Sleep Advances, 2022, 1–14

https://doi.org/10.1093/sleepadvances/zpac021
Advance Access Publication Date: 24 June 2022
Original Article

ORIGINAL ARTICLE

Low-intensity scheduled morning exercise for adolescents with a late chronotype: a novel treatment to advance circadian phase?

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Abstract

Study Objectives: During adolescence, an interplay between biological and environmental factors leads to constrained sleep duration and timing. The high prevalence of sleep deprivation during this developmental period is a public health concern, given the value of restorative sleep for mental, emotional, and physical health. One of the primary contributing factors is the normative delay of the circadian rhythm. Therefore, the present study aimed to evaluate the effect of a gradually advanced morning exercise schedule (30 min shift each day) completed for 45 min on 5 consecutive mornings, on the circadian phase and daytime functioning of adolescents with a late chronotype, compared with a sedentary control group.

Methods: A total of 18 physically inactive male adolescents aged 15–18 years spent 6 nights at the sleep laboratory. The morning procedure included either 45 min walking on a treadmill or sedentary activities in dim light. Saliva dim light melatonin onset, evening sleepiness, and daytime functioning were assessed during the first and last night of laboratory attendance.

Results: The morning exercise group had a significantly advanced (earlier) circadian phase (27.5 min ± 32.0), while sedentary activity resulted in a phase delay (–34.3 min ± 53.2). Morning exercise also led to higher evening sleepiness in the earlier hours of the night, but not at bedtime. Mood measures improved slightly in both study conditions.

Conclusions: These findings highlight the phase-advancing effect of low-intensity morning exercise among this population. Future studies are needed to test the transference of these laboratory findings to adolescents' real life.

Statement of Significance

This is the first study to examine the phase-advancing potential of low-intensity morning exercise, and also the first study to include physically inactive adolescents with a late chronotype. This is in contrast to previous research, which has almost exclusively implemented moderate-to-vigorous exercise among healthy and aerobically fit adults to achieve circadian phase shifts. Yet, adolescents with an extreme evening preference are unlikely to engage in moderate-to-vigorous exercise in the morning, particularly if they are typically inactive. The findings of the present experiment show support for the phase-advancing effect of 45 min of morning exercise compared with sedentary controls. Hence, this adds to the scarce literature on the phase-advancing effect of morning exercise and supports the newly established PRC of exercise by Youngstedt and colleagues (2019).

Key words: chronotherapy; dim light melatonin onset; evening sleepiness; evening vigilance; mood; physical exercise

Submitted: 2 March, 2022; **Revised:** 3 June, 2022

Introduction

Today's adolescents face a common chronic health problem: sleep restriction [1]. Research overwhelmingly supports the fundamental role of sleep in adolescents' emotional and cognitive development, which may have important implications for success and wellbeing in adulthood [2–4]. Of the many transitions that occur during adolescence, changes to sleep may have the most pervasive impact, with inadequate and ill-timed sleep being linked to poor physical health, including a greater current and future risk of becoming overweight [5, 6], poor psychosocial functioning, low academic performance, and risk-taking behaviors [7, 8].

A recent health report on the sleep patterns of Australian children and adolescents shows that sleep duration declines on average by 15 min per year from the age of 12–17 years [9], with a similar pattern reported worldwide [10]. While European adolescents average 8.44 h of sleep on school nights, this drops to 7.64 h among Asian adolescents and 7.46 h for North American adolescents. Bedtime autonomy paired with psychosocial pressure (e.g. socializing with friends and homework) contribute to later bedtimes across adolescence, while school-start rise times remain the same—or become even earlier [11]. Sleep need, in contrast, does not significantly decrease during adolescence, with ~9.2 h of sleep per night required for optimal cognitive and emotional functioning [12, 13], regardless of age or maturational stage [14].

Besides the psychosocial changes that contribute to later bedtimes and inadequate sleep duration, one of the primary contributors is the ubiquitous delay of the circadian rhythm (an individual's natural internal sleep–wake cycle that repeats roughly every 24 h) [15–17]. For example, the onset of melatonin secretion (a sleep-promoting hormone) becomes later with increasing age and pubertal status [18]. As a result, sleep timing becomes progressively later from childhood (age 10 years) until early adulthood (~21–22 years)—with this delayed midpoint of sleep known as a "late chronotype" [16, 19]. However, solutions are available to manage or even reverse this late chronotype—each with its own pros and cons.

While there are challenges and an ongoing debate about later school start times for high school students, alternative interventions to advance circadian and sleep timing in young people are needed. Morning bright light therapy and evening exogenous melatonin administration are recommended treatments for children and adolescents with a delayed circadian rhythm [20]. However, sufficient ambient light may not always be available, particularly in countries with limited daylight hours [21]. Poor compliance with bright light and/or melatonin scheduling may arise as an additional barrier to treatment [22–24]. One untested chronotherapy among adolescents is scheduled to exercise—as a movement that has the potential to phase advance the human circadian rhythm—independently of bright light [25–27]. Most research on exercise as a non-photic time cue has focused on facilitating adjustment to night work. Therefore, a bulk of evidence supports the role of nocturnal exercise in delaying circadian timing in adults [28–31].

Until recently, the field lacked consensus about "when" exercise exerts phase advances to circadian and sleep timing [27]. However, a phase response curve to exercise has now been established [32], reflecting both morning and evening phase advance portions—similar to the response to bright light [33]. Importantly for the present study, large (42–54 min) phase advances in the melatonin rhythm have been observed in response to 1 h of moderate exercise completed at 07:00 am over 3 consecutive days in normally entrained adults [32]. These results and a recently published trial [34] suggest that exercise completed shortly after awakening has the greatest phase-advancing effect for later chronotypes (morning exercise [10 h after dim light melatonin onset; DLMO]: 0.54 ± 0.29 h vs. evening exercise [20 h after DLMO]: 0.46 ± 0.25 h after 5 days). Furthermore, gradual advancement of exercise timing (e.g. 20–30 min) may facilitate phase advances [35]. This protocol overlaps with that of the sleep schedule, which is a core component of bright light therapy for adolescents and involves shifting bedtime and wake time by 30 min per day [23].

Scheduled morning exercise does not require expensive equipment and can be tailored to the individual's preference (e.g. running vs. cycling, inside vs. outside), making it affordable, easier to comply with, and accessible to most young people. Additionally, a health professional does not necessarily need to provide oversight. In this regard, scheduled exercise has many benefits over other chronotherapies. In addition to its sleep-promoting effect [36], scheduled morning exercise may also improve adolescents' wellbeing by reducing daytime sleepiness, enhancing mood, and improving cognitive performance [36, 37].

The present study addresses this gap in the literature by assessing the effect of a gradually advanced morning exercise schedule (30 min earlier per day), completed for 45 min on 5 consecutive mornings, on circadian timing (i.e. DLMO) and daytime functioning (i.e. daytime sleepiness, evening sleepiness, and mood) in adolescents with a late chronotype. The effect of the exercise intervention was compared with a control group that also woke up 30 min earlier each morning, but remained sedentary for 45 min after waking up. Moreover, previous studies investigating the chronotherapeutic effect of exercise have exclusively used moderate-to-vigorous exercise bouts in a predominantly aerobically fit adult population [28, 31, 32, 34, 35, 38, 39]. Yet, adolescents with an extreme evening preference are unlikely to engage in an intervention that involves a moderate-to-vigorous exercise in the morning, especially if they are typically physically inactive. With this in mind, this is the first study to examine the phase-shifting effect of low-intensity morning exercise among physically inactive adolescents with a late chronotype, in a light-controlled environment (i.e. to isolate the effect of exercise). We hypothesized that adolescents undertaking scheduled morning exercise would show earlier circadian timing, greater evening sleepiness, lowered evening vigilance, and improved mood, relative to adolescents in the sedentary group.

