

Circadian temperature amplitude moderates changes in sleep duration during
adolescence

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Tiivistelmä – Referat – Abstract <p><i>Aims of the study.</i> The aim of this study was to examine normative development of sleep patterns and circadian rhythmicity during adolescence. Previous studies have found that sleep duration shortens across the lifespan, and especially adolescents' sleep timing shifts later due to physiological and psychological factors. Sleep patterns in adolescence are connected to individual's endogenous circadian rhythms, usually measured by delayed melatonin secretion in the evening. There is a lack of understanding how sleep patterns are related to circadian body temperature rhythms during adolescence.</p> <p><i>Methods.</i> This study was part of SleepHelsinki! cohort study of the Sleep & Mind Research Group. Adolescents' sleep patterns were measured with actigraphies, whereas circadian body temperature was measured from the skin surface. Circadian temperature rhythmicity was inspected by circadian period length, the mesor of skin surface temperature and the amplitude of daily changes within the rhythm. Baseline measurements were measured from 215 (71.6 % girls) adolescents aged 16–18 years. At one-year follow-up, 156 (76.3 % girls) adolescents were measured again. Mixed models for repeated measures were used to examine changes over the year in sleep patterns and endogenous circadian temperature rhythm, separately for both girls and boys. Sex differences were tested with one-way variance analysis. Linear and ordinal regressions were used to predict sleep and circadian rhythm over the year.</p> <p><i>Results and conclusions.</i> Over the year, adolescents' sleep duration became longer during the week, while weekend sleep shortened. However, this change was only significant for girls. Sleep schedule became more delayed for both girls and boys during the week, as sleep onset, midpoint and offset occurred at a later time. Circadian rhythm changed for boys, as their average skin surface temperature increased, and their circadian temperature amplitude became smaller. Boys also had significantly lower circadian temperature amplitude than girls at the follow-up. Compared to boys, girls were 5.85 times more likely to have a high circadian temperature amplitude at the follow-up measurement. Changes in sleep length during the week was moderated by temperature amplitude, with higher circadian amplitude predicting sleep duration to become longer. Still, the likelihood to have long sleep duration was affected by past sleep duration.</p>		
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Tiivistelmä – Referat – Abstract <p><i>Tavoitteet.</i> Tämän tutkimuksen tavoitteena oli tarkastella unen ja sisäisen vuorokausirytmien normatiivista kehittymistä nuoruuden aikana. Aiemmissä tutkimuksissa on löydetty, että unen kokonaismäärä vähenee ikääntymisen myötä, ja erityisesti nuoruudessa unen ajoitus muuttuu myöhemmäksi, mihin vaikuttaa sekä fysiologisia että psykologisia tekijöitä. Uni on yhteydessä nuorten sisäiseen vuorokausirytmiiin, mitä on yleisesti tutkittu melatoniinin erityksen kautta. On epäselvää, kuinka uni on yhteydessä kehon lämpötilan vuorokausirytmiiin nuoruudessa.</p> <p><i>Menetelmät.</i> Tämä tutkimus oli osa Sleep & Mind -tutkimusryhmän SleepHelsinki! kohorttitutkimusta. Nuorten unta mitattiin aktigrafialla, kun taas lämpötilan vuorokausirytmii sisälsi ihon pintalämpötilan mittaamisen. Lämpötilan vuorokausirytmiiä tarkasteltiin rytmien pituudella, ihon keskiarvoisella lämpötilalla sekä vuorokauden aikana tapahtuvan vaihtelun suuruudella eli amplitudilla. Lähtömittaukseen osallistui 215 (71.6 % tyttöjä) nuorta (16–18 vuotta). Vuoden kuluttua 156 (76.3 % tyttöjä) nuorta osallistui seurantamittaukseen. Muutoksia unessa ja lämpötilan vuorokausirytmiiissä testattiin toistettujen mittausten sekamallilla, mikä tehtiin erikseen tytöille ja pojille. Sukupuolten välisiä eroja testattiin yksisuuntaisella varianssianalyysillä. Seurantamittauksen unta ja vuorokausirytmiiä ennustettiin lähtömittauksen arvoilla ja sukupuolella; testaukseen käytettiin sekä lineaarisia että ordinaalista regressiomalleja.</p> <p><i>Tulokset ja johtopäätökset.</i> Vuoden aikana nuorten unen kokonaismäärä kasvoi viikon aikana, mutta viikonlopun unen määrä väheni. Tämä muutos oli kuitenkin tilastollisesti merkitsevä pelkästään tytöillä. Unen ajoitus myöhästyi sekä tytöillä että pojilla, sillä unen alkamisaika, keskikohta sekä heräämisaika muuttuivat myöhäisemmiksi. Ihon lämpötilan vuorokausirytmii puolestaan muuttui pelkästään pojilla, sillä heidän keskiarvoinen iholämpötilansa kasvoi ja lämpötilan vaihtelun suuruus väheni. Seurantamittauksessa poikien lämpötilan amplitudi oli myös selvästi pienempi kuin tytöillä, ja tytöillä oli 5.85-kertainen todennäköisyys korkeaan amplitudiin poikiin verrattuna. Lämpötilan amplitudi oli yhteydessä viikon unen määrään, sillä lämpötilan amplitudi vaikutti muutoksiin unen määrässä: korkeampi amplitudi lämpötilassa ennusti unen määrän kasvamista. Kuitenkin aiempi unen määrä vaikutti todennäköisyyteen, että nuorella oli pitkä unen kesto vuoden kuluttua.</p>			
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Table of contents

1	Introduction	1
1.1	Circadian rhythm	1
1.2	Circadian rhythms are connected to sleep	2
1.3	Development of sleep and circadian rhythms in youth	4
1.3.1	How do sleep patterns change?.....	4
1.3.2	How do circadian rhythms change in relation to sleep?	6
1.3.2.1	Circadian temperature rhythm in youth	8
1.4	Research questions	9
2	Methods	10
2.1	Procedure	10
2.2	Participants	11
2.3	Measurements	11
2.3.1	Sleep actigraphy	11
2.3.2	Skin surface temperature	12
2.4	Statistical analysis	12
3	Results	13
3.1	Development of sleep and circadian rhythm over a year.....	13
3.2	Associations between sleep patterns and circadian temperature	15
3.3	Likelihoods to have better sleep patterns and higher circadian amplitude	17
4	Discussion.....	24
4.1	Main findings.....	24
4.1.1	Changes in sleep and circadian rhythm	24
4.1.2	Sleep duration and circadian amplitude are connected.....	26
4.2	Strengths & limitations	29
4.3	Conclusions	30
	References	31
	Appendix 1	37
	Appendix 2	39

Acronyms used in this paper:

EEG = electroencephalogram

EMG = electromyogram

EOG = electro-oculography

SCN = suprachiasmatic nucleus

CBT = core body temperature

NREM = non-rapid eye movement

POAH = preoptic area of the hypothalamus

SOL = sleep onset latency

DSPD = delayed sleep phase disorder

GnRH = gonadotropin-releasing hormone

1 Introduction

Adolescents go through psychological, physical, and social changes over the pubertal years, and sleep is commonly acknowledged to have an effect on children's and adolescents' development. Sleep is important for physical growth in both children and adolescents, as growth hormone secretion is most prominent during sleep (Steiger, 2003). Along with physical growth, youth also experience changes in brain structures and activation, which in turn affect the sleep structure (Kurth, Olini, Huber & LeBourgeois, 2015). Besides brain development, multitude of biological and psychological factors in youth can have an effect on sleep patterns. Research on sleep and endogenous circadian rhythm during adolescence gives insight on what could be regarded as normative development. Furthermore, acknowledging normative trends enables to determine what in turn could be regarded as abnormal development, requiring appropriate treatment to ensure healthful development.

Sleep is such an essential part of life that it has been a topic of interest for over two millennia, from Aristotle's discourse in "On sleep and sleepiness" (*De somno et vigilia*, part of *Parva Naturalia*, 350 BC), to contemporary research methods. Polysomnography – which often includes measurements of electroencephalogram (EEG) for brain activity, electromyogram (EMG) for muscle activity and electro-oculography (EOG) for eye movements – is a comprehensive method to study sleep (Scammell, Arrigoni & Lipton, 2017). Polysomnography gives detailed insight into the physiological changes during sleep, but due to its methodological complexity, it is not ideal for measuring sleep in a natural setting. Sleep diaries and surveys can be used to collect information about sleep patterns at home, but actigraphic measurements can be used to measure the sleep-wake rhythm more objectively (Ancoli-Israel et al., 2003). Actigraphy is used to measure body movements over extended periods of time, while not losing ecological validity (Ancoli-Israel et al., 2003).

1.1 Circadian rhythm

A circadian rhythm refers to an endogenous cycle that can be entrained to the environment (Kuhlman, Craig & Duffy, 2018). In order to be considered "circadian" (from Latin *circa* "about" and *die* "day"), the rhythm has to have a period length close to 24 hours, causing it to repeat daily even without environmental inputs. Circadian rhythmicity is indicated by biological markers, such as body temperature, melatonin, and cortisol, which are interconnected with each other (Kuhlman et al., 2018; Dijk & Duffy, 2020). Circadian rhythms are studied by assessing the phase of the cycle, which indicates what time a marker reaches its maximum/minimum or onset/offset values (Dijk & Duffy,

2020). By assessing the amplitude of the cycle, we know how large changes rhythms go through on a daily basis (Dijk & Duffy, 2020).

The circadian period is naturally a little longer than 24 hours in humans, and as a result the rhythm needs daily adjustment to the Earth's light-dark cycle (Kuhlman et al., 2018). Environmental cues, such as light exposure, food intake, environmental temperature and social factors guide the entrainment, making sure the circadian clock is synchronized with the environmental clock (Kuhlman et al., 2018). However, entrainment is not set in stone, and the environmental cues can also cause a phase-shift. The phase can be delayed or advanced, but exposure to the cue must happen during certain parts of the phase for it to shift (Kuhlman et al., 2018). Failure to entrain to environment or a shift in the timing causes misalignment, where the circadian clock is either too early or too late compared to environmental time (Kuhlman et al., 2018).

Circadian rhythmicity is controlled by a pacemaker, the suprachiasmatic nucleus (SCN), which is located in the lower part of the hypothalamus (Zee & Manthena, 2007). The SCN can adjust its activity accordingly to the light-dark cycle, and therefore regulate the biological markers (Zee & Manthena, 2007). Light, especially blue wavelength light can be processed in the SCN through the retinohypothalamic tract, and when light exposure declines, the SCN will release melatonin from the pineal gland (Zee & Manthena, 2007), which entrains the circadian melatonin rhythm to the environmental time.

Circadian rhythmicity is dependent on genetic regulation, and different gene variations can cause individual differences in circadian cycles and entrainment (Jones et al., 2016). Genetic variations and environmental factors affect chronotypes, the subjective experience of one's circadian rhythm (Roenneberg et al., 2007; Jones et al., 2016). Chronotypes are also referred to as circadian preference, as a person's subjective experience of the rhythm will affect what time of the day they feel most active, for example morning-oriented individuals feel most efficient during the morning hours (Roenneberg et al., 2007).

1.2 Circadian rhythms are connected to sleep

Sleep-wake cycle is regulated by two endogenous processes – sleep homeostasis and circadian rhythmicity (Borbély, 1982; Borbély, Daan, Wirz-Justice & Deboer, 2016). Sleep homeostasis, also referred to as S process in the two-process model, regulates the propensity of sleep (Borbély, 1982). Sleep homeostasis consists of accumulation of sleep pressure during wakefulness, and the release of sleep pressure during sleep. Without sleep, the pressure will continue to accumulate, and prolonged

wakefulness will cause sleep deprivation (Borbély, 1982). Sleep deprivation is followed by recovery sleep, which is more intense than regular sleep (Borbély, 1982, Borbély et al., 2016).

