Chronic sleep reduction in adolescents

Dewald, J.F.

Citation for published version (APA):
“The best cure for insomnia is to get a lot of sleep.”

-William Claude Fields-
Gradual sleep extension improves sleep and depressive symptoms in adolescents with chronic sleep reduction

Julia F. Dewald-Kaufmann¹, Frans J. Oort¹,², Anne Marie Meijer¹

¹ Research Institute of Child Development and Education, Faculty of Social and Behavioral Sciences, University of Amsterdam, Nieuwe Prinsengracht 130, 1018 VZ Amsterdam, The Netherlands

² Department of Medical Psychology, Academic Medical Centre, University of Amsterdam Meibergdreef 9, 1105 AZ Amsterdam, The Netherlands
Abstract

Objective: Sleep problems are common and persistent during adolescence and can have negative effects on adolescents’ mood. However, research investigating the effects of sleep extension on adolescents’ sleep and depressive symptoms is still lacking. The present study aims to investigate the effects of gradual sleep extension in adolescents with chronic sleep reduction on objectively measured sleep, self-reported sleep problems, and depressive symptoms.

Method: 55 adolescents with chronic sleep reduction (mean age: 15.44 years; 85.5% female) were included in the study. Participants were randomly assigned to either a sleep extension group (gradual sleep extension by advancing bedtimes in the evening) or to a control group (no instruction). Sleep was measured with actigraphy during three weeks, the first week was the baseline week, the last two weeks were the experimental weeks. Other outcome variables were self-reported sleep problems 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 and depressive symptoms diminished significantly. In addition, symptoms of circadian rhythm sleep disorder and sleep quality seemed to improve.

Conclusion: Gradual sleep extension has beneficial effects on sleep, self-reported sleep problems, and depressive symptoms in adolescents with chronic sleep reduction. The present approach has significant clinical and educational implications as it shows that advancing bedtimes can extend sleep and improve depressive symptoms.
7.1. Introduction

Sleep problems, including insufficient and/or poor sleep, are common and persistent during adolescence\(^1,2,3,4\). Many adolescents sleep less than their individual sleep need over a long period of time, often causing chronic sleep reduction\(^5,6\), which can result in severe daytime impairments\(^7-12\). Sleep deprivation studies show that especially mood can be affected by reduced sleep\(^13\). To date, a bidirectional link between sleep and depression has been reported\(^14\). However, longitudinal studies suggest that sleep problems are a potential risk factor for the development of depressive symptoms, whereas less evidence was found for the opposite relationship\(^15,16\).

Despite evidence for the negative effects of sleep problems, 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., 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\(^17-20\). Still, later school start times in high schools are often not supported by the society\(^21\).

An alternative approach to extend sleep is to advance bedtimes in the evening\(^22\). As the circadian system changes only 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 timings and jet-lag like symptoms\(^23\). To overcome these adverse effects, the present study combines gradual sleep extension during school nights with the prevention of bedtime shifts during weekends. As outlined above, many adolescents sleep less than their individual sleep need and consequently suffer from chronic sleep reduction\(^6\). It can therefore be expected that especially adolescents with chronic sleep reduction would benefit from extended sleep times. Furthermore, since earlier bedtimes and longer sleep times are associated with less depressive symptoms\(^24\), we assume that advancing bedtimes may improve sleep and reduce depressive symptoms of adolescents with chronic sleep reduction. Therefore, we aim (1) to study whether adolescents suffering from chronic sleep reduction are capable to extend their sleep by advancing their bedtimes, and (2) to investigate whether this approach affects sleep (e.g., total sleep time (TST)), self-reported sleep problems (e.g., insomnia symptoms, sleep quality), and depressive symptoms.
7.2. Method

7.2.1. Participants

60 adolescents agreed to participate, two adolescents dropped out of the study during the experiment. We excluded one individual because of a technical failure during the data collection and two because of unreliable data. Data of 55 adolescents (mean age: 15.44 years (range from 12.76 to 18.52 years), 85.5 % female) were analyzed. 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 type (67.3%), 9.1 % as a morning type, and 23.6% reported being something in between.

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 circadian preference (t(53) = 1.87, p = .07). Furthermore, the proportion of boys and girls was not significantly different in the two groups (p = .37, Fisher’s exact test), however, the number of boys was small in both groups (sleep extension group: n = 5; control group: n = 3).