Methods

Participants

A total of 62 male adolescents made contact with the Flinders University Sleep and Circadian Research Laboratory in response to advertisements on social media (Figure 1). Of these, 28 male adolescents met inclusion criteria and 23 were randomly assigned to either morning exercise or sedentary condition (M_{age} = 16.4 years, SD = 1.0). The randomized controlled trial was approved by the Southern Adelaide Clinical Human Research Ethics Committee (SAC HREC application number OFR

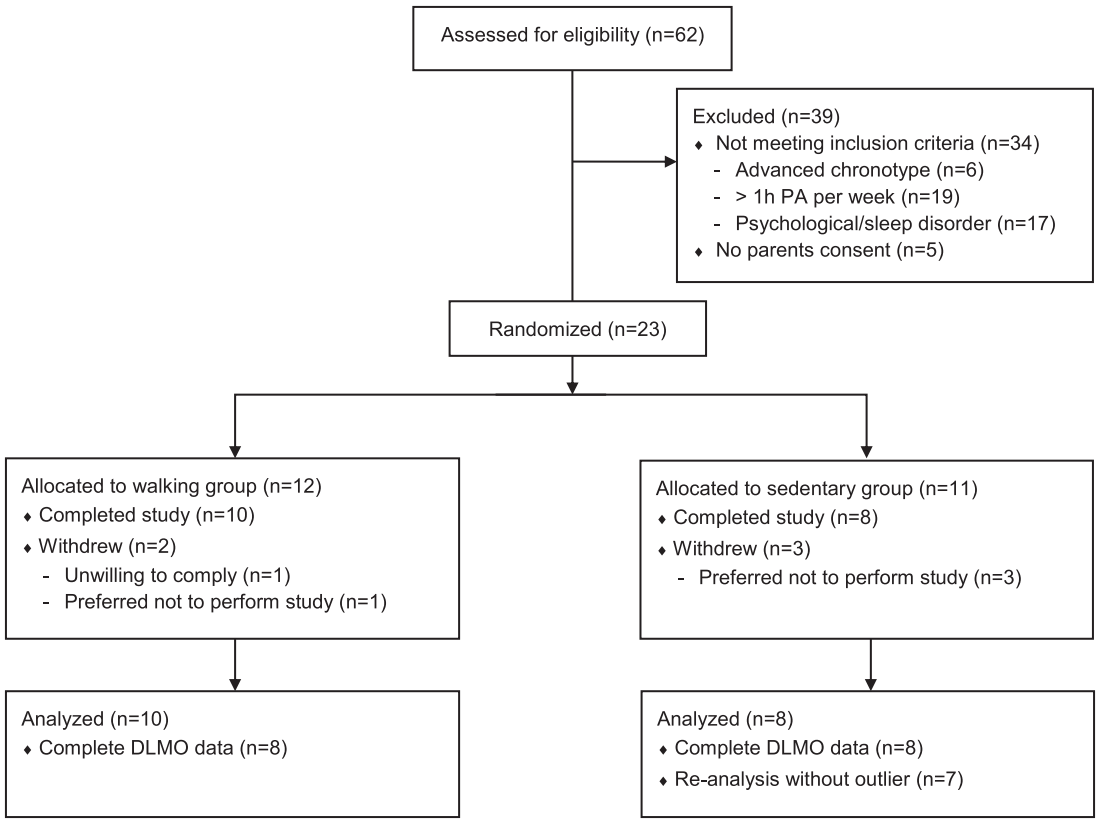

Figure 1. CONSORT diagram for this randomized trial. A total of 23 participants were randomized to a walking or sedentary group and 5 participants subsequently withdrew. While 18 participants completed the study, 16 were included in the analytic data set, as the DLMO could not be determined for 2 exercise participants. One participant had already exceeded the threshold level of 4.3 pg/mL at the time of the first baseline sample, which was taken 4 h prior to bedtime. The second participant had fluctuating melatonin levels at follow-up, which made it impossible to calculate the true onset. DLMO, dim light melatonin onset; PA, physical activity.

100.16-HREC/16/SAC/90). Written informed consent was provided by all study participants and their parents/caregivers, after inviting them to the sleep laboratory for familiarization with the environment and experimental procedures. Following completion of the study, adolescents were reimbursed with an AUD$50 gift voucher for their participation.

To meet inclusion criteria, interested adolescents had to score as of late chronotype on the Munich Chronotype Questionnaire ($MS_{Fsc} \geq 4.28$) [40, 41], and must not have traveled across time zones in the 2 months prior to the study. Further inclusion criteria were: male, aged between 15 and 18 years, physically inactive (<60 min physical activity per week), absence of evidence of sleep apnea and other pediatric sleep disorders (Paediatric Sleep Questionnaire; PSQ) [42], as well as psychological disorders, which have been linked to altered circadian rhythms (i.e. bipolar disorder [43], autism [44], and ADHD) [45]. Exclusion criteria included having physical conditions that needed medical advice before being physically active (e.g. resting heart rate [RHR] > 90 BPM), intake of psychopharmaceuticals, as some antidepressants impact the biotransformation of melatonin [46, 47]. Female adolescents were not included in this study, as the primary outcome was the circadian phase, and inconsistent evidence exists as to whether circadian rhythms change across the menstrual cycle [48–50]. To overcome this issue, previous research has suggested including females during their luteal phase, which occurs between ovulation and the beginning of their menstrual cycle [51]. Since we only had 1 week available during a 2-week holiday period, we were concerned about fitting the laboratory procedure within this biological timeframe, yet, remaining aware of the limitation that this causes.

Physical inactivity is defined as the non-achievement of physical activity guidelines [52], which leaves a great deal of room for interpretation. The review of 42 trials by Bennett and colleagues [53] showed that the large range of "inactive" samples in the literature increases the risk of response to training bias, making it difficult to understand and generalize findings. Ideally, the cut-point for defining a physically inactive lifestyle in research studies should be similar to public health prevalence statistics, but also not too low, making it difficult to recruit individuals for research trials [53]. For adolescents, the current WHO 2020 guidelines on *Physical Activity and Sedentary Behavior* [54] recommend at least 60 min of moderate-to-vigorous physical activity per day. Therefore, the present study adopted the recommended cut-point of <60 min physical activity per week from Bennet and colleagues [53] to define a physically inactive lifestyle.

Laboratory protocol

Study period and location

Data collection occurred from July 2016 to April 2017 during 1 regular school week (pre-week), immediately followed by 1 week of school holidays (sleep laboratory week). The laboratory week involved 6 consecutive nights and 5 consecutive mornings at the Flinders University Sleep and Circadian Research Laboratory. The sleep laboratory contains 4 bedrooms, allowing groups of

4 to be assessed per week. Light exposure was dim (<10 lux) throughout the experiment in the sound-attenuated laboratory. In the 72 h before and during saliva collection, adolescents were asked to avoid caffeine, nicotine, alcohol, and foods thought to affect habitual melatonin secretion (e.g. chocolate, bananas, and tomatoes) [55].

