**Low-Intensity Scheduled Morning Exercise for Adolescents with a Late
Chronotype: A Novel Treatment to Improve Sleep Health?**

¹Lang, C., ²Richardson, C., ³Short, M., & ³Gradisar, M.

*¹Department of Sport, Exercise, and Health, Section Sport and Psychosocial Health, University
of Basel, Basel, Switzerland*

*²School of Psychological Science, Centre for Sleep Science, University of Western Australia,
Perth, Australia.*

³College of Education, Psychology and Social Work, Flinders University, Adelaide, Australia.

Correspondence should be addressed to Christin Lang, Department of Sport, Exercise and
Health, University of Basel, Basel, Switzerland. Email: christin.langunibas.ch

Abstract

Study Objectives. During adolescence, an interplay between biological and environmental factors leads to constrained sleep duration and timing. Given the importance of restorative sleep for cognitive, emotional, and physical health during this developmental period, the high prevalence of sleep deprivation is a public health concern. 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 minutes on 5 consecutive mornings, on circadian timing and daytime functioning of adolescents with a late chronotype, compared to a sedentary control group.

Methods. 19 sedentary male adolescents aged 15-18 years spent one week of school holiday at the sleep laboratory. Morning procedure included either 45 min walking on a treadmill or sedentary activities in dim light. Saliva dim light melatonin onset (DLMO), evening sleepiness and daytime functioning were assessed during the first and last night of laboratory attendance.

Results. The morning exercise group had significantly advanced (earlier) circadian timing ($27.5 \text{ min} \pm 32.0$), while sedentary activity resulted in a phase delay ($-34.3 \text{ min} \pm 53.2$). Morning exercise also led to higher evening sleepiness in the earlier hours of the night, but not at bedtime. Mood measures reduced meaningfully in both study conditions.

Conclusions. These findings highlight the preventative 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.

Keywords. chronotherapy, dim light melatonin onset, evening sleepiness, evening vigilance, mood, physical exercise

Statement of Significance

This is the first study to examine the phase-advancing potential of not only low-intensity morning exercise, but also among sedentary 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 sedentary.

The findings of the present experiment show support for the phase-advancing effect of 45-min of morning exercise compared to sedentary controls. Hence, adding to the scarce literature on the phase-advancing effect of morning exercise and supporting the newly established PRC of exercise by Youngstedt and colleagues (2019).

1. Introduction

Today's adolescents face a common chronic health problem: sleep restriction ¹. Research overwhelmingly supports a fundamental role of sleep for 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}, psychosocial functioning, academic performance and risk taking behaviours ^{7,8}.

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

Besides the psychosocial changes that contribute to later bedtimes and inadequate sleep duration, two of the primary contributing factors are the ubiquitous delay and lengthening of the circadian rhythm (an individual's natural internal sleep-wake cycle that repeats roughly every 24 hours); 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 mid-point of sleep known as a 'late chronotype' ^{16,19}. However, solutions are available to manage or even reverse this late chronotype – each with their 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 exercise - as movement has the potential to phase advance the human circadian rhythm - independently of bright light ^{25–27}. Most research 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 hour of moderate exercise completed at 7am over 3 consecutive days in adults ³². These results and a recent published trial ³⁴ suggest that exercise completed shortly after awakening has the greatest phase-advancing effect for later chronotypes ($0.54 \pm 0.29\text{h}$ vs. $0.46 \pm 0.25\text{h}$ after 5 days). Furthermore, gradual advancement (e.g., 20–30 min) of the timing of exercise may accelerate phase advances ³⁵. This protocol overlaps with the protocol for sleep scheduling, which is a core component of bright light therapy for adolescents and involves shifting bed and wake time by 30 minutes daily 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 the benefits to sleep, scheduled morning exercise may also have positive flow on effects for adolescents' wellbeing, including reduced daytime sleepiness and improved mood and cognitive performance ^{36,37}.

