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The effects of sleep extension and sleep hygiene advice on sleep and depressive symptoms in adolescents: a randomized controlled trial

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Objective: Sleep problems are common and persistent during adolescence and can have negative effects on adolescents' mood. To date, studies that investigate the effects of sleep extension on adolescents' sleep and depressive symptoms are still lacking. This study aims to investigate the effects of gradual sleep extension combined with sleep hygiene advice in adolescents with chronic sleep reduction on objectively measured sleep, self-reported sleep problems and depressive symptoms. **Methods:** Fifty-five adolescents with chronic sleep reduction (mean age: 15.44 years; 85.5% females) were included in the study. Participants were randomly assigned to either a sleep extension group (gradual sleep extension by advancing bedtimes in the evening and receiving sleep hygiene advice) or to a control group (no instruction). Sleep was measured with actigraphy during three weeks, the first week was the baseline week, and the last two weeks were the experimental weeks during which sleep was extended. Other outcome variables were self-reported sleep problems (daytime sleepiness, symptoms of insomnia and circadian rhythm sleep disorder) and depressive symptoms, which were assessed before and after the experimental manipulation. **Results:** During the third week of the experiment, adolescents in the sleep extension group had earlier bedtimes, earlier sleep onsets, spent more time in bed and slept longer than adolescents in the control group. Their chronic sleep reduction, insomnia symptoms and depressive symptoms diminished significantly. In addition, there was a trend of improved circadian rhythm sleep disorder symptoms and sleep quality. **Conclusion:** Gradual sleep extension combined with sleep hygiene advice seems to have beneficial effects on sleep, self-reported sleep problems and depressive symptoms of adolescents with chronic sleep reduction. Although we cannot distinguish between the effects of sleep extension and sleep hygiene advice, the results suggest that advancing bedtimes can extend sleep and improve depressive symptoms. **Keywords:** Adolescents, sleep, chronic sleep reduction, sleep extension, depression.

Introduction

Sleep problems, including insufficient and/or poor sleep, are common and persistent during adolescence (Gibson et al., 2006; Liu & Zhou, 2002; Pagel, Forister, & Kwiatkowki, 2007; Russo, Bruni, Lucidi, Ferri, & Violani, 2007). Although it has been demonstrated that adolescents report to require an average sleep time of approximately 9 hr/night (Mercer, Merritt, & Cowell, 1998), the majority of the adolescent population sleeps less (Mercer et al., 1998; Gibson et al., 2006). This was supported by a recent study showing a reduction in sleep during adolescence (Leger, Beck, Richard, & Godeau, 2012). Insufficient sleep in adolescents is often caused by an interaction of biological (e.g. puberty, circadian or homeostatic changes) and environmental factors (e.g. early school start times, social pressure, academic workload). Consequently, many adolescents sleep less than their individual sleep need over a long period of time, often causing chronic sleep reduction (Loessl et al., 2008; Meijer, 2008), which can result in severe daytime impairments (Curcio, Ferrara, & De Gennaro, 2006; Dewald, Meijer, Oort, Kerkhof, & Bögels, 2010; Fallone, Owens, & Deane, 2002; Wolfson & Carskadon, 2003). Sleep deprivation and sleep restriction studies in children and adults show that especially mood can be affected by reduced sleep (Dagys et al., 2012; Dinges et al., 1997; Gruber, Cassoff, Frenette, Wiebe, & Carrier, 2012). To date, a bidirectional link between sleep and depression has been suggested (Gregory & Sadeh, 2012). However, longitudinal studies indicate that insufficient sleep is a potential risk factor for the development of depressive symptoms, whereas less evidence was found for the opposite relationship (Clarke & Harvey, 2012; Gregory et al., 2005; Gregory, Rijsdijk, Lau, Dahl, & Eley, 2009; Meijer, Reitz, Dekovic, Van den Wittenboer, & Stoel, 2010). This finding is supported by recent results demonstrating that sleep extension significantly improved children's emotional lability, whereas the opposite was found for sleep restriction (Gruber et al., 2012).

Despite the existing evidence for the negative effects of sleep loss on daytime functioning and particularly mood, only few studies investigated possibilities to extend adolescents' sleep times. Beneficial effects on sleep (e.g. sleep duration, satisfaction with sleep) and daytime functioning (e.g.

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mood, school absenteeism, academic performance) are reported in a limited number of studies in which school start times were delayed, providing individuals with the opportunity to obtain more sleep (O'Malley & O'Malley, 2008; Owens, Belon, & Moss, 2010; Wahlstrom, 2002). Still, later school start times in high schools are often not supported by the society (Kirby, Maggi, & D'Angiulli, 2011).

An alternative approach to extend sleep is advancing bedtimes in the evening (Fallone, Acebo, Arnedt, Seifer, & Carscadon, 2001; Sadeh, Gruber, & Raviv, 2003). Although the results from these studies show rather small effects, sleep extension resulted in less fatigue, less sleepiness and better cognitive performance on some cognitive performance tasks. One reason for the small effects could be that participants were healthy children presumably obtaining sufficient sleep and extending their sleep would not necessarily improve their daytime functioning. Therefore, it can be expected that especially adolescents with chronic sleep reduction would benefit from extended sleep times (Loessl et al., 2008; Meijer, 2008). Furthermore, since earlier bedtimes and longer sleep times are associated with less depressive symptoms (Gangwisch et al., 2010), we assume that advancing bedtimes may not only improve sleep but also reduces depressive symptoms of adolescents with chronic sleep reduction.