Pre-laboratory procedures

In consultation with each participant, we established a fixed sleep–wake schedule for both school days and weekend days based on the indicated sleep–wake times from the Munich Chronotype Questionnaire (MCTQ) [56]. One week prior to their laboratory stay, adolescents were instructed to adhere to their fixed baseline sleep schedule and to abstain from any exercise involving more than 60 min of moderate-to-vigorous PA (MVPA) per week. Reminders were sent via text messages, and compliance was checked by a sleep and activity diary. For sleep, a deviation of ±30 min on a maximum of 3 days was accepted. Bedtime and wake time minus time to fall asleep (sleep onset latency [SOL]) were used to calculate total sleep time (TST). TST, SOL, and mid-sleep time from the pre-week sleep log were aggregated separately for school nights and weekend nights and presented in Table 1. Nights were defined as weekday nights if participants went to school the next day; weekend nights were Friday and Saturday. Weekly physical activity levels were not allowed to exceed the threshold of 60 min MVPA. In the daily activity log, MVPA was defined as "any physical activity that you have done continuously for at least 10 min, for recreation, sport, exercise, leisure, or to get to or from places, which made you breathe harder and sweat."

Laboratory procedures

Study participants arrived on a Sunday evening (05:00 pm) for baseline assessments at the sleep laboratory (Figure 2). Daytime sleepiness and mood were assessed via self-report questionnaires upon arrival (~05:30 pm). To estimate evening sleep propensity, sustained vigilance and perceived sleepiness were assessed four times during the evening, using the Karolinska Sleepiness Scale (KSS) and the Go/No-go task at 3, 2, 1, and 0 h before bedtime. At other times, participants interacted with each other and staff members. Board/card games and television were provided in the communal lounge room. Mobile phones were not allowed. Participants were monitored to ensure wakefulness until their set bedtime. Meals and snacks were provided at set times consistently for all study participants, with no access to food at other times. Free access was provided to water, while caffeine consumption was prohibited.

With some exceptions, the laboratory protocol used in the present study is similar to the bright-light treatment protocol for delayed sleep-wake phase disorder (DSWPD) [32] that has been used for many years in clinical practice and research [57–60]. Briefly, the bright-light treatment protocol involves (1) letting the patient sleep in followed by bright-light treatment for 1 h, (2) advancing rise time by 30–60 min per day as the human circadian rhythm shifts earlier in response to bright light, (3) treatment advancement stops at desired wake-up time, and (4) bright light continues for at least 1–2 weeks to stabilize circadian timing [57, 58]. The present study replaced bright light with exercise as a chronotherapeutic agent. Accordingly, participants were allowed to sleep in on their first morning at the sleep laboratory, followed by their assigned morning activity. From that point onward, the wake-up time and morning activity progressively advanced by 30 min each day. Contrary to the bright-light treatment protocol, treatment advancement in the present experiment ended after the participant's laboratory stay. A fixed bedtime was maintained from the pre-week (school night) to avoid the possibility of a phase shift resulting from an advanced sleep time.

Morning activities included either 45-min treadmill walking or 45-min sedentary behavior (i.e. watching TV on a dimmed screen). We used the Karvonen formula, which is widely used in rehabilitation and exercise fields, to standardize exercise intensity for each individual by measuring RHR upon awakening [61, 62]. The Karvonen formula provides general "rule-of-thumb" target heart rates, with light-intensity exercise being defined as activity using 30%–40% of the heart rate reserve (HRR) [63]: 220 – age = HR_{max}; HR_{max} – RHR = HRR; $HR_{lower\ limit}$ (30%): (HRR * 0.3) + RHR = … bpm; $HR_{upper\ limit}$ (40%): (HRR * 0.4) + RHR = … bpm. In the present study, RHR was measured with a wrist-worn heart rate monitor connected to a chest strap (Polar rs100). Shortly after, adolescents started their allocated morning activity in dim light (<10 lux; approx. 15 min after waking). Participants' heart rates were monitored throughout the exercise to maintain this zone of intensity. When the participant's heart rate deviated from the target zone, an alarm was sounded and the speed was adjusted accordingly. Post hoc analyses showed that exercise timing occurred on average 12.32 h after baseline DLMO.

After the completion of the morning protocol and the opportunity to take a shower (approx. 10:00 am–01:00 pm), participants were picked up by their parents to spend the afternoon at home. The participants returned to the laboratory by 05:00 pm every evening, to ensure that daylight exposure remained constant for all participants irrespective of seasonal variations. This procedure improved acceptance by both participants and the primary caretaker. During the day, participants were not allowed to nap or exercise. As in the pre-week, compliance was monitored through the use of an ActiGraph Gt3x device worn on the nondominant wrist, as well as a daily activity log (data not included here).

Measures

Munich Chronotype Questionnaire (MCTQ)

The Munich Chronotype Questionnaire (MCTQ) was used as a screening tool to identify late chronotype adolescents. The self-rated questionnaire estimates chronotype based on the midpoint between average sleep onset and offset on school-free days (mid-sleep on free days, MSF), corrected for "oversleep" due to the sleep debt that individuals accumulate over a school week (MS_{Fsc}) [64]. This proxy for chronotype is based on the assumption that sleep timing on school-free days is highly influenced by an individual's circadian clock. Therefore, chronotype (MS_{Fsc}) can only be calculated when participants can sleep in on their school-free days. Specifically, the MCTQ asks about bedtime, time spent in bed awake before deciding to turn off the lights, how long it takes to fall asleep, wake-up time, and out-of-bedtime for both school- and free days. Validation against the gold standard for assessing circadian phase (DLMO) was high [65–68]. Based on data from 29,500 electronic questionnaires, Kühnle [41] classified an MS_{Fsc} score of ≥4.28 as late chronotype

Table 1. Baseline characteristics of study participants

Characteristics	Morning exercise group		Sedentary group			
	M ± SD	(Range)	M ± SD	(Range)	F	p
Age in years	16.20 ± 1.23	(15 – 18)	16.75 ± 0.71	(16 – 18)	1.26	0.279
BMI	23.41 ± 4.82	(17.28 – 32.41)	23.60 ± 5.84	(17.67 – 33.81)	0.01	0.940
7-day sleep diary						
Bedtime (hh:mm)						
School night	23:46 ± 1:12	(22:30 – 2:06)	0:24 ± 0:51	(23:13 – 1:52)	1.56	0.230
Weekend night	1:13 ± 1:48	(23:00 – 4:15)	2:03 ± 1.47	(23:54 – 4:27)	1.14	0.301
Wake-up time (hh:mm)						
School night	7:54 ± 1:31	(6:30 – 11:24)	7:53 ± 1:46	(6:15 – 12:00)	0.00	0.979
Weekend night	10:06 ± 0:55	(9:30 – 12:10)	10:58 ± 0:54	(10:00 – 12:30)	4.07	0.061
First morning at laboratory*	10:45 ± 1:31	(8:19 – 13:40)	10:28 ± 1:18	(9:19 – 13:00)	0.01	0.928
SOL (min)						
School night	24.4 ± 22.87	(2 – 72)	12.49 ± 12.24	(2 – 36)	1.75	0.204
Weekend night	29.83 ± 35.09	(4 – 41)	10.78 ± 12.53	(4 – 120)	2.12	0.165
TST (hh:mm)						
School night	8:01 ± 1:17	(6:18 – 10:22)	7:27 ± 2:04	(5:17 – 11:50)	0.53	0.478
Weekend night	8:54 ± 1:24	(6:45 – 10:30)	9:07 ± 1:32)	(5:42 – 10:43)	0.10	0.756
MCTQ						
Mid-sleep time (hh:mm)						
School night (MSW)	3:53 ± 1:15	(2:37 – 6:14)	4:09 ± 0:33	(3:06 – 6:05)	0.24	0.692
Weekend night (MSF)	5:38 ± 1:14	(4:15 – 8:12)	6:24 ± 0:53	(5:17 – 7:47)	2.10	0.166
MS_{Fsc} (chronotype)	5.40 ± 1.27	(4.28 – 8.27)	5.76 ± 1:15	(4.34 – 7.23)	0.35	0.564
DLMO (h)	23:11 ± 1:20	(21:36 – 0:49)	23:04 ± 2:02	(20:31 – 2:31)	0.21	0.886
Daytime sleepiness (PDSS)	14.90 ± 2.56	(11 – 18)	16.25 ± 4.03	(9 – 22)	0.75	0.399
Mood (DASS)						
Stress	9.40 ± 5.42	(2 – 18)	9.43 ± 9.91	(0 – 30)	0.50	0.826
Depression	8.44 ± 6.69	(0 – 18)	11.71 ± 5.82	(8 – 14)	2.59	0.127
Anxiety	7.40 ± 5.74	(0 – 16)	8.00 ± 5.54	(4 – 20)	0.17	0.682