7.2.2. Procedure

Half of the data were collected in spring 2011 and the other half in autumn 2011. Participants from a preceding survey 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)\textsuperscript{6}, which is considered to indicate high chronic sleep reduction\textsuperscript{25}.

We obtained active informed consent from adolescents and parents. Sleep was monitored during the experiment using actigraphy (see description under Measurements). Additionally, 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 diary. 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. A personal sleep schedule was sent to each participant in the sleep extension group and was individually explained over the telephone. The experimental week started on a Sunday night, however, in order to overcome weekend effects, participants in the sleep extension group were also asked not to sleep in on Sunday morning. After the three 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.

![Diagram](image-url)

**Figure 1.** Graphic illustration of the experiment

7.2.3. **Experimental manipulation**

7.2.3.1. **Sleep extension group**

Participants received a personal sleep schedule in which bedtimes, light-off times, and rise times were provided for each day. Bedtimes, light-off times, and rise times were based on their mean bedtimes, light-off times, and rise times that they reported during the baseline week. The bedtime/light-off time for the first night was 10 minutes earlier than their mean bedtime/light off time. Bedtimes/light-off times were advanced by five minutes (gradual sleep extension) each night. 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 one hour. Additionally, we provided an overview of sleep hygiene rules for the sleep extension group, which included limiting the use of social media, drinks with caffeine,
and napping behavior as well as optimizing the sleep environment (e.g., temperature, light/dark, silence).

7.2.3.2. Control group

The control group did not receive any instructions about their sleep.

7.2.4. Measurements

7.2.4.1. Chronic sleep reduction

Chronic sleep reduction was measured with the Chronic Sleep Reduction Questionnaire (CSRQ) consisting of 20 items (e.g., ‘I am a person who does not get enough sleep’), which refer to the previous two weeks. Each question has three ordinal response categories, with higher scores indicating more chronic sleep reduction. The CSRQ appears to be a reliable and valid measurement for chronic sleep reduction.

7.2.4.2. Sleep

7.2.4.2.1. 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. 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 x TST/TIB): percent of uninterrupted night sleep. Nocturnal activity data were logged at one minute 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.

7.2.4.3. Self-reported sleep problems

7.2.4.3.1. Daytime sleepiness

Daytime sleepiness was measured using a pediatric modification of the Epworth Sleepiness Scale (ESS) which consists of eight items. Participants 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’. 

136
Cronbach’s alphas in the present study were .84 and .82 at the pre- and posttest, respectively.

7.2.4.3.2. Sleep quality
Sleep quality was assessed with a sleep quality questionnaire 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 five-point Likert scales. Cronbach’s alphas in the present study were .77 and .75 at the pre- and posttest, respectively.

7.2.4.3.3. Insomnia
Insomnia was measured with a scale of the Holland Sleep Disorder Questionnaire (HSDQ) measuring different sleep disorders. It consists of seven items (e.g., ‘I feel fatigued during the day’) with five-point Likert scales. The item that also measures narcolepsy (‘During daytime I may perform ‘on the automatic pilot’, without any recollection of the event’) was excluded. Cronbach’s alphas in the present study were .72 and .79 at the pre- and posttest, respectively.

7.2.4.3.4. Circadian rhythm sleep disorder
Circadian rhythm sleep disorder was measured with a subscale of the HSDQ consisting of six items (e.g., ‘I usually fall asleep in the morning hours) with five-point Likert scales. Cronbach’s alphas in the present study were .70 and .80 at the pre- and posttest, respectively.

7.2.4.4. Depressive symptoms
Depressive symptoms were assessed with the Dutch version of the ‘Children’s Depression Inventory’ (CDI), which is based on the Beck Depression Inventory (BDI) for adults. 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. Cronbach’s alphas in this study were .72 and .77 at the pre- and posttest, respectively.
7.2.5. Analyses

7.2.5.1. Effects on sleep (actigraphy)

To examine changes in sleep and the effects of the experimental manipulation, 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, all participants that 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 versus autumn) as control variables. As the number of boys was rather small in both groups, gender was not included as control variable.

