. *Sleep*. 2021;**44**(12). doi: 10.1093/sleep/zsab180
13. Owens JA, Dearth-Wesley T, Herman AN, Whitaker RC. Drowsy driving, sleep duration, and chronotype in adolescents. *J Pediatr*. 2019;**205**:224–229. doi: 10.1016/j.jpeds.2018.09.072
14. Carskadon MA, Wolfson AR, Acebo C, Tzischinsky O, Seifer R. Adolescent sleep patterns, circadian timing, and sleepiness at a transition to early school days. *Sleep*. 1998;**21**(8):871–881. doi: 10.1093/sleep/21.8.871
15. Wittmann M, Dinich J, Merrow M, Roenneberg T. Social jetlag: misalignment of biological and social time. *Chronobiol Int*. 2006;**23**(1):497–509. doi: 10.1080/07420520500545979
16. Roenneberg T, Allebrandt KV, Merrow M, Vetter C. Social jetlag and obesity. *Curr Biol*. 2012;**22**(10):939–943. doi: 10.1016/j.cub.2012.03.038
17. Talbot LS, McGlinchey EL, Kaplan KA, Dahl RE, Harvey AG. Sleep deprivation in adolescents and adults: changes in affect. *Emotion*. 2010;**10**(6):831–841. doi: 10.1037/a0020138
18. Wolfson AR, Carskadon MA. Sleep schedules and daytime functioning in adolescents. *Child Dev*. 1998;**69**(4):875–887.

19. Gangwisch JE, Babiss LA, Malaspina D, Turner JB, Zammit GK, Posner K. Earlier parental set bedtimes as a protective factor against depression and suicidal ideation. *Sleep.* 2010;**33**(1):97–106. doi: 10.1093/sleep/33.1.97
20. Pallesen S, Saxvig IW, Molde H, Sorensen E, Wilhelmsen-Langeland A, Bjorvatn B. Brief report: behaviorally induced insufficient sleep syndrome in older adolescents: prevalence and correlates. *J Adolesc.* 2011;**34**(2):391–395. doi: 10.1016/j.adolescence.2010.02.005
21. Palmer CA, Alfano CA. Sleep and emotion regulation: an organizing, integrative review. *Sleep Med Rev.* 2017;**31**:6–16. doi: 10.1016/j.smrv.2015.12.006
22. Carskadon MA, Harvey K, Dement WC. Acute restriction of nocturnal sleep in children. *Percept Mot Skills.* 1981;**53**:103–112. doi: 10.2466/pms.1981.53.1.103
23. Randazzo AC, Muehlbach MJ, Schweitzer PK, Walsh JK. Cognitive function following acute sleep restriction in children ages 10-14. *Sleep.* 1998;**21**(8):861–868.
24. Yang CM, Spielman AJ, D'Ambrosio P, Serizawa S, Nunes J, Birnbaum J. A single dose of melatonin prevents the phase delay associated with a delayed weekend sleep pattern. *Sleep.* 2001;**24**:272–281. doi: 10.1093/sleep/24.3.272
25. Fallone G, Acebo C, Arnedt JT, Seifer R, Carskadon MA. Effects of acute sleep restriction on behavior, sustained attention, and response inhibition in children. *Percept Mot Skills.* 2001;**93**(1):213–229. doi: 10.2466/pms.2001.93.1.213
26. Taylor A, Wright HR, Lack LC. Sleeping-in on the weekend delays circadian phase and increases sleepiness the following week. *Sleep Biol Rhythms.* 2008;**6**:172–179. doi: 10.1111/j.1479-8425.2008.00356.x
27. Preckel F, Lipnevich AA, Schneider S, Roberts RD. Chronotype, cognitive abilities, and academic achievement: a meta-analytic investigation. *Learn Individ Differ.* 2011;**21**(5):483–492. doi: 10.1016/j.lindif.2011.07.003
28. Lin WH, Yi CC. Unhealthy sleep practices, conduct problems, and daytime functioning during adolescence. *J Youth Adolesc.* 2015;**44**(2):431–446. doi: 10.1007/s10964-014-0169-9
29. Tonetti L, Natale V, Randler C. Association between circadian preference and academic achievement: a systematic review and meta-analysis. *Chronobiol Int.* 2015;**32**(6):792–801. doi: 10.3109/07420528.2015.1049271
30. Phillips AJK, Clerx WM, O'Brien CS, *et al*. Irregular sleep/wake patterns are associated with poorer academic performance and delayed circadian and sleep/wake timing. *Sci Rep.* 2017;**7**(1):1–13.