The present study addresses this gap in the literature by evaluating the effect of a gradually advanced morning exercise schedule (30 min earlier/day), completed for 45 minutes on 5 consecutive mornings, on the circadian timing (i.e., dim light melatonin onset) and daytime functioning (i.e., sleepiness, vigilance, mood) of adolescents with a late chronotype. The effect of the exercise intervention was compared to a control group, who also woke up 30 minute

earlier each morning, yet were sedentary for 45 minutes after awakening. Moreover, previous studies investigating the chronotherapeutic effect of exercise have exclusively implemented moderate-to-vigorous exercise bouts among 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 moderate-to-vigorous exercise in the morning, particularly if they are typically sedentary. With this in mind, this is the first study to examine the phase shifting effect of low-intensity morning exercise among sedentary adolescents with a late chronotype, in a light-controlled environment (i.e., to isolate the effect of exercise). We hypothesised 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 control group.

2. Methods

2.1 Participants

Sixty-two 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 ($M_{\text{age}} = 16.4$ yrs, $SD = 1.0$). The randomized controlled trial was approved by the Southern Adelaide Clinical Human Research Ethics Committee (SAC HREC application number OFR 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. As reimbursement for their participation, adolescents received an AUD\$50 gift voucher upon completing the study.

To meet inclusion criteria, interested adolescents had to report a late chronotype on the Munich Chronotype Questionnaire (MCTQ) ⁴⁰, and must not have travelled across time zones in the 2 months prior to the study. Further inclusion criteria were: male, aged between 15-18 years, sedentary (<60 min physical activity / week), absence of evidence of sleep apnea and other pediatric sleep disorders (Paeditric Sleep Questionnaire; PSQ) ⁴¹, as well as psychological disorders, which have been linked to altered circadian rhythms (i.e., bipolar disorder, ⁴²; autism

⁴³; and ADHD, ⁴⁴. Exclusion criteria included having physical conditions that needed medical advice before being physically active (e.g., resting heart rate > 90 BPM), intake of psychopharmaceuticals, as some antidepressants impact the biotransformation of melatonin ^{45,46}. Female adolescents were not recruited, as modification in circadian rhythms across the menstrual cycle require an assessment during the luteal phase ⁴⁷.

While clear cut-points for minutes or days per week to meet physical activity (PA) recommendations are available from the CDC/ACSM guidelines (American College of Sports Medicine, 2018; U.S. Department of Health and Human Services, 2018), the definition for a sedentary lifestyle is less clear. The review of 42 trials by Bennett and colleagues⁵⁰ showed that the large range of “sedentary” 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 sedentary 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 ⁵⁰. Therefore, the present study adopted the recommended cut-point of <60 min PA per week from Bennet and colleagues⁵⁰ to define a sedentary lifestyle.

2.2 *Pre-laboratory Procedures*

One week prior to their laboratory stay, adolescents were instructed to maintain their habitual sleeping pattern on a fixed baseline sleep schedule, and to abstain from any exercise that involved > 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, and activity monitor (ActiGraph GT3X+; ActiGraph, Pensacola, USA). In the 72 hours before and during saliva collection, adolescents were asked to avoid caffeine, nicotine, alcohol, and foods thought to impede habitual melatonin secretion (e.g., chocolate, bananas, tomatoes) ⁵¹.

2.3 *Laboratory Protocol*

Data collection occurred from July 2016 to April 2017 during one week of school holidays, involving 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. Study participants arrived on a Sunday evening (17:00 h) for baseline assessments (Figure 2). Cognitive test batteries, including PDSS, DASS-21, Go/NoGo-task and Karolinska Sleepiness Scale (KSS) were completed at 3h, 2h, 1h, and 0h before bedtime to measure vigilance and sleepiness throughout the evening. At other times, participants interacted with each other and staff members. Board/card games and a television were provided in the communal lounge room. Mobile phones were disallowed. 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.