7.2.5.2. Effects on self-reported sleep problems and depressive symptoms (questionnaires)

All 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.

7.3. Results

7.3.1. Effects on sleep (actigraphy)

Table 1 gives means and standard deviations for the sleep variables for the baseline week, the last week, the baseline weekend, and the last weekend. Results from the linear mixed model analyses are presented in Table 2. The sleep extension group and the control group did not differ on any of the sleep variables during the baseline week. We did not find seasonal effects, indicating that the group being tested in autumn did not differ significantly on the sleep variables from the group being tested in spring. In comparison to younger participants, older participants had later bedtimes, later sleep onset times, they woke and got up later, and had shorter SOLs.

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.

Participants in the sleep extension group had earlier bedtimes and, although their SOLs increased significantly, also earlier sleep onset times during the second and third week than participants in the control group (see Table 2 and Figure 2). Therefore, adolescents in the sleep extension group also spent more time in bed and slept longer. 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. These changes indicate that their sleep schedule was advanced. The two groups did not differ significantly in sleep efficiencies and WASO times.

![Figure 2. Changes in bedtimes and sleep onset times for the sleep extension group and the control group separately](image-url)
Table 1. Means and standard deviations of sleep variables for the sleep extension and for the control group (actigraphy)

<table>
<thead>
<tr>
<th></th>
<th>Sleep extension group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline week</td>
<td>Week 3</td>
</tr>
<tr>
<td>Bedtime (hrs:min)</td>
<td>23:12 (00:46)</td>
<td>22:28 (00:52)</td>
</tr>
<tr>
<td>Sleep onset (hrs:min)</td>
<td>23:28 (00:44)</td>
<td>22:59 (00:51)</td>
</tr>
<tr>
<td>Wake up time (hrs:min)</td>
<td>7:41 (00:41)</td>
<td>7:27 (00:40)</td>
</tr>
<tr>
<td>Rise time (hrs:min)</td>
<td>7:46 (00:42)</td>
<td>7:28 (00:39)</td>
</tr>
<tr>
<td>Sleep onset latency (hrs:min)</td>
<td>00:16 (00:14)</td>
<td>00:31 (00:21)</td>
</tr>
<tr>
<td>Time in bed (hrs:min)</td>
<td>8:33 (00:38)</td>
<td>9:03 (00:43)</td>
</tr>
<tr>
<td>Total sleep time (hrs:min)</td>
<td>6:56 (00:32)</td>
<td>7:09 (00:36)</td>
</tr>
<tr>
<td>Wake time after sleep onset (hrs:min)</td>
<td>1:18 (00:21)</td>
<td>1:19 (00:20)</td>
</tr>
<tr>
<td>Sleep efficiency (%)</td>
<td>81.24 (5.12)</td>
<td>79.15 (4.65)</td>
</tr>
</tbody>
</table>
Table 2. Effects of gradual sleep extension on sleep variables (actigraphy)