As the circadian system can only change slowly, adolescents' sleep should be extended gradually. Moreover, many adolescents compensate insufficient sleep during the week by extending their sleep in the weekends, resulting in irregular sleep patterns and jet lag-like symptoms (Crowly & Carskadon, 2010; Dahl & Lewin, 2002). To overcome these adverse effects, this study combines gradual sleep extension during school nights with the prevention of bedtime shifts during weekends. In addition, we provided sleep hygiene advice with the aim to optimize adolescents' (sleep) environment and consequently helping participants to fall asleep.

Summarizing, we aim (a) to study whether adolescents suffering from chronic sleep reduction can extend their sleep time by advancing their bedtimes in combination with sleep hygiene advice, and (b) to investigate whether the experimental manipulation affects self-reported sleep problems (e.g. insomnia symptoms, sleep quality) and depressive symptoms.

Method

Participants

Participants were recruited from high schools in and around Amsterdam, the Netherlands. In total, 954 students were screened in a preceding survey. Adolescents were included in the experiment if their age was between 12 and 19 years and if they had a score of ≥40 on the Chronic Sleep Reduction Questionnaire (CSRQ; Meijer, 2008), which is equal to about one standard deviation above the mean and is considered to indicate high chronic sleep reduction (Dewald, Short, Gradisar, Oort, & Meijer, 2012). Previous research demonstrated that chronic sleep reduction measured with the CSRQ includes adolescents with insomnia (De Bruin, Oort, Bögels, & Meijer, 2013) and with Delayed Sleep Phase Syndrome (DSPS; Van Maanen, Dewald-Kaufmann, Smits, Oort, & Meijer, 2013) and is associated with sleep debt (Dewald et al., 2012). From the preceding survey 149 adolescents met the inclusion criteria and 60 adolescents agreed to participate. Two adolescents dropped out of the study during the experiment. We had to exclude three other individuals because of errors during data collection (technical failure, inconsistent data, not wearing the actigraph during the night). Data of 55 adolescents [mean age: 15.44 years (range from 12.76 to 18.52 years), 85.5% females] were analysed. All fathers (in 7.3% information was missing) and mothers (in 10.9% information was missing) were born in the Netherlands. In 83.6% of the families both parents were employed and in 16.4% only one parent was employed. More than half of the adolescents considered themselves as an evening chronotype (67.3%), 9.1% as a morning chronotype and 23.6% reported being something in between. Chronotype was measured with one question: 'Some people feel very active in the morning (extremely active in the morning), whereas others feel very active in the evening (extremely active in the evening). Somebody can also consider himself/herself to be a little bit more active in the morning than in the evening (moderate morning active) and somebody else may not feel different in the morning than in the evening. Please indicate your preference'. Participants could choose between five answer categories, ranging from extreme morning active to extreme evening active.

The two groups (sleep extension group: $n = 28$, control group: $n = 27$) did not differ significantly in age [$t(53) = -1.37$, $p = .18$], self-reported sleep need [$t(53) = .95$, $p = .35$] and chronotype [$t(53) = 1.87$, $p = .07$]. The proportion of boys was small in both groups (sleep extension group: $n = 5$; control group: $n = 3$), but not significantly different ($p = .37$, Fisher's exact test).

Procedure

Half of the data have been collected in spring 2011 and the other half in autumn 2011. The study was approved by the Ethical Committee of the Research Institute of Child Development and Education, University of Amsterdam. We obtained active informed consent from adolescents and parents.

Sleep was monitored during the experiment using actigraphy (see description under Measurements). In addition, adolescents completed online sleep diaries. The baseline week started on a Friday night. Adolescents' sleep diaries were daily checked and

participants were contacted by telephone when inconsistencies were observed or when they had not filled in their sleep dairy. During the baseline week participants filled in online questionnaires on sleep problems and depressive symptoms (pretest). After the baseline week participants were randomly assigned to the sleep extension group or to the control group (using the SPSS 'sample' procedure). A personal sleep schedule was sent to each participant in the sleep extension group on Friday, making sure that they received it on Saturday morning. In addition, the sleep schedule was sent to participants via e-mail. On Saturday all participants were called and their sleep schedule was individually explained over the telephone. After the baseline week, the first experimental week started on a Sunday night, however, to overcome weekend effects, participants in the sleep extension group were also asked not to sleep on Sunday morning. After the 3 weeks (1 baseline week and 2 experimental weeks), participants completed online questionnaires on sleep problems and depressive symptoms (posttest). All participants received a 30 Euro gift voucher and a summary of their actigraphy data of the baseline week. Schools, parents and participants received a summary of the study results. Figure 1 illustrates the design of the study.