Following a week of baseline sleep at home, participants were allowed to sleep-in on their first morning and from then on gradually advanced their wake-up time by 30 min on each subsequent day^{52,53}, but keeping their bedtimes fixed. Resting heart rate (RHR) was measured with a wrist-worn heart rate monitor connected to a chest strap (Polar rs100). RHR is used in the Karvonen formula to determine individual exercise intensity ranges, which is widely used in the fields of rehabilitation and physical training^{54,55}. The Karvonen formula provides general 'rule-of-thumb' target heart rates, with light-intensity exercise being defined as activity using 30% to 40% of the heart rate reserve (HRR)⁵⁶: $220 - \text{age} = HR_{max}$; $HR_{max} - RHR = HRR$; $HR_{lower\ limit\ (30\%)}: (HRR * 0.3) + RHR = \dots bpm$; $HR_{upper\ limit\ (40\%)}: (HRR * 0.4) + RHR = \dots bpm$. 20 min after the individual wake-up time, adolescents started their allocated morning activity in dim light (<10 lux). Morning activities either included 45-min treadmill walking or 45-min sedentary behavior (i.e., watching TV on a dimmed screen). Post-hoc analyses showed that exercise timing occurred on average 12 hours after baseline DLMO. Participants were allowed to leave the sleep laboratory thereafter, and return at 17:00 on the same evening. In this respect, daylight exposure remained stable between all participants, independently of seasonal variation.

2.4 Measures

2.4.1 Dim light melatonin onset (DLMO)

Salivary DLMO samples were taken half-hourly in dim light (<10 lux) using salivettes (Sarstedt, Newton, NC, USA) starting 4 hours before and finishing 2 hours after adolescents'

typical bedtime ¹⁵. Participants were seated for at least 5 min before and during each saliva sample, to minimize the masking effects 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)⁵⁷ was used to measure melatonin in the saliva. The mean intra-assay coefficient was 4.5 %. The inter-assay coefficients of variation for low and high levels of salivary melatonin were 8.8 % (5.2 – 12.9) and 88.3 % (50.7 – 104.5), respectively. 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 ⁵⁸. 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.

2.4.2 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?*”) ⁵⁹. Daytime sleepiness is a common symptom reported by adolescents with late chronotype ^{10,60–62}. Responses to each item are measured on a 5-point Likert scale (e.g., 0 = “*Never*”, 4 = “*Always*”). Total scores range from 0-32, with higher scores indicating higher sleepiness. A total cut off score of 20 has been recommended for clinical samples ⁶³. Drake and colleagues⁵⁹ reported good internal consistency (Cronbach α = 0.80); which was confirmed in the current study (Cronbach α = 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 on mood. The depression subscale consists of 7 items measuring depression. 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”*)⁶⁴. Multiplying the score by two yields a total score between 0 and 42. The DASS-21 has been shown to be a valid and reliable measure of depression, with adequate Cronbach’s alpha internal consistencies of 0.88, as well as satisfactory discriminant validity and convergent validity when compared to other measures of depression⁶⁴. This was confirmed in the present study (Cronbach’s alpha 0.76).

2.4.3 Evening sleepiness and vigilance

*The Karolinska Sleepiness Scale (KSS)*⁶⁵ was used to measure subjective sleepiness in the 3 hrs 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/NoGo 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 control⁶⁶, but has previously been used as a measure of sustained evening vigilance^{67,68}.

2.4.4 Sleep and activity log

Adolescents completed a daily sleep and activity log prior to and during the laboratory week. In the evenings, they were asked to indicate any PA performed continuously for at least 10 min, separately for walking, moderate, and vigorous intensity PA. In the mornings, they

indicated their bed- and wake-up time. Additionally, an 8-point visual analogue scale asked about sleep quality (1 = *very bad sleep quality*; 8 = *very good sleep quality*). 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.

2.5 Statistical analysis

All statistical analyses were performed using SPSS 26.0 (IBM Corporation, NY, USA). Test results with an alpha level $<.05$ were reported as statistically significant. Due to the small sample size, effects sizes were additionally considered when interpreting results⁶⁹. Independent-samples *t*-tests were used to compare baseline characteristics between groups. Exercise effects on circadian phase shift were analyzed with one-way ANOVA. Changes in daytime functioning were analyzed using repeated measure ANOVAs (time x group), whereas due to the repeated measure design, changes in evening sleepiness and vigilance were analyzed with Linear Mixed Modelling (LMM)⁷⁰ with the factors intervention phase (baseline vs. follow-up), time (3h, 2h, 1h, 0h before bedtime), and group (morning exercise vs. sedentary). For effect size, Cohen's *d* was calculated to establish the magnitude of within- and between-group differences. Cohen's *d* was calculated as $d = (M_1 - M_2) / (SD_{\text{pooled}})$.

