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Original Article

Clinical characterization of insomnia in adolescents – an integrated approach to psychopathology

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ABSTRACT

Objectives/Background: Insomnia in adolescence is common and debilitating yet it remains poorly understood. Here, we examine the complexity of clinical, behavioral, and psychosocial factors characterizing insomnia in adolescents.

Methods: Ninety-five adolescents (16–19 years) with (N = 47, 31 female) and without (N = 48, 28 female) insomnia symptoms participated. In the insomnia group, 26 (20 female) met full DSM-5 criteria for insomnia disorder, while 21 (11 female) met partial criteria. Participants completed a clinical interview and assessments of clinical, behavioral, and psychosocial dimensions associated with insomnia. GLMs and network analyses were used to evaluate group and sex differences in severity and inter-relationships between symptoms.

Results: Adolescents with insomnia symptomatology had lower sleep hygiene and thought control, more depressive symptoms and dysfunctional sleep-related cognitions, and more substance use as a coping behavior than healthy controls. They also indicated higher neuroticism, stress levels, and sleep stress reactivity ($p < 0.05$), but no difference in adverse childhood experiences, than controls. Girls compared to boys with insomnia reported lower sleep quality, and more pre-sleep cognitive activity and sleep stress reactivity ($p < 0.05$). Compared to healthy girls, girls in the insomnia group reported lower sleep hygiene and higher agreeableness. Network analyses confirmed profound group differences in network topology, with the insomnia group having different levels of centrality and relationships between clinical characteristics compared with controls.

Conclusions: Findings highlight clinical and sex-specific characteristics of adolescent insomnia, with network analyses revealing a complex interplay between clinical, behavioral, and psychosocial domains. Adolescents with insomnia symptoms, particularly girls, may benefit from interventions to improve negative cognition, mood, and stress, and behavioral strategies to counteract sleep-related maladaptive behaviors.

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1. Introduction

Insomnia disorder frequently emerges during adolescence, following different trajectories in boys and girls [1,2], toward a higher prevalence in female adolescents after menarche (risk ratio of 1.41) [1,3]. Insomnia in adolescence tends to be chronic [4,5] and increases risk for adverse health outcomes including substance use and severe medical and psychiatric conditions [5]. Despite being the most prevalent sleep disorder in the teenage years [2,5], affecting up to 23% [1,2] of adolescents, insomnia in adolescents is still under-diagnosed, under-treated, and poorly characterized [6,7]. Part of the challenge lies in the heterogeneity of the disorder [8], requiring the investigation and interpretation of the multidimensionality of insomnia symptomatology.

Work mostly done in adult populations has shown that insomnia is characterized by a range of clinical, behavioral, and psychosocial aspects, which may play a role in its onset and development [9–11]. For example, personality characteristics such as neuroticism have been frequently associated with sleep problems [12] and insomnia

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disorder [13] in adults. Adult insomnia is also frequently accompanied by depressed mood [14,15], and psychophysiological arousal [16], characterized for example by uncontrollable thoughts at bedtime [17]. It is also associated by increased stress levels and an altered stress reactivity [18,19] as well as childhood trauma [20].

Insomnia can have its roots in adolescence [6], emerging on the top of biopsychosocial changes accompanying this vulnerable life stage, which include profound maturation in sleep and circadian processes [6,21]. Although reasons are not yet fully understood, research suggests a number of factors associated with the increased susceptibility to insomnia in adolescence, including changes in the homeostatic sleep pressure during wakefulness and in the endogenous circadian oscillator, resulting in a reduction in sleep drive and delayed sleep/wake rhythms [22]. Adolescents have substantial school obligations, including homework load, as well as several sport and social activities, including social media use and other screen time activities, which can interfere with sleep timing. They also have constraints, notably early school start times, which conflicts with a progressive developmental delay in bedtimes, further interfering with adolescents' sleep times [6]. Notably, adolescence is characterized by unique yet intense stresses relating to school, peers, parenting, and future uncertainty [23]. Stress and the vulnerability to stress are key factors associated with the development of insomnia [18], and school stress has been specifically linked with insomnia disorder in adolescents [5]. Whether individuals with insomnia perceive similar stressors as more stressful than their healthy counterparts [24] or they adopt different coping strategies, has yet to be determined.

Current research suggests that insomnia in adolescents tends to have similar physiological and psychosocial vulnerability factors as in adults. In a recent review, Blake and colleagues [22] highlighted associations between psychological (eg, negative attributional styles, worry, or dysfunctional beliefs and attitudes about sleep) and social (eg, impaired social interactions or family stress) factors that may increase the vulnerability to insomnia disorder in teens. Among the few studies investigating potential alterations in physiological domains of arousal in adolescents with insomnia, current research suggests that similar to adults, insomnia in adolescence is characterized by the presence of higher cortical high-frequency EEG activity [25], cognitive and emotional arousal [26] and stress [27]. Personality factors associated with poor sleep in adults have also been reported in adolescents. For example, high levels of neuroticism were associated with later bedtimes and shorter sleep duration, as well as sleep difficulties in a sample of 183 adolescents [28].

Overall, compared to the adult literature, features of insomnia in adolescents have been less well studied, and frequently investigated as isolated clinical aspects of the disorder, resulting in a poor characterization of the disorder in adolescent populations. More research is needed to provide a broader and integrative picture of various clinical, behavioral, and psychosocial aspects of the disorder as well as their interplay in the context of adolescence. Only recently have network approaches emerged as useful and well-established components for studying psychological constructs and their interrelationships in health psychology [29–31], providing a beneficial complement or alternative to more traditional methods of analysis with the potential to uncover hidden factors in complex symptom networks. They offer integrative approaches to study the structure of psychopathological pathways underlying mental disorders [31], and reveal symptom dynamics not visible with conventional methods. In the current study we comprehensively investigated clinical (nocturnal and diurnal clinical manifestations), behavioral (mood, stress, or personality related factors), and psychosocial (environmental factors including academic and social obligations) features of insomnia in a sample of older adolescents. To explore the relationships between these symptoms in more detail and to examine group differences in the interconnections between multi-symptoms, we also adopted a network approach to specifically examine group differences in network topology (eg, centrality measures).

2. Methods

2.1. Sample

Ninety-five participants (16–19 years old; 59 female) in a study evaluating insomnia, sleep, and cardiovascular health in adolescence (R01HL139652), formed the final sample. The study was approved by Advarra International Institutional Review Board. All adults consented to participate; minor participants provided written assent along with consent from a parent/legal guardian.

All participants were 10–12th grade high-school students. They were recruited from the San Francisco Bay Area high schools and communities based on the following criteria: free from severe current mental (eg, Major Depressive Disorder, Post-Traumatic Stress Disorder) and medical conditions (eg, Heart Diseases, Diabetes, Seizures), and no current use of medications known to affect sleep (eg, hypnotics) and/or the cardiovascular system (eg, antihypertensives). Further exclusion criteria were time-zone travel within the past month or pregnancy (females). All participants were post-pubertal as determined by the pubertal development scale (PDS) [32], and 16 females were taking hormonal birth control or fertility medications. While there is a strong female-prevalence of insomnia in adolescence, recruitment efforts were targeted towards equal distribution of sexes across groups.

All participants underwent a personal structured clinical interview [33] with a trained CA licensed clinical psychologist (DP), including a detailed sleep assessment for DSM-5 insomnia disorder, accounting for adolescent-specific characteristics (eg, shifted and erratic sleep and wake timing, sleep opportunities) in the clinical assessment of the disorder (see Refs. [6,7]). Twenty-six participants (20 female) met DSM-5 clinical criteria: (i) significant difficulty initiating sleep, and/or (ii) significant difficulty maintaining sleep, and/or (iii) waking too early and having difficulty returning to sleep, on at least 3 nights per week, with a duration of 3 months or more despite adequate opportunity to sleep, along with significant distress or impairment (DSM 5 Insomnia Disorder criterion B: *The sleep disturbance causes clinically significant distress or impairment in social, occupational, educational, academic, behavioral, or other important areas of functioning*). An additional 21 participants (11 female) who met all DSM-5 criteria (all reporting significant clinically relevant distress and impairment associated with their insomnia) but were below the DSM-5 threshold for insomnia duration and/or frequency were also included in the insomnia group (insomnia). In total, 47 participants (31 female) were included in the insomnia group. Forty-eight participants (28 female) without insomnia symptoms were included in the healthy control group (control).

None of the participants suffered from sleep disorders (sleep-disordered breathing, leg-movement disorders) other than insomnia as evaluated by the clinical assessment. This was also confirmed by a laboratory clinical polysomnographic evaluation in a subgroup (N = 63) of the sample performed before the COVID-19 pandemic-related home-stay regimen.