Experimental manipulation

Sleep extension group. Participants received a personal sleep schedule in which bedtimes, light-off times and rise times were provided for each day. We used their mean bedtimes, light-off times and rise times as reported in the sleep diaries to calculate their starting bedtimes/light-off times. During the following school nights their bedtimes/light-off times were advanced by 5 min (gradual sleep extension) each day. The bedtime/light-off time for the first night was 10 min earlier than their mean bedtime/light-off time. In other words, at the end of the experiment adolescents' bedtimes/light-off times were 55 min earlier than their bedtimes/light-off times during the baseline week. Advancing sleep by approximately 1 hr was inspired by previous research (Gruber et al., 2012; Sadeh et al., 2003). Bedtimes/light-off times during the weekends were equal to the Friday night before the weekend and participants were allowed to delay their rise time by a maximum of 1 hr compared to their rise time on school nights. In addition, together with their individual sleep schedule, they received an overview of sleep hygiene rules, such as limiting the use of social media (e.g. 'do not play video games or watch television 1 hr prior to your bedtime'), consuming only limited drinks with caffeine (e.g. 'make sure that you don't drink coffee, tea, cola, or cocoa after 8 PM'), napping behaviour (e.g. 'don't nap during the day. In case you need to take a nap, make sure that it does not take longer than 30 min and don't nap 4 hr before your bedtime') and optimizing the sleep environment (e.g. temperature, light/dark, silence) ('e.g. make sure that you dim the light at least 1 hr prior to your bedtime. In the morning immediately open the curtains'). Providing these rules aimed at optimizing their (sleep) environment and consequently helping participants to fall asleep.

Control group. The control group did not receive any instructions about their sleep. Participants in the control group were simply asked to wear the actigraph and fill in the sleep diaries, just as during the baseline week.

Measurements

Chronic sleep reduction. Chronic sleep reduction was measured with the Chronic Sleep Reduction Questionnaire (CSRQ; Meijer, 2008), consisting of 20 items (e.g. 'I am a person who does not get enough sleep') which refer to the previous 2 weeks. Each question has three ordinal response categories, with higher scores indicating more chronic sleep reduction. Total scores can range from 20 to 60. The CSRQ

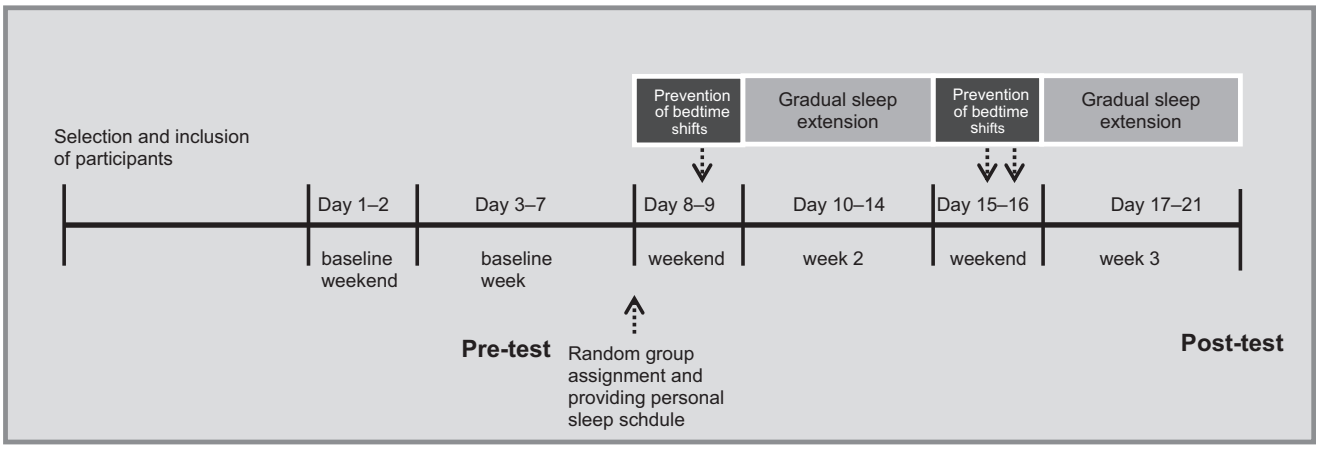

Figure 1 Graphic illustration of the experiment

appears to be a reliable and valid measurement for chronic sleep reduction (Dewald et al., 2012).

Sleep

Actigraphy. Participants' sleep was monitored using AW4 actiwatches (Cambridge Neurotechnology Ltd., Cambridge, UK). Actigraphy is known to be a reliable and valid measure to study sleep in a natural environment (Kushida et al., 2001; Morgenthaler et al., 2007). Participants were instructed to wear the actiwatch on their nondominant wrist when going to bed and to remove it after getting up. We assessed: (a) sleep onset latency (SOL): time between individuals' bedtime and sleep onset, (b) time in bed (TIB): time between participants' bedtime and rise time, (c) total sleep time (TST): number of minutes that individuals actually slept, (d) wake time after sleep onset (WASO): wake time between sleep onset and wake up time in the morning and (e) sleep efficiency (defined as 100 × TST/TIB): percentage of the time in bed which is spent asleep. Nocturnal activity data were logged at 1 min epochs and scored with the Actiwatch Sleep Analysis 7 software. As recommended by the manufacturer we used the medium sensitivity sleep algorithm which corresponds well with polysomnographic estimates (Morgenthaler et al., 2007).