Data are presented as mean (M) and standard deviation (SD); baseline participant characteristics for the morning exercise and sedentary groups were compared using one-way ANOVA; SOL, sleep onset latency; TST, total sleep time; MCTQ, Munich Chronotype Questionnaire; MSW, mid-sleep time on school days; MSF, mid-sleep time on free days; MS_{Fsc}, mid-sleep time on free days corrected for sleep debt on school days; DLMO, dim light melatonin onset, PDSS; Pediatric Daytime Sleepiness Scale, DASS; Depression Anxiety Stress Scale-21.
*Participants were allowed to sleep-in on their first morning and from there onwards, gradually advanced wake-up time by 30 min on each subsequent day.

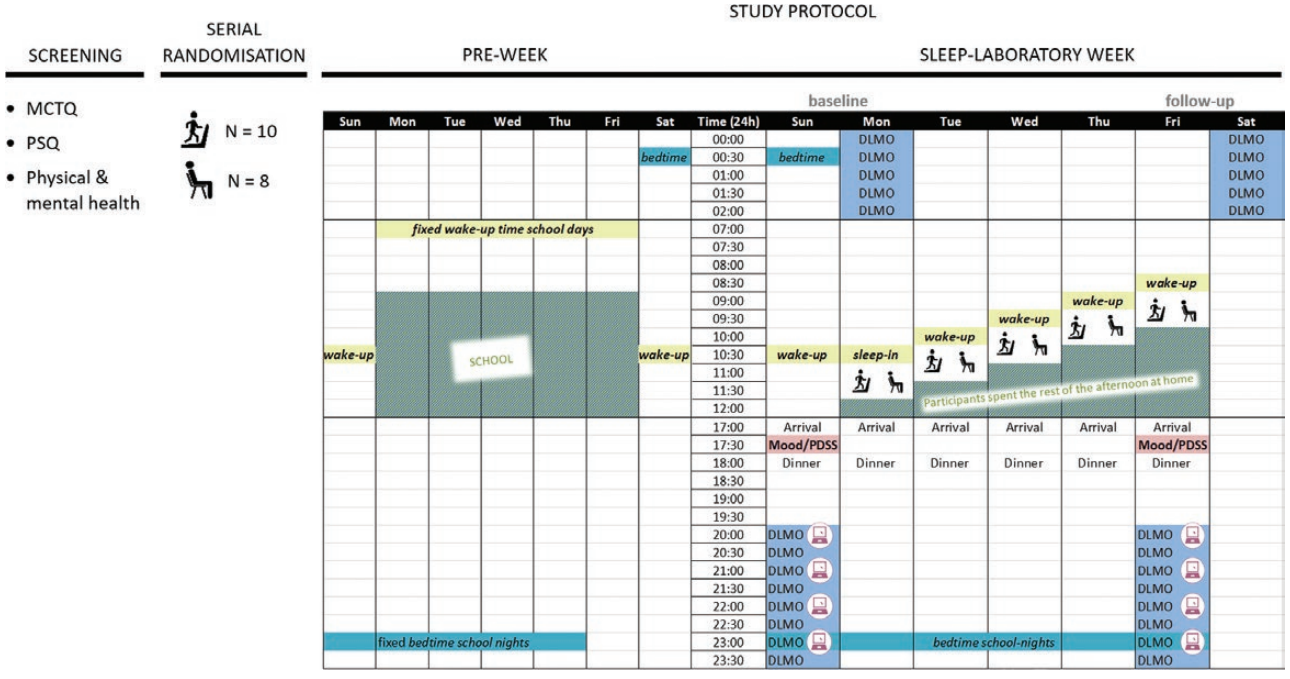

Figure 2. Study protocol. A late chronotype was screened via Munich Chronotype Questionnaire (MCTQ); the absence of sleep apnea and other sleep disorders via the Paediatric Sleep Questionnaire (PSQ). Schedules for pre-week and sleep laboratory week (DLMO, computerized Go/No-go task, KSS) are based on an individual's average bedtime and rise time. After the morning protocol (morning exercise vs. sedentary activity), participants were picked-up by their parents and spent the afternoon at home. They returned to the laboratory at 05:00 pm each day.

and ≥7.25 as an extreme late chronotype. Therefore, interested participants had to score ≥4.28 to be included in the present study.

Dim light melatonin onset

Salivary DLMO samples were taken half-hourly in dim light (<10 lux) using salivettes (Sarstedt, Newton, NC, USA) starting 4 h before and finishing 2 h after adolescents' typical bedtime [15]. Participants were seated for at least 5 min before and during each saliva sample, to minimize the masking effect of physical movement on endogenous melatonin production. Food and water were consumed only after saliva collection to reduce contamination or dilution of the sample. Participants were instructed to place the swab in their mouth and accumulate saliva for 2 min. After collection, samples were stored frozen at –20°C. This procedure was replicated during follow-up assessments. For analysis, samples were thawed and centrifuged for 10 min at 2500 rpm, the swabs removed from the casing, and the supernatant retained. A sensitive (4.3 pM) direct radioimmunoassay (RIA) using reagents from Buhlmann Laboratories AG (Allschwil, Switzerland) [69] was used to measure melatonin in the saliva. The intra-assay coefficient of variation (CV) was <10% at all times (mean = 4.5%). The inter-assay CV was 8.8% at 12.9 ppm and 13.1% at 104.5 ppm. The functional least detectable dose of the assay was 1.0 pg/mL. DLMO was calculated by linear interpolation across time points when melatonin concentration increased to 4.0 pg/mL or above [70]. The difference between follow-up DLMO relative to baseline DLMO (in minutes) was calculated as an indicator of circadian phase shift. Thus, phase advances were expressed as positive values and phase delays as negative values.

Daytime functioning

The Pediatric Daytime Sleepiness Scale (PDSS) is an 8-item self-report scale of daytime sleepiness (e.g. "How often do you fall asleep or feel drowsy in class?") [71]. Daytime sleepiness is a common symptom reported by adolescents with late chronotypes [10, 60, 72, 73]. Responses to each item are measured on a 5-point Likert scale (e.g. 0 = "Never", 4 = "Always"). Total scores range from 0 to 32, with higher scores indicating higher sleepiness (a score of ≥20 indicates clinically relevant daytime sleepiness [74]). The internal consistency of the data was good (Cronbach $\alpha = 0.80$). The PDSS has been shown to be sensitive to chronobiological treatment and was used to measure changes in daytime functioning [10].