Of the initial sample recruited (N = 122), 27 participants were excluded because of the following reasons: non-compliance, medical problems including current or history of psychiatric disorders, severe current medical conditions affecting brain function or behavior including diabetes, cancer, neurological diseases (eg, seizure disorders) recurrent migraine, cardiovascular diseases (eg,

Hypertension) and traumatic brain injury with loss of consciousness >30 min, and due to loss to follow-up because of the start of COVID-19 quarantine and isolation measures, resulting in a final sample of N = 95 participants. A comprehensive dropout scheme is provided in Supplementary Fig. 1.

2.2. Procedures

All participants completed a phone screen, to be informed about the study and to assess their general eligibility, and a face-to-face visit to undergo a detailed clinical evaluation. Sixty-three of the participants completed the clinical assessment via an in-person visit (between 01.04.2019 and 03.04.2020) at SRI International. Due to the COVID-19 pandemic stay-home policy implementation, thirty-two participants completed the clinical assessment via remote face-to-face clinical interviews via secure "Zoom" platform (Zoom Video Communications, Inc.) between 07.15.2020 and 02.01.2021.

2.3. Questionnaire battery

All adolescents completed standardized validated questionnaires including measures of insomnia symptoms, sleep quality and habits, sleep-related cognitions, stress and stress reactivity, mood, personality & childhood trauma, coping and emotion-regulation behavior. All questionnaires were delivered, and data collected, via a secure REDCap web-based electronic platform.

2.3.1. Insomnia symptoms, sleep quality and habits

Insomnia symptoms (past 2 weeks) were assessed using the insomnia severity index (ISI) [34], a validated instrument to determine insomnia severity with higher scores indicating higher insomnia severity. Habitual sleep (past month) was measured using the Pittsburgh sleep quality index (PSQI) [35], which is a reliable self-rating questionnaire to assess sleep quality and disturbances (eg, I wake up in the middle of the night or early morning). Higher scores indicated more sleep disturbances. The morningness–eveningness questionnaire (MEQ) [36] was included to evaluate circadian typology (eg, alertness and activeness in the morning vs. evening times), with higher scores indicating a higher alertness or activeness in the morning, and lower scores indicating a higher alertness or activeness in the evening. The questionnaire did not refer to a specific time, but rather, to the 'last few weeks'. Participants also completed the adolescent sleep hygiene scale (ASHS) [37] to assess common sleep habits (eg, routines before bed or typical sleeping conditions) in the past month. This scale contains 28 items summarized in 9 categories, with higher scores indicating a better sleep hygiene: physiological domain (eg, I go to bed feeling hungry), cognitive domain (eg, I go to bed and think about things I need to do), emotional domain (eg, I go to bed feeling upset), sleep environment (eg, I fall asleep while watching television), daytime sleep (eg, During the day, I take a nap that lasts >1 h), substances (eg, After 6 pm, I drink beer, or other drinks with alcohol), bedtime routine (eg, I use a bedtime routine), sleep stability (eg, On weekends, I stay up >1 h past my usual bedtime), and bed/bedroom sharing (eg, I sleep alone). Participants completed the Cleveland Adolescent Sleepiness Questionnaire (CASQ) [38] to determine their daytime sleepiness and alertness 'during a usual week,' with higher scores indicating higher levels of daytime sleepiness or alertness.

2.3.2. Sleep-related cognitions

While some of the above-mentioned questionnaires included subscales related to cognitive sleep-related cognitions (eg, ASHS), several additional instruments were administered to specifically assess sleep-related cognition and cognitive processes. To determine pre-sleep cognitive activity (past 7 nights), all adolescents completed the Glasgow content of thoughts inventory (GCTI) [39]. The GCTI is comprised of 25 items that determine thoughts that keep one awake before sleeping, divided into 5 subscales: Thoughts about sleep related anxiety (eg, *how bad you are at sleeping*), thoughts about reflection and planning (eg, *things you have to do tomorrow*), thoughts about general worries (eg, *your health*), thoughts about the environment (eg, *noises you hear*), and thoughts about negative affect (eg, *how frustrated/annoyed you feel*). A higher score indicated more disturbing thoughts at bedtime. Negative attitudes about sleep (eg *worry about the next night's sleep*) were assessed using the dysfunctional beliefs and attitudes about sleep scale (DBAS-16) [40], which is a reliable instrument to determine insomnia-related cognitions including dysfunctional expectations or appraisals about sleep. Higher scores indicated more negative attitudes about sleep. The questionnaire did not refer to a specific time window in the past, but to general attitudes. Strategies to control negative thoughts before sleep were assessed with the revised version of the thought control questionnaire for insomnia (TCQI-R) [17]. The TCQI-R is comprised of 35 items and 6 subscales: aggressive suppression (eg, *I punish myself for thinking the thought*), cognitive distraction (eg, *I think about something else instead*), reappraisal (eg, *I analyze the thought rationally*), behavioral distraction (eg, *I occupy myself with work instead*), social avoidance (eg, *I avoid discussing the thought*), and worry (eg, *I focus on different negative thoughts*). Higher scores indicated a more frequent use of each strategy to control negative thoughts before sleep. The questionnaire did not refer to a specific time window in the past, but to general attitudes.

2.3.3. Stress & stress reactivity

Perceived stress levels (past month) were assessed using the perceived stress scale (PSS) [41], with higher scores indicating higher subjective stress levels. Common adolescent-specific stressors (past year) were measured using the adolescent stress questionnaire (ASQ) [23], which is comprised of 58 items and 10 subscales (stress of home life, school performance, school attendance, romantic relationships, peer pressure, teacher interaction, future uncertainty, school/leisure conflict, financial pressure, and emerging adult responsibility). Items included adolescent-specific stressful situations such as *not being taken seriously*, *keeping up with school work*, or *being hassled for not fitting in*. Higher scores indicated higher stress levels in each domain. Additionally, participants completed the perceived stress reactivity scale (PSRS) [42] to assesses their reactivity to different stressful conditions. The PSRS includes 23 items and five subscales (prolonged reactivity, reactivity to work overload, reactivity to social conflict, reactivity to failure, and reactivity to social evaluation) with higher scores indicating higher perceived stress for each category. Items assessed reactions to stressful situations in the past (no specific time frame) (eg, *When I make a mistake: a. In general, I remain confident, b. I sometimes feel unsure about my abilities, c. I often have doubts about my ability*). The Ford insomnia response to stress test (FIRST) [43] was used to assess how likely participants are to experience sleep issues after a stressful situation (no specific time frame) (eg, *after an argument, after getting bad news during the day*), with higher scores indicating a higher likelihood of experiencing sleep issues.

2.3.4. Mood

All participants completed the Beck Depression Inventory (BDI-II) [44], which is a validated instrument to assess depressive symptoms (past 2 weeks). The BDI includes items on common feelings of depression, for example feelings of guilt, failure, or suicidal thoughts, with higher scores indicating higher depression

symptoms. The State-Trait Anxiety Inventory (STAI-Y2) [45] was administered to determine trait anxiety levels (no specific time frame). Items were assessed as a self-report on a 4-point scale (eg, *I feel nervous and restless*) with higher scores indicating more anxiety symptoms.

2.3.5. Personality

The NEO Five-Factor Inventory (NEO-FFI) [46] was used to determine five personality traits, neuroticism, extraversion, openness, agreeableness, and conscientiousness, with higher scores indicating a higher degree of expression in the respective domain (no specific time frame).

2.3.6. Childhood trauma

The Adverse Childhood Experiences (ACE) [47] questionnaire was administered to determine negative childhood experiences such as abuse and neglect. Items refer to the first 18 years of life and were self-rated as *yes* or *no* (eg, *Did you often feel that you didn't have enough to eat, had to wear dirty clothes, and had no one to protect you?*). If participants answered "yes" to any of the items, a clinician was involved to assess acute abuse in the family.

2.3.7. Coping & emotion-regulation behavior

The COPE inventory (COPE) [48] was used to assess adolescent's coping mechanisms to potentially stressful conditions on 15 subscales: Positive reinterpretation and growth, mental disengagement, focus on & venting of emotions, seeking of instrumental social support, active coping, denial, turning to religion, humor, behavioral disengagement, restraint coping, seeking of emotional social support, substance use, acceptance, suppression of competing activities, and planning. The items assessed reactions when individuals faced difficult or stressful situations and were self-rated on a 4-point scale. Higher scores indicate a higher usage of each coping mechanism. The questionnaire did not refer to a specific time window in the past, but to general coping mechanisms. Emotional regulation (no specific time frame) was assessed with the Difficulties in Emotion Regulation Scale (DERS) [49]. The scale is comprised of 36 items that are summarized in a total score and 6 subscales (nonacceptance of emotional responses, difficulties engaging in goal-directed behaviors, impulse control difficulties, lack of emotional awareness, limited access to emotion regulation strategies, and lack of emotional clarity). Participants were advised to self-rate how often the statements (eg, *When I'm upset, I become out of control*) apply to them. Higher scores indicate a worse emotional regulation.