Self-reported sleep problems

Daytime sleepiness. Daytime sleepiness was measured using a paediatric modification of the Epworth Sleepiness Scale (ESS; Johns, 1991), which consists of eight items that have to be rated on 4-point response scales. Total scores can range from 0 to 24, with higher scores indicating more daytime sleepiness. Participants are asked to rate how likely they are to doze in different situations (e.g. 'sitting and reading'; 'watching TV'). The last item 'in a car while stopped for a few minutes in traffic' was replaced with 'doing homework or taking a test' (Anderson, Storfer-Isser, Taylor, Rosen, & Redline, 2009). The cut-off score for clinical daytime sleepiness is highly discussed in the literature. Whereas originally a cut-off score of 10 has been suggested (Johns & Hocking, 1997; Johns, 2002), some research recommends to use a higher cut-off score of 11 or 12 (Vignatelli et al., 2003). Cronbach's alphas in this study were .84 and .82 at the pre- and posttest respectively.

Sleep quality. Sleep quality was assessed by a sleep quality questionnaire (Meijer & Van den Wittenboer, 2004) consisting of seven questions measuring problems with falling asleep, maintaining sleep, reinitiating sleep and waking up (e.g. 'I felt well rested when I woke up this morning'). Answers are rated on 3-point Likert scales. Total scores can range from 7 to 21, with higher scores indicating better sleep quality. Cronbach's alphas in this study were .77 and .75 at the pre- and posttest respectively.

Insomnia. Insomnia was measured with a subscale of the Holland Sleep Disorder Questionnaire (HSDQ; Kerkhof et al., 2012) measuring different sleep disorders. It consists of eight items (e.g. 'I feel fatigued during the day') with 5-point Likert scales. The total score is calculated using the mean score across the items. Therefore, the total score can range from 1 to 5, with higher scores indicating more severe insomnia symptoms. A score of 3.68 can be used as a clinical cut-off score. Cronbach's alphas in this study were .78 and .81 at the pre- and posttest respectively.

Circadian rhythm sleep disorder. Circadian rhythm sleep disorder was measured with a subscale of the HSDQ (Kerkhof et al., 2012) consisting of six items (e.g. 'I usually fall asleep in the morning hours) with 5-point Likert scales. The total score is calculated using the mean score across the items. Therefore, the total score can range from 1 to 5, with higher scores indicating more symptoms of a circadian rhythm sleep disorder. A score of 3.41 can be used as a clinical cut-off score. Cronbach's alphas in this study were .70 and .80 at the pre- and posttest respectively.

Depressive symptoms

Depressive symptoms. Depressive symptoms were assessed with the Dutch version of the 'Children's Depression Inventory' (CDI; Kovacs, 2002). The CDI includes 27 items, each consisting of three statements that are graded in severity (e.g. 'I am sad once in a while', 'I am sad many times', 'I am sad all the time'). The higher the assigned value (ranging from 0 to 2), the more severe the symptom is. The total score can range from 0 to 54. A cut-off score of 19 can be used to indicate clinical depression. Cronbach's alphas in this study were .72 and .77 at the pre- and posttest respectively.

Analyses

Effects on sleep (actigraphy). To examine changes in sleep we used linear mixed model analyses. The daily measured observations are considered as nested within subjects. As mixed model analyses allow inclusion of participants with incomplete data (Snijders & Bosker, 1999) all participants who provided baseline data (regardless of missing data at one or more assessment points) were included in the analyses. We fitted a model with a random intercept (to account for individual differences at baseline) and regression coefficients that represent deviations from baseline in the second and third week and in the weekends (representing time effects

during the three weeks of the experiment). To test whether the two groups varied in changes in sleep we added interaction effects (representing additional experimental effects in the sleep extension group). All analyses included age and season (spring vs. autumn) as control variables. As the number of boys was small in both groups gender was not included as control variable.

Effects on self-reported sleep problems and depressive symptoms (questionnaires). All outcome variables were transformed into standardized z-scores. To test changes in the outcome variables from the pre- to the posttest, we used linear mixed model analyses. The pretest was used as reference time point, meaning that regression coefficients represent deviations from the pretest. To test whether the two groups varied in changes on self-reported sleep problems and/or depressive symptoms we also added interaction effects. Again, age and season were included as control variables in all analyses.

Results

Effects on sleep (actigraphy)

Table 1 gives a descriptive overview of the sleep variables by providing means and standard deviations for the baseline week, the last week, the baseline weekend and the last weekend. Results from the linear mixed model analyses, testing changes and differences within and between groups, are presented in Table 2. The results show that during the baseline week, the sleep extension group and the control group did not differ on any of the sleep variables (see 'sleep extension group baseline week' in Table 2), and that there were no season effects, indicating that the experimental group and the control group started with similar sleep schedules, and that there were no significant differences between participants being tested in autumn and participants being tested in spring. Consistent with previous studies, older participants had later bedtimes, later sleep onset times, they woke and got up later and had shorter SOLs than younger participants (see Table 2). In comparison to the baseline week, bedtimes, sleep onset times, wake up times and rise times were delayed during the baseline weekend, resulting in longer TIBs and TSTs. Furthermore, SOLs were significantly shorter during the baseline weekend than during the baseline week.