The Depression, Anxiety, and Stress Scale (DASS-21) was used to assess secondary outcomes of morning exercise and/or circadian phase shift in mood. Seven items represent each subscale. In the depression subscale, items address hopelessness, self-deprecation, low levels of positive affect, and devaluation of life. In the anxiety subscale, items refer to physiological stimulation and subjective awareness of anxious affect. Items of the stress subscale include difficulty relaxing, tension, impatience, irritability, and restlessness. Participants were asked to indicate how much each statement applied to them over the past week, using a 4-point Likert scale, ranging from 0 ("Did not apply to me at all") to 3 ("Applied to me very much, or most of the time") [75]. Multiplying the score by two yields a total score between 0 and 42 [75]. In the present study, the internal consistency of the data was acceptable (Cronbach's alpha 0.76).

Evening sleepiness and vigilance

The KSS [76] was used to measure subjective sleepiness in the 3 h before bedtime. The KSS consists of a visual analogue scale, spanning 9 levels from 1 (extremely alert) to 9 (very sleepy, great effort keeping awake, fighting sleep). Adolescents were asked to circle the number that represents their current perceived level of sleepiness.

Go/No-Go task. Objective evening vigilance was measured immediately after each KSS rating. The computerized Go/No-go task (E-Prime v1.2, Psychology Software Tools, Inc., Pittsburgh, PA, USA, 2006) measures sustained attention in relation to inhibitory functions and consists of two visual stimuli presented in random order. Adolescents pressed the space bar within 500 ms if the letter "M" was shown on the screen (Go stimuli). If the letter "W" was shown, they were instructed not to press any buttons (No-go stimuli). A total of 80% of "M" letters were shown, in a quasi-random sequence. Approximately 200 "M" letters were shown during 8 min. Analyses were performed with commission error (falsely pressing the button in "No-go" trials), as well as reaction time (RT) of correct Go-trials. The latter outcome is usually not considered as an index of inhibitory control [77] but has previously been used as a measure of sustained evening vigilance [78, 79].

Sleep log

Adolescents completed a daily sleep log prior to the laboratory week. In the mornings, they indicated their bed- and wake-up time, as well as SOL. Nights were defined as weekday nights if they went to school the next day; weekend nights were Friday and Saturday. To compute data, weekday and weekend sleep parameters were aggregated separately.

Statistical analysis

All statistical analyses were performed using SPSS 28.0 (IBM Corporation, NY, USA). One-way ANOVA was used to compare baseline characteristics between groups. In order to determine the effects of exercise on circadian phase, mood, daytime sleepiness, evening sleepiness, and vigilance, change scores for the time and each group (exercise vs. sedentary) were calculated and analyzed using one-way ANOVA. Baseline scores of the respective change scores were used as covariates (except for DLMO). Due to the repeated measurement design, we also investigated the overall effects of evening sleepiness and vigilance using Linear Mixed Modelling (LMM) [80], with the repeated-measure factors day (baseline vs. follow-up) and time (3, 2, 1, and 0 h before bedtime), as well as the between-group factor (exercise vs. sedentary).

Test results with an alpha level <0.05 were reported as statistically significant. Due to the small sample size, effects sizes were additionally considered when interpreting results [81]. For one-way ANOVA, partial eta square is reported. Following Cohen [82] an effect size of $\eta^2 = 0.01$ indicates a small effect ([S] i.e. negligible practical importance), $\eta^2 = 0.06$ indicates a medium effect ([M] i.e. moderate practical importance), and $\eta^2 = 0.14$ indicates a large effect ([L] i.e. crucial practical importance). For LMM analyses, Cohen's *d* was used as the effect size to measure the magnitude of within- and between-group differences. Cohen's *d* was calculated as $d = M_1 - M_2/(SD_{pooled})$.

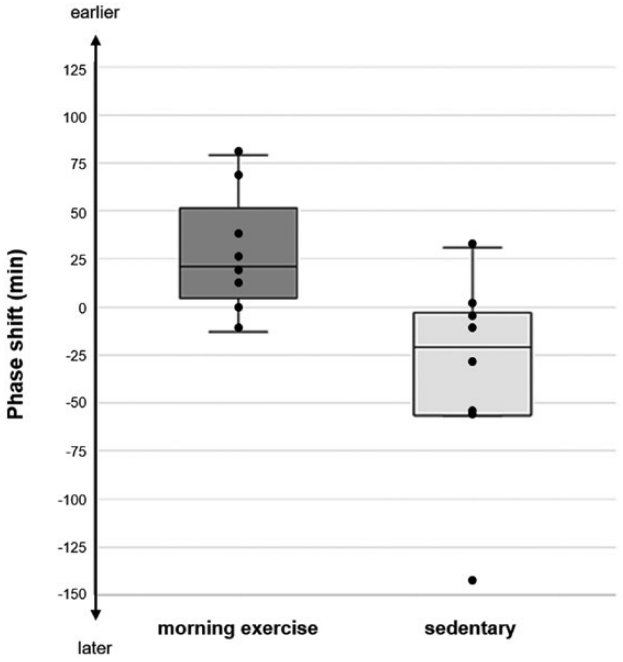

Figure 3. Over a 5-day period, low-intensity morning exercise, in contrast to sedentary activity, advanced the circadian phase of physically inactive adolescents with a late chronotype.

Results

Sample characteristics

Baseline characteristics of the study sample are presented in Table 1. There were no significant differences between groups with regard to age, BMI, sleep duration, and SOL on school and weekend nights, as reported in a daily sleep log the week prior to commencing the sleep laboratory. Groups did not differ by chronotype, mid-sleep time on both school and weekend nights, and DLMO upon laboratory entry. Regarding daytime functioning, no significant differences between daytime sleepiness and mood were found.

Effect of low-intensity morning exercise on DLMO

A total of 16 participants were included in the analysis, as the DLMO could not be determined for 2 exercise participants. One participant had already exceeded the threshold level of 4.3 pg/mL at the time of the first baseline sample, which was taken 4 h prior to bedtime. The second participant had fluctuating melatonin levels at follow-up, which made it impossible to calculate the true onset.

Scheduled morning exercise over 5 consecutive days had a significant phase-advancing effect on DLMO of 27.5 min (±32.0), while sedentary activity resulted in a DLMO phase delay of –34.3 min (±53.2), $F(1,13) = 7.92$, $p = 0.014$, $\eta^2 = 0.361$. However, analyses were re-run after the exclusion of an outlier in the control group (–144 min). Here, the sedentary activity group shows a slightly reduced DLMO phase delay of –18.6 min (±12.0), yet with a similarly large effect size in favor of the exercise intervention $F(1,12) = 7.81$, $p = 0.015$, $\eta^2 = 0.375$. Figure 3 represents the average and individual circadian phase shift for the morning exercise group ($N = 8$) and sedentary group ($N = 8$) highlighting inter-individual differences in the degree of phase shift.

Treatment effect on evening sleep propensity

Evening sleep propensity was assessed by measuring perceived evening sleepiness and objective evening vigilance.

Perceived evening sleepiness

Figure 4 illustrates the results of perceived sleepiness at 3, 2, 1, and 0 h before bedtime on baseline and follow-up night. Table 2 provides an overview of the descriptive statistics for each time point as well as an inferential statistic for change scores. The LMM (day × time × group) did not show an overall effect on perceived evening sleepiness that would imply consistent higher evening sleepiness scores across all time points following morning exercise, $F(3,81) = 0.52$, $p = 0.67$, $d = 0.03$. However, pairwise comparisons indicate that morning exercisers were more sleepy 2 h before their fixed bedtime on the follow-up night than their sedentary peers, $F(1,13) = 5.13$, $p = 0.039$, $\eta^2 = 0.255$; however, these differences diminished at 1 and 0 h before bedtime (Figure 4).

