Original Article

The independent relationships between insomnia, depression, subtypes of anxiety, and chronotype during adolescence

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ABSTRACT

Objectives: To investigate the independent effects of depression and subtypes of anxiety on insomnia, and vice versa, and the independent effect of chronotype on insomnia, depression, and subtypes of anxiety.

Methods: In all, 318 South Australian high school students from grades 7–11 (age range, 12–18 years; mean, 14.97 ± 1.34) participated in this cross-sectional study. Validated self-report questionnaires were used to assess insomnia, depression, subtypes of anxiety, and chronotype.

Results: After confounder variables were controlled, insomnia predicted depression and panic disorder (PD), whereas insomnia was predicted by depression and generalized anxiety disorder (GAD). Obsessive–compulsive disorder (OCD), separation anxiety (SAD), and social phobia (SP) were not significantly related to insomnia. Eveningness predicted the models in which depression and PD predicted insomnia and vice versa. Evenness also predicted the models in which insomnia was predicted by OCD, SAD, and SP.

Conclusions: Insomnia independently predicts depression and is predicted by depression and GAD, but not by other forms of anxiety. The independent prediction of insomnia on PD is unlikely to be clinically significant. Chronotype independently predicts and hence is a risk factor for insomnia and depression, but not subtypes of anxiety. Theoretical and clinical implications are discussed.

1. Introduction

Insomnia is a widespread sleep problem in the general population, with a lifetime prevalence rate of ~11% in adolescents aged 13–16 years [1]. Recent studies have consistently found an association between insomnia and depression, and insomnia and anxiety disorders in adolescent populations [2,3], which is expected since the presence of insomnia is a possible criterion for the diagnosis of depression and various anxiety disorders, and since these disorders contain overlapping neurobiological, psychological, and social risk factors [4–11]. Insomnia comorbid with anxiety or depression can further intensify the problematic outcomes associated with each disorder, such as alcohol and drug misuse during adolescence [12].

Despite their well-documented association, various aspects and mechanisms of the relationships between insomnia and anxiety, and insomnia and depression are unclear. First, the significance of the relationship between insomnia and anxiety may differ across anxiety disorders. A recent study found an association between insomnia and separation anxiety disorder (SAD), and between insomnia and generalized anxiety disorder (GAD), but not between insomnia and social anxiety disorder [13]. Indeed, some associations between sleep difficulties and anxiety disorders (GAD) are expected given the symptom overlap for diagnosis. Another study found that youths with GAD reported higher rates of trouble sleeping overall than youths with SAD, social phobia (SP), and obsessive–compulsive disorder (OCD) [14]. Second, the independent effects of insomnia on anxiety or depression, and vice versa, remain unclear. Anxiety may account for some or all of the relationship between insomnia and depression, whereas depression may account for some or all of the relationship between insomnia and anxiety due to the common underlying neurobiological, psychological, and social factors mentioned above. Finally, chronotype, defined as an individual’s natural inclination for mornings (morningness) or evenings (eveningness), could uniquely predict insomnia, anxiety and depression. Recent studies suggest that an eveningness chronotype predicts higher levels of insomnia, anxiety, and depression during adolescence [15–18]. These studies, however, have yet to demonstrate whether eveningness predicts insomnia beyond depression and anxiety, anxiety beyond insomnia and depression, or depression beyond insomnia and anxiety. Such findings would suggest that eveningness is an
independent risk factor for insomnia, anxiety and depression. These findings would be particularly important, because adolescents tend to develop a preference for evenings due to circadian rhythm and environmental changes [19], which sometimes develops into delayed sleep phase syndrome [20]. This syndrome affects ~8% of adolescents [21], and has been associated with insomnia, anxiety, and depression [22].