Circadian rhythmicity (C process) controls the timing and consolidation of sleep to an optimal time of day (Borbély, 1982; Dijk & Czeisler, 1995). As the SCN generates the endogenous circadian rhythm, the SCN needs to be synchronized with environmental time for optimal sleep consolidation (Zee & Manthena, 2007). Circadian rhythmicity is also in continuous interaction with the homeostatic process. Thalamocortical networks, including the SCN, are important for both processes (Zee & Manthena, 2007; Borbély et al., 2016). The SCN is influenced by sleep pressure: activity in the suprachiasmatic area has been found to decrease as the sleep pressure increases (Borbély et al., 2016), creating a feedback cycle between the rhythmic and homeostatic processes.

Biological markers have different rhythms for optimal synchronizations. Melatonin is an important hormone for sleep regulation as it induces sleep (Brzezinski, 1997). Melatonin levels stay low for a significant part of the day and start to rise soon after it begins to get dark (Brzezinski, 1997). After falling asleep, melatonin reaches its maximum after midnight, after which it begins to dissipate from the body (Brzezinski, 1997). On the other hand, core body temperature (CBT) rises across the day, reaching its peak hours before going to bed, and then declines especially during non-rapid eye movement (NREM) sleep (Harding, Franks & Wisden, 2019). Besides melatonin, skin temperature changes induce sleep, as warming up distal body parts (hands, feet) will cause sleepiness (warm-bath effect; Harding et al., 2019). These microclimates of skin warmth increase activity in the preoptic area of the hypothalamus (POAH), which is crucial for NREM initiation (Harding et al., 2019).

The circadian melatonin and body temperature rhythms are inversely connected with each other. The decline in CBT coincides strongly with the rise in melatonin levels (Cagnacci, Elliot & Yen, 1992; Benloucif et al., 2005). The association between melatonin, body temperature, and sleep is strongest during the early morning (Benloucif et al., 2005), when the CBT begins to rise and melatonin levels decline before waking up (Cagnacci et al., 1992). This has also been shown in experimental studies, in which manipulating melatonin levels also influences body temperature. It has been found that administering melatonin causes a decrease in body temperature, whereas suppressing melatonin levels causes body temperature to rise (Cagnacci et al., 1992). These influences are only present for the length of the manipulation, and body temperature will go back to normal once the manipulation over.

Besides being connected to the timing of sleep, body temperature can affect other features of sleep. People sleep for shorter time when sleep is initiated close to the minimum CBT, but in contrast sleep

is longer when falling asleep happens close after the peak in CBT (Czeisler, Weitzman, Moore-Ede, Zimmerman & Kronauer, 1980; Zulley, Wever & Aschoff, 1981; Dijk & Czeisler, 1995). Interestingly, the sleep-wake cycle and body temperature rhythm are continuously attempting to be synchronized, so that sleep falls on the declining curve of CBT (Zulley et al., 1981). If desynchronized, variations in sleep duration helps the two cycles to become synchronized again (Zulley et al., 1981). Furthermore, the connection between sleep duration and temperature cycle has been found to be stronger than the connection between sleep duration and the length of prior wakefulness (Czeisler et al., 1980). Sleep is also more efficient when it falls upon the declining curve of the CBT, although falling asleep is faster closer to the minimum CBT (Dijk & Czeisler, 1995).

The connection between sleep and circadian rhythms can also be examined by phase angles of entrainment. Phase angles are measured by the temporal interval between sleep onset/offset and a phase marker (minimum/maximum, onset/offset) (Duffy, Dijk, Hall & Czeisler, 1999). With CBT, the later the minimum temperature is, the closer it is to waking up (Duffy et al., 1999; Baehr, Revelle & Eastman, 2000). Moreover, the timing of the minimum CBT is connected to body temperature at bedtime and how steep the decline is during sleep. For people who reach their CBT minimum early in the night, their CBT has already declined significantly by the time they go to bed, whereas people with later minimums have higher bedtime CBTs, and they experience a much steeper decline in their body temperature during their sleep (Baehr et al., 2000).

The phase of CBT rhythm has also been found to be connected to chronotypes. People who evaluate themselves as morning-oriented have earlier body temperature minimums compared to evening-oriented people (Horne & Östberg, 1977; Duffy et al., 1999; Baehr et al., 2000). Morning types also have a sharper body temperature increase during the morning (Duffy et al., 1999). Melatonin secretion is connected to chronotype the same way as body temperature, with morning types having earlier melatonin onsets (Duffy et al., 1999). Additionally, chronotypes are also associated with sleep patterns (Roenneberg et al., 2007), as evening types usually have a later sleep schedule than morning or intermediate types (Horne & Östberg, 1977; Karan et al., 2021).

1.3 Development of sleep and circadian rhythms in youth

1.3.1 How do sleep patterns change?

Newborns spend the majority of the day asleep, but during the first year of life, daytime sleeping decreases significantly while sleep consolidates to nighttime (Iglowstein, Jenni, Molinari & Largo, 2003). Nocturnal sleep duration increases until the age of one, up to an average of 11.7 hours, and

thereafter it decreases gradually (Iglowstein et al., 2003). The decrease in duration continues across childhood (Spruyt, O'Brien, Cluydts, Verleye & Ferri, 2005) and adolescence (Strauch & Meier, 1988; Wolfson & Carskadon, 1998; Loessl et al., 2008). On average, 13-year-olds sleep for 8.5 hours each night, which then declines to approximately 8.1 hours at 16 years and to 7.3 hours for 17-year-olds and older (Loessl et al., 2008; Maslowsky & Ozer, 2014). Sleep duration has been found to increase temporarily when entering adulthood (Maslowsky & Ozer, 2014), but overall, sleep duration declines throughout the lifespan (Thorleifsdottir, Björnsson, Benediktsdottir, Gislason & Kristbjarnarson, 2002; Evans et al., 2021). However, sleep durations within individuals stay quite stable across the lifespan (Thorleifsdottir et al., 2002), meaning those who sleep less during earlier years of life are more likely to sleep even less as they age than individuals who sleep longer.

In contrast to earlier years of life, children's and adolescents' sleep patterns are affected by school schedules. While sleep duration has been found to decrease for both the weekdays and weekends as children get older (Wolfson & Carskadon, 1998), comparing sleep duration between weekdays and weekends show that sleep is longer during the weekend on average (Loessl et al., 2008). The difference between weekday and weekend sleep duration appears after the age of nine (Thorleifsdottir et al., 2002). Average sleep duration for weekdays is 8.04 hours, and 9.51 hours for weekend days (Loessl et al., 2008), which means adolescents have almost a two-hour difference in sleep duration between weekdays and weekends. The desynchronization between week and weekend sleep is regarded as social jet lag because the difference is caused by societal demands, such as work or school (Wittmann, Dinich, Merrow & Roenneberg, 2006). Social jet lag has been found to be connected to health behaviour, as more social jet lag is associated with more depressed mood, intrapsychic imbalance, smoking, alcohol consumption and caffeine intake (Wittmann et al., 2006).

A closer look at sleep schedules reveals that children and adolescents go to bed later as they age (Wolfson & Carskadon, 1998; Thorleifsdottir et al., 2002; Loessl et al., 2008). The change in bedtime is one of the most robust changes in sleep schedule (Evans et al., 2021). Sleep onset latency (SOL), which refers to the temporal interval between going to bed and falling asleep, has been self-reported to become longer with age (Russo, Bruni, Lucidi, Ferri & Violani, 2007), but actigraphic measurements have shown that adolescents have a shorter SOL than children (Rensen et al., 2020). SOL relates to sleep efficacy, which is often measured by how much time is spent sleeping out of the total time in bed. Actigraphy based research has shown that sleep efficacy increases with age in youth, which indicates that adolescents might have an easier time falling and staying asleep than preschool- and school-aged children (Rensen et al., 2020).

While bedtimes become delayed with age whether it is a weekday or weekend (Wolfson & Carskadon, 1998), SOL and wake-up time vary depending on the day of the week. In one study, children reported that they are able fall asleep faster during the weekend than on weekdays, more specifically schooldays (Spruyt et al., 2005). Overall, youth tend to wake up later with age (Evans et al., 2021), but wake-up times on schooldays stay quite consistent in childhood and adolescence (Thorleifsdottir et al., 2002). During the weekend wake-up times occur later, as children and adolescents can sleep longer in the morning compared to weekdays, and the difference between weekday and weekend wake-up times becomes larger with age (Thorleifsdottir et al., 2002; Spruyt et al., 2005).

Sleep patterns clearly change during childhood and adolescence, but the changes are apparent even within weekdays. This can lead to adolescents not getting enough sleep. The National Sleep Foundation and Finnish Current Care Guidelines (Käypä Hoito) recommend that school-aged children should optimally sleep 9–11 hours each night, whereas adolescents should sleep 8–10 hours (Hirshkowitz et al., 2015). In a Finnish School Health Promotion study, the percentage of adolescents who slept less than 8 hours in 2019 was 34.8 within 15–16-year-olds (8. and 9. graders) and 42.4 within 17–18-year-olds in upper secondary school, and this percentage increased in 2021 (40.3 % of 15–16-year-olds, 47.9 % of 17–18-year-olds; Finnish Institute for Health and Welfare, 2021). Even more vocational schoolers sleep less than 8 hours compared to upper secondary schoolers (52.2 % in 2019 & 52.9 % in 2021; Finnish Institute for Health and Welfare, 2021). The percentage of individuals with a short sleep duration (< 6 hours) have been found to increase from 1.1 % to 8.5 % in adolescence, while at the same time the number of adolescents with long sleep duration (> 10 hours) decreases from 4.5 % to 1.6 % (Maslowsky & Ozer, 2014). Subjectively evaluated chronic tiredness has been found to increase with age during adolescence, but there has also been a growing trend in tiredness among adolescents from the mid-1990's to late 2000's (Kronholm et al., 2015).

1.3.2 How do circadian rhythms change in relation to sleep?

In infancy, the sleep-wake cycle begins to develop at approximately 44 postconceptional weeks, or 1 month after due date (Shimada, Segawa, Higurashi & Akamatsu, 1993; Shimada et al., 1999). The circadian rhythms of biological markers take longer to develop. CBT rhythm has been found to develop by 2.5 months after due date in full term infants (Guilleminault et al., 1996) and by 3 months after due date in preterm infants (Mirmiran, Baldwin & Ariagno, 2003). Melatonin secretion is minuscular before 3 months of age, but 1–3-year-old toddlers have the highest melatonin concentration levels compared to any other developmental stage (Brzezinski, 1997).

Development of circadian rhythm in children and adolescents has largely been studied by examining changes in melatonin secretion. Overall, melatonin secretion becomes delayed with age during childhood and adolescence (Carskadon, Acebo, Richardson, Tate & Seifer, 1997; Carskadon, Wolfson, Acebo, Tzischinsky & Seifer, 1998; Crowley, Acebo, Fallone & Carskadon, 2006; Crowley et al., 2014). Changes in melatonin secretion give a physiological explanation as to why children tend to go to sleep later when they get older (Carskadon et al., 1997). Age also influences melatonin levels, as adolescents have lower melatonin levels than children (Rensen et al., 2020). There has been some speculation whether this decrease could be caused by an increase in body mass during puberty (Brzezinski, 1997), but it could also partially explain why older adolescents have less interdaily stability in their sleep-wake cycles (Rensen et al., 2020).

As expected, youth with later melatonin onset also go to sleep later than those with early melatonin onset, causing them to have a shorter sleep duration (Carskadon et al., 1997; Carskadon et al., 1998). The phase angle between melatonin onset and sleep onset also becomes longer with age, as older adolescents tend to stay awake for longer after the melatonin onset (Crowley et al., 2006; Crowley et al., 2014). This could be caused by changes in accumulation of sleep pressure, which has been hypothesized to slow down during adolescence (Crowley et al., 2014). Consequently, the phase angle is affected by the timing of melatonin secretion: those with early melatonin onset tend to stay up longer after the onset, resulting in an even longer phase angle than for those who have a later onset (Crowley et al., 2006). Despite clear changes in melatonin secretion at group-level, individual differences are quite prominent. As with sleep duration, melatonin secretion appears to be relatively stable within individuals (Carskadon et al., 1998), and there are also clear individual differences in developmental trajectories (Crowley et al., 2014).