Regarding our research question, we found between-group differences in changes in sleep (see Table 2, under 'additional experimental effects' in the sleep extension group). In the second and third week, participants in the sleep extension group had earlier bedtimes and, although their SOLs increased significantly also earlier sleep onset times (see

Table 1 Actigraphy in the first and last week of the experiment: means and standard deviations for the sleep extension and control group

	Sleep extension group				Control group			
	Baseline week	Week 3	Baseline weekend	Weekend 3	Baseline week	Week 3	Baseline weekend	Weekend 3
Bedtime (hr:min)	23:12 (00:46)	22:28 (00:52)	01:14 (02:10)	23:01 (01:01)	23:02 (00:50)	23:06 (00:45)	00:09 (00:52)	00:21 (01:32)
Sleep onset (hr:min)	23:28 (00:44)	22:59 (00:51)	01:26 (02:08)	23:20 (00:58)	23:27 (00:40)	23:35 (00:42)	00:29 (00:53)	00:42 (01:29)
Wake up time (hr:min)	7:41 (00:41)	7:27 (00:40)	9:53 (01:49)	8:20 (00:53)	7:35 (00:46)	7:37 (00:45)	09:19 (01:09)	09:48 (01:22)
Rise time (hrs:min)	7:46 (00:42)	7:28 (00:39)	9:56 (01:50)	8:25 (00:52)	7:39 (00:48)	7:38 (00:45)	09:25 (01:12)	09:54 (01:22)
Sleep onset latency (hrs:min)	00:16 (00:14)	00:31 (00:21)	00:12 (00:09)	00:19 (00:18)	00:24 (00:23)	00:29 (00:20)	00:21 (00:21)	00:22 (00:22)
Time in bed (hr:min)	8:33 (00:38)	9:03 (00:43)	8:42 (01:14)	9:24 (01:00)	8:36 (00:49)	8:34 (00:43)	09:16 (01:18)	09:33 (01:39)
Total sleep time (hr:min)	6:56 (00:32)	7:09 (00:36)	7:18 (01:08)	7:35 (00:55)	6:54 (00:45)	6:49 (00:42)	07:30 (01:07)	07:40 (01:20)
Wake time after sleep onset (hr:min)	1:18 (00:21)	1:19 (00:20)	1:09 (00:18)	1:25 (00:24)	1:15 (00:24)	1:14 (00:21)	01:20 (00:32)	01:26 (00:28)
Sleep efficiency (%)	81.24 (5.12)	79.15 (4.65)	83.75 (4.04)	80.64 (5.80)	80.61 (6.82)	79.70 (5.55)	81.01 (7.87)	80.35 (5.24)

The values are expressed as mean (*SD*).

Table 2 Effects of gradual sleep extension on sleep variables (actigraphy): results from the linear mixed model analyses