This study, therefore, adds to the previous literature by investigating in one study the independent effect of depression and various types of anxiety on insomnia, and vice versa, along with the effect of chronotype on insomnia, depression, and subtypes of anxiety that is independent from other predictors. The anxiety subtypes assessed in the current study include GAD, OCD, panic disorder (PD), SAD and SP. It was hypothesized that insomnia would be independently related to some mental health problems, for example to depression, GAD and SAD, but not to SP. It was also hypothesized that chronotype would independently predict insomnia beyond depression and anxiety, depression beyond insomnia and anxiety, and subtypes of anxiety beyond insomnia and depression. The independent effect was defined as the amount of variance of a dependent variable ($Y$) that is explained by an independent variable ($X$) after controlling for confounder variables ($C_i$). Insomnia, anxiety subtypes, and depression were used as predictor and outcome variables, as recent studies have suggested that insomnia, anxiety, and depression are bidirectionally related [23].

Understanding the pathways of the relationship between insomnia and anxiety subtypes, and between insomnia and depression can inform public health campaigns and clinical interventions for each disorder, and also enhance the understanding of the interaction between these disorders.

2. Methods

2.1. Participants

Three-hundred and eighteen South Australian secondary school students aged 12 – 18 (mean, 14.96; standard deviation, 1.34) participated in the study. Of these, 164 (51.6%) students were male and 154 (48.4%) were female. Nine students were in grade 7, 102 were in grade 8, 66 in grade 9, 52 in grade 10, and 89 in grade 11. The study was voluntary, and required student and parental consent for participation. Participants were eligible for this study if their parents consented, were in grades 7 – 11, and were fluent in English. This study was approved by the Human Research Ethics Committee (HREC) from the University of Adelaide, the Department for Education and Child Development (DECD), and Catholic Education South Australia (CESA).

2.2. Measurements

A questionnaire composed of several inventories was used to assess adolescent sleep and mental health. Demographic questions included date of birth, gender, and socio-economic status as assessed by post code [24]. Personal questions such as previous sleep or mental health problems, previous therapy for sleep or mental health problems, previous or current disabilities/chronic illnesses (e.g. asthma, diabetes, deafness, etc.), current medications that may affect sleep or mental health, and the frequency of drug and alcohol consumption were adaptations from the School Sleep Habits Survey (SSHS) [25]. Total sleep time, bed time, and rise time on weekdays and weekends were also reported, the latter two of which were used to calculate total bed time. These questions were asked to give an account of the general characteristics of the sample.

Insomnia was measured by the Insomnia Severity Index (ISI) [26], a seven-item inventory that assesses the severity of subjective symptoms and consequences of insomnia based on the DSM-IV [27]. Each item was scored on a 0 – 4 Likert scale. Total scores were calculated by the sum of each item and ranged from 0 to 28. Higher scores indicate more severe insomnia. Morin [26] provides a scoring guideline: 0 – 7, no clinically significant insomnia; 8 – 14, sub-threshold insomnia; 15 – 21, moderate clinical insomnia; 22 – 28, severe clinical insomnia. The symptoms assessed included difficulty falling sleep, difficulty staying asleep, and problems waking up too early, while the consequences assessed included impaired quality of life, worry/distress about current sleep patterns, and perceived interference of sleep problems with daily life. Although designed for adults, the ISI has been widely used in the adolescent population [28 – 30]. Furthermore, a recent study has validated the ISI in an adolescent population, reporting significant correlations with clinical ratings of insomnia, the Sleep–Wake Habits Questionnaire, General Mental-Health Questionnaire, Epworth Sleepiness Scale, smoking habits, alcohol use, number of naps per week, and academic performance [31]. Cronbach’s $\alpha$ = 0.83, and a two-week test–retest reliability of 0.79 for the ISI in an adolescent population [31] were also reported.