Besides age, sex also affects melatonin secretion. Boys have been found to have later melatonin onsets than girls (Carskadon et al., 1998). Boys have also reported going to bed at later time than girls (Spruyt et al., 2005; Loessl et al., 2008), which could be explained by the differences in melatonin secretion. Despite this, findings on sex differences in sleep patterns are not clear, as some studies have not found any differences between sex (Thorleifsdottir et al., 2002; Rensen et al., 2020), and some studies contradict each other. Boys have reported to sleep longer (Maslowsky & Ozer, 2014), but actigraphic measurements show that boys sleep less than girls (Short, Gradisar, Lack, Wright & Carskadon, 2012). This could be caused by the fact that boys move more in their sleep (Meltzer et al., 2019), which can distort measurements.

1.3.2.1 Circadian temperature rhythm in youth

The circadian rhythm of body temperature and its development in youth has been studied sporadically. From those that have researched this topic previously, Andrade, Benedito-Silva and Menna-Barreto's (1992) longitudinal study examined how chronotypes are connected to age, sleep schedule and oral body temperature in adolescents. Eveningness was associated with later sleep schedule and body temperature maximum, but the relationship between chronotype and body temperature was only noticeable at the second measurement time (Andrade et al, 1992). While most of the participants were intermediate types (79.0 % to 82.2 %), Andrade and colleagues (1992) did not find age to affect chronotypes, which could have been due to small sample size ($n = 62$) and skewness in the age range (77.4 % aged 12–13 years). Eveningness has been found to increase among 13-year-olds and older, albeit chronotypes are relatively stable within individuals in adolescents (Russo et al., 2007; Karan et al., 2021).

Martínez-Lozano and colleagues (2020) found similar results for temperature rhythms in eight- to 12-year-old children. Age did not affect chronotypes, but evening types had a later sleep-wake and temperature cycles compared to morning types (Martínez-Lozano et al., 2020). They also had a less robust circadian rhythm, causing evening types to have more day-to-day variation in their rhythm (Martínez-Lozano et al., 2020). Within daily body temperatures, evening types had a higher morning body temperature but lower evening body temperature. Therefore, evening types had smaller body temperature amplitude than morning types (Martínez-Lozano et al., 2020). Differences in circadian rhythm could be explained by light exposure, as evening-oriented children were exposed to light at later hours compared to morning-oriented children (Martínez-Lozano et al., 2020), which affects melatonin secretion.

The association between circadian temperature rhythm and sleep has also been examined directly. Lipsanen, Kuula, Elovainio, Partonen and Pesonen (2021) used data-driven clustering methods and found three distinct profiles of circadian temperature rhythms among adolescents aged 16 to 18 years. These clusters were significantly different from each other, not only based on circadian temperature rhythm but also sleep patterns (Lipsanen et al., 2021). The cluster with highest circadian amplitude and the lowest mean temperature had the earliest sleep schedule, whereas the cluster with a moderate circadian amplitude and mean temperature had the most delayed sleep schedule (Lipsanen et al., 2012). The latter cluster had the highest percentage of boys compared to the other clusters, but the clusters did not differ significantly based on sex (Lipsanen et al., 2021). Furthermore, chronotypes differed between the aforementioned clusters, as the cluster with highest circadian amplitude had

more morning-oriented adolescents, which is congruent to the findings in children (Martínez-Lozano et al., 2020).

Circadian rhythms of melatonin and body temperature have prominent effects on sleep-wake rhythm during childhood and adolescence, as described above. Despite the apparent changes in melatonin rhythm, it is less clear if the body temperature rhythm changes in youth. However, the circadian body temperature rhythm has been found to go through changes during adulthood. The circadian phase becomes advanced with age, as body temperature reaches its minimum earlier in older adults than it does in younger adults (Baehr et al., 2000). On average, women also reach their temperature minimum earlier than men (Baehr et al., 2000). Besides the timing of the rhythm, the amplitude of body temperature is also affected by age in adults: a smaller difference between the peak and minimum body temperature is associated with older age (Baehr et al., 2000). The timing and the amplitude are connected to each other, as adults with larger amplitude tend to have later CBT minimums in their circadian rhythm (Baehr et al., 2000). This is contradictory to the findings on temperature amplitudes and chronotypes in children (Martínez-Lozano et al., 2020) and adolescents (Lipsanen et al., 2021).

While the delays in circadian melatonin rhythm and sleep schedule during childhood and adolescence seem prominent, this phase-shift is not necessarily a sign of a sleep-related disorder. People with delayed sleep phase disorder (DSPD) have even later sleep schedule (bedtime, sleep onset and wake-up time) than healthy sleepers, but they also have later circadian phases and longer circadian periods, causing them to have difficulties maintaining a regular sleeping schedule (Micic et al., 2013). Nonetheless, temporal misalignment between endogenous circadian rhythm and environmental time is a risk factor for physical and mental health (Kuhlman et al., 2018). Sleep and circadian disruptions are connected to a multitude of psychiatric disorders, including mood disorders, anxiety disorders and substance use (Wulff, Gatti, Wettstein & Foster, 2010). Disruptions are also commonly reported to precede the onset of these disorders, making sleep and circadian rhythms practical targets for treatment to prevent psychiatric disorders (Wulff et al., 2010).

1.4 Research questions

The aims of the study are to examine how sleep patterns and circadian body temperature rhythm develop during adolescence over a one-year period. The specific research questions are:

1. Do adolescents' sleep patterns and circadian body temperature rhythm change in a year?

Hypothesis: Based on previous studies, the youth's sleep is expected to become shorter while their sleep schedule becomes delayed. Previous studies have also shown that melatonin secretion goes

through changes during adolescence. When taken into account that melatonin and body temperature rhythms are connected, it can be expected that body temperature rhythm changes during adolescence. Furthermore, since some studies have found sex differences in sleep patterns and circadian rhythms, the possible changes might differ between the sexes.

2. How are the possible changes in sleep patterns and circadian temperature rhythm associated? More precisely, are there moderating factors for these changes?

Hypothesis: Based on the fact that adolescents' sleep patterns change when their circadian melatonin rhythm changes, the changes in sleep patterns are expected to be accompanied with circadian temperature changes. In a previous study from the same SleepHelsinki! data, Lipsanen and colleagues (2021) found that adolescents' circadian temperature amplitude is associated with their sleep schedule, as adolescents with higher circadian amplitude had earlier timed sleep schedules. Based on this, sleep and circadian amplitude were expected to moderate each other in the current study.

3. What increases the likelihood for adolescents to have better sleep patterns and higher circadian temperature amplitude?

Hypothesis: Given the fact that sleep patterns and circadian rhythms have been found to be stable within individuals, it can be expected that the likelihoods for better sleep patterns and higher circadian amplitude are dependent of past patterns and rhythm.

2 Methods

2.1 Procedure

The data was collected as a part of SleepHelsinki! cohort study of the Sleep & Mind Research Group at the University of Helsinki. The study was initiated in autumn of 2016. The first phase included an online survey, which was used to gather information about sleep, health, and behaviour. In the second phase, baseline measurements were gathered for sleep actigraphy and circadian skin temperature at the same time. The baseline measurements were conducted between November 2016 and December 2017. Follow-up measurements were conducted one-year after the baseline measurements, from January 2018 to November 2018. All participants gave informed consent for the study. The study was approved by The Hospital District of Helsinki and Uusimaa Ethics Committee for gynecology and obstetrics, pediatrics, and psychiatry (Decision number 50/13/03/03/2016), and all its procedures followed the guidelines of Helsinki Declaration and its later amendments.

2.2 Participants

Adolescents born in 1999 and 2000, who resided in Helsinki and spoke Finnish as their first language ($N = 10\,476$), were identified using the Finnish Population Register Centre. From these adolescents, $n = 7539$ were invited to participate in the study (72.0 % of the total sample). Approximately 50 % were girls. Out of those invited, 1411 adolescents responded to the online survey, with $n = 1374$ (18.0 % of 7539) providing valid responses (66.0 % girls).

For the second study phase, a random sample of $n = 552$ adolescents were invited to participate in the baseline measurement. From those, adolescents who did not receive any sleep-related intervention during the study ($n = 346$) were selected for these analyses. Participant is defined as an adolescent who had given valid measurements for sleep actigraphy, circadian temperature or both. For the baseline measurement, $n = 215$ adolescents aged 16 to 18 ($M = 16.86$ years, $SD = 0.61$), with 71.6 % of participants being girls, gave valid measurements. The one-year follow-up measurements were completed by $n = 156$ adolescents, with 76.3 % of participants being girls.

2.3 Measurements

2.3.1 Sleep actigraphy

Actigraphy is used as a behavioral analysis method to measure body movements throughout the day and night. Actigraphy has been found to be more objective and reliable than sleep diaries, but less reliable than polysomnography (Ancoli-Israel et al., 2003). Due to actigraphy being noninvasive, actigraphic measurements permit sleep-wake rhythms to be studied at home, in a natural sleeping environment.

In the present study, The GENEActiv (Original, Kimbolton, UK) actigraphy devices were worn on the wrist of the non-dominant hand. Participants were asked to wear actigraphies for 10 days for both baseline ($M = 8.18$ days, $SD = 1.35$) and follow-up measurements ($M = 8.17$ days, $SD = 1.53$). Participants were instructed to follow their usual sleep schedule, and any unusual events, such as difficulty falling asleep during a certain night, were marked down by the participants. Typical sleep pattern was detected from the 10-day loggings to form average values. Variables derived from actigrams included sleep onset and offset, sleep length (i.e., the duration between sleep onset and offset), and midpoint of the sleep episode. Weekday and weekend sleep had separated variables. On top of the estimates of sleep duration, sleep length during week was deducted from weekend sleep length to examine social jet lag.

2.3.2 Skin surface temperature

For the present study, circadian skin surface temperatures were measured using wireless ThermoChron iButton (DS1922 L, Maxim Integrated, San Jose, CA, USA) devices. The iButton sensors were attached on top of the radial artery with medical tape, and participants were advised to mark down any occasion they removed the device from use. The iButtons have the accuracy of ± 0.5 °C between -10 °C and $+65$ °C, and it can log temperature data at a desired frequency. For this study, the iButtons recorded temperature once each minute. Participants were instructed to follow their normal daily activities, such as physical activity and food intake. Measurements of skin surface temperature have been found to be a reliable way to evaluate circadian temperature rhythmicity (Sarabia, Rol, Mendiola & Madrid, 2008).

Participants were asked to wear the iButton for 10 days for both baseline and follow-up measurements. Because of difficulties wearing the iButton, variables for circadian rhythm were derived from three days of measurements. Variables that were used in the present analyses include averages for circadian period length, the mesor of the skin surface temperature (C°) and the amplitude of the rhythm (C°). They were calculated as previously reported (Lipsanen et al., 2021): circadian period was calculated as the duration of one cycle, mesor as the mean temperature value for the rhythm, and amplitude as half of the variation within a cycle.

2.4 Statistical analysis

Statistical analyses were done with the IBM SPSS Statistics program (IBM Corp. 2021, Windows version 28.0). Before analyses, outliers were identified for each relevant variable. Values over three standard deviations from the mean were classified as outliers and then deleted, after which visual examination was conducted to confirm normal distribution for each variable. One participant was excluded from the analyses due to multiple outliers across different variables and both measurement times. Missing and deleted values were not replaced nor imputed in order to avoid biasing the analyses. Statistical significance was set to $p < .05$ for all analyses.

In response to the first research question, differences between measurement times were examined with repeated measures mixed models, and pairwise comparisons with Bonferroni adjustment were used to determine significant changes for both sexes separately. Sex differences were also examined for both baseline and follow-up by using one-way variance analysis (ANOVA). Linear regression models were formed to predict changes in certain variables. In Model 1, follow-up values were predicted by corresponding baseline values and sex.

Linear regression was also used to address the second research question. In addition to Model 1, Model 2 was formed for sleep length, social jet lag and circadian temperature amplitude, because those variables had significant Pearson's correlation with the opposite type of variable. Model 2s were used to examine associations between sleep and circadian rhythm. Model 2s included interactions between selected variables in order to examine moderating effects. Interactions were selected based on correlations. Variance inflation factor (VIF) values were examined for each linear regression model to ensure that variables had no multicollinearity.