2.4. Statistical analyses

For the analyses, statistical outliers were defined as those exceeding 4 standard deviations from the mean, in each of the dependent variables. Participants meeting this criterion were excluded from analyses of the (sub)scales "substance use" (ASHS, $n = 2$), "bedroom sharing (ASHS, $n = 2$), "stress of emerging adult responsibility" (ASQ, $n = 1$), "stress of financial pressure" (ASQ, $n = 1$), "turning to religion" (COPE, $n = 1$), "impulse or control difficulties" (DERS, $n = 1$), and for the ACE total score ($n = 1$).

To analyze frequency distribution for sex, race and ethnicity, a Chi-Square test was used. Generalized linear models (GLM, R version 3.6.2, lmer package) were performed to analyze group (insomnia, control) and sex (boys, girls) differences within the sample. To control for the influence of the COVID-19 pandemic, reflected in the differences in onsite (pre-COVID)-versus-remote (after the COVID-related stay-home policy implementation) data collection, 'remote data collection' was included as a covariate in the statistical model. For categorical outcome variables, generalized mixed-effect models with Poisson distribution were employed, and similar factors and covariates were entered in the statistical models. Significance level was set to 0.05.

To investigate the relationship among clinical, behavioral, and psychosocial aspects of insomnia, a network analysis approach was used. Individual's characteristics (total scores for each of the measures outlined in the *Questionnaire battery* section) were used to represent nodes of the network, with edges indicating the relationships among them. After standardization and outlier detection (values falling outside 4 standard deviation from the mean were replaced by the group-level mean; two values for "adverse childhood experiences" and "dysfunctional beliefs and attitudes about sleep" were replaced), significant ($p < 0.05$) partial correlations (based on Spearman's rho $-\ \rho\ -$ the significant correlations between pairs of symptoms that remain when all other symptoms are controlled for) were used to estimate the network, as suggested by others [31]. Partial correlation can encode the basic network structure, "the causal skeleton" excluding the spurious correlations that might be the result of a common effect or chain structures within the network. To calculate pairwise correlations, we used a non-regularized method from the Python package Pingouin [50]. Python's NetworkX package [51] was used to estimate the weighted $-$ undirected concentration graph [52]. NetworkX was also used to compute the metrics of nodes and create the graphs. While both the negative and positive correlations were included in the network construction, we used the absolute ρ values to calculate the node positions in the network visualization. Self-loops, ie, edges connecting a node to itself, were removed from the networks. The position of the nodes in the network is based on the Fruchterman–Reingold algorithm [53], which calculates the optimal layout so that strongly correlated symptoms cluster in the middle, whereas symptoms with weaker connections to other symptoms are placed further apart.

To examine the connectivity of the networks, we applied the Girvan–Newman hierarchical community detection method [54] (available in NetworkX), which is based on centrality notions of the network and is widely used for "community" detection in social and biological graphs [55].

To visualize group differences in network topology, we compared the network structure of the control group with that of the insomnia group by projecting the clusters identified in the control network on to the insomnia network. The following centrality measures were computed for both insomnia and control participants: (1) Degree centrality, which measures the number of edges attached to a node. It is used to determine what nodes are most connected. In this study, degree centrality of a given node is computed as the fraction of nodes connected to it. It is normalized by dividing by the maximum possible degree; (2) Betweenness centrality, which measures the number of times that a given node is part of the shortest path between two other nodes. It can be used to find nodes that act as a bridge between different parts of the network. In this study, betweenness centrality of a given node is computed as the sum of the fraction of all pairs of shortest paths that pass through it. The value is normalized by the maximum possible value, ie, the maximum number of shortest paths of the complete graph; (3) Closeness centrality, which measures the mean distance from one node to any other node. The more central a node is, the closer it is to all the other nodes. Here we refer to it in its normalized form.

The data that support the findings of this study are openly available in [56].

3. Results

3.1. Sample demographics

Sample demographics are presented in Table 1. Adolescents in the insomnia group were significantly (mean discrepancy = 0.49 years) older than adolescents in the control group ($p < 0.05$). There were more adolescents with a Hispanic decent in the control group (10.64% in insomnia versus 27.08% in controls).

3.2. Clinical, behavioral, and psychosocial aspects

Mean and standard errors for each of the questionnaires, and the significant models and factors are provided in Table 2. Interaction effects are presented in Fig. 1, and group effects are presented in Fig. 2.

3.2.1. Group differences

In contrast to healthy controls, adolescents in the insomnia group experienced: more depressive symptoms (BDI, $p < 0.01$); higher levels of neuroticism (NEO, $p < 0.05$) and openness (NEO, $p < 0.05$); higher insomnia severity scores (ISI, $p < 0.01$); poorer sleep quality (PSQI, $p < 0.01$); higher sleep stress reactivity (FIRST, $p < 0.01$); more daytime alertness (CASQ, $p < 0.01$) and daytime sleepiness (CASQ, $p < 0.05$) and lower sleep hygiene (ASHS total score, $p < 0.01$), particularly characterized by a worse sleep stability; and higher cognitive and emotional arousal (ASHS, $p < 0.01$). Adolescents in the insomnia group reported more dysfunctional beliefs and attitudes about sleep (DABS, $p < 0.01$); and more disturbing cognitive activity before sleep than adolescents in the control group (GCTI total score, $p < 0.01$). Disturbing cognitive activity before sleep focused on thoughts about the environment ($p < 0.01$), sleep-related anxiety ($p < 0.01$), general worries ($p < 0.01$), reflection and planning ($p < 0.01$), and negative affect ($p < 0.01$). Additionally, adolescents in the insomnia group reported using more strategies to control these negative thoughts in the night (TCQI total score, $p < 0.01$), especially by using thought management attempts like reappraisal (eg, *analyzing the thought rationally*) or social avoidance (eg, *keeping thoughts to themselves*) ($p < 0.01$). Adolescents in the insomnia group (TCQI, $p < 0.01$), particularly girls ($p < 0.01$), indicated attempts to suppress their major unwanted intrusive thoughts more with minor other worries (eg, *focusing on different negative thoughts or worry about other minor things instead*). Adolescents in the insomnia group perceived school attendance (ASQ, $p < 0.05$) as more stressful with a higher stress reactivity to work overload as compared to controls (PSRS, $p < 0.05$), and reported greater stress levels induced by peer pressure (ASQ, $p < 0.05$), school performance (ASQ, $p < 0.01$), school-leisure conflict (ASQ, $p < 0.05$), and future uncertainty (ASQ, $p < 0.01$) than adolescents in the control group. Notably, adolescents in the insomnia group reported a higher usage of substances to cope with these stresses than controls (COPE, $p < 0.05$). There were no group differences in adverse childhood experiences.

3.2.2. Interaction effects

Girls in the insomnia group perceived their sleep quality as poorer (PSQI, $p < 0.05$) and reported more pre-sleep cognitive activity concerned with reflection and planning (GCTI, $p < 0.01$) compared to boys in the insomnia group. Additionally, girls in the insomnia group reported being more likely to experience sleep issues after a stressful situation compared to boys in the insomnia group (FIRST, $p < 0.05$). While all adolescents in the insomnia group reported lower sleep hygiene and higher levels of agreeableness, girls in the insomnia group in particular reported lower successful sleep hygiene in the cognitive domain (ASHS, $p < 0.05$) and higher agreeableness (NEO, $p < 0.05$) compared to their healthy peers.

3.2.3. Sex differences

Irrespective of the group, girls reported a lower sleep quality (PSQI, $p < 0.05$) and higher levels of aggressive suppression (eg, *punishing themselves for the thoughts*) to control unwanted thoughts before bedtime (TCQI, $p < 0.05$). Girls also reported experiencing more daytime sleepiness (CASQ, $p < 0.05$) with more physiological factors contributing to poor sleep hygiene (ASHS, $p < 0.05$) than boys. Additionally, girls reported lower levels of openness (NEO, $p < 0.05$), greater stress levels from school/leisure conflict (ASQ, $p < 0.05$), peer pressure (ASQ, $p < 0.05$), school performance (ASQ, $p < 0.05$), future uncertainty (ASQ, $p < 0.01$), and a higher reactivity to stress induced by social evaluation (PSRS, $p < 0.05$) and work overload (PSRS, $p < 0.05$) compared to boys. To cope with these daily stresses, girls indicated focusing more on the venting of emotions (eg, *getting upset and letting the emotions out*) ($p < 0.05$), the seeking of instrumental social support (eg, *getting advice from others*) ($p < 0.05$) and the use of emotional social support (eg, *discussing their feelings with others*) ($p < 0.01$).