	Bedtime β (*SE*)	Sleep onset β (*SE*)	Wake up time β (*SE*)	Rise time β (*SE*)	Sleep onset latency β (*SE*)	Time in bed β (*SE*)	Total sleep time β (*SE*)	Wake time after sleep onset β (*SE*)	Sleep efficiency β (*SE*)
Time effects[a]									
Baseline week (intercept)	18.69 (1.13)	19.80 (1.11)	4.67 (.79)	4.74 (.81)	.97 (.31)	10.30 (.94)	8.22 (.89)	.90 (.59)	81.37 (8.45)
	$p < .01$	$p < .01$	$p < .01$	$p < .01$	$p = <.01$	$p < .01$	$p < .01$	$p = .13$	$p < .01$
Week 2	.18 (.07)	.08 (.08)	−.08 (.12)	−.08 (.12)	−.11 (.04)	−.34 (.13)	−.16 (.11)	−.05 (.04)	.61 (.56)
(vs. baseline week)	$p = .02$	$p = .33$	$p = .52$	$p = .51$	$p = .01$	$p = .01$	$p = .16$	$p = .24$	$p = .27$
Week 3	.15 (.08)	.22 (.09)	.05 (.12)	.04 (.12)	.06 (.05)	−.11 (.14)	−.15 (.11)	−.05 (.05)	−.90 (.62)
(vs. baseline week)	$p = .07$	$p = .02$	$p = .69$	$p = .73$	$p = .23$	$p = .42$	$p = .20$	$p = .31$	$p = .15$
Baseline weekend	1.10 (.22)	1.01 (.22)	1.93 (.23)	1.98 (.24)	−.09 (.05)	.76 (.23)	.66 (.20)	.12 (.06)	.78 (.71)
(vs. baseline week)	$p < .01$	$p < .01$	$p < .01$	$p < .01$	$p = .05$	$p < .01$	$p < .01$	$p = .07$	$p = .27$
Weekend 2	.25 (.30)	.25 (.30)	.27 (.31)	.28 (.31)	−.00 (.05)	−.11 (.37)	−.02 (.31)	−.04 (.09)	−.34 (.83)
(vs. baseline weekend)	$p = .40$	$p = .42$	$p = .38$	$p = .37$	$p = .97$	$p = .77$	$p = .95$	$p = .64$	$p = .68$
Weekend 3	.22 (.26)	.27 (.26)	.47 (.28)	.45 (.28)	−.01 (.06)	.39 (.27)	.20 (.24)	.10 (.08)	−.68 (.86)
(vs. baseline weekend)	$p = .39$	$p = .30$	$p = .10$	$p = .10$	$p = .89$	$p = .15$	$p = .40$	$p = .20$	$p = .43$
Additional experimental effects in the sleep extension group[b]									
Sleep extension group	.02 (.19)	−.11 (.19)	.07 (.17)	.08 (.18)	−.11 (.06)	.04 (.20)	.09 (.18)	.04 (.10)	.68 (1.40)
baseline week	$p = .93$	$p = .56$	$p = .69$	$p = .63$	$p = .08$	$p = .86$	$p = .60$	$p = .72$	$p = .63$
(vs. control group baseline week)									
Sleep extension group week 2	−.47 (.10)	−.29 (.12)	−.16 (.16)	−.18 (.16)	.18 (.06)	.39 (.18)	.22 (.16)	.01 (.06)	−.71 (.76)
(vs. control group week 2)	$p < .01$	$p = .01$	$p = .32$	$p = .27$	$p < .01$	$p = .03$	$p = .17$	$p = .83$	$p = .35$
Sleep extension group week 3	−.83 (.11)	−.62 (.12)	−.26 (.17)	−.25 (.17)	.17 (.07)	.60 (.20)	.38 (.16)	.04 (.07)	−1.32 (.85)
(vs. control group week 3)	$p < .01$	$p < .01$	$p = .12$	$p = .14$	$p = .02$	$p < .01$	$p = .02$	$p = .54$	$p = .12$
Sleep extension group	.78 (.32)	.80 (.32)	.09 (.33)	.03 (.34)	.03 (.06)	−.69 (.33)	−.40 (.29)	−.23 (.09)	1.16 (1.01)
baseline weekend	$p = .02$	$p = .02$	$p = 78$	$p = .92$	$p = .64$	$p = .04$	$p = .16$	$p = .01$	$p = .26$
(vs. control group baseline weekend)									
Sleep extension	−1.07 (.42)	−.96 (.42)	−.86 (.42)	−.81 (.43)	.04 (.07)	.40 (.50)	.06 (.43)	.16 (.12)	−1.31 (1.16)
group weekend 2	$p = .01$	$p = .03$	$p = .04$	$p = .06$	$p = .59$	$p = .43$	$p = .89$	$p = .16$	$p = .26$
(vs. control group weekend 2)									
Sleep extension	−2.17 (.36)	−2.09 (.37)	−1.65 (.39)	−1.61 (.39)	.08 (.09)	.52 (.38)	.24 (.34)	.17 (.11)	−1.84 (1.21)
group weekend 3	$p < .01$	$p < .01$	$p < .01$	$p < .01$	$p = .34$	$p = .17$	$p = .47$	$p = .13$	$p = .13$
(vs. control group weekend 3)									
Control variables									

(continued)

Table 2 (continued)

	Bedtime β (*SE*)	Sleep onset β (*SE*)	Wake up time β (*SE*)	Rise time β (*SE*)	Sleep onset latency β (*SE*)	Time in bed β (*SE*)	Total sleep time β (*SE*)	Wake time after sleep onset β (*SE*)	Sleep efficiency β (*SE*)
Age	.28 (.07) $p < .01$	.23 (.07) $p = <.01$	.18 (.05) $p < .01$	.18 (.05) $p = .01$	−.04 (.02) $p = .05$	−.11 (.06) $p = .07$	−.08 (.06) $p = .13$	.02 (.04) $p = .55$	−.03 (.53) $p = .95$
Season	.08 (.19) $p = .69$	.10 (.19) $p = .62$	−.02 (.13) $p = .88$	−.02 (.14) $p = .89$	−.02 (.05) $p = .67$	−.10 (.16) $p = .54$	−.08 (.15) $p = .61$	−.01 (.10) $p = .91$	−.56 (1.42) $p = .69$

We use 'bedtime' as an example to show how the parameter estimates translate to 'expected' bedtimes (expected according to the mixed model analysis). In the control group, the expected bedtime is 18.69 (intercept) + .28 × 15.44 (average age) = 23.01 hr. In Weeks 2 and 3 their expected bedtimes are .18 and .15 hr later, and in Weekends 1, 2 and 3, their expected bedtimes are 1.10, .25 and .22 hr later. So, for example, the expected bedtime in Week 3 is: 23.01 + .15 = 23.16 hr. In the experimental group, the expected bedtime in the baseline week is .02 hr later than for participants from the control group (23.01 + .02 = 23.03 hr). In Weeks 2 and 3, the expected bedtimes in the experimental group are .47 and .83 hr earlier than in the control group, and in the baseline weekend, the expected bedtime is .78 hr later than in the control group, but in Weekends 2 and 3 expected bedtimes are 1.07 and 2.17 hr earlier than in the control group. Finally, overall, expected bedtimes of spring participants are .08 hr later than expected bedtimes of autumn participants (see 'control variables'). We should note that the estimated means that can be calculated on the basis of the maximum likelihood estimates of the model parameters may deviate from the observed means in Table 1, owing to the fact that incomplete cases are weighted differently in maximum likelihood estimation of models that are not saturated.