Sleep length, social jet lag and circadian amplitude were formed into three equal sized groups (tertiles) based on rank order. Therefore, adolescents were grouped into either the low/short/small, average/moderate or high/long/large tertile. Tertiles were formed for both baseline and follow-up, and the percentage of adolescents who stayed in the same tertile was calculated. In response to the third research question, ordinal regression was then used to determine likelihoods for follow-up tertile membership, based on baseline tertiles and sex. Besides corresponding baseline tertiles predicting follow-up tertiles, baseline circadian amplitude tertiles were used to predict follow-up sleep length and social jet lag tertiles, whereas baseline sleep length tertiles were used to predict follow-up circadian amplitude tertiles; this gives furthermore insight into the associations between sleep patterns and circadian amplitude (Research question 2). Because some participants' social jet lag was negatively valued, social jet lag was squared before grouping. Therefore, the interpretation of social jet lag tertiles was based on how large of a difference adolescents had between week and weekend sleep, but not whether they slept more during the week or weekend. If the ordinal regression model was not significant, a series of logistic regression was done to determine likelihoods more precisely.

3 Results

3.1 Development of sleep and circadian rhythm over a year

Descriptive values for sleep and circadian temperature variables are presented in Table 1. At the baseline, girls slept more during the week than boys, but after the year this difference disappeared. Girls slept more during the weekend than boys across the year, while girls also consistently feel asleep earlier than boys. For the circadian temperature variables, girls had a significantly higher circadian amplitude than boys at the follow-up.

In response to the first research question, baseline means were deducted from follow-up means (Table 1). Sleep duration during the week became longer over the year while weekend sleep became shorter, however these changes were only significant for girls (Table 1). Although the mean difference in week sleep duration was few minutes greater for boys, boys' sample was smaller than girls', and their

Table 1. Means, standard deviations (in parentheses) and valid n for sleep and circadian temperature variables. Baseline and follow-up values were compared between sexes to see if there were any sex differences, and if the possible differences were the same between measurement times. Difference between measurement times was examined for both sexes separately to determine significant changes in sleep patterns and circadian temperature rhythm.

Variables	Baseline		p	Follow-up		p	Follow-up – Baseline		p ^a
	Female M (SD) n	Male M (SD) n		Female M (SD) n	Male M (SD) n		Female Mean diff.	Male Mean diff.	
Sleep length, week (h:mm)	6:45 (0:51) 148	6:24 (0:56) 57	.014 (F > M)	6:56 (0:53) 109	6:39 (0:47) 31		0:11	0:15	.010 (F)
Sleep length, weekend (h:mm)	7:39 (1:04) 143	7:08 (1:09) 57	.003 (F > M)	7:18 (1:04) 106	6:48 (1:21) 29	.039 (F > M)	-0:21	-0:20	.010 (F)
Social jet lag (h)	0.94 (1.18) 141	0.65 (1.25) 55		0.34 (1.26) 106	0.21 (1.51) 29		-0.60	-0.44	<.001 (F)
Sleep onset, week (h:mm)	0:18 (1:14) 149	0:44 (1:19) 59	.030 (F < M)	0:29 (1:24) 109	1:07 (1:33) 31	.034 (F < M)	0:11	0:23	.012 (F); .016 (M)
Sleep onset, weekend (h:mm)	1:19 (1:37) 142	2:08 (1:31) 59	.001 (F < M)	1:31 (1:39) 106	2:24 (1:53) 30	.015 (F < M)	0:12	0:16	
WASO*, week (h)	0.90 (0.33) 148	0.90 (0.32) 58		0.96 (0.34) 107	0.99 (0.41) 31		0.06	0.09	.020 (M)
WASO*, weekend (h)	1.02 (0.40) 142	1.05 (0.45) 59		0.97 (0.39) 105	0.98 (0.33) 30		-0.05	-0.07	
Sleep midpoint, week (h:mm)	4:07 (1:07) 149	4:23 (1:02) 58		4:27 (1:18) 109	4:56 (1:33) 31		0:20	0:33	<.001 (F); .001 (M)
Sleep midpoint, weekend (h:mm)	5:40 (1:30) 142	6:11 (1:27) 59	.029 (F < M)	5:40 (1:24) 104	6:14 (1:32) 29		0:00	0:03	
Sleep offset, week (h:mm)	7:52 (1:08) 147	8:06 (1:10) 58		8:24 (1:23) 109	8:46 (1:42) 31		0:32	0:40	<.001 (F); .001 (M)
Sleep offset, weekend (h:mm)	10:01 (1:39) 143	10:12(1:45) 59		9:47 (1:33) 105	10:22 (1:51) 30		-0:14	0:10	
Circadian period (h)	24.11 (1.22) 128	24.31 (1.45) 42		24.21 (1.15) 108	24.62 (1.52) 28		0.10	0.31	
Circadian mesor (°C)	33.52 (0.68) 144	33.32 (0.65) 58		33.58 (0.62) 115	33.69 (0.53) 34		0.07	0.37	.003 (M)
Circadian amplitude (°C)	1.69 (0.65) 141	1.54 (0.68) 58		1.71 (0.60) 114	1.23 (0.42) 34	<.001 (F > M)	0.03	-0.31	.006 (M)

* WASO = wake after sleep onset

^a Based on pairwise comparisons with Bonferroni adjustment

measurement had a higher standard error for both week sleep (boys SE = 0.14, girls SE = 0.08) and weekend sleep (boys SE = 0.22, girls SE = 0.12). Because week and weekend sleep length developed to resemble each other, social jet lag became smaller, but the change was once again only significant for girls (Table 1). However, it should be noted that the standard deviations for social jet lag were quite large at both the baseline and follow-up, which insinuates that adolescents' social jet lag differs considerably from one another.

Sleep rhythm became delayed, as sleep onset, midpoint and offset occurred later at the follow-up for both boys and girls during the week (Table 1). Boys also woke up later during the weekend at the follow-up while girls woke up earlier, although neither change was statistically significant. Besides changes in sleep, boys also experienced significant changes in their circadian temperature mesor and amplitude: on average boys' mesor became higher while their amplitude lowered over the year (Table 1). Circadian period became slightly longer for both girls and boys, but this change was not significant.

Sleep length, social jet lag, midpoint of sleep episode and circadian temperature variables were selected for further analyses. Table 2 shows correlations between sleep length, midpoint of sleep episode, differences between weekend and week sleep, and circadian temperature variables for the entire sample, and the Appendix 1 separately for sexes. All the baseline values correlated significantly with the corresponding follow-up value (Table 2). Based on this, linear regression was performed to predict the changes in follow-up values with corresponding baseline values and sex (Model 1). Since there were some differences between sexes (Table 1), sex was included as an independent factor. Table 3 presents estimates for Model 1: baseline predicted follow-up values at a significant level for all variables. Sex was only a significant predictor for circadian amplitude, as girls were predicted to have higher amplitude than boys at follow-up (Table 3). Model 1 was significant for almost all variables, except for circadian period length and midpoint difference between weekend and week (Table 3). Sleep duration during week had greater explanatory power than weekend sleep (30.2 % & 10.6 %, respectively).

3.2 Associations between sleep patterns and circadian temperature

In response to the second research question, Model 2s were formed to examine associations between sleep patterns and circadian temperature. Model 2 was formed for follow-up sleep length during week, social jet lag and circadian amplitude as dependent variables, as these variables had a significant correlation with the opposite type of variable. Baseline circadian amplitude correlated

Table 2. Pearson's correlations for total sleep length, midpoint of sleep episode and circadian temperature variables, for both baseline and follow-up. Total sleep length and midpoint are separated into values for week, weekend and difference between weekend and week (social jet lag). Correlations between measurement times (diagonal of lower left segment) show the degree of relationship between corresponding baseline and follow-up values. Correlations between sleep and circadian variables indicate associations with each other.

Time	Variables	Baseline									Follow-up								
		Sleep length, week	Sleep length, weekend	Social jet lag	Midpoint, week	Midpoint, weekend	Midpoint (weekend - week)	Circadian period	Circadian mesor	Circadian amplitude	Sleep length, week	Sleep length, weekend	Social jet lag	Midpoint, week	Midpoint, weekend	Midpoint (weekend - week)	Circadian period	Circadian mesor	Circadian amplitude
Baseline	Sleep length, week	--																	
	Sleep length, weekend	.305**	--																
	Social jet lag	-.465**	.702**	--															
	Midpoint, week	-.110	-.215**	-.126	--														
	Midpoint, weekend	-.247**	.026	.221**	.605**	--													
	Midpoint (weekend - week)	-.249**	.215**	.406**	-.157*	.692**	--												
	Circadian period	.046	-.009	-.049	-.086	.066	.139	--											
	Circadian mesor	.015	.014	-.018	.028	-.010	-.032	-.157*	--										
	Circadian amplitude	.200**	.183*	.031	.017	.002	-.009	.069	-.600**	--									
Follow-up	Sleep length, week	.547**	.245**	-.186*	-.292**	-.264**	-.076	.067	.031	.175*	--								
	Sleep length, weekend	.234**	.313**	.137	-.075	-.019	.040	-.048	.075	.040	.227**	--							
	Social jet lag	-.142	.121	.250**	.127	.158	.087	-.094	.047	-.084	-.467**	.756**	--						
	Midpoint, week	-.219*	-.317**	-.143	.580**	.532**	.162	-.084	-.053	-.004	-.072	-.212*	-.144	--					
	Midpoint, weekend	-.197*	-.149	.014	.494**	.567**	.279**	-.150	-.028	-.004	-.173*	-.124	.004	.745**	--				
	Midpoint (weekend - week)	.005	.203*	.213*	-.045	.126	.195*	-.104	.030	-.001	-.154	.104	.198*	-.258**	.451**	--			
	Circadian period	-.031	-.022	.020	.100	.029	-.052	.187*	-.131	.102	-.055	-.099	-.053	.061	.063	.008	--		
	Circadian mesor	-.107	-.037	.031	.120	.108	.029	-.171	.402**	-.281**	-.045	.081	.104	.135	.130	.007	-.032	--	
	Circadian amplitude	.200*	.123	-.019	-.121	-.107	-.029	.048	-.243**	.440**	.229**	.032	-.124	-.041	-.096	-.085	-.064	-.584**	--

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Table 3. Follow-up values were predicted by corresponding baseline values and sex with linear regression. Regression coefficients (unstandardized B), p-values (in parentheses) for coefficients, model significance and R²s for Model 1.

Independents	Dependent follow-up variable								
	Sleep length, week B (p)	Sleep length, weekend B (p)	Social jet lag B (p)	Midpoint, week B (p)	Midpoint, weekend B (p)	Midpoint weekend - week B (p)	Circadian period B (p)	Circadian mesor B (p)	Circadian amplitude B (p)
Baseline value	0.54 (<.001)	0.32 (<.001)	0.28 (.004)	0.74 (<.001)	0.56 (<.001)	0.17 (.023)	0.18 (.046)	0.38 (<.001)	0.37 (<.001)
Sex	0.10 (.487)	0.26 (.278)	0.08 (.763)	-0.36 (.118)	-0.26 (.318)	0.01 (.997)	-0.38 (.273)	-0.15 (.189)	0.39 (<.001)
Model sig.	<.001	<.001	.014	<.001	<.001	.075	.072	<.001	<.001
R ²	0.302	0.106	0.063	0.345	0.327	0.039	0.045	0.172	0.273

* Male as reference for sex

significantly with follow-up sleep length for the entire sample ($r = .175$, $p < .05$), while the reverse was also significant: baseline sleep length correlated with follow-up circadian amplitude ($r = .200$, $p < .05$). Because of this, baseline sleep length and circadian amplitude were selected as additional predictors for each other. Because circadian amplitude and sleep length correlated at baseline ($r = .200$, $p < .01$), the interaction between these variables was included to predict follow-up sleep length and circadian amplitude. Baseline circadian amplitude also correlated with follow-up social jet lag once the correlations were examined separately for sexes (Appendix 1). This correlation was only significant for girls ($r = -.208$, $p < .05$). Therefore, the interaction between sex and circadian temperature amplitude was selected to predict follow-up social jet lag.