3.3. Network analysis

3.3.1. Centrality

Fig. 3 represents the networks, reflecting multi-symptom relations of the control group and the insomnia group. For both groups, the networks had 19 nodes, with 25 edges for controls and 36 edges for the insomnia group. In the insomnia group, the network degree centrality (reflecting the mean connection of a node with other nodes) is higher ($M = 0.18$, $SD = 0.13$) than in the control group ($M = 0.14$, $SD = 0.10$), with each node being connected to 2.51 (controls) and 3.23 (insomnia) other symptoms, on average. The average network betweenness centrality (the number of times that a given node is part of the shortest path between two other nodes) is lower in the insomnia group ($M_{insomnia} = 0.02$, $SD_{insomnia} = 0.03$, $M_{control} = 0.04$, $SD_{control} = 0.05$), while the average closeness (the mean distance from one node to any other node) is similar in both networks ($M_{insomnia} = 0.29$, $SD_{insomnia} = 0.18$, $M_{control} = 0.28$, $SD_{control} = 0.15$).

Clinical, behavioral, and psychosocial characteristics have different roles and importance, in the networks of the control group and the insomnia group, reflected in the centrality metrics. Severity of insomnia-like symptoms and neuroticism appear to be important in both networks (higher degree centrality), with the insomnia severity being the most connected node in the insomnia group network. In contrast, anxiety has the highest degree

Table 1
Sample demographics. Data are reported as numbers or mean ± standard deviation (SD), separately for adolescents with (insomnia) and without (controls) insomnia symptomatology.

	Controls	Insomnia	
Sample (No.)	48	47	
Sex (No., boys/girls)	20/28	16/31	
Age, y	18.2 (1.0)	17.7 (0.9)	[a]
BMI	22.5 (3.8)	21.3 (2.6)	
Race, No.			
- American Indian/Alaska Native	1	1	
- Asian	19	16	
- Native Hawaiian or Other Pacific Islander	1	0	
- Black or African American	0	1	
- White	27	33	
Ethnicity, No.			[a]
- Hispanic or Latino	13	5	
- NOT Hispanic or Latino	35	41	

[a] , $p < 0.05$.

Table 2
Clinical, behavioral, and psychosocial characteristics of the sample. Data are reported as mean and standard error (SE), separately for boys and girls with and without clinically relevant insomnia symptomatology. Generalized linear models were performed with group (insomnia, control) and sex (boys, girls) as fixed factors and remote data collection as a covariate.

Instrument	Control		Insomnia		p (group)	p (sex)	p (group*sex)
	Girls mean (SE)	Boys mean (SE)	Girls mean (SE)	Boys mean (SE)			
Insomnia symptoms, sleep quality and habits							
Insomnia severity index (ISI)							
- Total score	3.7 (0.7)	4.2 (0.8)	10.6 (0.7)	8.6 (1.0)	0.001		
Pittsburgh sleep quality index (PSQI)							
- Total score	3.7 (0.4)	4.1 (0.5)	8.3 (0.4)	5.9 (0.6)	0.001	0.012	0.004
Adolescent sleep hygiene scale (ASHS)							
- Total score	4.7 (0.1)	4.6 (0.1)	4.3 (0.1)	4.3 (0.1)	0.001		
- Physiological domain	5.3 (0.1)	5.1 (0.1)	5.2 (0.1)	4.8 (0.1)		0.035	
- Cognitive domain	4.2 (0.2)	3.9 (0.2)	3.1 (0.1)	3.5 (0.2)	0.001		0.044
- Emotional domain	5.2 (0.1)	5.2 (0.1)	4.5 (0.1)	4.7 (0.2)	0.001		
- Daytime sleep	5.2 (0.2)	5.0 (0.3)	4.8 (0.2)	4.9 (0.3)			
- Sleep environment	5.4 (0.1)	5.4 (0.1)	5.3 (0.1)	5.3 (0.1)			
- Bedtime routine	2.1 (0.3)	2.3 (0.3)	2.2 (0.2)	3.1 (0.3)			
- Sleep stability	4.2 (0.1)	3.8 (0.2)	3.7 (0.1)	3.6 (0.2)	0.017		
- Substances	5.9 (0.1)	5.9 (0.1)	5.9 (0.1)	6.0 (0.1)			
- Bed/bedroom sharing	3.6 (0.1)	3.5 (0.1)	3.6 (0.1)	3.5 (0.1)			
Morningness–eveningness questionnaire revised (MEQ-R)							
- Total score	51.8 (1.4)	47.7 (1.7)	49.7 (1.4)	48.6 (1.9)			
Cleveland adolescent sleepiness questionnaire (CASQ)							
- Total score	36.9 (1.2)	36.6 (1.4)	37.7 (1.1)	33.5 (1.5)			
- Sleepiness statements	19.8 (1.0)	17.9 (1.1)	22.3 (0.9)	19.3 (1.3)	0.05	0.020	
- Alertness Statements	12.9 (0.8)	11.3 (0.9)	14.6 (0.7)	15.8 (1.0)	0.001		
Sleep-related cognitions							
Dysfunctional beliefs and attitudes about sleep (DBAS)							
- Total score	31.8 (2.3)	31.7 (2.8)	44.7 (2.2)	40.0 (3.1)	0.001		
Glasgow content of thoughts inventory (GCTI)							
- Total score	36.0 (1.8)	36.9 (2.2)	55.9 (1.7)	50.9 (2.4)	0.001		
- Sleep-related anxiety	10.5 (0.7)	10.3 (0.8)	16.9 (0.7)	10.1 (0.9)	0.001		
- Reflection & planning	7.1 (0.4)	7.6 (0.5)	11.2 (0.4)	9.7 (0.5)	0.001		0.031
- General worries	5.7 (0.4)	6.2 (0.5)	9.6 (0.4)	9.1 (0.5)	0.001		
- Thoughts about environment	4.2 (0.3)	4.3 (0.4)	6.1 (0.3)	4.7 (0.5)	0.001		
- Negative affect	4.2 (0.3)	4.4 (0.3)	6.1 (0.3)	5.7 (0.4)	0.001		
Thought control questionnaire insomnia – revised (TCQI-R)							
- Total score	64.4 (1.5)	63.9 (1.8)	74.5 (1.5)	57.9 (2.0)	0.001	0.018	
- Aggressive suppression	12.7 (0.4)	11.6 (0.5)	13.0 (0.4)	12.1 (0.6)		0.030	
- Cognitive distraction	11.2 (0.5)	11.5 (0.6)	11.2 (0.4)	11.4 (0.7)			
- Reappraisal	14.0 (0.6)	14.1 (0.7)	16.9 (0.6)	16.0 (0.9)	0.001		
- Behavioral distraction/suppression	9.6 (0.5)	10.4 (0.6)	11.3 (0.5)	10.1 (0.7)			
- Social avoidance	6.3 (0.3)	6.3 (0.3)	7.6 (0.3)	7.5 (0.4)	0.001		
- Worry	10.7 (0.5)	9.9 (0.6)	14.4 (0.5)	11.7 (0.7)	0.001	0.001	
Stress and stress reactivity							
The Ford insomnia response to stress test (FIRST)							
- Total score	15.7 (0.8)	15.7 (0.9)	24.2 (0.7)	20.5 (1.0)	0.001	0.008	0.028
Perceived stress reactivity scale (PSRS)							
- Total score	22.5 (4.0)	20.6 (3.9)	23.8 (5.0)	22.2 (5.8)			
- Prolonged reactivity	4.7 (0.3)	4.6 (0.4)	4.5 (0.3)	3.9 (0.4)			
- Reactivity to work overload	3.7 (0.4)	2.8 (0.5)	5.0 (0.4)	4.2 (0.5)	0.005	0.040	
- Reactivity to social conflict	5.1 (0.3)	4.8 (0.3)	4.9 (0.2)	5.1 (0.3)			
- Reactivity to failure	4.7 (0.2)	4.7 (0.3)	4.1 (0.2)	4.9 (0.3)			
- Reactivity to social evaluation	4.4 (0.3)	3.7 (0.4)	5.2 (0.3)	4.1 (0.4)		0.010	
Perceived stress scale (PSS)							
- Total score	22.6 (0.9)	20.6 (1.4)	23.7 (0.8)	22.2 (1.2)			
Adolescent stress questionnaire (ASQ)							
- Stress of home life	21.5 (1.5)	19.9 (1.8)	23.3 (1.4)	20.5 (2.0)			
- Stress of school performance	15.0 (1.0)	13.4 (1.2)	19.5 (1.0)	16.1 (1.3)	0.001	0.014	
- Stress of school attendance	4.8 (0.3)	4.9 (0.4)	6.2 (0.3)	5.2 (0.5)	0.009		
- Stress of romantic relationships	6.8 (0.7)	7.9 (0.8)	7.9 (0.6)	8.3 (0.9)			
- Stress of peer pressure	10.5 (0.6)	9.2 (0.8)	12.7 (0.6)	10.4 (0.8)	0.11	0.11	
- Stress of teacher interaction	9.4 (0.7)	8.9 (0.8)	10.5 (0.6)	9.6 (0.9)			
- Stress of future uncertainty	8.0 (0.5)	6.4 (0.5)	10.9 (0.4)	7.7 (0.6)	0.001	0.001	
- Stress of school/leisure conflict	10.2 (0.6)	9.2 (0.7)	12.2 (0.6)	10.1 (0.8)	0.032	0.022	
- Stress of finance	6.8 (0.5)	6.8 (0.6)	6.7 (0.5)	6.9 (0.7)			
- Stress of emerging adult responsibility	4.9 (0.3)	4.6 (0.4)	4.4 (0.3)	3.7 (0.4)			
Mood							
Beck depression inventory (BDI)							
- Total score	2.7 (0.8)	2.4 (0.9)	7.7 (0.7)	6.0 (1.0)	0.001		
State-trait anxiety inventory (STAI-Y2)							
- Total score	41.8 (1.2)	43.5 (1.5)	43.0 (1.2)	41.6 (1.6)			
Personality							
NEO five-factory inventory (NEO-FFI)							