<table>
<thead>
<tr>
<th>Time effects*</th>
<th>Bedtime</th>
<th>Sleep onset</th>
<th>Wake up time</th>
<th>Rise time</th>
<th>Sleep onset latency</th>
<th>Time in bed</th>
<th>Total sleep time</th>
<th>Wake time after sleep onset</th>
<th>Sleep efficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline week (intercept)</td>
<td>18.69 (.13) p &lt; .01</td>
<td>19.80 (.11) p &lt; .01</td>
<td>4.67 (.79) p &lt; .01</td>
<td>4.74 (.81) p &lt; .01</td>
<td>.97 (.31) p &lt; .01</td>
<td>10.30 (.94) p &lt; .01</td>
<td>8.22 (.89) p &lt; .01</td>
<td>.90 (.59) p = .13</td>
<td>81.37 (8.45) p &lt; .01</td>
</tr>
<tr>
<td>Week 2 (vs. baseline week)</td>
<td>.18 (.07) p = .02</td>
<td>.08 (.08) p = .03</td>
<td>-.08 (.12) p = .52</td>
<td>-.08 (.12) p = .51</td>
<td>-.11 (.04) p = .01</td>
<td>-.34 (.13) p = .01</td>
<td>-.16 (.11) p = .16</td>
<td>-.05 (.04) p = .24</td>
<td>.61 (.56) p = .27</td>
</tr>
<tr>
<td>Week 3 (vs. baseline week)</td>
<td>.15 (.08) p = .07</td>
<td>.22 (.09) p = .02</td>
<td>.05 (.12) p = .69</td>
<td>.04 (.12) p = .73</td>
<td>.06 (.05) p = .33</td>
<td>-.11 (.14) p = .42</td>
<td>-.15 (.11) p = .20</td>
<td>-.05 (.05) p = .31</td>
<td>-.90 (.62) p = .15</td>
</tr>
<tr>
<td>Baseline weekend (vs. baseline week)</td>
<td>1.10 (.22) p &lt; .01</td>
<td>1.01 (.22) p &lt; .01</td>
<td>1.93 (.23) p &lt; .01</td>
<td>1.98 (.24) p &lt; .01</td>
<td>-.09 (.05) p = .05</td>
<td>.76 (.23) p &lt; .01</td>
<td>.66 (.20) p &lt; .01</td>
<td>.12 (.06) p = .07</td>
<td>.78 (.71) p = .27</td>
</tr>
<tr>
<td>Weekend 2 (vs. baseline weekend)</td>
<td>.25 (.30) p = .40</td>
<td>.25 (.30) p = .40</td>
<td>.27 (.31) p = .38</td>
<td>.28 (.31) p = .37</td>
<td>-.00 (.05) p = .97</td>
<td>-.11 (.37) p = .77</td>
<td>-.02 (.31) p = .95</td>
<td>-.04 (.09) p = .64</td>
<td>-.34 (.83) p = .68</td>
</tr>
<tr>
<td>Weekend 3 (vs. baseline weekend)</td>
<td>.22 (.26) p = .39</td>
<td>.27 (.26) p = .30</td>
<td>.47 (.28) p = .10</td>
<td>.45 (.28) p = .10</td>
<td>-.01 (.06) p = .89</td>
<td>.39 (.27) p = .15</td>
<td>.20 (.24) p = .40</td>
<td>.10 (.08) p = .20</td>
<td>-.68 (.86) p = .43</td>
</tr>
</tbody>
</table>

Additional experimental effects in the sleep extension group

| Sleep extension group baseline week (vs. control group baseline week)         | .02 (.19) p = .93 | -.11 (.19) p = .56 | .07 (.17) p = .69 | .08 (.18) p = .63 | -.11 (.06) p = .08 | .04 (.20) p = .86 | .09 (.18) p = .60 | .04 (.10) p = .72 | .68 (.140) p = .63 |
| Sleep extension group week 2 (vs. control group week 2)                      | -.47 (.10) p < .01 | -.29 (.12) p = .01 | -.16 (.16) p = .32 | -.18 (.16) p = .27 | .18 (.06) p < .01 | .39 (.18) p = .03 | .22 (.16) p = .17 | .01 (.06) p = .83 | -.71 (.76) p = .35 |
| Sleep extension group week 3 (vs. control group week 3)                      | -.83 (.11) p < .01 | -.62 (.12) p < .01 | -.26 (.17) p = .12 | -.25 (.17) p = .14 | .17 (.07) p = .02 | .60 (.20) p < .01 | .38 (.16) p = .02 | .04 (.07) p = .54 | -.13 (.85) p = .12 |
| Sleep extension group baseline weekend (vs. control group baseline weekend) | .78 (.32) p = .02 | .80 (.32) p = .02 | .09 (.33) p = .78 | .03 (.34) p = .92 | .03 (.06) p = .64 | -.69 (.33) p = .04 | -.40 (.29) p = .16 | -.23 (.09) p = .01 | 1.16 (.101) p = .26 |
| Sleep extension group weekend 2 (vs. control group weekend 2)               | -.107 (.42) p = .01 | -.96 (.42) p = .03 | -.86 (.42) p = .04 | -.81 (.43) p = .06 | .04 (.07) p = .59 | .40 (.50) p = .43 | .06 (.43) p = .89 | .16 (.12) p = .16 | -.13 (.116) p = .26 |
| Sleep extension group weekend 3 (vs. control group weekend 3)               | -.217 (.36) p < .01 | -.209 (.37) p < .01 | -.165 (.39) p < .01 | -.161 (.39) p < .01 | -.08 (.09) p = .34 | .52 (.38) p = .17 | .24 (.34) p = .47 | .17 (.11) p = .13 | -.184 (.121) p = .13 |

