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Original Article

Autistic traits and sleep in typically developing adolescents

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ABSTRACT

Objective: Diagnosed autism spectrum disorders have been associated with a high prevalence of sleep problems, other psychiatric disorders and social deficits in adolescence. However, little is known about the possible connection between subclinical autistic traits and sleep. This study explored whether adolescents with elevated levels of subclinical autistic traits are at heightened risk for sleep problems.
Methods: This study used data from the community cohort born in 1998. The sample consisted of 157 (57% girls) 17-year-old adolescents. Autistic traits were assessed using the Autism Spectrum Quotient (AQ). The Beck Anxiety Inventory (BAI), Beck Depression Inventory (BDI), and the Adult ADHD Self-Report Scale were utilized to control for comorbid psychiatric symptoms. Sleep was measured with actigraphy and sleep quality was self-rated using the Pittsburgh Sleep Quality Index (PSQI). Associations between autistic traits and sleep were examined using logistic regression analysis.
Results: Elevated levels of autistic traits were significantly associated with shorter weekday sleep duration. Moreover, autistic traits remained an independent predictor of short sleep duration when comorbid psychiatric symptoms were controlled for (OR 1.14; 95% CI: 1.03–1.26).
Conclusions: The results suggest that subclinical autistic traits should be considered as a possible underlying mechanism affecting adolescent sleep.

1. Introduction

Poor and insufficient sleep is a common finding among children and adolescents with autism spectrum disorders (ASD) [1–3]. As sleep problems have the potential to markedly impair daytime functioning [3,4], they may place an additional burden on individuals with deficits in social functioning [5]. Individuals with elevated levels of autistic traits but without a formal ASD diagnosis show more psychiatric difficulties [6–8], and may additionally suffer from sleep problems.

To the best of our knowledge, only one study [5] has investigated the associations between sleep and autistic traits in children (aged 7–9) and adolescents (aged 11–13) in the general population who have not had a formal ASD diagnosis. As opposed to our cross-sectional approach, Sivertsen et al., [5] adopted a longitudinal perspective and found that autism spectrum problems defined as scoring above a threshold on the Autism Spectrum Screening Questionnaire (ASSQ) remained an independent risk factor for parent-rated sleep problems even after controlling for demographics, mental retardation and comorbid psychiatric risk factors like emotional problems, hyperactivity and conduct problems. However, more information is needed at the subclinical level of autistic traits using objective measures of sleep. Our cross-sectional study is not able to capture the developmental perspective of Sivertsen et al., [5]. Despite this, we provide the first insight into the associations between subclinical autistic traits and sleep in a specific adolescent age cohort.

Focusing on both older adolescents and young adults with diagnosed ASD, a small actigraphy (n = 10) study by Øyane and Bjorvatn [9] found that 80% of the individuals met the criteria for sleep disturbance as indicated by low sleep efficiency (<85%) or sleep latency more than 30 min, as well as short total sleep time and a long wake after sleep onset (WASO). Essentially similar results were obtained in a larger study by Wiggs and Stores [10], although this study comprised children and adolescents of a younger and wider age range (5–16 years). Other individual studies have shown varying results, indicating longer sleep latency but no

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difference in total sleep time, sleep efficiency and actual time awake during the night, comparing an ASD group with typically developing individuals [11,12]. Baker et al., [13] compared sleep patterns of adolescents diagnosed with high-functioning ASD (IQ > 70) at a mean age of 15.5 to those of typically developing adolescents. Both subjective and objective measurement of sleep supported the notion that adolescents with ASD are more likely to experience disrupted sleep than typically developing adolescents.

Internalizing behavior (especially depressive and anxiety symptoms) is related to sleep disturbance in ASD individuals [2]. Recent studies support the view that anxiety in particular is related to sleep problems in adolescents with [14]. From this viewpoint, the lack of differences in objectively measured sleep patterns between typically developing individuals and individuals with ASD in some previous studies [11,12,15,16] and discrepancies in results could be explained by the presence of comorbid psychiatric symptoms. Tani et al., [16] concluded that the subjective experience of poor sleep quality in ASD individuals was explained by anxiety symptoms. Previous research suggests that the associations between autistic traits and comorbid symptoms including sleep disruption are most likely complex and bidirectional [2,3,17]. Thus, the role of anxiety as a possible mediator in the relationship between autistic traits and sleep needs to be studied further.

Evidence from the general population has shown increased rates of both depression and anxiety among individuals with elevated autistic traits [7]. More recently, studies have also demonstrated significant associations between autistic traits and depression and anxiety symptoms in student populations [18,19].

In view of this evidence, it is important to explore whether individuals with high levels of subclinical autistic traits also are at heightened risk for sleep loss and disruptions of sleep as indicated by their sleep patterns. Moreover, comorbid psychiatric symptoms and sleep may interact in complex ways in general adolescent populations [20] and in clinical populations [3]. Therefore, the role of common comorbid symptoms related to attention-deficit/hyperactivity disorder (ADHD), anxiety and depression are important to take into account when assessing sleep.