(*continued on next page*)

Table 2 (*continued*)

Instrument	Control		Insomnia		p (group)	p (sex)	p (group*sex)
	Girls mean (SE)	Boys mean (SE)	Girls mean (SE)	Boys mean (SE)			
- Neuroticism	17.9 (1.0)	16.4 (1.2)	20.1 (0.9)	18.7 (1.3)	0.041		
- Extraversion	28.1 (0.9)	29.1 (1.)	29.6 (0.8)	28.0 (37.7)			
- Openness	24.1 (0.8)	26.6 (1.0)	26.2 (0.8)	28.2 (1.1)	0.031	0.020	
- Agreeableness	24.0 (1.1)	25.4 (1.4)	29.7 (1.1)	28.1 (1.5)	0.001		0.048
- Conscientiousness	28.9 (1.1)	29.4 (1.2)	30.7 (1.0)	31.2 (1.4)			
Childhood trauma							
Adverse childhood experiences (ACE)							
- Total score	0.3 (0.1)	0.9 (0.2)	0.3 (0.1)	0.4 (0.2)			
Coping & emotion regulation behavior							
The COPE inventory (COPE)							
- Positive reinterpretation & growth	11.8 (0.5)	11.3 (0.6)	12.6 (0.5)	11.1 (0.6)			
- Mental disengagement	8.4 (0.4)	8.2 (0.4)	9.4 (0.3)	8.2 (0.5)			
- Focus on & venting of emotions	8.1 (0.6)	6.6 (0.7)	9.3 (0.5)	7.3 (0.7)		0.005	
- Seeking social support – instrumental	10.1 (0.6)	9.4 (0.7)	11.3 (0.6)	9.4 (0.8)		0.039	
- Active coping	10.0 (0.4)	10.2 (0.5)	10.6 (0.4)	11.1 (0.6)			
- Denial	4.5 (0.2)	4.8 (0.3)	4.6 (0.2)	4.7 (0.3)			
- Turning to religion	5.3 (0.4)	5.6 (0.5)	5.9 (0.4)	4.6 (0.6)			
- Humor	8.4 (0.7)	8.8 (0.8)	8.7 (0.6)	10.8 (0.9)			
- Behavioral disengagement	5.4 (0.3)	5.3 (0.4)	5.8 (0.3)	4.9 (0.4)			
- Restraint coping	8.6 (0.4)	8.4 (0.5)	9.2 (0.4)	8.7 (0.5)			
- Seeking social support – emotional	10.8 (0.7)	9.3 (0.8)	11.6 (0.6)	7.3 (0.9)		0.001	
- Substance use	5.4 (0.4)	5.6 (0.5)	5.9 (0.4)	4.6 (0.6)	0.041		
- Acceptance	10.3 (0.5)	11.2 (0.6)	11.0 (0.5)	11.4 (0.7)			
- Suppression of competing activities	7.8 (0.4)	8.3 (0.5)	8.5 (0.4)	8.9 (0.5)			
- Planning	10.5 (0.6)	9.9 (0.7)	11.2 (0.5)	11.6 (0.7)			
Difficulties in emotion regulation scale (DERS)							
- Total score	78.7 (2.7)	80.2 (3.2)	79.1 (2.6)	76.7 (3.6)			
- Nonacceptance of emotional responses	9.7 (0.7)	10.5 (0.8)	9.8 (0.6)	10.0 (0.9)			
- Difficulties engaging in goal-directed behavior	13.1 (0.7)	12.2 (0.8)	13.4 (0.7)	13.5 (0.9)			
- Impulse control difficulties	10.1 (0.5)	9.7 (0.7)	11.0 (0.5)	9.5 (0.7)			
- Lack of emotional awareness	16.7 (1.2)	21.2 (1.4)	18.5 (1.1)	17.9 (1.5)			
- Limited access to emotion regulation strategies	14.1 (0.7)	14.6 (0.8)	14.6 (0.7)	14.4 (0.9)			
- Lack of emotional clarity	12.0 (0.5)	11.4 (0.6)	11.7 (0.5)	11.3 (0.6)			

centrality in the control group network, while if ranked, it only reaches the ninth place in importance in the insomnia group network. Greater importance for personality traits of agreeableness, openness, and extraversion, as well as daytime sleepiness, characterizes the network of control group compared to that of the insomnia group. However, greater importance for adverse childhood experiences, circadian rhythm typology, emotion regulation difficulties, and perceived stress characterize the network of the insomnia group, compared to that of the control group. The evaluation of the closeness of nodes suggests a different network structure in adolescents in the insomnia group compared to controls. Insomnia symptoms severity, sleep stress reactivity, adverse childhood experiences and circadian rhythm type are the most central features in the insomnia group network, while most of these factors are not central in the control group network. The betweenness centrality further emphasize the roles of the insomnia severity in the insomnia group network, and the extraversion in the control group network. However, high betweenness is not necessarily associated with high degree centrality or closeness. The fact that we can see an overlap in these metrics highlight the importance of these symptoms.

3.3.2. Structure

Separate concentration networks were generated for adolescents with and without insomnia symptomatology. The visualization of symptom networks conveys the results of the analysis showing patterns of connections and the restructuring of these interactions in the clinical condition. Excluding the isolated nodes, the Girvan–Newman algorithm detected 2 main clusters in the control group network and 2 clusters in the insomnia group network. The main clusters are mixed-domain subsets of the nodes and may correspond to a broad-scope structuring of the data. To identify structural network differences, the clusters of the control group network were projected on the insomnia group network (see Fig. 3). Adverse childhood experiences and circadian typology are negatively correlated to each other and represent a separate cluster with relatively low overall centrality in the control group network. However, they show high degree centrality and a positive association in the insomnia group network. In insomnia, there is a strong negative correlation between adverse childhood experiences and insomnia severity, and a strong positive association between adverse childhood experiences and the circadian typology, with more negative childhood experiences being associated with more morningness. Alternatively, these nodes are unrelated in healthy controls.

Perceived stress and emotion regulation difficulties are isolated nodes in the control group, while they are connected in the insomnia group network. Additionally, personality factors including agreeableness, openness and neuroticism are closely connected and central in the control group network, while there is a complete rearrangement of these factors in the insomnia group. On the other hand, neuroticism is central and negatively correlated to circadian typology in the insomnia group, while the opposite pattern is seen in the control group network, displaying a large gap and no connection between these two nodes. Additionally, openness and extraversion are isolated in the insomnia group.