The Morningness–Eveningness Scale for Children (MESC) [32], a 10-item adaptation of the Composite Scale of Morningness [33], was used to assess adolescents’ orientation towards morning and evening chronotypes. Seven items are scored on a Likert scale from 1 to 4, and three items are scored on a Likert scale from 1 to 3. Total scores are calculated by the sum of each item and range from 10 to 42. Lower scores indicate a tendency towards eveningness. The MESC successfully discriminates between morningness and eveningness in adolescents [34]. In accordance with previous research [16,18], evening and morning types were defined as below the 10th and above the 90th percentile, respectively, and scores in between were identified as neither type (cut-off scores were <20 for eveningness and >33 for morningness). Previous studies have reported good internal consistency, with Cronbach’s $\alpha$ of 0.73 [16], 0.82 [34], and 0.82 [35] in Italian, Spanish, and Australian adolescents, respectively. Good test–retest reliability (0.78) [36] and external validity have been reported [34], and the MESC has been shown to predict daytime functioning, academic achievement, and various behavioral outcomes in adolescents [16,35]. Two items were altered to keep the language consistent with the Australian population and the age of the sample. First, the phrase ‘gym class’ was changed to ‘sports class’. Second, the item ‘Your parents have decided to let you set your own bed time. What time would you pick?’ was changed to ‘What time would you prefer to go to bed?’

Adolescents who met the following criteria were deemed likely to suffer from delayed sleep phase syndrome [1,37]; a minimum of 1 h shift in bed and rise times from weekdays to the weekend; moderate, severe or very severe complaints of difficulty falling asleep; no or mild complaint of difficulty maintaining sleep; and not at all easy to wake up in the morning.

Subtypes of anxiety and depression were assessed by the Revised Child Anxiety Depression Scale (RCADS) [38], a 47-item self-report questionnaire that is an adaption of the Spence Children’s Anxiety Scale [39] and corresponds to diagnostic categories of DSM-IV. Each item is scored on a 0 – 3 Likert scale, with higher scores corresponding to more severe depression or anxiety. Sub-scales are provided for GAD (six items, scores ranging from 0 to 18), PD (nine items, scores ranging from 0 to 27), OCD (six items, scores ranging from 0 to 18), SAD (seven items, scores ranging from 0 to 21), SP (nine items, scores ranging from 0 to 27), and major depressive disorder (MDD; 10 items, scores ranging from 0 to 30). An overall scale for anxiety (37 items, scores ranging from 0 to 111) is also provided. Scales are calculated by the sum of each item. The RCADS users guide (available on the University of California, Los Angeles website [40]) converted raw scores into standardized T scores: 65 – 69 indicates borderline clinical threshold; $\geq$ 70...
indicates above clinical threshold. A recent study reported the following Cronbach’s α-values in Hawaiian adolescents: SP, 0.81; PD, 0.85; GAD, 0.80; MDD, 0.76; SAD, 0.78; OCD, 0.71 [38]. The same study provided strong support for the structural, convergent, and discriminant validity of the RCADS. The RCADS has also been validated in Australian adolescents; De Ross et al. [41] provided support for the internal consistency for MDD, anxiety overall, and anxiety subscales. Good convergent validity was also demonstrated [41] with moderate to strong correlations between the subscales of RCADS with scores on the Revised Manifest Anxiety Scale (RCMAS) [42] and the Children’s Depression Inventory (CDI) [43]. Indeed, the RCADS has been extensively used in the adolescent population [44,45], including in studies that have assessed the relationship between sleep problems, anxiety, and depression [46].

3. Results

3.1. Descriptive statistics

Tables 1–3 report descriptive statistics for demographic information, sleep and mental health variables, and frequencies of clinically significant and sub-threshold mental health and insomnia cases. Approximately 25% of adolescents reported suffering from past sleep or mental health problems, whereas ~17% reported previous treatment for sleep or mental health problems. Approximately 20% reported other medical issues, whereas 14% reported previous or ongoing treatment for such issues. Insomnia was the most frequently identified clinically significant problem (11.19%), whereas MDD was the most common mental health problem (8.39%).

Correlations are reported in Table 4. ISI was moderately to highly correlated with each subscale from the RCADS. The results also depicted small to medium correlations between chronotype and each subscale from the RCADS, except for GAD, where no significant correlation was found. The difference between total sleep time on weekends and weekdays was significantly correlated with an eveningness chronotype, insomnia, depression, PD, OCD, and SP.