To date, the majority of the research has focused on the clinical extremes of autistic traits and associated problems, although studying the full range of traits could give better insight on how the disorder itself is conceptualized [55]. This study is the first to explore the associations between subclinical autistic traits and sleep in a community sample of adolescents using objective measurements of sleep. Based on previous research in clinical populations, we hypothesized that elevated levels of autistic traits in ASD populations are associated with shorter total sleep time, prolonged sleep latency and reduced sleep efficiency as indicated by a meta-analysis [21]. Additionally, we hypothesized that in ASD populations there would be a later sleep midpoint, as indicated by studies related to circadian rhythm [22], and that the associations are independent after controlling for comorbid mood and anxiety symptoms. Furthermore, explored whether anxiety mediates the relationship between autistic traits and sleep, as it has been suggested that anxiety is related to sleep problems in individuals with ASD [11,12,14–16,23].

2. Material and methods

2.1. Participants

This study used data from the latest follow-up of the longitudinal Glycyrrhizin in Licorice (GLAKU) project [24,25], which is a prospective cohort study on child development. The adolescents studied came from an urban community cohort initially comprising 1049 healthy infants born in 1998 in Helsinki City Maternity Hospital. Originally, the focus of the GLAKU project was focused on the effects of maternal licorice consumption during pregnancy on the development of the child [25]. Objective measurements of sleep quality and quantity were obtained for the first time during the follow-up at the age of eight in 2006 [26]. During the follow-up at age 12 in 2009–2011, 354 children took part in a sleep measurement period [27]. Of that group, 279 adolescents were invited, and 197 (70.6%) participated in the follow-up at age 17. The current study utilized follow-up data from this phase. Of the initial sample (n = 197), 40 adolescents were excluded due to incomplete data on measures of autistic traits, psychiatric symptoms or sleep (less than four nights measured by actigraphy). Thus, the final study sample included 157 adolescents (89 girls, 68 boys), with a mean age of 16.90 years (SD = 0.12 years).

When compared to the original community cohort (n = 892), the current study sample (n = 157) did not differ regarding gestational age, length at birth, birth weight, mother's licorice consumption or alcohol consumption during pregnancy or the number of boys and girls. However, the adolescents in this study had significantly older mothers ($p < 0.05$) than in the initial cohort. When compared to the adolescents who were invited but did not participate (n = 122), the current sample did not differ regarding gestational age, length at birth, birthweight, mother's licorice consumption or alcohol consumption during pregnancy, the number of boys and girls or body mass index (BMI). However, adolescents participating in this study had significantly older mothers ($p < 0.05$) than the non-participants. The Ethical Committee of the Helsinki University Central Hospital approved the study protocol, and each adolescent provided written informed consent.

2.2. Demographic and background variables

Parental education, sex and participants' body mass index (BMI) were all included in the analyses as covariates. Earlier studies have linked mother's consumption of glycyrrhizin in licorice during pregnancy to externalizing symptoms and attention problems [24] but not on sleep quantity or quality or autistic traits. Thus, it was not included as a covariate in the current study. Parental education (highest self-reported level of either parent) was used as an index of socioeconomic status and classified as follows: (1) secondary or lower (2) vocational or (3) university degree. Socioeconomic condition has been shown to be related to sleep disturbances and short sleep duration in adolescents [28]. In addition, significant sex differences have been found with regard to sleep patterns, sleep quality and quantity among typically developing adolescents [29]. BMI was calculated using the formula weight (kg)/height^2 (m^2). There exists evidence that BMI is related to variability in total sleep time [30].

2.3. Objective measurement of sleep by actigraph

Objective measurement of sleep was obtained using wrist actigraphy (Actiwatch AW7, Cambridge Neurotechnology Ltd., UK) with medium sensitivity and 1-min epochs. We used the validated [31] Actiwatch algorithm which automatically determines sleep to start when immobile data has been recorded for 10 min after the registered bed time. The scoring procedure of the activity data was described in more detail previously [32]. Participants wore actigraphs on their non-dominant wrist continuously for an average of 8.36 nights (SD = 1.76; range 4–17). All participants were asked to complete a daily sleep diary and press the event marker on the actigraph every night when they decided to start sleeping (bedtime) and every morning when they woke up (wake-up time). Participants were also instructed to report all exceptions to daily routine (eg, traveling or illness) and time intervals when the device

was not in use. Actigraphy measurements were conducted over typical school months, during which adolescents had school five days a week. If there were any sleepovers, illness, or any other disruption from everyday life, that night was excluded from analyses for this study. The adolescents lived with both parents in most of the cases: out of all the participants, 73% lived with both parents. Twenty-six percent of the adolescents' parents had divorced. Due to the relatively high occurrence of participants with only one valid weekend night measurement, only weekday measurements were included in the analyses. However, to provide a view of the participants' sleep patterns during the whole week, descriptive statistics are reported for both weekend and weekday nights.