31. Lin L SG, Boursier J, Santisteban JA, Gruber R. Sleep duration is associated with academic achievement of adolescent girls in mathematics. *Nat Sci Sleep*. 2020;**12**:173–182.
32. Alfonsi V, Palmizio R, Rubino A, *et al*. The association between school start time and sleep duration, sustained attention, and academic performance. *Nat Sci Sleep*. 2020;**12**:1161–1172. doi: 10.2147/NSS.S273875
33. Crowley SJ, Velez SL, Killen LG, Cvengros JA, Fogg LF, Eastman CI. Extending weeknight sleep of delayed adolescents using weekend morning bright light and evening time management. *Sleep.* 2023;**46**(1). doi: 10.1093/sleep/zsac202
34. Crowley SJ, Carskadon MA. Modifications to weekend recovery sleep delay circadian phase in older adolescents. *Chronobiol Int.* 2010;**27**(7):1469–1492. doi: 10.3109/07420528.2010.503293
35. Misiunaite I, Eastman CI, Crowley SJ. Circadian phase advances in response to weekend morning light in adolescents with short sleep and late bedtimes on school nights. *Front Neurosci.* 2020;**14**(13831):99. doi: 10.3389/fnins.2020.00099
36. Crowley SJ, Eastman CI. Human adolescent phase response curves to bright white light. *J Biol Rhythms.* 2017;**32**(4):334–344. doi: 10.1177/0748730417713423
37. Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. *Appl Psychol Meas.* 1977;**1**(3):385–401. doi: 10.1177/014662167700100306
38. Ishihara S. *Tests for Colour-Blindness*. 24 Plates Edition ed. Tokyo, Japan: Kanehara Shuppan Co., LTD.; 1972.
39. Klerman EB, Gershengorn HB, Duffy JF, Kronauer RE. Comparisons of the variability of three markers of the human circadian pacemaker. *J Biol Rhythms.* 2002;**17**(2):181–193. doi: 10.1177/074873002129002474
40. Buxton OM, L'Hermite-Baleriaux M, Turek FW, Van Cauter E. Daytime naps in darkness phase shift the human circadian rhythms of melatonin and thyrotropin secretion. *Am J Physiol Regul Integr Comp Physiol.* 2000;**278**:R373–R382. doi: 10.1152/ajpregu.2000.278.2.R373
41. Crowley SJ, Suh C, Molina TA, Fogg LF, Sharkey KM, Carskadon MA. Estimating the dim light melatonin onset of adolescents within a 6-h sampling window: the impact of sampling rate and threshold method. *Sleep Med.* 2016;**20**:59–66. doi: 10.1016/j.sleep.2015.11.019
42. Meltzer LJ, Walsh CM, Traylor J, Westin AML. Westin AM. Direct comparison of two new actigraphs and polysomnography in children and adolescents. *Sleep.* 2012;**35**(1):159–166. doi: 10.5665/sleep.1608
43. Acebo C, Sadeh A, Seifer R, *et al*. Estimating sleep patterns with activity monitoring in children and adolescents: how many nights are necessary for reliable measures? *Sleep.* 1999;**22**(1):95–103. doi: 10.1093/sleep/22.1.95
44. Gradisar M, Dohnt H, Gardner G, *et al*. A randomized controlled trial of cognitive-behavior therapy plus bright light therapy for adolescent delayed sleep phase disorder. *Sleep.* 2011;**34**(12):1671–1680. doi: 10.5665/sleep.1432
45. Richardson C, Cain N, Bartel K, Micic G, Maddock B, Gradisar M. A randomised controlled trial of bright light therapy and morning activity for adolescents and young adults with Delayed Sleep-Wake Phase Disorder. *Sleep Med.* 2018;**45**:114–123. doi: 10.1016/j.sleep.2018.02.001