Generalized estimation equations were run with and without participants who were identified with delayed sleep phase syndrome tendencies [complete data available for 300 participants, n = 18 (6%)]. No differences were found, and therefore all available data were retained.

3.2. Depression and insomnia

The depression scale contained an item similar to insomnia, which was thought to possibly inflate the regression coefficients of the relationship between insomnia and depression. The overall pattern of results remained the same when analyses were conducted with and without the insomnia-based MDD item. Therefore, the original MDD scale was retained.

The analyses of depression and insomnia included 302 participants. Depression had a significant independent effect on insomnia (β = 0.526; 95% CI, 0.406–0.645) and vice versa (β = 0.3767; 95% CI, 0.276–0.477). Chronotype and anxiety uniquely predicted insomnia (chronotype β = −0.210; 95% CI, −0.306 to −0.113; anxiety β = 0.040; 95% CI, 0.0017–0.063) and depression (chronotype β = −0.103; 95% CI, −0.169 to −0.036; anxiety β = 0.167; 95% CI, 0.137–0.196).

3.3. GAD and insomnia

A total of 307 participants was included in the analyses for the relationship between GAD and insomnia. Chronotype and GAD were not associated before potential confounders were controlled. Therefore, chronotype was not used to predict GAD.

Significant independent effects were found when GAD predicted insomnia (β = 0.258; 95% CI, 0.025–0.336) but not when insomnia predicted GAD (β = 0.116; 95% CI, −0.005 to 0.236). Chronotype and depression predicted insomnia (chronotype β = −0.227; 95% CI, 0.330 to −0.124; depression β = 0.522; 95% CI, 0.385–0.659), and depression predicted GAD (depression β = 0.245; 95% CI, 0.158–0.345).
The analyses for the relationship between OCD and insomnia included 303 participants. A significant independent effect was not found when OCD predicted insomnia (95% CI, −0.117 to 0.080) nor when insomnia predicted OCD (95% CI, −0.050 to 0.034); Depression and chronotype predicted insomnia (depression $\beta = 0.168$; 95% CI, 0.499–0.738; chronotype $\beta = −0.194$; 95% CI, −0.286 to −0.103); whereas depression but not chronotype uniquely predicted OCD (depression $\beta = 0.400$; 95% CI, 0.351–0.449; chronotype 95% CI, −0.041 to 0.096).

### 3.4. OCD and insomnia

The analyses for the relationship between OCD and insomnia included 303 participants. A significant independent effect was not found when OCD predicted insomnia (95% CI, −0.117 to 0.080) nor when insomnia predicted OCD (95% CI, −0.050 to 0.034); Depression and chronotype predicted insomnia (depression $\beta = 0.168$; 95% CI, 0.499–0.738; chronotype $\beta = −0.194$; 95% CI, −0.286 to −0.103); whereas depression but not chronotype uniquely predicted OCD (depression $\beta = 0.400$; 95% CI, 0.351–0.449; chronotype 95% CI, −0.041 to 0.096).

### 3.5. Panic disorder and insomnia

The sample size for the analyses of the relationship between PD and insomnia was 304. An independent significant effect was found when insomnia predicted PD (independent $\beta = 0.064$; 95% CI, 0.007–0.121) but not when PD predicted insomnia (95% CI, −0.003 to 0.201). Depression and chronotype predicted insomnia (depression $\beta = 0.562$; 95% CI, 0.463–0.661; chronotype $\beta = −0.199$; 95% CI, −0.290 to −0.108); depression, not chronotype, predicted PD (depression $\beta = 0.464$; 95% CI, 0.395–0.532; chronotype 95% CI, −0.010 to −0.081).