[a]The time effects (representing time effects during the 3 weeks of the experiment) refer to both groups.

[b]For the sleep extension group the additional experimental effects have to be added.

Figure 2 Changes in bedtimes and sleep onset times for the sleep extension group and the control group separately

Table 2 and Figure 2). Adolescents in the sleep extension group also spent more time in bed and slept longer during the third week. Furthermore, participants in the sleep extension group went to bed earlier, fell asleep earlier and woke and got up earlier during the second and third weekend; however, these changes did not affect their TST during the second and third weekend. The two groups did not significantly differ in sleep efficiencies and WASO times.

Effects on sleep problems and depressive symptoms (questionnaires)

Table 3 gives means and standard deviations of self-reported sleep problems and depression at the pre- and the posttest for the two groups separately. Results from the linear mixed model analyses show that the two groups did not differ significantly from each other at the pretest (all $p > .05$). However, the group effect almost reached significance for daytime sleepiness, demonstrating that the sleep extension group experienced less daytime sleepiness at baseline than the control group ($\beta = -.48$, $SE = .25$, $p = .06$). Furthermore, no significant effects for age and season were found for any of the outcome variables (all $p > .05$). In comparison to the control group, in the sleep extension group chronic sleep reduction ($\beta = -.89$, $SE = .26$, $p < .01$), insomnia symptoms ($\beta = -.53$, $SE = .22$, $p = .02$) and depressive symptoms ($\beta = -.41$, $SE = .16$, $p = .01$) decreased significantly from the pre- to the posttest. Although not significant at the .05 level, we found a tendency that sleep quality ($\beta = .33$, $SE = .18$, $p = .06$) improved and that circadian rhythm sleep disorder symptoms decreased ($\beta = -.34$, $SE = .19$, $p = .08$). At the posttest the two groups did not differ

Table 3 Means and standard deviations of self-reported sleep problems and depressive symptoms for the sleep extension group and the control group

	Sleep extension group		Control group		Significance test[a]
	Pretest Mean (*SD*)	Posttest Mean (*SD*)	Pretest Mean (*SD*)	Posttest Mean (*SD*)	β (standard error), probability
Self-reported sleep problems					
Sleep quality	13.25 (3.39)	14.46 (2.55)	12.96 (2.65)	13.31 (2.54)	.33. (.18), $p = .06$
Chronic sleep reduction	41.88 (4.11)	37.04 (6.53)	42.21 (3.60)	41.69 (4.59)	−.89 (.26), $p < .01$
Insomnia	3.06 (.80)	2.78 (.73)	3.19 (.65)	3.12 (.78)	−.53 (.22), $p = .02$
Circadian rhythm sleep disorder	2.71 (.90)	2.36 (.79)	2.92 (.68)	2.81 (.98)	−.34 (.19), $p = .08$
Daytime sleepiness	9.04 (5.03)	7.50 (4.32)	10.92 (4.67)	10.42 (4.57)	−.22 (.17), $p = .21$
Depressive symptoms	12.17 (4.53)	11.18 (5.48)	12.04 (4.92)	12.81 (5.05)	−.41 (.16), $p = .01$

[a]These regression coefficients can be interpreted as standardized between-group difference in mean gain scores, with .20, 50 and 80 being considered small, medium and large effects (Cohen, 1988).

significantly on self-reported daytime sleepiness ($\beta = -.22$, $SE = .17$, $p = .21$). Regression coefficients can be interpreted as effect sizes with .20, .50 and .80 indicating small, medium and large effect sizes (Cohen, 1988), meaning that we found a large effect size for changes in chronic sleep reduction, a medium effect size for insomnia symptoms and small effect sizes for the other outcome variables.

Discussion

The present experimental study aimed to investigate the effects of gradual sleep extension in combination with sleep hygiene advice on adolescents' sleep, self-reported sleep problems and depressive symptoms. During the last week of the experiment, adolescents in the sleep extension group had earlier bedtimes, earlier sleep onset times, longer TIBs and longer TSTs than adolescents in the control group. The changes in bedtimes during school nights and during the weekends indicate that adolescents in the sleep extension group developed a more regular sleep schedule. Based on these results, it can be concluded that adolescents are capable to advance their bedtimes in the evening at least for a limited time period and thereby extend their sleep.

Adolescents in the sleep extension group slept on average about 13 min longer during the experimental week than during the baseline week. This is less than the half hour of additional sleep that has been reported by Sadeh et al. (2003). However, in opposite to the findings by Sadeh et al. (2003), we did not find negative effects on participants' sleep efficiency. Therefore, the results of this study clearly indicate that extending sleep gradually is important to extend sleep without negatively affecting other aspects of sleep. It also has to be mentioned that the average total sleep times cannot directly be compared to previous findings (Gruber et al., 2012; Sadeh et al., 2003) as participants in these studies had the opportunity to sleep about 1 hr longer for several days, whereas participants in this study reached that opportunity not before the last day of the experiment. It would be of interest to extend the study period after the third week, during which adolescents are asked to keep the sleep schedule of the last day; these data, once sleep extension was established, could then be compared with the findings by Gruber et al. (2012) and Sadeh et al. (2003).