3. Results

3.1 Sample characteristics

Baseline characteristics of the study sample are presented in *Table 1*. There were no significant differences between groups with regard to age ($F(1,17) = 4.32$), BMI ($F(1,17) = .09$), as well sleep duration ($F(1,17) = .81 / .10$ (school / weekend), sleep onset latency ($F(1,17) = 2.48 / 2.12$), and sleep quality ($F(1,17) = .08$ 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 ($F(1,17) = .35$, mid-sleep time on both school ($F(1,17) = .81$ and weekend nights ($F(1,17) = .18$, and DLMO upon laboratory entry ($F(1,17) = 1.69$). Regarding daytime functioning, no significant differences for daytime sleepiness ($F(1,17) = .99$ and mood (stress ($F(1,17) = .90$; depression ($F(1,17) = 1.13$; anxiety ($F(1,17) = .45$) were found.

3.2 Does timed low-intensity morning exercise phase-advance DLMO in late chronotype adolescents?

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), $F(1,16) = 7.92$, $p = .014$, $d = 1.45$. However, analyses were re-run after 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 similar large effect size in favor of the walking intervention $F(1,15) = 7.81$, $p = .015$, $d = 1.41$. *Figure 3* represents the average and individual circadian phase shift for the morning exercise ($N = 8$) and sedentary group ($N = 7$), highlighting inter-individual differences in the degree of phase-shift.

3.3 Does evening sleepiness increase following treatment?

Table 2 provides an overview of the descriptive statistics for each measuring time point. Baseline differences between groups were not observed $F(1,17) = .927$, $p = .35$, $d = -.46$. Sleepiness on the baseline night increased equally in both groups from 3h to 0h before bedtime, as shown in *Figure 4*. Comparing baseline and follow-up night, there were no overall effects (group x time x day), that would indicate consistently higher evening sleepiness scores in favor of morning exercise, $F(3,81) = 0.52$, $p = .67$, $d = .03$. However, the effect size from pairwise comparisons indicate that morning exercisers were sleepier 2 hours before their fixed bedtime in the follow-up night, compared to their peers who remained sedentary, $F(1,17) = 5.13$, $p = 0.39$, $d = .59$, yet significant between group differences were not observed 1 hour before bed, nor at bedtime.

3.4 Does evening vigilance decrease following treatment?

Objective evening vigilance, reflected by false positive NoGo trials (go on NoGo trials) and RT (Go trials) of the computerized Go/No-Go task is represented in *Figure 5* and *Figure 6*, respectively. *Table 2* provides an overview of the descriptive statistics for each measuring time point.

False positive NoGo trials (NoGo errors %). There were no significant baseline differences (3h before bedtime) for false positive NoGo trials between groups $F(1,17) = .30$, $p =$

.59, $d = -.23$. This was consistent for all subsequent assessments that night at 2h, 1h, and 0h before bedtime (all $p > .05$). Comparing baseline and follow-up night, there were no overall effects (group x time x day), that would indicate a higher error percentage (i.e. worse evening vigilance) in favor of morning exercise, $F(3,81) = 0.70$, $p = .557$, $d = .26$. However, pairwise comparisons at follow-up night indicate a trend in that morning exercisers produced more errors 1h before their fixed bedtime, compared to their peers who remained sedentary, $F(1,17) = 4.08$, $p = 0.062$, $d = 1.15$.

Reaction time. No significant baseline differences (3h before bedtime) between groups were observed $F(1,17) = .090$, $p = .77$, $d = -.14$. Both groups showed increasing reaction times (i.e. worse evening vigilance) throughout the evening, $F(3,15) = 2.5$, $p = .07$, $d = 0.18$. Comparing baseline and follow-up night, there were no overall effects (group x time x day), that would indicate a continuous slower reaction time in favor of morning exercise, $F(3,84) = 0.34$, $p = .80$, $d = -.16$.

In summary, evening vigilance in the last hour before bedtime appears to be somewhat higher in the sedentary condition in contrast to their peers who exercised in the morning. However, these between group differences levelled out at bedtime.

3.5 Does daytime functioning improve following scheduled exercise?

Mood. Controlling for baseline values, adolescents in both groups reported improved mood on all subscales at the end of their laboratory stay (Table 2); depression $F(2,16) = 3.05$, $p = 1.04$, $d = .88$; anxiety $F(2,16) = 4.68$, $p = .05$, $d = 1.03$; stress: $F(2,16) = 1.08$, $p = .32$, $d = .51$.