### 3.6. Separation anxiety and insomnia

In all 303 participants were included for the analyses of the relationship between SAD and insomnia. A significant independent effect was not found when SAD predicted insomnia (95% CI, −0.028 to 0.276), nor when insomnia predicted SAD (95% CI, −0.016 to 0.113). Depression and chronotype predicted insomnia (depression $\beta = 0.581$; 95% CI, 0.479–0.684; chronotype $\beta = −0.200$; 95% CI, −0.294 to −0.106), whereas depression but not chronotype uniquely predicted SAD (depression $\beta = 0.239$; 95% CI, 0.161–0.318; chronotype 95% CI, −0.058 to 0.061).

### 3.7. Social phobia and insomnia

The sample size for the analyses of the relationship between SP and insomnia was 304. The results failed to show a significant independent effect when SP predicted insomnia (95% CI, −0.048 to 0.143), and vice versa (95% CI, −0.065 to 0.191). Depression and chronotype uniquely predicted insomnia (depression $\beta = 0.578$; 95% CI, 0.481–0.676; chronotype $\beta = −0.197$; 95% CI, −0.285 to −0.109); whereas depression but not chronotype predicted SP (depression $\beta = 0.663$; 95% CI, 0.522–0.804; chronotype 95% CI, −0.051 to 0.168).

### 4. Discussion

The first aim of the study was to investigate the independent relationship between insomnia and depression, and insomnia and different subtypes of anxiety. The results were consistent with previous adolescent studies that reported a positive association between insomnia and depression [2,3], insomnia and GAD, but not between insomnia and SP [13]. This study, however, adds to these findings by showing that insomnia is related to depression and GAD after chronotype and anxiety or depression (respectively) are controlled. Higher levels of insomnia were significantly predicted by higher levels of depression, and, conversely, higher levels of insomnia significantly predicted higher levels of depression and GAD.

The relationships observed between insomnia, GAD, and depression may be at least partially explained by abnormalities in neurotransmitters and brain structures such as dopamine, hypocretin-1, serotonin, the brainstem, and thalamus, which are associated with the sleep–wake cycle, anxiety, and depression [4–9]. Consequently, insomnia, anxiety, and depression may have overlapping courses of development, and hence contribute to the development of and result from one another [11,49,50]. Psychological and social factors that are common during adolescence such as increased autonomy, and psychosocial (eg, peer groups), familial, and educational stressors [10,11] may also predispose adolescents to the development of insomnia, anxiety, and depression.

Whereas associations between insomnia, OCD, SAD, and SP were evident, the relationships were no longer significant when depressive symptoms were controlled for. In contrast, a previous study reported an association between insomnia and SP [13], and suggested that depression has a large yet partial mediation effect.
Table 1
Demographic and personal history.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self-report of past mental health symptoms</td>
<td>Depression</td>
<td>22</td>
<td>6.9</td>
</tr>
<tr>
<td></td>
<td>Anxiety</td>
<td>21</td>
<td>6.6</td>
</tr>
<tr>
<td></td>
<td>Sleep problems</td>
<td>25</td>
<td>7.9</td>
</tr>
<tr>
<td></td>
<td>Depression, anxiety</td>
<td>4</td>
<td>1.3</td>
</tr>
<tr>
<td></td>
<td>Depression, anxiety and insomnia</td>
<td>5</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>8</td>
<td>2.5</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>233</td>
<td>73.3</td>
</tr>
<tr>
<td>Self-report of past treatment for symptoms of depression, anxiety, sleep problems</td>
<td>Psychologist</td>
<td>11</td>
<td>3.5</td>
</tr>
</tbody>
</table>