Evening vigilance

Figure 5 illustrates the results of evening vigilance based on error %, that is, false positive No-go trials (Go on No-go trials) and RT for Go trials of the computerized Go/No-Go task. Table 2 provides an overview of the descriptive statistics for each time point as well as an inferential statistic for change scores.

Error %. The LMM (day × time × group) did not show an overall effect for error % that would imply consistent higher error % across all time points following morning exercise, $F(3,81) = 0.70$, $p = 0.560$, $d = 0.26$. Pairwise comparisons indicate a trend that morning exercisers produced more errors 1 h before their fixed bedtime than their sedentary peers, $F(1,13) = 4.08$, $p = 0.062$, $\eta^2 = 0.214$.

Reaction time. The LMM (day × time × group) did not show an overall effect for RT that would imply a consistent lower RT across all time points following morning exercise, $F(3,84) = 0.34$, $p = 0.80$, $d = -0.16$. Pairwise comparisons show no significant differences between groups, which reinforces the observed trend in error % 1 h before bedtime since RT did not improve.

Overall, evening vigilance in the last hour before bedtime tends to be slightly higher among sedentary participants in comparison to those who exercised in the morning. However, these group differences leveled out at bedtime.

Treatment effect on daytime functioning

Table 2 provides an overview of the descriptive statistics for each measuring time point as well as an inferential statistic for change scores.

Mood. Morning exercise did not lead to improved mood after 5 days, as both groups scored lower on all symptom subscales at the end of their laboratory stay (time effect). However, there was a significant drop in anxiety scores among participants of the sedentary group, leading to a significant time × group effect in favor of sedentary morning activity, anxiety: $F(1,13) = 4.54$, $p = 0.051$, $\eta^2 = 0.245$; depression: $F(1,13) = 2.40$, $p = 0.147$, $\eta^2 = 0.167$; stress: $F(1,13) = 0.97$, $p = 0.341$, $\eta^2 = 0.065$.

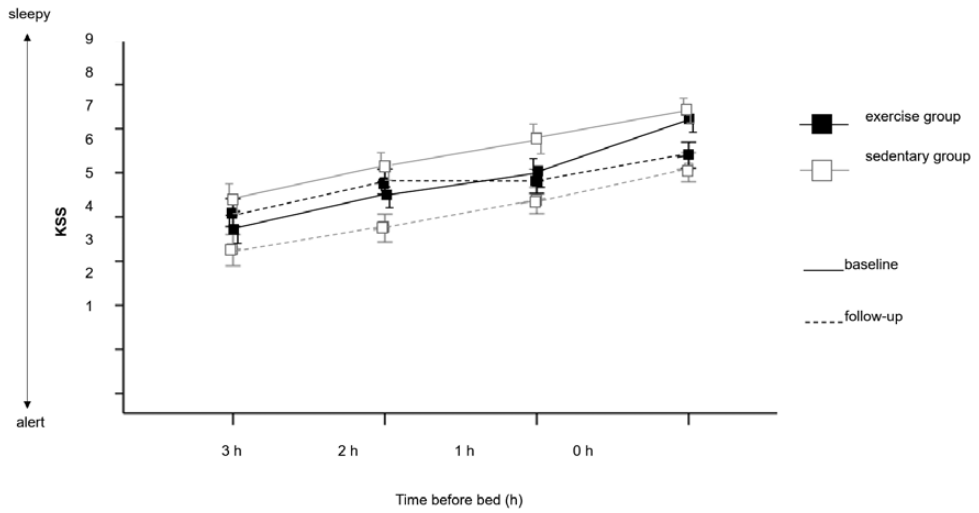

Figure 4. Evening sleepiness. Time course of the KSS (KSS; mean values ± SEM). 1 = extremely alert; 5 = neither alert nor sleepy; 9 = very sleepy, great effort to keep awake. Morning exercisers were more sleepy 2 h before their bedtime at follow-up than their sedentary peers; however, these differences diminished at 1 and 0 h before bedtime.

Daytime Sleepiness. There was no significant time × group interaction effect in daytime sleepiness following 5 days of morning exercise, $F(1,13) = 2.56$, $p = 0.130$, $\eta^2 = 0.146$.

Discussion

The present study is the first to demonstrate the phase-advancing potential of low-intensity morning exercise. After 5 mornings of treadmill walking in dim light for 45 min, circadian timing advanced by 27.5 min in comparison to sedentary controls, which were delayed by 34.3 min. Hence, this adds to the scarce literature on the phase-advancing effect of morning exercise [32, 34, 35], and supports the newly established PRC of exercise by Youngstedt and colleagues [32]. It is also the first study that included physically inactive adolescents with a late chronotype. This is in contrast to previous research, which has almost exclusively implemented moderate-to-vigorous exercise among healthy and aerobically fit adults to achieve circadian phase shifts [20, 28, 39, 83, 84].

In individuals with circadian rhythm sleep–wake disorders, a threshold of a 30-min circadian phase-change has been determined to be clinically significant by the Task Force of the American Academy of Sleep Medicine [82]. Given that the present study only included 5 days of exercise conducted shortly after awakening, an advancement of 27.5 min highlights the potential of timed morning exercise as an alternative treatment approach. Unlike previous research with normal sleepers, in which morning exercise took place at 7:00 am [32], the exercise session in this sample began between 08:45 am and 01:30 pm on the first morning, and 6:45 am and 11:30 am on the last morning. Though it may not be morning by most definitions, 7:00 am for normal chronotypes may correspond to 10:00 am or 12:00 pm for late chronotypes. As a consequence and to avoid previous situations where exercise was timed in the phase-delay region of the phase response curve to exercise [31, 34], the present sample was allowed to sleep on their first morning. This approach is based on principles from the bright-light treatment protocol, in order to increase the likelihood that scheduled morning exercise occurs after an individual's lowest core body temperature point (~2 h before natural wake-up time) [85, 86], which marks the crossover point between an individual's circadian phase-advancing and phase-delaying region [58, 87].

In the present sample, a phase-delaying effect specifically occurred in the sedentary condition. Considering a human's circadian rhythm naturally delays in dim light conditions [88], a circumstance that often exacerbates a late chronotype, specifically among adolescents engaging in sedentary activities during school holidays [10, 89] (or as shown recently, during COVID-19 lockdowns), our findings highlight the effect of morning exercise on circadian timing among young people. We found a conservative phase difference of 46.1 min between groups after 1 week, which when extrapolated to typical 2-week school holiday periods would result in a phase difference of around 1.5 h. Our findings suggest that adolescents who are consistently sedentary in the morning are at risk of clinically significant phase delays that will conflict with their school start times—possibly placing them at risk for developing DSWPD [90–92]. Thus, correctly timed morning exercise may provide some protection to move these at-risk adolescents away from such a conflict.

However, we also observed individual differences, with 70% of adolescents showing a phase advance in response to scheduled morning exercise, and 75% of adolescents showing a phase delay in response to morning sedentary activity. Further replication studies are needed to identify which adolescents benefit (and which ones do not) from scheduled morning exercise. Since the time of rising on the first morning set the starting point for the allocated morning activity, it cannot be ruled that some adolescents awoke earlier than they would have at home. Youngstedt's 7:00 am morning exercise group began to exercise on average 10.83 h after the onset of melatonin. No phase-shifting effect was observed for 10:00 am, which would have occurred approximately 13.83 h after melatonin onset [32]. The most recent study demonstrating the phase-advancing proportion of morning exercise timed the starting point 10 h after melatonin onset [34]. In the present study, exercise on the first morning occurred on average 12.32 h after melatonin onset and was gradually advanced to around 10.32 h on the last morning.