As indicated by the drop in the betweenness and closeness of extraversion in the insomnia group network, this variable appears to be losing its mediator/transmission role. Anxiety symptoms have the highest degree centrality in the control group network. Interestingly, results however indicate that anxiety symptoms have a high betweenness centrality in both networks, showing that it frequently appears along the trajectory between other pairs of nodes, and it is similarly important in the association of symptoms

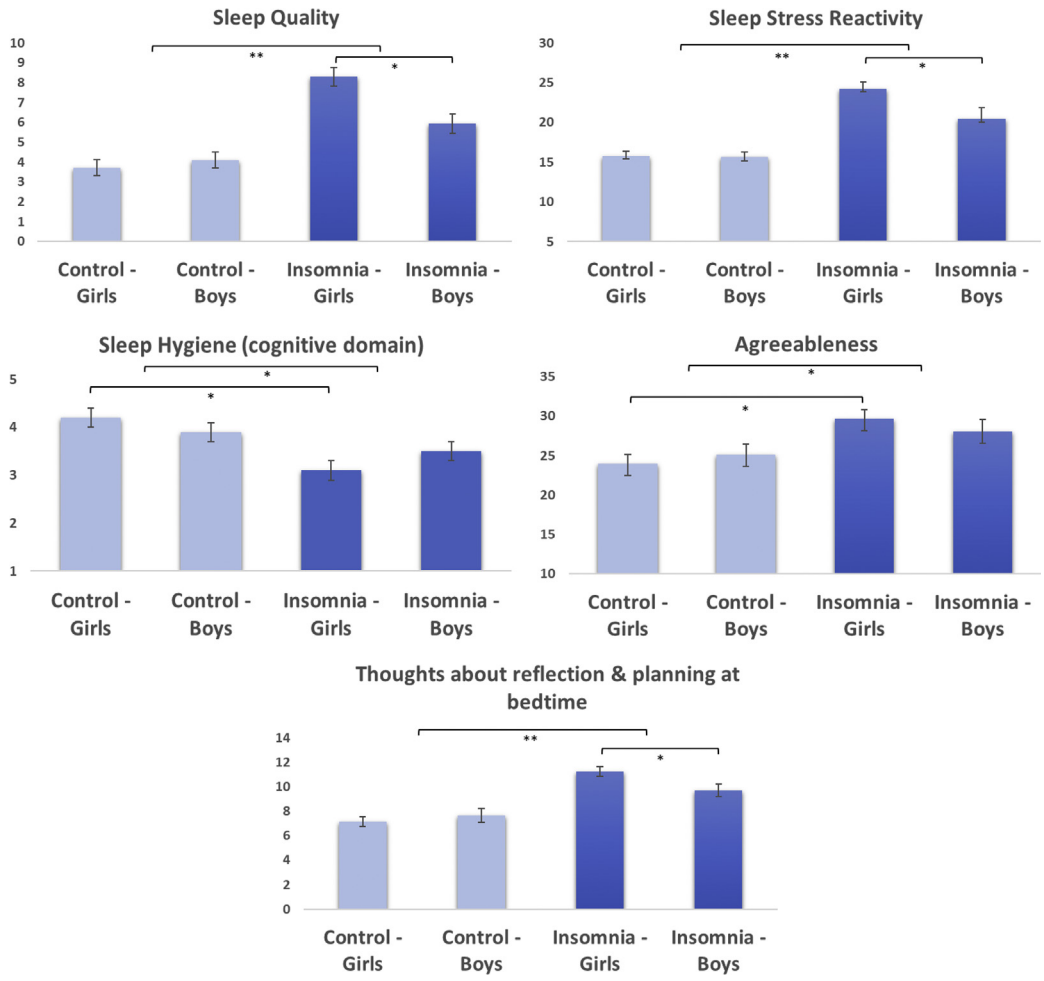


Fig. 1. Interaction effect analysis for the sleep quality (PSQI), sleep stress reactivity (FIRST), thoughts about reflection and planning at bedtime (GCTI), sleep hygiene in the cognitive domain (ASHS), and agreeableness (NEO) in adolescents with and without insomnia symptomatology, provided separately for boys and girls. Adolescents with insomnia, particularly girls, indicate a lower sleep quality, higher vulnerability to experience insomnia symptoms after a stressful situation, more thoughts related to reflection and planning before bedtime, lower sleep hygiene, and higher agreeableness. *p < 0.05; **p < 0.01.

as insomnia severity, which shows an increase in its betweenness in the insomnia group network.

3.4. Exploratory analysis: clinical vs. subclinical insomnia

Additional exploratory analyses were conducted to investigate for potential differences in adolescents with clinically relevant insomnia symptomatology not meeting the DSM-5 threshold for insomnia duration and/or frequency (subclinical insomnia) and adolescents meeting full DSM-5 criteria for insomnia disorder (clinical insomnia). Generalized linear mixed-effect models were performed with group (insomnia, subclinical insomnia) and sex (boys, girls) as fixed factors and remote data collection as a covariate.

Results indicated little differences within the subgroups. Compared to individuals who met all criteria for insomnia disorder according to DSM-5 standards, those with subclinical insomnia symptomatology showed similar symptoms and impairments, but sometimes to a reduced degree of severity. Particularly, those with a diagnosed clinical insomnia disorder exhibited higher insomnia severity (ISI, $p < 0.01$), lower sleep quality (PSQI, $p < 0.01$), a higher vulnerability to situational insomnia (FIRST, $p < 0.01$), lower sleep hygiene practices (ASHS, $p < 0.01$), higher stress levels (ASQ, $p < 0.05$), more thoughts that keep them awake before sleeping (GCTI, $p < 0.05$), and more attempts to control unwanted thoughts at bedtime (TCQI-R, $p < 0.05$) compared to individuals not meeting the threshold for insomnia symptom duration and/or frequency. A detailed description of the subgroup sample characteristics is provided in Supplementary Table 1.

4. Discussion

This study investigated the clinical, behavioral, and psychosocial aspects of insomnia in adolescence, and explored the relationships and representation of these factors in individuals with and without clinically relevant insomnia symptomatology. Adolescents with insomnia symptomatology reported worse sleep-related behaviors, higher neuroticism scores, lower moods, and higher perceived stress levels, mostly related to school-, peer- and future uncertainty stress. Further, girls compared to boys with insomnia exhibited a higher likelihood to experience sleep issues after a stressful situation, lower perceived sleep quality overall, and more pre-sleep cognitive activity. In general, girls in the insomnia group reported lower sleep hygiene and higher agreeableness than their healthy peers. Exploratory analysis revealed minor differences between adolescents who did not meet the DSM-5 threshold for duration and/or frequency of insomnia and adolescents who met the full DSM-5 criteria for insomnia disorder, with adolescents in the

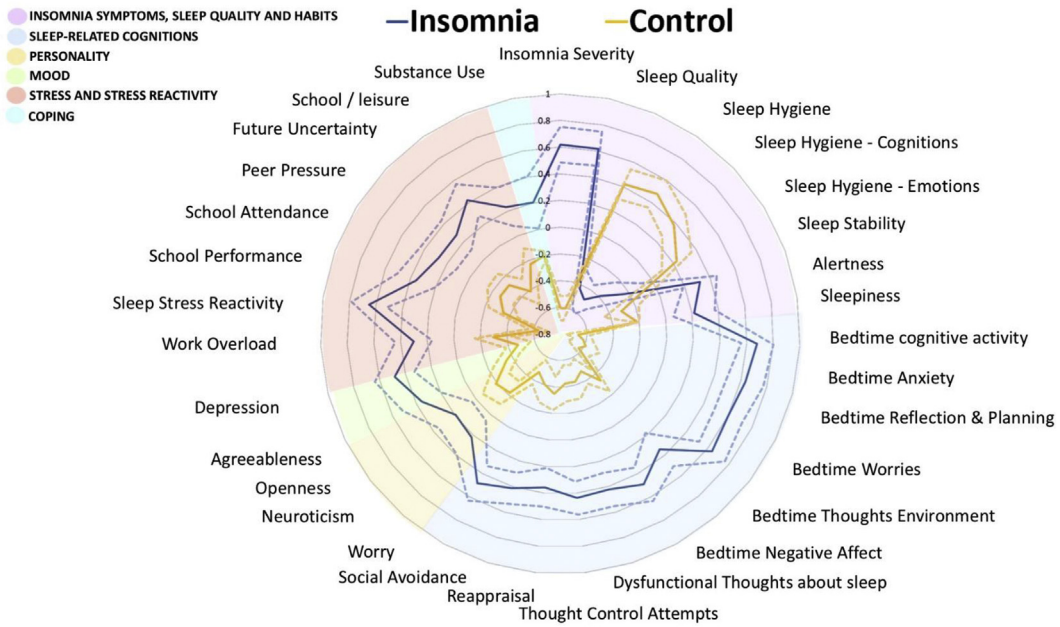


Fig. 2. Significant group differences in adolescents with and without clinically relevant insomnia symptomatology. Adolescents in the insomnia group indicated poorer sleep quality and habits, greater dysfunctional pre-sleep cognitions and arousal, higher depressive symptoms, lower thought control, higher stress levels and sleep-stress-reactivity, higher substance use as coping mechanism, and higher neuroticism, openness, and agreeableness levels than adolescents in the control group ($p < 0.05$).