Table 2
Descriptive statistics for sleep and mental health variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Missing data</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insomnia</td>
<td>312</td>
<td>6</td>
<td>7.63</td>
<td>5.28</td>
<td>0–25</td>
</tr>
<tr>
<td>Major depressive disorder</td>
<td>314</td>
<td>4</td>
<td>7.62</td>
<td>4.69</td>
<td>0–28</td>
</tr>
<tr>
<td>Generalized anxiety disorder</td>
<td>314</td>
<td>4</td>
<td>5.63</td>
<td>3.20</td>
<td>0–16</td>
</tr>
<tr>
<td>Obsessive-compulsive disorder</td>
<td>313</td>
<td>5</td>
<td>4.36</td>
<td>3.01</td>
<td>0–17</td>
</tr>
<tr>
<td>Panic disorder</td>
<td>314</td>
<td>4</td>
<td>6.55</td>
<td>4.37</td>
<td>0–24</td>
</tr>
<tr>
<td>Separation anxiety</td>
<td>314</td>
<td>4</td>
<td>3.68</td>
<td>3.15</td>
<td>0–14</td>
</tr>
<tr>
<td>Social phobia</td>
<td>314</td>
<td>4</td>
<td>6.69</td>
<td>4.40</td>
<td>0–25</td>
</tr>
<tr>
<td>Morningness–eveningness</td>
<td>315</td>
<td>3</td>
<td>27.17</td>
<td>5.41</td>
<td>10–40</td>
</tr>
<tr>
<td>Total sleep time weekday</td>
<td>313</td>
<td>5</td>
<td>8.31</td>
<td>1.27</td>
<td>4–11.5</td>
</tr>
<tr>
<td>Total sleep time weekend</td>
<td>314</td>
<td>4</td>
<td>9.43</td>
<td>1.72</td>
<td>4.5–15</td>
</tr>
<tr>
<td>Total time in bed weekday</td>
<td>316</td>
<td>2</td>
<td>8.70</td>
<td>1.10</td>
<td>4–11.5</td>
</tr>
<tr>
<td>Total time in bed weekend</td>
<td>311</td>
<td>7</td>
<td>9.67</td>
<td>1.48</td>
<td>3.5–13.5</td>
</tr>
</tbody>
</table>

ADHD, attention deficit/hyperactivity disorder.

The independent effect of chronotype on insomnia and depression may be explained by symptoms that are common to depression, insomnia, and an evening preference during adolescence such as persistent sleep deprivation, sleep displacement, difficulty adjusting to social constraints and an alternating lifestyle are also related to poor sleep and mental health [16]. CarSkadon et al. [53] and Kaneita et al. [11] suggested that daily pressures when SP predicts insomnia [51]. These studies used different methodologies; the items used in the current study, as opposed to the other [51], are based on DSM-IV [52] criteria and hence may better represent the symptoms that are assessed for a clinical diagnosis of insomnia, depression, and subtypes of anxiety. Furthermore, the previous study used an overall sleep construct that assessed insomnia via one item [13], whereas the current study used a validated seven-item instrument that is specific to insomnia. An explanation for the current study’s findings could be that symptoms of depression and GAD may lead to significant and persistent distress and cognitive arousal at night, whereas symptoms of PD, SAD, and SP are triggered by particular stimuli that may not be present nocturnally [52].

The findings from this study also suggest that depression has a stronger role in either the development or maintenance of sleep and anxiety symptoms than insomnia. Depression, not insomnia (or chronotype), predicted each anxiety variable. Furthermore, insomnia predicted each anxiety subtype before but not after depression had been entered into the models, suggesting that depression may explain the relationship between insomnia and anxiety subtypes. Nevertheless, this study improves upon current clinical theories by indicating that insomnia has a stronger relationship with depression than anxiety subtypes, that insomnia is independently related to some but not other anxiety subtypes, and that depression may be a mediating factor between insomnia and subtypes of anxiety. Such findings, paired with the high correlations between insomnia and all anxiety subtypes before covariates were controlled, further consolidate the notion of a complex and intertwined relationship between insomnia, anxiety, and depression [49].
of life accumulate during adolescence and hence promote later bed times, poorer sleep, and more mental health problems in older adolescents. Furthermore, Russo et al. [18] found that later bed and rise times occur with older age on the weekends, indicating that rise times are dictated by school schedule. Consequently, total sleep time on school nights decreases with age but remains constant on the weekend, suggesting that sleep deprivation may occur during the week [18]. The results of this study showed a similar pattern.