Table 4 presents estimates for how well independent variables predicted the follow-up values. Model 2 was significant for all predicted variables (Table 4). Sleep length during the week was predicted significantly by baseline sleep length ($B = 0.53$, $SE = 0.07$, $p < .001$), and the interaction between sleep length and circadian amplitude ($B = 0.23$, $SE = 0.12$, $p < .049$): circadian amplitude moderated the association between baseline and follow-up sleep duration. On the other hand, circadian amplitude was predicted at a significant level by baseline amplitude ($B = 0.36$, $SE = 0.07$, $p < .001$) and sex ($B = 0.37$, $SE = 0.10$, $p < .001$), while sleep length and the interaction were not statistically significant predictors (Table 4). Baseline social jet lag was the only significant predictor for follow-up social jet lag in Model 2 (Table 4).

3.3 Likelihoods to have better sleep patterns and higher circadian amplitude

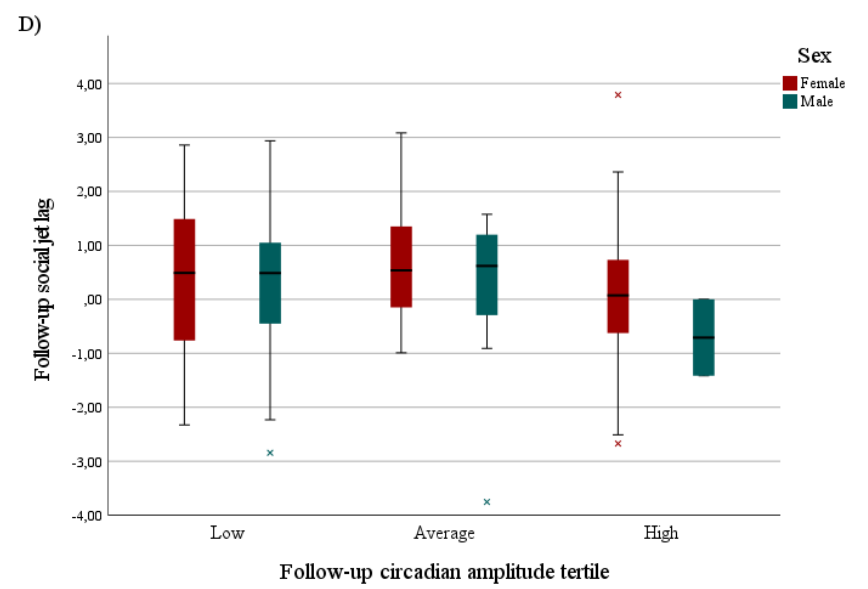
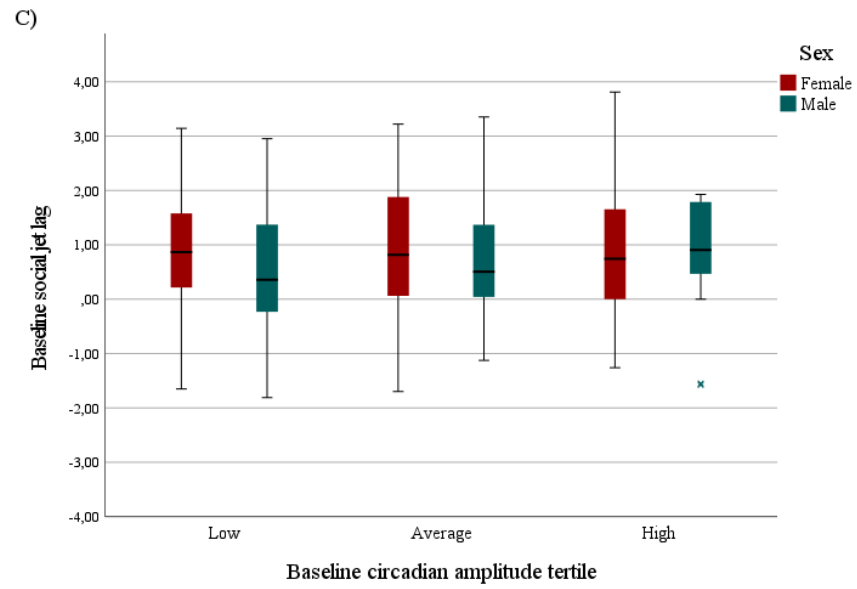
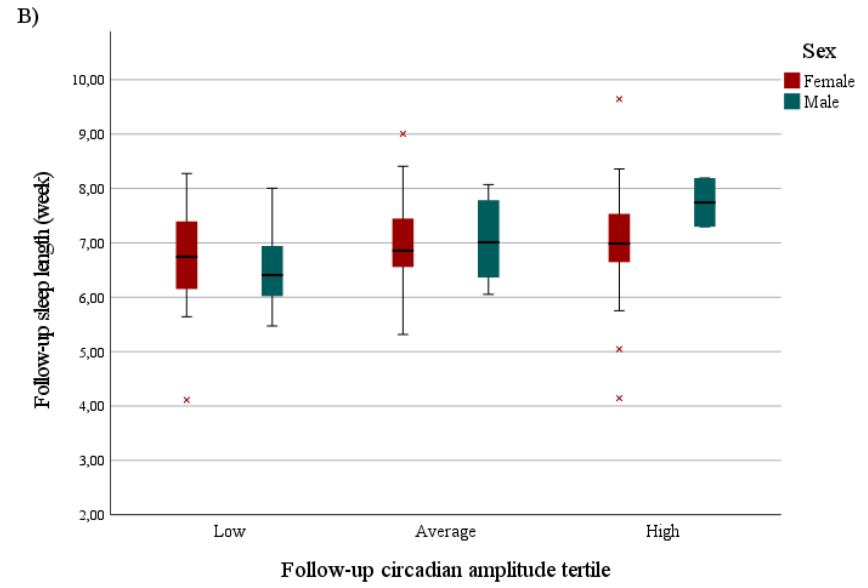
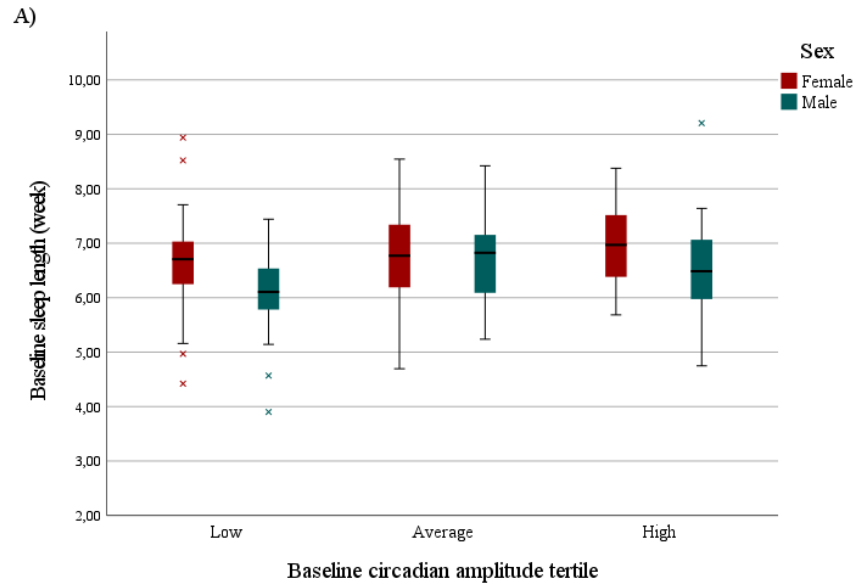
Adolescents were grouped into tertiles based on their rank order in sleep length, social jet lag and circadian amplitude. Tertiles were formed for both baseline and follow-up. Figure 1 shows mean values for sleep length, social jet lag and circadian amplitude, when they are examined within sleep length or circadian amplitude tertiles. The differences between tertiles seem to appear only at the follow-up: the baseline tertiles are quite similar with each other within all three variables (Figure 1A,

Table 4. Follow-up values for sleep length (week), social jet lag and circadian temperature amplitude were predicted with baseline values and sex to determine what influences the changes in the dependent variables. Regression coefficients (unstandardized B), standard errors (in parentheses), p-values, model significance and R²s for Model 2.

Dependent follow-up variable	Independent baseline variable	Model 2	
		B (SE)	p
Sleep length, week			
	Sleep length, week	0.53 (0.07)	<.001
	Sex ^a	0.07 (0.15)	.630
	Circadian amplitude	0.08 (0.10)	.437
	Sleep length*Circadian amplitude	0.23 (0.12)	.049
	Model sig.	<.001	
	R ²	0.347	
Social jet lag			
	Social jet lag	0.23 (0.10)	.025
	Sex ^a	0.02 (0.26)	.938
	Circadian amplitude	0.32 (0.34)	.349
	Circadian amplitude*Sex	-0.64 (0.40)	.113
	Model sig.	.044	
	R ²	0.076	
Circadian amplitude			
	Circadian amplitude	0.36 (0.07)	<.001
	Sex ^a	0.37 (0.10)	<.001
	Sleep length, week	0.09 (0.05)	.095
	Sleep length*Circadian amplitude	0.01 (0.09)	.931
	Model sig.	<.001	
	R ²	0.293	

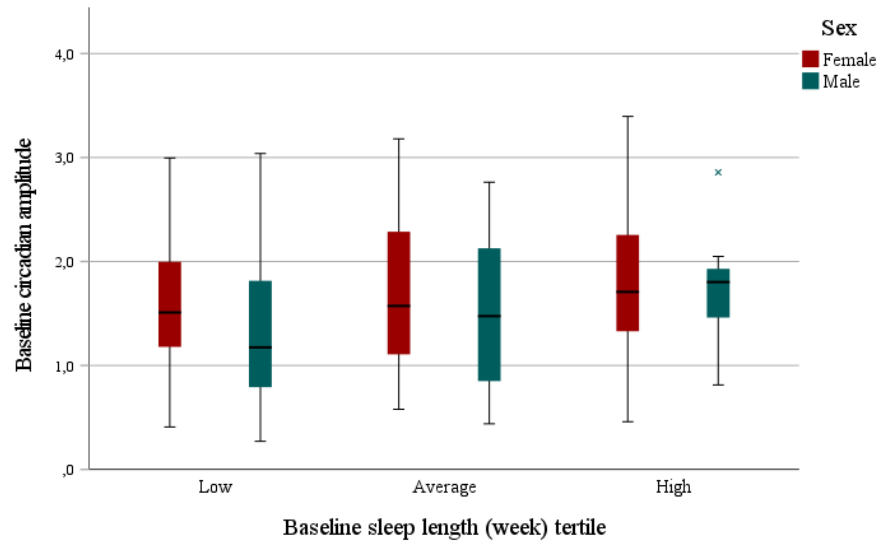
^aMale as reference for sex

C & E), whereas the follow-up tertiles for sleep length (Figure 1B) and social jet lag (Figure 1D) differ more from each other. Especially boys' follow-up circadian amplitude tertiles appear to differ from each other. Boys seem to sleep for longer within each circadian amplitude tertile (Figure 1B), which was also apparent from correlations, as boys' follow-up sleep length and follow-up circadian amplitude had a significant correlation ($r = .576$, $p < .001$), whereas girls did not (Appendix 1). Furthermore, boys' with high circadian amplitude at follow-up had different social jet lag than other tertiles (Figure 1D), whereas at the baseline the circadian amplitude tertiles had quite similar social jet lags (Figure 1 C). Some participants stayed in the same tertile between baseline and follow-up, but tertiles did not completely remain the same. From baseline to the follow-up, 51.8 % of participants



(Continued)

E)



F)

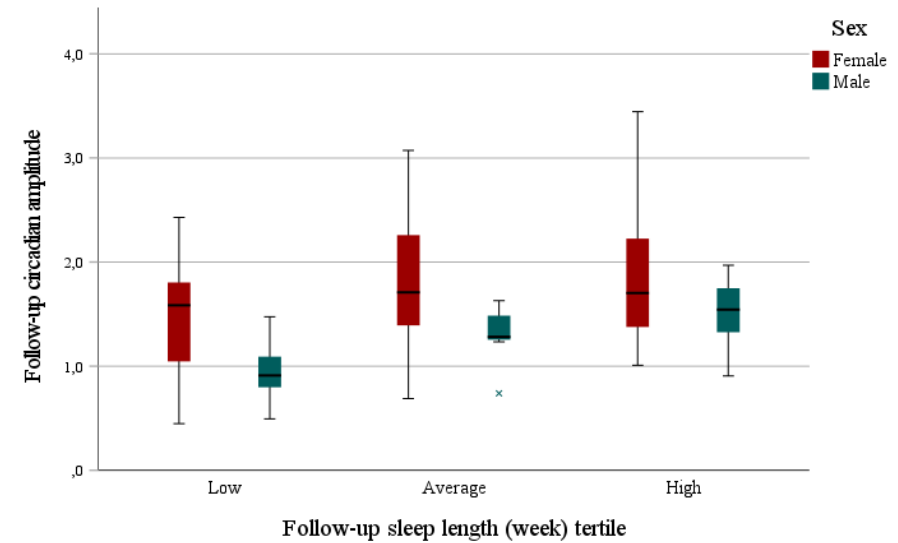


Figure 1. Means and 95 % confidence intervals for sleep length (A & B), social jet lag (C & D) and circadian amplitude (E & F). Means were grouped by sex and circadian amplitude or sleep length tertile. Means and tertiles were graphed separately for baseline (left column) and follow-up (right column). Figures show how the association between sleep length and circadian amplitude, or social jet lag and circadian amplitude differ between the two measurement times.

stayed in the same sleep length tertile, 44.4 % of participants stayed in the same social jet lag tertile, and 53.2 % of participants stayed in the same circadian amplitude tertile.