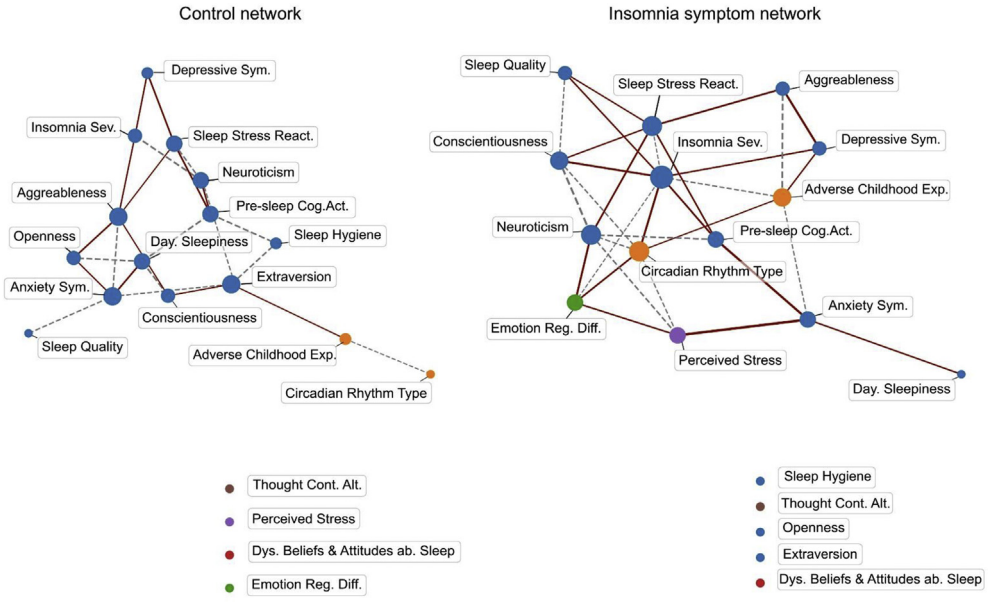


Fig. 3. Illustration of networks for N = 95 post-pubertal adolescents with and without insomnia symptomatology. Nodes in the network represent the symptoms associated with the insomnia condition. The edges between connecting the nodes represent connection strength between these symptoms based on partial correlations (ρ). Isolated nodes are listed at the bottom. The direction of the ρ is indicated by the color of the line (red: positive, blue: negative). The higher the correlation, the thicker the edge. The position of the nodes in the network is based on the Fruchterman–Reingold algorithm [53], which calculates the optimal layout so that strongly correlated symptoms cluster in the middle, whereas symptoms with weaker connections to other symptoms are placed further apart. The node colors are defined by the clustering (Girvan–Newman algorithm, [54]) of the control network. The sizes of nodes indicate the degree centrality with larger nodes representing a higher degree centrality in the networks. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

subclinical group mostly experiencing similar symptoms and impairments, albeit, sometimes to a lesser extent.

Based on network analysis of the interconnections between all these characteristics, adolescents with insomnia symptomatology showed differences in the centrality, closeness and betweenness of their symptoms, indicating different network topology in terms of clinical, behavioral, and psychosocial characteristics than controls. For example, our results showed that insomnia severity has the highest centrality in the insomnia network, being significantly related with several other critical factors in insomnia pathophysiology, like adverse childhood experiences, stress vulnerability to situational insomnia, sleep quality, pre-sleep cognitive activity, and circadian typology. While the current cross-sectional dataset cannot separate cause from consequences, future studies applying

network approached to longitudinal data can be used to investigate directionality in these relationships, expanding our capability of discovering patterns of clinical, behavioral, and psychosocial factors underlying insomnia. This approach could uncover hidden factors and pathways involved in the development of insomnia, which could advance understanding of its etiology and better management of the disorder.

Insomnia disorder has been well described and characterized in adult samples, and our results indicate comparable symptoms in adolescents. Insomnia is characterized by recurrent perceived sleep complaints [33] and as expected, adolescents with insomnia symptoms reported a variety of disturbed sleep-related symptoms, behaviors and cognitions including a lower sleep quality, more dysfunctional beliefs and attitudes about sleep and poorer sleep hygiene practices; for example inconsistent bed and wake-patterns on weekdays or weekends. They also reported higher pre-sleep cognitive (eg, thinking about things they need to do before bedtime) and emotional (eg, going to bed upset) arousals. Current literature describes chronic hyperarousal in particular as a core element that plays a role in the onset and development of insomnia [16,57]. Hyperarousal may be characterized by increased levels of physiological and cognitive activity (eg, worry). In fact, consideration of cognitive processes and other mental activities has previously been described as particularly important for a better understanding of insomnia disorder [58]. Our findings underscore that adolescents with insomnia symptomatology indeed experienced more thoughts that kept them awake before sleep than healthy controls, potentially contributing to the development and manifestation of the disorder in adulthood. They specifically reported pre-sleep cognitive activities related to general worry or sleep-related anxiety, suggesting that these cognitive disruptions affected their ability to fall asleep and get a good night's sleep. In addition, adolescents with insomnia symptomatology reported more attempts to control these unwanted thoughts than healthy children, using a variety of strategies; most of which, however, appear to have little success. For example, adolescents with insomnia symptomatology reported replacing unwanted thoughts before bedtime with other negative thoughts (TCQI factor: worry), which is an unhelpful strategy for improving sleep impairments [17]. Summarized, these findings suggest that adolescents with insomnia symptomatology would benefit greatly from sleep hygiene education, as well as from cognitive strategies aimed at facilitating their falling asleep process. Our findings suggest that adolescents with insomnia clinically relevant symptomatology are somewhat ineffective at managing disruptive or dysfunctional thoughts before bedtime, suggesting that they would likely benefit from specific interventions to help them regulating their bedtime and wake-up time, and improve sleep-related behaviors and cognitions.

Additionally, we found sex-insomnia interaction effects that could offer potential insights for gender-specific therapy and diagnostic approaches: girls with insomnia symptomatology were especially affected by distracting pre-sleep cognitive activity, specifically by thoughts associated with reflection and planning that kept them awake before sleep, compared with boys with insomnia. They also reported a higher vulnerability to situational insomnia under stressful conditions (sleep stress reactivity) indicating a higher sensitivity to stress represented in sleep impairments than boys. Interestingly, girls with insomnia symptoms also generally exhibited lower sleep hygiene in the cognitive domain, for example, reflecting on the day's events or thinking about their responsibilities before bed, than girls without insomnia symptoms. These results are in line with current literature, that indicated a female vulnerability to sleep-specific stressors in adolescents [59]. Girls, specifically those with insomnia symptomatology, also reported a poorer sleep quality than boys, including boys with insomnia symptomatology, although insomnia severity scores in the insomnia group did not differ according to sex. Previous work highlighted that women in general report a lower sleep quality than men [60], which is consistent with our findings of subjectively lowest sleep quality in girls with insomnia symptoms. While girls with insomnia symptomatology scored higher agreeableness than female controls, their scores were still within normal ranges. Reasons for this difference could be varied and remain to be further explored. Findings highlight some differences between girls and boys with insomnia and indicate sex-specific clinical features of insomnia disorder in teens, while emphasizing the importance of considering sleep issues with regard to psychosocial factors such as stressful daytime situations and personality traits. Girls especially tend to suffer from stressful daytime situations and transfer these tensions into their sleep, making them particularly vulnerable for developing insomnia symptoms under stressful conditions.