Daytime Sleepiness. Daytime sleepiness increased slightly in the exercise condition, whereas scores dropped notably in the sedentary condition, leading to a significant difference between groups, $F(2,16) = 3.54$, $p = .07$, $d = .89$.

4. Discussion

This is the first study to examine the phase-advancing potential of not only low-intensity morning exercise, but also among sedentary 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 ^{20,28,39,71,72}.

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-in on their first morning. This ensured that scheduled morning exercise occurred after an individual's lowest core body temperature point (~2 hours before natural wake-up time) ^{73,74}, which marks the crossover point between an individual's circadian phase-advancing and phase-delaying region ^{53,75}.

The findings of the present experiment show support for the phase-advancing effect of 45-min of morning exercise among young people with a late chronotype, compared to sedentary controls (+27.5 min vs. -34.3 min). Hence, adding to the scarce literature on the phase-advancing effect of morning exercise ^{32,34,35}, and supporting the newly established PRC of exercise by Youngstedt and colleagues ³². Given that the human circadian rhythm naturally delays in dim-light conditions ⁷⁶, a situation that often exacerbates a late chronotype, particularly for adolescents during school holidays ^{10,77}, these findings highlight the preventative effect of morning exercise among this population. In the present sample, a phase delaying effect specifically occurred in the sedentary condition. In this respect, a phase difference of 64.8 min between groups confers a clinically significant finding - particularly, when extrapolated to a 2-week school holiday period (i.e., a potential difference in circadian timing up to 2 hours across 2 weeks of school holidays). Interestingly, individual differences were observed, 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.

The shift towards circadian evening preference may also be related to increased mood difficulties observed during the onset of adolescence ⁷⁸⁻⁸⁰, 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 to their early chronotype peers, independently of sleep duration ⁸¹⁻⁸³. Dolsen and Harvey ⁸⁴ 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 affect 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 measures, as depression, anxiety, and stress symptoms 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 ^{85,86}. Yet, proposing higher exercise levels in the morning to a group of adolescents classified as late chronotypes, particularly with a sedentary lifestyle, may raise the barrier to the implementation of this intervention.

The nature of the gradually advanced wake-up schedule paired with the fixed bedtimes may have induced a slight sleep restriction over the course of the week, yet, all daytime sleepiness scores ranged below the clinical cut-off of 20. Interestingly, while daytime sleepiness scores dropped significantly in the sedentary condition, it remained stable in the exercise condition, suggesting that not only a phase shift but also morning exercise itself plays a key role in building up sufficient sleep propensity for initiating sleep at a decent clock time. This highlights the reported benefit of regular exercise on adolescent's sleep health ^{36,87–89}.

A further aim of the study was to investigate whether positive phase shifts resulted in greater evening sleepiness and reduced evening vigilance before bedtime. Evening sleepiness (KSS) in the present study 2 hours before bedtime was slightly higher among morning exercisers compared to 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 ⁹⁰. Participants of the advanced group reported significantly higher sleepiness scores in the earlier hours of the night, compared to participants with a prescription designed to delay circadian phase. A slightly different pattern was observed for evening vigilance (Go/NoGo), with lower performance in the exercise group 1 hour before bedtime. Yet once again, the sedentary group reached similar levels at bedtime. Thus, our results partially confirming earlier findings among healthy young men ⁶⁷, where reaction times in the Go/NoGo tasks were significantly improved, while evening increase in endogenous melatonin levels were suppressed after a 5-hours evening exposure to a white LED-backlit screen. As participants of the present study had to adhere to a forced wakefulness protocol until their fixed

bedtime, it remains speculative to what extent the small effects at 2 and 1 hour before bedtime would have translated into earlier bedtimes.