Control variables

| 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 = .81 | -.01 (.10) p = .91 | -.56 (.142) p = .69 |

Note. * The time effects (changes in sleep during the three weeks of the experiment) refer to both groups. For the sleep extension group the additional experimental effects have to be added.
7.3.2. Effects on sleep problems and depressive symptoms (questionnaires)

Table 3 gives means and standard deviations 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, $p < .01$) and depressive symptoms ($\beta = -.41$, SE = .16, $p = .01$) decreased significantly from the pre- to the posttest. Additionally, we found effects on sleep quality ($\beta = .33$, SE = .18, $p = .06$) and circadian rhythm sleep disorder symptoms ($\beta = -.34$, SE = .19, $p = .08$), however, these effects were not significant at the .05 level. At the posttest the two groups did not differ significantly on self-reported daytime sleepiness and insomnia (all $p > .05$).

Regression coefficients can be interpreted as effect sizes with .20, .50, and .80, indicating small, medium, and large effect sizes.\(^{35}\)

<table>
<thead>
<tr>
<th></th>
<th>Sleep extension group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pretest</td>
<td>Posttest</td>
</tr>
<tr>
<td><strong>Self-reported sleep problems</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep quality</td>
<td>13.25 (3.39)</td>
<td>14.46 (2.55)</td>
</tr>
<tr>
<td>Chronic sleep reduction</td>
<td>41.88 (4.11)</td>
<td>37.04 (6.53)</td>
</tr>
<tr>
<td>Insomnia</td>
<td>3.20 (.78)</td>
<td>2.96 (.83)</td>
</tr>
<tr>
<td>Circadian rhythm sleep disorder</td>
<td>2.71 (.90)</td>
<td>2.36 (.79)</td>
</tr>
<tr>
<td>Daytime sleepiness</td>
<td>9.04 (5.03)</td>
<td>7.50 (4.32)</td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>12.17 (4.53)</td>
<td>11.18 (5.48)</td>
</tr>
</tbody>
</table>

7.4. Discussion

The present experimental study aimed to investigate the effects of gradual sleep extension on adolescents’ sleep, self-reported sleep problems, and depressive symptoms. During the third week, adolescents in the sleep extension group had earlier bedtimes, earlier sleep onset times, longer TIBs, and longer TSTs than during the baseline week. 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 and sleep onset times in the evening and thereby extend their sleep.

Adolescents’ chronic sleep reduction and depressive symptoms were significantly diminished at the posttest. Concerning the ongoing debate about the bi-directional relationship between sleep and depression[^14^], the results from this study support evidence from longitudinal research, indicating that improvements in sleep cause improvement in depressive symptoms, which is highly relevant for clinical practice. The finding that adolescents in the sleep extension group also reported better sleep quality and less circadian rhythm sleep disorder symptoms shows that the experiment did indeed change aspects of adolescents’ circadian system, bringing it in better alignment with our society (e.g., school start times). Consequently, 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.

The experimental manipulation did not affect daytime sleepiness and insomnia. The non-significant effect on sleepiness is in line with results showing 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[^36^]. It can be speculated that the absent effect of insomnia is caused by an interaction of the increased SOLs and the extended TSTs. In other words, adolescents slept longer and therefore should have improved on one insomnia symptom (obtaining insufficient sleep), however, at the same time another symptom, namely the time they needed to fall asleep, was negatively affected. Still, more research is needed to shed more light on this complex interplay between different insomnia 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 the present study. Second, we could not investigate gender effects due to the small number of boys in the study. Finally, although we provided sleep hygiene rules to the sleep extension group, we could not check whether participants followed these rules. These aspects should be examined in future research.
In conclusion, this study is the first experimental study showing that gradual sleep extension has beneficial effects on sleep, self-reported sleep problems, and depressive symptoms in adolescents with chronic sleep reduction. This finding has significant clinical and educational implications as it may be an attractive alternative to the delay of school start times. Furthermore, professionals should consider gradual sleep extension as a possible treatment method to improve depressive symptoms in adolescents with chronic sleep reduction.
7.5. References