In order to address the third research question, follow-up tertiles were predicted with ordinal regression, with baseline tertiles and sex as independent factors to determine likelihoods to have better sleep patterns and higher circadian amplitude. The ordinal regression model included the same variables as Model 2, sans the interactions. Estimates to belong in the highest tertile at follow-up are presented in Table 5, and odds ratios (OR) are depicted in Appendix 2. The ordinal regression model was significant for sleep length and circadian amplitude, but not for social jet lag (Table 5). Tertiles with average or short sleep duration were significantly less likely to have long sleep lengths at the follow-up (OR = 0.21, $p < .001$ & OR = 0.10, $p < .001$, respectively) compared to participants who were in the long sleep tertile at baseline. Baseline circadian amplitude tertiles or sex did not affect the likelihood to have long sleep at follow-up (Table 5), although boys' high circadian amplitude tertile appeared to have different changes than any other tertile, as their sleep duration was at a slight downward slope (Figure 2A).

Social jet lag seems to grow for girls with low baseline circadian amplitude, while boys with low circadian amplitude seem to have greater social jet lag than other boys (Figure 2B). Despite this, likelihood to have large social jet lag did not differ within any baseline tertiles or sex (Table 5). Moderate social jet lag did not differ from the large social jet lag tertile at the follow-up, as it had a threshold estimate of 0.01, while small social jet lag tertile had a threshold estimate of -1.40 . Given the fact that follow-up social jet lag tertiles were similar to each other, proportional odds assumption was not met ($\chi^2 = 11.78$, $df = 5$, $p = .038$). Series of logistic regressions revealed that low circadian amplitude tertile had a significant likelihood to have moderate social jet lag at follow-up compared to high circadian amplitude tertile (OR = 3.28, $p = .021$). There were no other tertile differences.

Having high circadian amplitude at follow-up was significantly predicted by sex, as girls were 5.85-fold ($p < .001$) more likely to have high circadian amplitude than boys. Low circadian amplitude and short sleep tertiles were less likely to have high circadian amplitude at the follow-up compared to high circadian amplitude and long sleep tertiles (OR = 0.09, $p < .001$ & OR = 0.42, $p = .047$, respectively). Figure 2C shows the association between sleep tertiles and circadian amplitude changes. For girls, the three tertiles stay quite stable across the year, but for boys circadian amplitude appears to lower in each sleep tertile, which supports the changes in boys' circadian amplitude (Table 1).

Table 5. Ordinal regression estimates for sleep length during week, social jet lag and circadian temperature amplitude, when predicting follow-up tertiles (highest as reference) with baseline tertiles (highest as reference) and sex (male as reference). Odds ratios (OR) indicate how likely it is for adolescents within certain tertile/sex to have long sleep length, large social jet lag or high circadian amplitude compared to the reference tertile/sex.

Baseline tertile	Estimate	SE	Wald	df	p	OR	95% CI
Follow-up sleep length, week							
Long sleep length	0.00					1.00	
Average sleep length	-1.58	0.43	13.26	1	<.001	0.21	[0.09; 0.48]
Short sleep length	-2.27	0.47	23.21	1	<.001	0.10	[0.04; 0.26]
High amplitude	0.00					1.00	
Average amplitude	0.17	0.42	0.15	1	.694	1.18	[0.52; 2.71]
Low amplitude	-0.37	0.45	0.66	1	.418	0.69	[0.29; 1.68]
Male	0.00					1.00	
Female	0.44	0.42	1.09	1	.297	1.55	[0.68; 3.55]
Model fit (χ^2)	34.99			5	<.001		
R ² (Nagelkerke)	0.271						
Follow-up social jet lag							
Large social jet lag	0.00			0		1.00	
Moderate social jet lag	-0.35	0.42	0.70	1	.403	0.71	[0.31; 1.60]
Small social jet lag	-0.68	0.41	2.82	1	.093	0.51	[0.23; 1.12]
High amplitude	0.00			0		1.00	
Average amplitude	-0.35	0.40	0.78	1	.378	0.70	[0.32; 1.54]
Low amplitude	0.03	0.42	0.01	1	.943	1.03	[0.45; 2.36]
Male	0.00			0		1.00	
Female	-0.31	0.40	0.60	1	.439	0.74	[0.34; 1.60]
Model fit (χ^2)	4.07			5	.539		
R ² (Nagelkerke)	0.036						
Follow-up circadian amplitude							
High amplitude	0.00			0		1.00	
Average amplitude	-0.69	0.41	2.92	1	.088	0.50	[0.23; 1.11]
Low amplitude	-2.40	0.49	23.83	1	<.001	0.09	[0.04; 0.24]
Long sleep length	0.00			0		1.00	
Average sleep length	-0.34	0.41	0.68	1	.410	0.71	[0.32; 1.60]
Short sleep length	-0.86	0.43	3.94	1	.047	0.42	[0.18; 0.99]
Male	0.00			0		1.00	
Female	1.77	0.46	14.48	1	<.001	5.85	[2.35; 14.51]
Model fit (χ^2)	53.59			5	<.001		
R ² (Nagelkerke)	0.371						

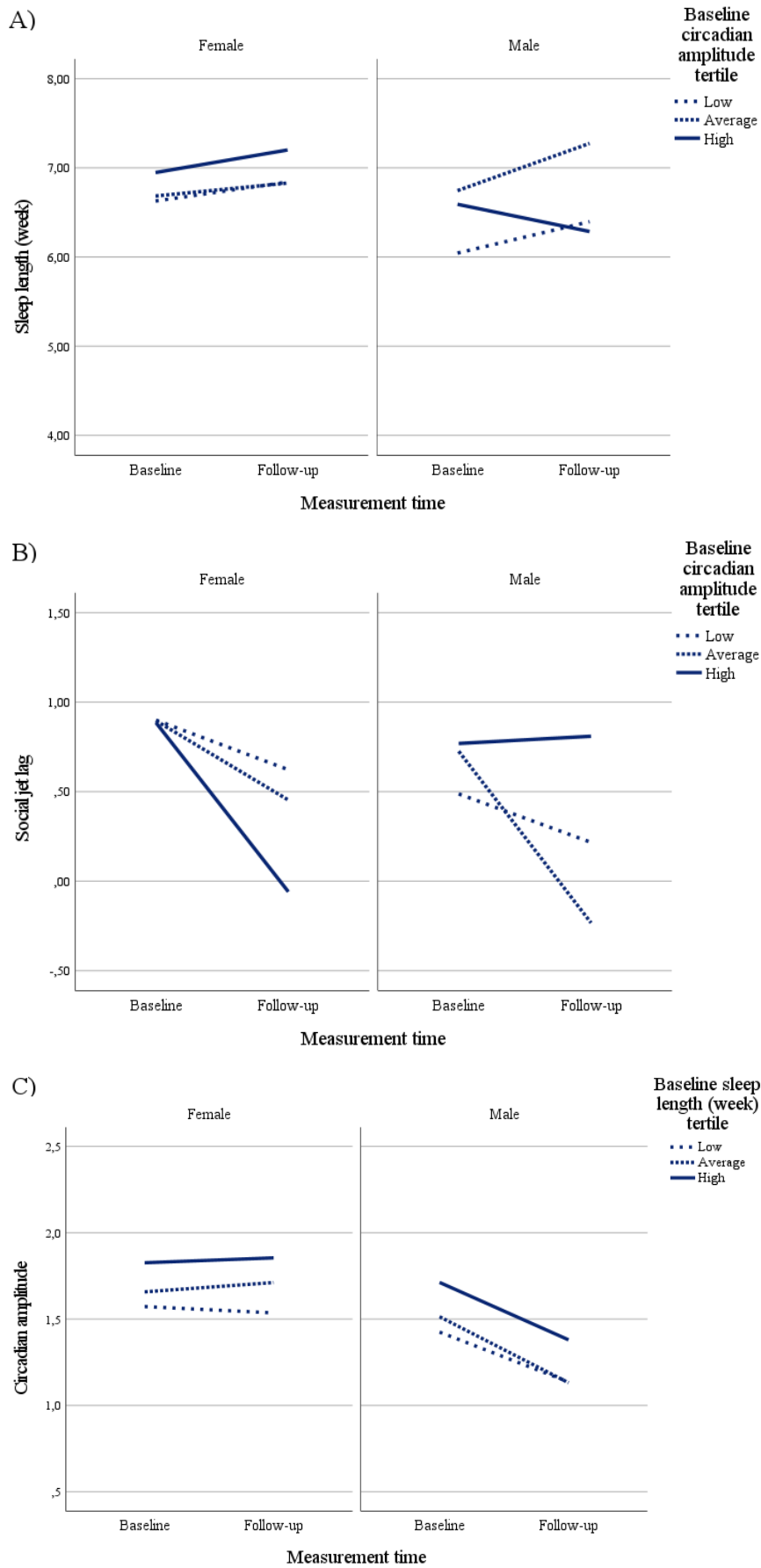


Figure 2. How sleep length (A), social jet lag (B), and circadian amplitude (C) change from baseline to follow-up within a certain baseline tertile. Each graph is divided by sex.

4 Discussion

4.1 Main findings

The aim of this study was to examine how sleep patterns and circadian temperature rhythm change over a one-year period in adolescents aged 16–18 years. There were changes in both week and weekend sleep durations, especially for girls. Most prominent change was in the sleep-wake rhythm, as adolescents' sleep episodes became more delayed during the week. Besides sleep, only boys experienced changes in their circadian temperature mesor and amplitude. The average skin surface temperature rose for boys, while their daily change in temperature became smaller. Adolescents' circadian amplitude moderated changes in sleep duration during the week, as higher circadian amplitude predicted longer sleep after the year. Still, adolescents who slept long during the week had an increased likelihood to also have a long sleep duration after the year, when compared to adolescents who had a shorter sleep duration.