5. Limitations and future directions

Due to the challenge of finding motivated, sedentary, late chronotype adolescents to spend one 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 (68 min), and a reliable advancement for the exercise condition, and a reliable delay in the sedentary condition. Further, for the Go/NoGo 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 (Figures 4 and 5). We did not follow-up 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. Finally, since there is a second phase-advance portion in the afternoon (1-4pm) from exercising in entrained individuals ³², future research could investigate the effect of mid-late afternoon exercise alone, or in combination with morning exercise. Moreover, whether there is an additive effect of morning bright light therapy in combination with low-intensity exercise would provide important basic and applied scientific knowledge, as the combination has been shown to produce greater phase shifts in a single day ³¹ and could be implemented as a 'walking to school' intervention in countries where ambient light is abundant.

6. Conclusion

Scheduled low-intensity morning exercise has been shown in the present study to advance the circadian timing of sedentary adolescents with a late chronotype. Despite this change in adolescents' underlying circadian biology, no immediate nor 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 adolescents' real life.

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Author contributions

Conceptualization: C.L., M.G.; Data acquisition: C.L.; Data preprocessing: C.L.; Data analysis: C.L., M.S.; Methodology: C.L., M.G., M.S.; Results interpretation: C.L., C.R., M.G.; Wrote the manuscript: C.L., *Revised and edited the manuscript: All authors.*

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List of figure captions

Figure 1. CONSORT diagram for this randomized trial. Twenty-three participants were randomized to walking or sedentary group and five participants subsequently withdrew. While 18 participants completed the study, 16 participants were included in the analytic data set (DLMO could not be determined at one time point in two participants), yet, re-analyses without an outlier resulted in a final sample of 15. DLMO = dim light melatonin onset; PA = physical activity

Figure 2. Study Protocol.

Figure 3. Individually timed low-intensity morning exercise vs. sedentary activity over 5 days phase advances the internal circadian rhythm of sedentary adolescents with late chronotype.

Figure 4. Evening Sleepiness. Time course of the Karolinska Sleepiness Scale (KSS) (mean values $\pm$ SEM). 1 = extremely alert; 5 = neither alert nor sleepy; 9 = very sleepy, great effort to keep awake.

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Figure 2. Study Protocol. Following a baseline week at home, study participants arrived at the sleep laboratory on a Sunday evening. Baseline assessments took place on the night of arrival, follow-up assessments on the subsequent Friday night. Cognitive test batteries included the Go/NoGo-task (vigilance), the Karolinska Sleepiness Scale (KSS), psychological questionnaires regarding daytime functioning. Dim light melatonin onset (DLMO) was assessed as a marker of circadian phase position. For the intervention, participants completed either 45 min low-intensity exercise or sedentary activity 20 minutes upon awakening on 5 consecutive mornings. Participants were allowed to leave the lab after completion of their morning activity and 17:00 h.

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Table 1. Baseline characteristics of study participants

Characteristics	Morning Exercise Group		Sedentary Group		F - value
	M ± SD	(range)	M ± SD	(range)	
Age in years	16.20 ± 1.23	(15-18)	16.75 ± 0.71	(16-18)	4.32
BMI	23.41 ± 4.82	(17.28-32.41)	23.60 ± 5.84	(17.67-33.81)	.09
TST (h)					
school night	8:01 ± 1:17	(6:18-10:22)	7:27 ± 2:04	(5:17-11:50)	.81
weekend night	8:54 ± 1:24	(6:45-10:30)	9:07 ± 1:32)	(5:42-10:43)	.10
Mid-sleep time (hh:mm)					
school night (MSW)	3:53 ± 1:15	(2:37-6:14)	4:09 ± 0.56	(3:06-6:05)	.81
weekend night (MSF)	5:38 ± 1:14	(4:15-8:12)	6:24 ± 0:53	(5:17-7:47)	.18
MSFsc (chronotype)	5:24 ± 1:16	(4:20-8:16)	5:46 ± 1:15	(4:21-7:14)	.35
Extreme late type	20.0 %		50.0 %		
Moderate late type	30.0 %		12.5 %		
Slight late type	50.0 %		37.5 %		
DLMO (h)	23:11 ± 1:20	(21:36-0:49)	23:04 ± 2:02	(20:31-2:31)	1.69
SOL (min)					
School night	24.4 ± 22.87	(2-72)	12.49 ± 12.24	(2-36)	2.48
weekend night	29.83 ± 35.09	(4 – 41)	10.78 ± 12.53	(4 – 120)	2.12
Sleep quality	5.28 ± 0.91	(3.40-6.60)	5.86 ± 0.82	(4.75-7.00)	.08
Daytime sleepiness (PDSS)	14.90 ± 2.56	(11-18)	16.25 ± 4.03	(9-22)	.99
Mood (DASS)					
Stress	9.4 ± 5.42	(2-18)	9.43 ± 9.91	(0-30)	.90
Depression	8.44 ± 6.69	(0-18)	11.71 ± 5.82	(8-14)	1.13
Anxiety	7.40 ± 5.74	(0-16)	8.00 ± 5.54	(4-20)	.45