Generally, a decrease in sleep efficiency can be expected when sleep is extended. Interestingly, this was not the case in our experimental group. This finding may be explained by the fact that the amount of extra sleep in the last week was almost equivalent to the extra SOL in the last week. It can therefore be concluded that, once fallen asleep, adolescents with chronic sleep reduction seem to sleep very well when given the opportunity to do so. This is also supported by the finding that WASOs did not change significantly during the experimental weeks. Therefore, it seems that this group tries to catch up for a sleep debt, which has been related to chronic sleep reduction (Dewald et al., 2012).

In the experimental group, adolescents' chronic sleep reduction and insomnia symptoms were significantly diminished at the posttest, showing that these adolescents experienced subjective improvements in sleep, which was not the case for adolescents in the control group. Furthermore, depressive symptoms decreased significantly in the experimental group but not in the control group. Concerning the ongoing debate about the bidirectional relationship between sleep and depression (Gregory & Sadeh, 2012), the results from this study support evidence from longitudinal research (Gregory et al., 2005, 2009; Meijer et al., 2010), indicating that improvements in sleep are associated with improvement in depressive symptoms, which is highly relevant for clinical practice. Furthermore, our results support the protective effect of earlier bedtimes for depressive symptoms (Gangwisch

et al., 2010). The finding that adolescents in the sleep extension group also reported better sleep quality and less circadian rhythm sleep disorder symptoms suggests that the experiment might have changed aspects of adolescents' circadian system, bringing it in better alignment with our society (e.g. school start times). However, it is important to notice that these effects did not reach significance at a .05 level and should therefore be carefully interpreted.

The experimental manipulation did not affect daytime sleepiness. The nonsignificant effect on sleepiness is in line with results, which show that self-reported sleepiness is not affected by longer sleep duration, which may be explained by limited awareness due to subjective measures such as self-reports (Vedaa, Saxvig, Wilhelmsen-Langeland, Bjorvatn, & Pallesen, 2011). In addition, Fallone et al. (2001) found similar subjective sleepiness ratings for a group with restricted sleep and a group with optimized sleep, whereas objective measures showed clear differences. Similarly, daytime sleepiness decreased significantly after sleep extension when measured by parent reports (Gruber et al., 2012). Taken together these results support the idea that self-reported sleepiness ratings may not be the most representative measure for the effects of changes in sleep. Carskadon and colleagues found in their famous summer camp study that adolescents are still sleepy during the day, even when they obtain optimal sleep during the night (described in Carskadon, 1999). This daytime sleepiness is thought to be due to rapid body and brain development that occur during adolescence.

In summary, it can be concluded that gradual sleep extension during school nights, combined with the prevention of bedtime shifts during weekends and sleep hygiene rules does not only improve adolescents sleep but also positively affects depressive symptoms.

Notwithstanding the strengths of the present experiment, such as the experimental manipulation in individuals' home environment and use of objective and subjective measures, some limitations have to be outlined: First, the posttest was conducted at the end of the experiment, but no follow-up measurement was included in the study design. Therefore, the question whether the achieved changes persist over a longer time period cannot be answered with this study. Such data are highly needed to answer the question whether adolescents are capable to fight their natural tendency of delaying bedtimes over a longer time period. Second, due to the small number of boys in the study we could not investigate gender effects. Third, because participants received specific instructions about their sleep and they were aware of the fact that their sleep was monitored, expectations and placebo effects could affect their behaviour and consequently the results. Fourth, although the sleep hygiene rules, which formed part of the intervention, were intended to improve adolescents' sleep environment and prevent negative effects of the advanced bedtimes, we cannot disentangle the effects of the personal sleep schedule and the sleep hygiene rules. More research on this topic is needed. As an aside we note that we could not check whether participants took notice and followed sleep hygiene rules. Fifth, future research should also address the underlying reasons for adolescents' chronic sleep reduction. This is especially important as it can be assumed that different groups of adolescents (e.g. chronic sleep reduction as a result of insomnia problems vs. behaviourally induced sleep debt) might respond to the intervention differently.

Conclusions

In conclusion, this study is the first experimental study showing that gradual sleep extension in combination with sleep hygiene advice has beneficial effects on sleep, self-reported sleep problems and depressive symptoms of adolescents with chronic sleep reduction. This finding has significant clinical implications as it may be an alternative to the delay of school start times as a means of providing adolescents with more opportunity for sleep. The approach presented here is, however, more in line with the time schedule and the demands of our society, which can be seen as an additional advantage. Furthermore, professionals should consider gradual sleep extension as a possible treatment method to improve depressive symptoms in adolescents with chronic sleep reduction.

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Key points

- Previous research has demonstrated a relationship between sleep and mood.
- This is the first randomized controlled trial that investigates the effect of sleep extension on adolescents' depressive symptoms.
- This experimental study demonstrates that sleep extension, by advancing bedtimes in the evening, combined with sleep hygiene advice, does not only improve adolescents' objectively measured sleep and self-reported sleep problems, but also diminishes their depressive symptoms.
- The finding that adolescents' sleep and depressive symptoms can be improved by advancing bedtimes has significant scientific and clinical implications as it may be an alternative to the delay of school start times.

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