The current study has public health and clinical implications. Given that depression and GAD independently predict insomnia, prevention and treatment plans for insomnia may also focus more broadly on depression and GAD. Similarly, since insomnia independently predicts depression, prevention and treatment plans for depression might also consistently focus on improving sleep. Eaton et al. [54] estimated that 47% of cases of depression could have been prevented had sleep problems been successfully treated one year previously. Furthermore, Ohayon and Roth [55] found that insomnia was a precursor for relapse of anxiety and depression. Further, since chronotype and anxiety predicted insomnia and depression, prevention and treatment plans for depression and insomnia could simultaneously focus on anxiety and the evening-ness chronotype. Finally, given the importance of depression, prevention and treatment plans for various subtypes of anxiety disorders should also consider depression. Therefore, interventions that focus on mental health, sleep, and circadian rhythms might prevent the development or help alleviate symptoms of insomnia, depression, and the subtypes of anxiety.

The current study contained some limitations. First, directionality could not be inferred due to the cross-sectional methodology used, and knowledge about the etiological aspect of the relationship between these problems remains unclear. This is particularly relevant to the finding that GAD predicted insomnia, but that insomnia did not predict depression. From such a finding an association may only be inferred, and longitudinal studies are needed for a clearer understanding of the direction of this relationship. Future studies might therefore assess the longitudinal relationships between insomnia and subtypes of anxiety, and between insomnia and depression after accounting for potential covariates, thereby expanding the current knowledge base of the direction and hence etiological relationship between these problems.

Second, this study is based on self-reported sleep and mental health symptoms rather than methods allowing a clinical diagnosis. Regarding mental health problems, comprehensive interviews are required to detect the presence of other disorders that may confound the results, such as adjustment or conduct disorder. Nevertheless, the RCADS has been extensively used in the adolescent population [44,45], including in studies that have assessed the relationship between sleep problems, anxiety, and depression [46]. Furthermore, recent studies have shown inconsistencies between subjective and objective reports of poor sleep in pediatric populations diagnosed with MDD [56,57] and anxiety disorders [57]. The same studies also showed that subjective and objective reports of sleep were more similar in the general population than in youths diagnosed with anxiety or depression [56,57]. Indeed, the current study assessed the general population, in which prevalence rates of clinically significant insomnia, depression, and SP were similar to those found in other studies on the general population [2,58,59]. The RCADS and ISI are also measures of mental health disorders and insomnia based on the DSM-IV diagnostic criteria [52]. Future studies could use objective measures and/or clinical methods of assessment for insomnia, depression, and subtypes of anxiety.

Moreover, other sleep problems were not assessed. However, disorders such as obstructive sleep apnoea are unlikely to confound the results due to the relatively low prevalence rates (range, from 0.4% [60] to 2.9% [61]) in pediatric populations. In any case, future studies could use polysomnography and sleep diaries to detect other sleep disorders, and might even assess biological measures that are directly relevant to chronotype and therefore might better detect delayed sleep phase syndrome such as core body temperature and dim-light melatonin onset.

5. Conclusions

This study adds to the current literature by assessing the independent relationship between insomnia and depression, and between insomnia and anxiety across subtypes of anxiety, while investigating the effects of chronotype on insomnia, anxiety, and depression beyond potential confounders. The general adolescent population was the targeted sample, and psychometrically sound
measures based on the DSM-IV [52] criteria for insomnia, subtypes of anxiety and depression were used. The results suggested that insomnia is independently related to symptoms of depression and GAD, but not to the other subtypes of anxiety. Furthermore, an evening preference uniquely predicted and hence may be an independent risk factor for insomnia and depression, but not GAD, PD, OCD, SAD, or SP. Prevention and treatment efforts for insomnia and depression should potentially consider and concurrently focus on mental health, sleep, and the eveningness chronotype, whereas prevention and treatment efforts for anxiety subtypes may consider also focusing on insomnia and depression.

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Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2014.03.019.

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