Data are presented as mean ± SD; baseline participant characteristics for the morning exercise and sedentary groups were compared using independent sample t-test; *p < .05, **p < .01; MSW = mid-sleep time on school days; MSF = mid-sleep time on free days; MSFsc = mid-sleep time on free days corrected for sleep debt on school days

Table 2. Mean and standard deviation of main outcome variables at baseline and follow-up night (N = 18)

Measures	Pre				Post			
	Exercise group		Sedentary group		Exercise group		Sedentary group	
	M	95% CI	M	95% CI	M	95% CI	M	95% CI
DLMO	23.18	22.16, 24.21	23.06	21.36, 24.77	22.36	21.49, 23.23	23.76	22.04, 25.48
Mood (DASS)								
Depression	8.44	3.30, 13.59	11.71	6.33, 17.10	4.60	0.49, 8.71	2.57	0.25, 4.89
Anxiety	7.40	3.29, 11.51	8.00	2.88, 13.12	6.80	1.83, 11.77	3.00	0.32, 5.68
Stress	9.40	5.52, 13.28	9.43	0.26, 18.60	6.80	1.88, 11.72	4.75	-1.37, 10.87
Daytime Sleepiness (PDSS)	14.90	13.07, 16.73	16.25	12.88, 19.62	15.10	11.70, 18.50	11.75	6.92, 16.58
Evening Sleepiness (KSS)								
3h before bedtime	4.80	3.73, 5.87	5.50	4.30, 6.70	5.10	4.03, 6.17	4.25	3.05, 5.45
2h before bedtime	5.60	4.53, 6.67	6.25	5.05, 7.45	5.80	4.73, 6.87	4.75	3.55, 5.95
1h before bedtime	6.10	5.03, 7.17	6.88	5.68, 8.07	5.80	4.73, 6.87	5.38	4.18, 6.57
0h before bedtime	7.30	6.23, 8.37	7.50	6.30, 8.70	6.40	5.33, 7.47	6.13	4.93, 7.32
Evening vigilance (Go/NoGo)								
Errors								
3h before bedtime	2.05	1.26, 2.84	2.33	1.45, 3.21	3.23	2.44, 4.01	2.63	1.75, 3.52
2h before bedtime	1.73	0.94, 2.51	1.76	0.88, 2.64	2.19	1.40, 2.98	2.44	1.56, 3.32
1h before bedtime	3.03	2.25, 3.82	2.20	1.31, 3.08	2.45	1.67, 3.24	1.37	0.49, 2.26
0h before bedtime	2.11	1.32, 2.90	2.06	1.18, 2.94	2.14	1.35, 2.93	2.51	1.63, 3.39
Reaction time								
3h before bedtime	312.30	286.29, 338.31	317.50	288.42, 346.58	313.10	287.09, 339.11	334.87	305.80, 363.95
2h before bedtime	312.60	286.59, 338.61	324.25	295.17, 353.33	312.60	286.59, 338.61	320.25	291.17, 349.33
1h before bedtime	327.20	301.19, 353.21	324.00	294.92, 353.08	314.60	288.59, 340.61	313.50	284.42, 342.58
0h before bedtime	333.60	307.59, 359.61	328.13	299.05, 357.20	318.50	292.49, 344.51	323.62	294.55, 352.70

Note. M = mean; 95% CI = confidence interval; DLMO: decimal time format; Mood: higher scores reflect worse mood; KSS: higher scores reflect higher evening sleepiness; Go/NoGo:

higher errors reflect lower evening vigilance, lower reaction time (ms) reflect lower evening vigilance; ANOVA: + p<0.1; *p<0.05; **p<0.01; ***p=0.001