Table 2. Descriptive and inferential statistic of main outcome variables (*N* = 18).

	Exercise group				Sedentary group						
	Pre		Post		Pre		Post		Statistical analyses		
Measures	M (*SD*)	95% CI	M (*SD*)	95% CI	M (*SD*)	95% CI	M (*SD*)	95% CI	*F*	*p*	η^2
DLMO	23.18 (1.34)	22.16, 24.21	22.36 (1.13)	21.49, 23.23	23.06 (2.04)	21.36, 24.77	23.76 (2.05)	22.04, 25.48	7.92	0.014	0.361
Mood (DASS)											
Depression	8.44 (6.69)	3.30, 13.59	4.60 (5.74)	0.49, 8.71	11.71 (5.82)	6.33, 17.10	2.57 (2.51)	0.25, 4.89	2.40	0.147	0.167
Anxiety	7.40 (5.74)	3.29, 11.51	6.80 (6.94)	1.83, 11.77	8.00 (5.54)	2.88, 13.12	3.00 (3.21)	0.32, 5.68	4.54	0.051	0.245
Stress	9.40 (5.42)	5.52, 13.28	6.80 (6.88)	1.88, 11.72	9.43 (9.91)	0.26, 18.60	4.75 (7.32)	–1.37, 10.87	0.97	0.341	0.065
Daytime Sleepiness (PDSS)	14.90 (2.56)	13.07, 16.73	15.10 (4.75)	11.70, 18.50	16.25 (4.03)	12.88, 19.62	11.75 (5.78)	6.92, 16.58	2.56	0.130	0.146
Evening Sleepiness (KSS)											
3 h before bedtime	4.80 (1.99)	3.73, 5.87	5.10 (2.33)	4.03, 6.17	5.50 (0.54)	4.30, 6.70	4.25 (1.49)	3.05, 5.45	1.68	0.214	0.101
2 h before bedtime	5.60 (1.78)	4.53, 6.67	5.80 (2.04)	4.73, 6.87	6.25 (0.71)	5.05, 7.45	4.75 (1.39)	3.55, 5.95	5.13	0.039	0.255
1 h before bedtime	6.10 (1.91)	5.03, 7.17	5.80 (1.87)	4.73, 6.87	6.88 (0.99)	5.68, 8.07	5.38 (1.41)	4.18, 6.57	1.32	0.268	0.081
0 h before bedtime	7.30 (1.57)	6.23, 8.37	6.40 (2.12)	5.33, 7.47	7.50 (0.76)	6.30, 8.70	6.13 (1.46)	4.93, 7.32	0.88	0.363	0.055
Evening vigilance (Go/NoGo)											
Error %											
3 h before bedtime	2.05 (1.04)	1.26, 2.84	3.23 (1.86)	2.44, 4.01	2.33 (1.15)	1.45, 3.21	2.63 (1.13)	1.75, 3.52	0.62	0.443	0.040
2 h before bedtime	1.73 (0.46)	0.94, 2.51	2.19 (0.77)	1.40, 2.98	1.76 (0.43)	0.88, 2.64	2.44 (1.47)	1.56, 3.32	0.18	0.676	0.012
1 h before bedtime	3.03 (1.97)	2.25, 3.82	2.45 (1.09)	1.67, 3.24	2.20 (0.85)	1.31, 3.08	1.37 (0.69)	0.49, 2.26	4.08	0.062	0.214
0 h before bedtime	2.11 (0.83)	1.32, 2.90	2.14 (1.26)	1.35, 2.93	2.06 (0.85)	1.18, 2.94	2.51 (2.30)	1.63, 3.39	0.30	0.590	0.020
Reaction time											
3 h before bedtime	312.30 (31.07)	286.29, 338.31	313.10 (47.32)	287.09, 339.11	317.50 (42.45)	288.42, 346.58	334.87 (43.04)	305.80, 363.95	0.91	0.356	0.057
2 h before bedtime	312.60 (37.17)	286.59, 338.61	312.60 (37.46)	286.59, 338.61	324.25 (48.07)	295.17, 353.33	320.25 (31.81)	291.17, 349.33	0.01	0.928	0.001
1 h before bedtime	327.20 (45.80)	301.19, 353.21	314.60 (37.92)	288.59, 340.61	324.00 (40.81)	294.92, 353.08	313.50 (35.22)	284.42, 342.58	0.00	0.967	0.000
0 h before bedtime	333.60 (48.03)	307.59, 359.61	318.50 (37.22)	292.49, 344.51	328.13 (37.37)	299.05, 357.20	323.62 (27.93)	294.55, 352.70	0.31	0.586	0.020

M, mean; SD, standard deviation; 95% CI, confidence interval; DLMO, decimal time format; Mood, higher scores reflect worse mood; PDSS, higher scores reflect higher daytime sleepiness; KSS, higher scores reflect higher evening sleepiness; Go/NoGo, higher errors reflect lower evening vigilance, lower RT (ms) reflect lower evening vigilance; ANOVA, one-way ANOVA with change scores and baseline scores as covariate.

Whether or not a phase-angle of 10 h is more sensitive needs to be investigated further.

During the onset of adolescence, the commonly observed shift toward an evening circadian preference may also contribute to greater mood difficulties [93–95], thus, highlighting the importance of low-barrier sleep-health interventions. Several studies have revealed that later chronotype adolescents show increased symptoms of depression, and less positive mood compared with their early chronotype peers, independently of sleep duration [96–98]. Dolsen and Harvey [99] further investigated this hypothesis among 163 adolescents with an evening circadian preference. Their findings partially support the proposed relationship between mood difficulties and evening preference, as higher negative effect was cross-sectionally associated with a later DLMO timing. However, the present study did not provide evidence that participants with an advanced circadian phase shift showed an advantage with regard to improved mood, as depression, anxiety, and stress symptoms were reduced meaningfully in both study conditions. Moreover, the vast majority of research in this field indicates that exercise intensity moderates the antidepressant effect significantly, in that at least moderate intensity is required to achieve measurable impact [100, 101]. Yet, proposing higher exercise intensities in the morning to adolescents classified as late chronotypes, particularly those with physically inactive lifestyles, may result in resistance to this intervention.

A gradual advance in wake-up time together with a fixed bedtime may have resulted in a mild sleep restriction during the experimental week, yet, sleep schedules were less restricted than during adolescents' regular school week. In addition, all daytime sleepiness scores ranged below the clinical cutoff of 20. Interestingly, while daytime sleepiness scores remained stable in the exercise condition, they dropped in the sedentary condition. Previous research has indicated that exercise plays a role in the regulation of homeostatic sleep regulation [102–104]. Sedentary behavior coupled with the circadian delay observed in the present control condition may explain the reduction in daytime sleepiness scores that