Indeed, stress or increased stress levels are generally strongly associated with insomnia disorder as well as with the onset of insomnia symptoms [18,19]; with previous studies additionally indicating that individuals with insomnia perceive similar life events as more stressful than their healthy counterparts [61]. It is possible that higher perceived stress levels during adolescence contribute to the development of the disorder and a manifestation during adulthood. While our findings highlight the impact of daytime stress on sleep in adolescents with insomnia symptomatology, particularly girls, our results also show significantly higher perceived stress levels in adolescents with insomnia symptomatology, particularly in the context of social or academic life. These findings underscore the importance of considering teen-specific stressors such as school attendance or -performance, peer pressure, or the uncertainty of their future – likely related to the uncertainty of college applications at this age – when describing clinically relevant insomnia symptoms in adolescents. Once again, our results suggest a higher vulnerability to adolescence-specific stress in girls triggered by, for example, social evaluation, indicating a strong female vulnerability to social and academic stress. Given that a critical issue in the study and treatment of adolescent insomnia is its recognition, our findings suggest that clinicians and medical societies should not only consider these adolescent-specific stressors in characterizing adolescent insomnia, but also raise awareness that girls are at higher risk of being strongly influenced by school- or society-related stresses, making them particularly vulnerable to developing stress-induced negative health outcomes. Notably, adolescents with insomnia in our study reported using more substances to cope with these daily stresses compared to their healthy peers. This finding, similar to their less effective thought control strategies, suggests that adolescents with sleep problems may not use their resources efficiently to cope effectively with aversive or stressful situations. Despite the higher burden of stress in the insomnia group, there were no differences in effective coping skills or emotion regulation behavior between adolescents with and without insomnia symptomatology, again emphasizing that adolescent-centered therapy services may consider teen specific worries and needs (eg, peer pressure) when approaching adolescent insomnia.

In line with previous work highlighting the risk relationship between depression and sleep disturbances in adolescents [62], in our study teenagers with insomnia symptomatology indicated more depressive symptoms than healthy controls. While our results indicate depressive symptoms on a subclinical level, they still suggest an increased psychological burden, which commonly accompanies sleep disturbances. Recent studies further indicate associations between insomnia disorder and other mental disorders, with more than 50% of adolescents with insomnia having had at

least one comorbid psychiatric disorder [1]. While our results are cross-sectional, these data urge the need for a better clinical characterization of adolescent insomnia to improve long-term adverse mental health outcomes.

Interestingly, in addition to differences in mood symptoms, individuals with insomnia symptomatology generally also show differences in their personality traits. We found that neuroticism was higher in the insomnia group, which is consistent with the literature [12,28]. However, our results also indicate that adolescents with insomnia symptoms have higher levels of agreeableness and openness than their healthy peers, which contradicts recent work showing the opposite relationship in adult populations [12,13]. Possibly, our findings differ because we focused on a distinctive age group of adolescents. Adolescence is accompanied by massive psychosocial changes, with personality traits still forming and developing during the maturation period. However, current research also indicates that personality structures can still change after maturation [63]. However, as mentioned above, social factors should be considered in personality formation. Due to the high peer pressure during adolescence – and our results showing that teenagers with insomnia symptomatology are more affected by this stress – we hypothesize that this group of adolescents attempts to better adapt to their environment and surroundings in order to avoid possible sources of stress. This hypothesis would also be consistent with our finding of ineffective coping strategies among teens with insomnia symptoms, suggesting that more open and adaptive behaviors may be used as strategies to deal with societal stressors in adolescence.

Beyond presence and severity of insomnia symptomatology, a network approach was used to uncover hidden factors entwined in the psychopathology of insomnia disorder. There were profound group differences within the interconnection of multi-symptoms, suggesting a restructured symptom network in adolescents with clinically relevant insomnia symptomatology compared to healthy teens. Notably, when evaluating group differences in network structure, the insomnia severity index in particular stood out. The importance of insomnia severity in the network analysis, while not reflecting the severity of the symptom itself, describes its relative position in the symptom space and emphasizes that many symptoms that do not necessarily differ between the two groups are related to it. Thus, unlike healthy controls, insomnia severity, which is not naturally present in healthy participants without insomnia and is therefore of greatest interest in the study of insomnia, played a central role in the insomnia group network and was closely related to factors such as adverse childhood experiences, stress susceptibility to situational insomnia, sleep quality, pre-sleep cognitive activity, and circadian typology. These results suggest the strong interconnection of these symptoms in adolescent insomnia, and emphasize the close relationship between sleep disturbances, stress, cognitions, and adverse childhood experiences. Additionally, perceived stress and emotion regulation difficulties appeared to be closely connected in adolescents with insomnia symptomatology, emphasizing links between the ability to self-regulate emotions and subjective stress levels. These findings support the use of centered therapies focused on emotion and stress management strategies as well as general coping, which could in turn target the core insomnia symptomatology (perceived difficult falling asleep and maintaining sleep and associated daytime dysfunctions).

Network analysis also suggested that extraversion seems to be losing its mediator role when clusters of controls were projected on the insomnia group network, indicating that this personality factor may play a role in the development of insomnia. It is possible that extraversion serves as a buffer for the development of insomnia symptoms, or at least mitigates such symptoms. Last, anxiety is a factor that plays an important role in both healthy and insomnia networks. While anxiety in healthy teens is particularly associated with subjective sleep quality and personality traits such as openness and extraversion, in the insomnia group, anxiety seems to be more associated with daytime sleepiness, stress and adverse childhood experiences. This emphasizes the distinctive role of anxiety in the network topology and indicates the importance of considering anxiety in the context of stress and adverse childhood experiences in adolescent insomnia.

Network analysis offers a promising and valuable alternative to traditional methods, in which symptoms of a disorder are usually considered to be the expression of some shared underlying factors, whereas the network approach considers the disorder as an expression of the causal interaction of these symptoms [31]. The main advantage of using network analysis, therefore, is to identify possible hidden but modifiable factors that play a central role in the development and expression of insomnia. While network analysis does not address causality, it emphasizes approaching insomnia disorder as a holistic concept that focuses not only on how clinical, behavioral, and psychosocial aspects predominate, but more importantly, how they interact and influence each other as well as how they are interconnected.

4.1. Limitations & future directions

Despite the high prevalence of insomnia disorder highlighted in the epidemiological literature, recruiting adolescents with clinical insomnia, as the primary disorder, without other current comorbid disorders such as major depression or anxiety disorder and in the context of the unique circumstances of adolescent sleep (eg, preference for later bedtimes, early school starts and subsequent sleep deprivation, challenging the DSM-5 criteria of “adequate opportunity to sleep”) was challenging. Our group recently highlighted the difficulty in diagnosing insomnia in adolescents (see Ref. [7]). The inclusion of sub-clinical insomnia participants (not reaching DSM-5 threshold for insomnia frequency/duration) may have increased the generalizability of the results but also deemphasizes the magnitude of the severity of the clinical representation of the insomnia disorder. Given the limited literature on the characteristics of insomnia in adolescence (for a review, see Ref. [6]), more research is needed to describe insomnia in the context of adolescence and to identify age-specific challenges to clearly identify and diagnose insomnia disorder in teenagers. While most of our participants underwent a polysomnographic recording night to assess other sleep disorders, a subsample of 32 subjects were unable to complete the laboratory portion of the protocol because of COVID-19 policy restrictions and were assessed for sleep disorders via remote clinical interviews. However, we controlled for the influence of the pandemic in the analyses to rule out any confounding effects that the onset of quarantine measurements might have had on our outcome variables. Future studies should evaluate the clinical representation of insomnia across the whole continuum of adolescents at risk for insomnia (vulnerability features), those with sub-clinical symptoms, as well as those meeting full diagnostic criteria for insomnia disorder, and across different age groups and regions as the current sample consisted mainly of non-Hispanic white and Asian adolescents from the San Francisco Bay area. Further, the current sample showed an overrepresentation of Hispanics or Latinos in the control group, which could potentially influence the results. Further research on insomnia in more diverse samples is warranted. Longitudinal models would enable more detailed exploration of the hidden processes involved in the development and differentiation of sub-clinical and clinical groups, possibly with exploration of tipping points critical to disorder development and progression.

Statement of significance

Research on the clinical, behavioral, and psychosocial features of adolescent insomnia is still in its early stages and a better characterization of the disorder is crucial to advance understanding of its etiology as well as to allow early recognition and treatment. Our results provide promising indications for research on early detection and treatment of insomnia in teenagers, which can significantly improve the long-term prognosis of the disorder. Besides classical insomnia-related features, we suggest that sex- and adolescence-specific factors such as social or academic stresses need to be considered in the context of adolescent insomnia. Also, network approaches may be promising to unveil hidden associations among clinical, behavioral, and psychosocial features of insomnia.

CRediT author statement

CRediT author statement. DY: Investigation, Conceptualization, Methodology, Formal analysis, Visualization, Project administration, Writing – review & editing; OK: Formal analysis, Writing – review & editing; DP: Investigation, Conceptualization, Methodology, Project administration, Writing – review & editing. FB: Conceptualization, Project administration, Funding acquisition, Writing – review & editing. MdZ: Conceptualization, Methodology, Formal analysis, Visualization, Project administration, Funding acquisition, Writing – original draft, Writing – review & editing.

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Data availability statement

The data that support the findings of this study are openly available in Ref. [56].

Conflict of interest

None declared.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at 10.1016/j.sleep.2022.03.010.

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: https://doi.org/10.1016/j.sleep.2022.03.010.

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