46. Kaplan KA, Mashash M, Williams R, Batchelder H, Starr-Glass L, Zeitzer JM. Effect of light flashes vs sham therapy during sleep with adjunct cognitive behavioral therapy on sleep quality among adolescents: a randomized clinical trial. *JAMA Netw Open.* 2019;**2**(9):e1911944–e1911944. doi: 10.1001/jamanetworkopen.2019.11944
47. Bjorvatn B, Pallesen S. A practical approach to circadian rhythm sleep disorders. *Sleep Med Rev.* 2009;**13**(1):47–60. doi: 10.1016/j.smrv.2008.04.009
48. Micic G, Richardson C, Cain N, *et al*. Readiness to change and commitment as predictors of therapy compliance in adolescents with Delayed Sleep-Wake Phase Disorder. *Sleep Med.* 2018;**55**:48–55. doi: 10.1016/j.sleep.2018.12.002
49. Bonnar D, Gradisar M, Moseley L, Coughlin A, Cain N, Short MA. Evaluation of novel school-based interventions for adolescent sleep problems: does parental involvement and bright light improve outcomes? *Sleep Health.* 2015;**1**(1):66–74. doi: 10.1016/j.sleh.2014.11.002
50. Crowley SJ, Fournier CL, Eastman CI. Late bedtimes prevent circadian phase advances to morning bright light in adolescents. *Chronobiol Int.* 2018;**35**(12):1748–1752. doi: 10.1080/07420528.2018.1504784
51. Burgess HJ. Evening ambient light exposure can reduce circadian phase advances to morning light independent of sleep deprivation. *J Sleep Res.* 2013;**22**(1):83–88. doi: 10.1111/j.1365-2869.2012.01042.x
52. Gooley JJ, Chamberlain K, Smith KA, *et al*. Exposure to room light before bedtime suppresses melatonin onset and shortens melatonin duration in humans. *J Clin Endocrinol Metab.* 2011;**96**(3):E463–E472. doi: 10.1210/jc.2010-2098

53. Cajochen C, Frey S, Anders D, *et al*. Evening exposure to a light-emitting diodes (LED)-backlit computer screen affects circadian physiology and cognitive performance. *J Appl Physiol*. 2011;**110**(5):1432–1438. doi: 10.1152/japplphysiol.00165.2011
54. van der Lely S, Frey S, Garbazza C, *et al*. Blue blocker glasses as a countermeasure for alerting effects of evening light-emitting diode screen exposure in male teenagers. *J Adolesc Health*. 2015;**56**(1):113–119. doi: 10.1016/j.jadohealth.2014.08.002
55. Wood B, Rea MS, Plitnick B, Figueiro MG. Light level and duration of exposure determine the impact of self-luminous tablets on melatonin suppression. *Appl Ergon*. 2013;**44**(2):237–240. doi: 10.1016/j.apergo.2012.07.008
56. Phillips AJK, Vidafar P, Burns AC, *et al*. High sensitivity and interindividual variability in the response of the human circadian system to evening light. *Proc Natl Acad Sci U S A*. 2019;**116**:12019–12024. doi: 10.1073/pnas.1901824116
57. Hirshkowitz M, Whiton K, Albert SM, *et al*. National Sleep Foundation's sleep time duration recommendations: methodology and results summary. *Sleep Health*. 2015;**1**(1):40–43. doi: 10.1016/j.sleh.2014.12.010
58. Berry KM, Berger AT, Laska MN, *et al*. Weekend night vs. school night sleep patterns, weight status, and weight-related behaviors among adolescents. *Sleep Health*. 2021;**7**(5):572–580. doi: 10.1016/j.sleh.2021.07.008