4.1.1 Changes in sleep and circadian rhythm

Adolescents slept more during the week over the year, but their weekend sleep shortened, which is contradictory to previous findings of both week and weekend sleep becoming shorter (Wolfson & Carskadon, 1998). These changes were only significant for girls, as boys had more variation among themselves. Changes in sleep duration were explained 30.2 % by past sleep duration when examining week sleep, but for weekend sleep duration only 10.6 % could be explained by past sleep. Weekend sleep is generally less hindered than week sleep, as adolescents do not have to adjust their sleep schedule to their school schedule. This could explain why weekend sleep was less stable than week sleep over time, and therefore weekend sleep would be more likely to change independent of past behaviour. The changes in weekday and weekend sleep were reflected as a decrease in social jet lag. On average, adolescents in this study had less than an hour of social jet lag, and after the year it decreased to less than a half an hour. However, social jet lag had quite large variations among adolescents: while some participants were sleeping more during the weekend than they did during the week, some adolescents had negative social jet lag, meaning their weekend sleep was shorter than week sleep. Predicting changes in social jet lag resembled predicting weekend sleep, as both had quite small explanatory powers. Because of this, social jet lag also appears to be more sensitive to changes that are independent of past sleep patterns.

As hypothesized, adolescents' sleep episodes shifted to a later time after a year, as they went to sleep later and woke up later during the week. Despite changes in both girls' and boys' sleep timing, girls always fell asleep earlier than boys, which is in line with previous studies (Spruyt et al., 2005; Loessl

et al., 2008). Bedtimes becoming delayed during adolescence is supported by previous studies (Wolfson & Carskadon, 1998; Thorleifsdottir et al., 2002; Loessl et al., 2008), as it is one of the most robust changes in sleep schedules (Evans et al., 2021). The shift in sleep rhythm was more prominent during the week than the weekend in the present study.

Since circadian period length did not become longer between the measurement times, it appears that the change in sleep-wake rhythm could be caused by a shift in the circadian phase, and not by changes in circadian period. Entrainment of circadian rhythm depends on timing of social cues: phase-response curve defines that the circadian phase is more likely to shift, if one is exposed to strong enough cues within few hours of circadian markers, such as melatonin or temperature peak (Kuhlman et al., 2018). While natural light is the most effective regulator for circadian rhythmicity, the SCN is especially susceptible to short-wavelength spectrum light, also known as blue light (Zee & Manthena, 2007), which is omitted from electronic devices. Circadian phase can be delayed by blue light, if blue light is seen at the later part of the phase-response curve. Moreover, media usage has also been found to be connected to short sleep durations (Twenge, Krizan & Hisler, 2017). The phase could be advanced by getting exposed to light during the morning, but given the fact that adolescents woke up later as they aged, this seems unlikely. However, it is important to note that the effects of electronic devices are not limited to adolescence, as the SCN is sensitive to blue light at any age. Additionally, the shift could be influenced more by the behavioral aspect of adolescents choosing to use media devices instead of sleeping, or by the physiologically arousing content of media that makes falling asleep more troublesome (e.g., LeBourgeois et al., 2017).

Adolescents' sleep timing could also be affected by changes in sleep homeostasis. Crowley and colleagues (2014) have theorized that adolescents accumulate sleep pressure slower than children and adults, resulting in a naturally longer waking period. Slower accumulation could be caused by the decrease in melatonin levels, as adolescents' melatonin secretion is smaller than children's (Rensen et al., 2020). As sleep homeostasis is in constant interaction with the circadian rhythm (Borbély, 1982), adolescents' attenuated sleep pressure could slowly delay the internal clock, especially since activity in SCN has been found to decrease when sleep pressure increases (Borbély, 2016). Therefore, adolescents' delayed sleep rhythm would still match their endogenous circadian rhythm, but not the societal time they are expected to follow.

From a behavioral standpoint, adolescents might stay up later for many reasons. Becoming independent from one's parents is one of the biggest developmental steps in youth, and adolescents are generally more likely to be able to choose their own bedtime compared to children, whose parents

might moderate their bedtime. Besides getting to choose their bedtimes, adolescents have reported schoolwork and working paid jobs as reasons why they do not sleep for long (Twenge et al., 2017). Adolescents are left to struggle with balancing school responsibilities and social life, while also trying to maintain a healthy sleep schedule. Furthermore, once sleep is delayed, adolescents might not be motivated to advance their sleep schedule to ensure enough sleep. Waking up early for school could help indirectly by exposing adolescents to light during the morning, which could advance their circadian phase. Although this seems to be nullified during the weekend, because adolescents wake up even later during the weekend than the week, (as shown in Table 1; Thorleifsdottir et al., 2002; Spruyt et al., 2005), presumably to recover from sleep deprivation that might have been caused by too little sleep on weekdays.

Besides changes in sleep patterns, only boys' average skin surface temperature increased while their circadian amplitude decreased. Despite sleep patterns and circadian temperature rhythm developing over a year long period, the likelihoods to have longer sleep length during the week and higher circadian amplitude were affected by past sleep duration and circadian amplitude. Adolescents with short or average sleep length were significantly less likely to have long sleep durations after a year than adolescents, who already had long sleep durations. This was expected, as in previous studies sleep duration has been found to be quite stable within individuals across the lifespan (Thorleifsdottir et al., 2002). Furthermore, adolescents who had low circadian amplitude were less likely to develop high amplitude within their circadian temperature rhythm. Both sleep length and circadian amplitude were quite stable over time, as approximately half of the participants were grouped into the same tertile at both measurement times (51.8 % and 53.2 %, respectively).

4.1.2 Sleep duration and circadian amplitude are connected

In a previous study from the SleepHelsinki! research project, Lipsanen and colleagues (2021) found that adolescents' circadian temperature amplitude is associated with sleep schedules, as adolescents with higher circadian amplitude had earlier timed sleep schedules. In the current study, circadian amplitude had a moderating effect on week sleep duration when examining changes over the year. Circadian amplitude affects sleep duration positively, as the higher the adolescents' circadian amplitude was, the longer the adolescents sleep becomes. While sleep did not moderate circadian amplitude changes, sleep length at baseline was still connected to how likely it was to have high circadian amplitude after the year: adolescents with short sleep length were 0.42 times less likely to have high circadian amplitude than those with long sleep durations.

In children, smaller circadian amplitude has been associated with evening-oriented chronotype (Martínez-Lozano et al., 2020). The result of the previous (Lipsanen et al., 2021) and the current study supports the effect of circadian amplitude on sleep is the same in adolescence as they are in childhood: during development those with higher circadian amplitude go to bed earlier and get more sleep compared to adolescents with smaller circadian amplitude. Interestingly, the direction of this association seems to change later in adulthood: instead of smaller circadian amplitude being connected to evening type, adults who have later timed circadian phases also have higher circadian amplitudes (Baehr et al., 2000). Still, the connection between eveningness and later sleep-wake rhythm remains the same (Horne & Östberg, 1997; Karan et al., 2021). It is unclear when exactly this change happens, or why it happens.

The association between sleep duration and circadian amplitude could be facilitated by melatonin. As stated earlier, the circadian rhythms of melatonin and CBT are inversely connected and manipulating melatonin levels causes changes in body temperature (Cagnacci et al., 1992; Benloucif et al., 2005). The thermoregulating properties of melatonin might account for $\pm 40\%$ of the changes in CBT amplitude (Cagnacci et al., 1992). Given the fact that melatonin is a sleep-inducing hormone (Brzezinski, 1997), there could be a three-way association between sleep, temperature amplitude and melatonin amplitude. While the current study did not measure adolescents' melatonin rhythm, one possible mechanism could be that individuals who produce higher levels of melatonin might have larger daily changes in their temperature: those with high levels of melatonin could have a larger drop in their body temperature compared to those whose melatonin levels are lower. It is speculated that the decrease in melatonin levels in adolescents (Rensen et al., 2020) is caused by the growth spurt during puberty (Brzezinski, 1997). It could be that the growing body mass and lowered melatonin levels cause smaller changes within the body temperature in adolescents. This could partially explain why there was sex differences in circadian temperature amplitude, as boys generally enter puberty later than girls (Vijayakumar, Op de Macks, Shirtcliff & Pfeifer, 2018), and therefore they experience the growth spurt, drop in melatonin level and the possible effects on body temperature amplitude later than girls.

Sex also affected the association between circadian temperature amplitude and sleep duration. Girls had higher circadian amplitude than boys after a year because boys' circadian amplitude decreased. Because of this, girls had 5.85-fold likelihood to have high circadian amplitude than boys. Higher circadian amplitude in girls might explain why girls slept more than boys, especially during the weekend. What causes sex differences in the connection between temperature amplitude and sleep? During puberty, the preoptic area of hypothalamus (POAH) becomes more active and releases more

gonadotropin-releasing hormone (GnRH), which in turn regulates the release of gonadal axis hormones (Vijayakumar et al., 2018). As stated, girls go through puberty at younger age than boys (Vijayakumar et al., 2018), but puberty is also different for sexes. Menstruation causes women to experience monthly fluctuations in predominantly female hormones, as estrogen levels rise during follicular phase until ovulation and then plummet during luteal phase, while progesterone levels stay low until the luteal phase. It has been found that the ratio between progesterone and estrogen in the luteal phase facilitates decreases in circadian amplitude and increases in temperature mesor (Cagnacci, Volpe, Paoletti & Melis, 1997). On top of hormone regulation, the medial POAH integrates thermoregulatory information from the body as it has thermosensitive neurons, and POAH's ventrolateral area is essential for NREM initiation (Harding et al., 2019). The POAH is connected to the SCN, as they are both located in the anterior part of the hypothalamus (Zee & Manthena, 2007). The rising hormone secretion activity in the POAH could possibly cause changes in its functions on body temperature and sleep, and furthermore, also in SCN's activity. If it does, girls would experience it earlier than boys due to different timing of puberty. On top of that, the SCN has estrogen receptor- α 's, which are more prominent in female SCN's (Kruijver & Swaab, 2002), so increase in estrogen secretion could directly affect the functioning of SCN in girls.

Besides normative development, circadian temperature amplitude also appears to play a role in sleep-related problems. Tomoda, Miike, Yonamine, Adachi and Shiraishi (1997) found that 12–18-year-old adolescents, who had been diagnosed with sleep disturbances (DSPD, non-24-hour sleep-wake syndrome, irregular sleep, or long sleep), had significantly lower skin temperature amplitudes than healthy controls. Tomoda and colleagues (1997) concluded that desynchronization between the sleep-wake rhythm and circadian rhythm can cause difficulties in daily activities, including school attendance which was a common demeanor among sleep disturbed participants. There is also a connection between circadian amplitude and depression. Lorenz, Spada, Sander, Riedel-Heller and Hegerl (2019) examined relative amplitude in healthy controls and depressed adults, who were not taking antidepressants. The depressed group had a lower CBT amplitude and less stable interdaily temperature rhythm than the control group (Lorenz et al., 2019). Although Lorenz and colleagues (2019) did not find any differences in sleep between the two groups, clinical depression has a strong connection to sleep, as sleep-related changes are classified as one of the possible symptoms of depression in DSM-5 and ICD-10, and sleep disruptions can precede mood disorders (Wulff et al., 2010).

Artificially changing skin temperature amplitude has also been found to affect sleep structure. Raymann, Swaab and Van Someren (2008) used a thermosuit to manipulate adult participants'

proximal and distal skin temperatures during sleep in an alternately and cyclical manner between the body parts. The thermosuit increased participants' skin temperature by 0.4 C°, which was sufficient to deepen sleep while also making waking up during the night less likely in both young and older adult participants (Raymann et al., 2008). The warming especially increased slow wave sleep and prolonged wake-up times in older adults with insomnia (Raymann et al., 2008). These results show that circadian amplitude presents an opportunity to develop novel treatments for sleep-related problems. In the future, treatments based on circadian amplitude should be developed more, especially ones that are targeted towards children and adolescents, in order to aid physical and mental health.

4.2 Strengths & limitations

The current study had a longitudinal structure, which made it possible to examine changes within the same individuals. However, one of the limitations was dropouts. There was also large fluctuation in sleep patterns and circadian rhythm between adolescents, resulting in quite a few outliers within multiple variables. One participant was excluded from the analyses, because at the follow-up they slept mainly during the daytime. It is unclear as to why this adolescent's sleep rhythm had shifted so drastically compared to their peers.