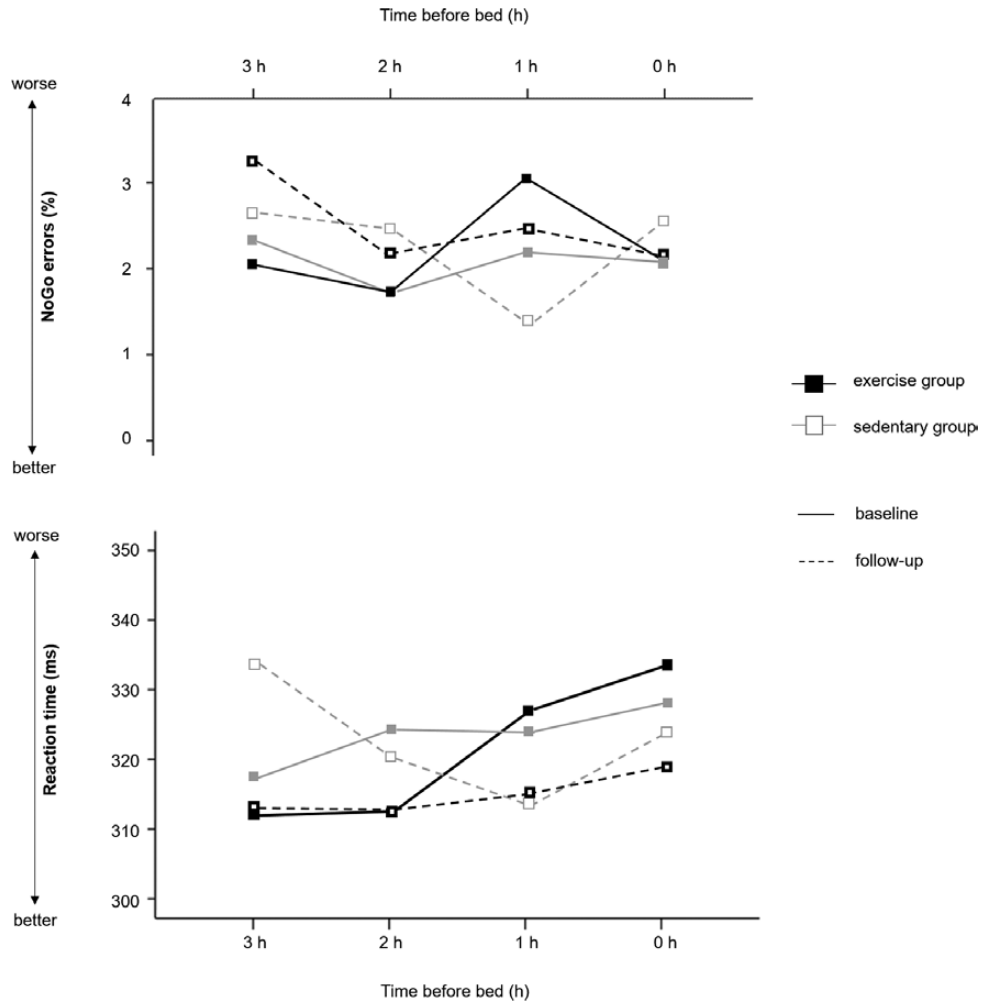

Figure 5. Go/No-Go task: time course of No-go errors (%), reflecting participant's accuracy, and RT for Go-trials. Accuracy should always be considered in relation to RT. The RT at follow-up 1 h before bedtime is similar between the two groups, however, it occurs with a great loss of accuracy in the exercise group. Error bars were excluded as inclusion led to visual overcrowding.

were assessed in the late afternoon. Whether low-intensity morning exercise may also be beneficial for building up sufficient sleep pressure to enable sleep at a decent clock time, however, could not be confirmed in the present study. So far, research on adolescents' sleep health emphasizes the general importance of regular exercise [36, 105–107].

A further aim of the study was to investigate whether phase advances would result in greater evening sleepiness and reduced evening vigilance before bedtime. Evening sleepiness (KSS) in the present study 2 h before bedtime was slightly higher among morning exercisers compared with their sedentary peers, post-intervention. This mirrors findings from a previous study, in which sleepiness scores were assessed in healthy young adults before and after undergoing a circadian phase shift protocol [108]. Participants of the advanced group reported significantly higher sleepiness scores in the earlier hours of the night, compared with participants with a prescription designed to delay the circadian phase. A slightly different pattern was observed for evening vigilance (Go/No-go), with lower performance in the exercise group 1 h before bedtime. Yet once again, the sedentary group reached similar levels at bedtime. As such, our results are partially in line with earlier findings in healthy young men [78], which revealed evening RTs in the Go/No-go task were sensitive to changes in the circadian phase. Since the participants of our study were required to adhere to a forced wakefulness protocol until their fixed bedtime, it remains uncertain whether or not the small effects found at 2 and 1 h before bedtime would have translated into earlier bedtimes.

Limitations and Future Directions

The study involved only male adolescents, which may limit the generalizability of the findings to female adolescents. Further, due to the challenge of finding motivated, physically inactive, late chronotype adolescents to spend 1 week of their school holidays in a sleep laboratory, the present study had a sample size that may be considered small. To this end, we supplemented inferential statistics with effect sizes that can be interpreted for their meaningfulness. For example, there is a clear and meaningful difference in circadian shifts between the two groups (61.8 min), with advancement for the morning exercise condition, and a delay in the sedentary condition. For the Go/No-go data, a larger sample will not yield a significant or meaningful effect as 31

out of 32 data points are similar between the groups and across time (Figure 5). However, further studies with a larger and more gender-balanced sample are necessary to confirm these findings.

According to the bright-light treatment protocol, treatment advancement proceeds until the targeted wake-up time is reached, and then continues for at least 1–2 weeks to stabilize circadian timing. Due to the nature of the study design, treatment advancement stopped at the end of the 5-day laboratory experiment. We also did not follow-up with adolescents to test the sustainability of the obtained circadian phase advancement and whether the observed findings are translated into earlier bedtimes and longer sleep durations on school nights. Therefore, a key challenge remains in linking such experimental results generated under highly-controlled laboratory conditions to the real world, where environmental stimuli are complex and feedback loops between physiology and behavior are interacting [1]. Lastly, as a second phase-advance portion has been reported from exercising in the afternoon (01:00 pm–04:00 pm) in entrained individuals [32], future research could investigate the effects of mid-late afternoon exercise alone, or in combination with morning exercise. However, one should consider that 07:00 am or 04:00 pm for normal sleeping individuals may equal 10:00 am or 06:00 pm among late chronotypes. Moreover, determining whether morning bright light therapy in conjunction with low-intensity exercise produces an additive effect among late chronotype adolescents would provide valuable basic and applied scientific knowledge. In entrained individuals, the combination has been shown to produce greater phase shifts in a single day [31] and therefore, could be implemented as a "walking to school" intervention in countries where ambient light is abundant.

Conclusion

Scheduled low-intensity morning exercise has been shown in the present study to advance the circadian timing of physically inactive adolescents with a late chronotype. Thus, confirming the previously established PRC for exercise [32], in which exercise—as a non-photic time cue—acted similarly to the PRC of bright light. Despite the change in adolescents' underlying circadian biology, no consistent changes in evening sleepiness and depression symptoms were apparent. We encourage future replication studies in this neglected area, so we can be more confident of the effects of scheduled morning exercise. The field will also benefit by incorporating follow-up assessments to witness any latent changes in secondary outcomes (e.g. depression symptoms), as well as discovering the transference of these laboratory findings to an adolescent's real life.

Funding

This work was supported by the Swiss National Science Foundation (Early Postdoc. Mobility Fellowship [P2BSP1-165373] to CL).

Acknowledgments

The first author would like to thank Dr. Harald Seelig for providing insightful discussions on methodological and statistical approaches.

Disclosure Statement

The authors have no conflict of interest to declare.

Author Contributions

Conceptualization by CL and MG; data acquisition by CL; data pre-processing by CL; data analysis by CL and MS; methodology by CL, MG, and MS; results interpretation by CL, CR, and MG. This manuscript was written by CL and all authors revised and edited the manuscript.

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