Among the sample, there was a clear difference in the number of girls and boys in this study, as 71.6 % and 73.6 % of the participants were girls. The small sample size of boys was especially apparent when the participants were divided into equal sized tertiles based on rank order. This resulted in some empty cells, as some tertiles did not have any boys. The small sample size of boys also affected the power of the analyses in this study, making the sex differences less reliable. The results on sex differences in sleep patterns might also be distorted due to the fact that boys have been found to move more during their sleep (Meltzer et al., 2019). Because of this, boys' actigraphies might not be as accurate as girls', which could have contributed to boys' having greater standard deviations and standard errors in their sleep duration measurements. However, whether or not boys moved more than girls was not assessed in the current study.

Girls were asked to log their menstruation cycle during the measurements, but because of a small number of recordings, the menstruation cycles were left out of these analyses. Menstruation has been found to affect sleep in women, especially in those who have physical symptoms such as cramps (Baker & Driver, 2007). The phase of the cycle has also been found to affect circadian rhythm in women, including temperature amplitude (Cagnacci et al., 1997). The underlying factors that cause

the differences in circadian temperature amplitude between girls and boys could be partially explained by the girls' menstruation cycles, but it could not be tested.

Time of year was also not accounted for in the analyses. Adolescents participated in this study across all seasons. Natural light exposure is different during different seasons, therefore it could have a different effect as a social cue for the circadian rhythm. Different times of the year also meant that some participants were waking up for school during the week, while others were on summer vacation and had more freedom in their sleeping schedule. In future studies, it could be interesting to account for the time of the year, as the seasons in Finland are quite different from each other.

Sleep and circadian rhythm were measured at the same time, which makes the association between sleep and circadian rhythm more ecologically valid. Although participants were asked to wear both the sleep actigraphy and iButton device for 10 days, adolescents had difficulties wearing the iButton. In the end, the circadian temperature variables were only averaged from three days of measurements. On the other hand, it was easy for the adolescents to wear the sleep actigraphy. Despite the difficulties, sleep and circadian temperature rhythm was measured in a natural setting, giving this study more ecological validity.

4.3 Conclusions

The current study examined adolescents' sleep patterns with sleep actigraphy and circadian rhythm through skin surface temperature. Adolescents aged 16 to 18 were remeasured after a year to see how the sleep-wake and circadian rhythm change over this time. The results show that adolescents' sleep rhythm shifts later over the year, and girls especially experience changes in how much they sleep, as they sleep more during the week and less during the weekend as they age. This resulted in girls' social jet lag to also shorten, but generally adolescents' social jet lag had quite large variation within individuals. Besides sleep, boys experience changes in their circadian rhythm, as their temperature mesor rises while simultaneously they are experiencing less variation in their temperature. Sleep and circadian rhythm were found to be connected: circadian temperature amplitude moderates changes in sleep duration across a year, as higher circadian amplitude influences sleep duration to become longer. This could partially explain differences in sleep lengths between sexes, as girls were more likely to have high circadian amplitude than boys, in addition to also sleeping more than boys. Still, the likelihood to have long sleep duration was affected by past sleep duration, as adolescents who slept more had greater likelihood to have long sleep duration after the year, when comparing them to adolescents who slept for shorter amounts of time.

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Appendix 1

Appendix 1. Pearson's correlations separated by sex. Correlations for total sleep time, midpoint of sleep and circadian temperature variables, for both baseline and follow-up measurements. Total sleep time and midpoint are separated by week, weekend and difference between weekend and week (social jet lag). Correlations between measurement times (diagonal of lower left segment) show the degree of relationship between corresponding baseline and follow-up values. Correlations between sleep and circadian variables indicate associations with each other.

Female		Baseline									Follow-up								
Variables		Sleep length, week	Sleep length, weekend	Social jet lag	Midpoint, week	Midpoint, weekend	Midpoint (weekend - week)	Circadian period	Circadian mesor	Circadian amplitude	Sleep length, week	Sleep length, weekend	Social jet lag	Midpoint, week	Midpoint, weekend	Midpoint (weekend - week)	Circadian period	Circadian mesor	Circadian amplitude
Baseline	Sleep length, week	--																	
	Sleep length, weekend	.285**	--																
	Social jet lag	-.469**	.713**	--															
	Midpoint, week	-.058	-.234**	-.172*	--														
	Midpoint, weekend	-.189*	.047	.192*	.589**	--													
	Midpoint (weekend - week)	-.202*	.265**	.402**	-.182*	.688**	--												
	Circadian period	.066	.009	-.041	-.100	.038	.141	--											
Circadian mesor	0.014	.012	-.001	.001	.050	.059	-.102	--											
Circadian amplitude	.173*	.171*	.022	.016	.024	.014	.041	-.590**	--										
Follow-up	Sleep length, week	.495**	.195*	-.204*	-.316**	-.285**	-.079	.094	.010	.232*	--								
	Sleep length, weekend	.188	.238*	.096	-.081	-.039	.020	-.015	.140	-.041	.238*	--							
	Social jet lag	-.170	.076	.230*	.150	.165	.074	-.087	.120	-.208*	-.499**	.723**	--						
	Midpoint, week	-.203*	-.274**	-.104	.546**	.543**	.198*	-.153	-.057	.015	-.056	-.151	-.096	--					
	Midpoint, weekend	-.164	-.065	.080	.452**	.576**	.317**	-.156	.006	.007	-.172	-.066	.063	.702**	--				
	Midpoint (weekend - week)	.024	.241*	.236*	-.041	.133	.195*	-.019	.077	-.008	-.164	.096	.203*	-.277**	.490**	--			
	Circadian period	.036	-.011	-.003	.176	.032	-.111	.179	-.113	.170	-.115	-.062	.028	.058	.087	.046	--		
	Circadian mesor	-.128	.018	.098	.101	.073	.003	-.153	.384**	-.223*	-.026	.102	.109	.105	.112	.024	-.118	--	
	Circadian amplitude	.163	.020	-.093	-.140	-.074	.026	.034	-.300**	.411**	.158	-.066	-.172	.015	-.056	-.096	-.020	-.614**	--

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

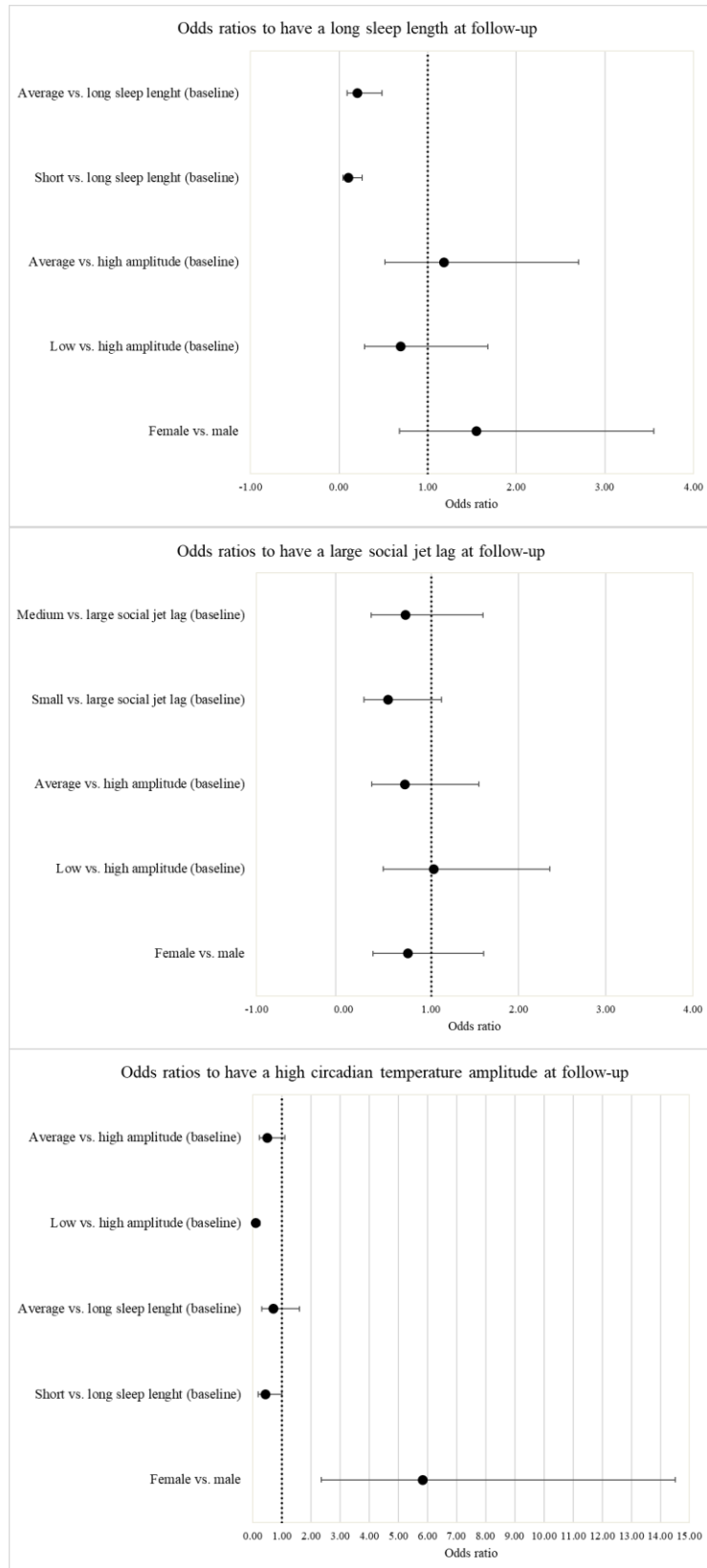
Appendix 1. *Continued.*

Male		Baseline									Follow-up									
	Variables	Sleep length, week	Sleep length, weekend	Social jet lag	Midpoint, week	Midpoint, weekend	Midpoint (weekend - week)	Circadian period	Circadian mesor	Circadian amplitude	Sleep length, week	Sleep length, weekend	Social jet lag	Midpoint, week	Midpoint, weekend	Midpoint (weekend - week)	Circadian period	Circadian mesor	Circadian amplitude	
Baseline	Sleep length, week	--																		
	Sleep length, weekend	.252	--																	
	Social jet lag	-.544**	.675**	--																
	Midpoint, week	-0.183	-.101	.030	--															
	Midpoint, weekend	-.309*	.102	.381**	.628**	--														
	Midpoint (weekend - week)	-.323*	.185	.487**	-.132	.689**	--													
	Circadian period	.023	-.023	-.053	-.060	.121	.129	--												
	Circadian mesor	-.070	-.067	-.110	.150	-.065	-.222	-.319*	--											
	Circadian amplitude	.208	.149	.018	.053	.024	-.014	.175	-.718**	--										
Follow-up	Sleep length, week	.691**	.335	-.209	-.137	-.085	.021	-.002	.046	-.055	--									
	Sleep length, weekend	.289	.441*	.211	.013	.165	.217	-.109	-.178	.200	.128	--								
	Social jet lag	-.109	.221	.305	.086	.196	.187	-.103	-.185	.208	-.419*	.847**	--							
	Midpoint, week	-.172	-.342	-.204	.679**	.461*	-.046	.072	.026	-.008	-.044	-.300	-.251	--						
	Midpoint, weekend	-.196	-.285	-.129	.615**	.493**	.063	-.190	-.060	.015	-.096	-.204	-.135	.843**	--					
	Midpoint (weekend - week)	-.056	.082	.125	-.071	.090	.197	-.459*	-.174	.046	-.096	.160	.198	-.237	.324	--				
	Circadian period	-.174	.037	.142	-.166	-.020	.151	.208	-.172	-.044	.200	-.125	-.227	.017	-.071	-.150	--			
	Circadian mesor	.046	-.169	-.193	.186	.222	.121	-.241	.522**	-.480**	-.086	.093	.132	.201	.147	-.091	.211	--		
	Circadian amplitude	.200	.198	.057	.099	-.057	-.181	.158	-.252	.505**	.576**	.153	-.151	-.037	-.035	.003	-.043	-.515**	--	

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Appendix 2



Appendix 2. Odds ratios with 95 % confidence intervals to have better sleep patterns and higher circadian amplitude. Value 1 marks odds ratios for reference (highest baseline tertile or male). Odds ratios that do not cover value 1 indicate significant difference between the reference and comparison.