Sleep parameters of interest included total sleep time, sleep latency, sleep efficiency, sleep midpoint and WASO. Total sleep time (TST) was determined by the Actiwatch software algorithm and indicates actual sleep time (assumed sleep time minus wake time). Sleep latency refers to a period between bedtime and actual sleep onset determined by the Actiwatch algorithm. Sleep efficiency was obtained by dividing total sleep time by the amount of time in bed (time between bedtime and wake-up time) and multiplied by 100. Sleep midpoint was used as an indication of sleep timing and calculated as the time point when an individual had slept half the amount of assumed sleep. Wake after sleep onset (WASO) is determined as the number of minutes spent awake during the night after falling asleep. WASO was calculated by subtracting actual sleep time from the assumed sleep time.

2.4. Self-report measures

Autistic traits were assessed using the Autism Spectrum Quotient (AQ). The AQ is a self-report questionnaire designed to measure the extent to which an individual with normal intelligence possesses autistic traits [33]. The AQ consists of 50 items which cover five domains (10 items each) relevant for autistic traits: social skills, attention switching, attention to detail, communication and imagination. Participants rated items on a 4-point Likert type scale ("definitely disagree," "slightly disagree," "slightly agree," "definitely agree"). This study followed the dichotomous scoring procedure recommended by Baron-Cohen et al., [33]: one point was given to responses indicating behaviors characteristic of ASD (mildly or strongly) whereas responses not referring to autistic-like-behavior scored zero points. Subscale scores were calculated by summing ten dichotomized items at a time. On subscales, a maximum of five missing values was allowed and replaced by the mean value of the participant's non-missing items. Total AQ scores (possible range 0–50) were calculated by combining all subscale scores. A cut-off point of 32 has been reported optimal for identifying clinically significant levels of autistic traits [33].

The AQ has shown good psychometric properties, including good test-retest and interrater reliabilities [33]. Internal consistency of the overall AQ score has been reported to be satisfactory (Cronbach's $\alpha = 0.81$) in a student sample [34] and adequate ($\alpha = 0.67$) in a non-clinical sample [35]. In our study, the AQ demonstrated adequate internal consistency for the total score (Cronbach's $\alpha = 0.74$).

Attention-deficit/hyperactivity (ADHD) symptoms were measured using the Adult ADHD Self-Report Scale (ASRS) [36]. The ASRS includes 18 questions which assess the frequency of ADHD symptoms over the past six months. The ASRS is comprised of two subscales: inattentiveness (nine items) and hyperactivity/impulsivity (nine items). In this study, each item was scored on a 5-point Likert scale ranging from zero ("never") to four ("very often"). Subscale scores (possible range 0–36) were obtained by summing the individual item scores and total scale score (possible range 0–72) by combining the two subscale scores. The ASRS has shown high internal consistency on the total scale (Cronbach's $\alpha = 0.88$) [37]. In the current study, internal consistency (Cronbach's α) was 0.84 for the total scale. Kessler et al., [36] have assessed the ability of the ASRS to discriminate clinical cases and non-cases and demonstrate a sensitivity of 57.2% and specificity of 96.5% when using a simple scoring system presented above. Since our focus was not on clinical cases and controls, retaining all the information provided by the scale was important. Using a dichotomized scoring system would yield higher sensitivity and specificity [36].

Depressive symptoms were assessed with the Beck Depression Inventory (BDI) [38]. The BDI is a self-administered measure comprising 21 items which cover a range of depressive symptoms that have been present over the past two weeks [38]. Each item is rated on a 4-point scale (0–3) regarding intensity of the symptoms, yielding a total score which can vary from zero to 63. The BDI has been found to have high internal consistency in non-clinical samples (Cronbach's $\alpha = 0.81$) [38]. In this study, internal consistency (Cronbach's α) was 0.92.

Anxiety symptoms were assessed using the Beck Anxiety Inventory (BAI), which has been developed to discriminate anxiety from depressive symptoms [39]. The BAI is composed of 21 self-rating items mainly reflecting physiological anxiety symptoms that have occurred over the past week including the day when the questionnaire is administered. Each item is rated on a 4-point scale ranging from zero ("not at all") to three ("severely") regarding the severity of symptoms, yielding a total score of 0–63. The BAI has shown high internal consistency (Cronbach's $\alpha = 0.91$) in a non-clinical sample of undergraduate students [40]. Also, in this study reliability was satisfactory (Cronbach's $\alpha = 0.84$).

Subjective sleep quality was assessed with the Pittsburgh Sleep Quality Index (PSQI) which is a self-administered questionnaire designed to measure sleep quality and disturbances over the past 1-month [41]. The PSQI consists of seven components (each scored from zero to three) measuring domains of subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication and daytime dysfunction. The component scores are summed to obtain a total PSQI score (possible range 0–21). A total PSQI score >5 is used to identify poor sleepers [41]. The PSQI has demonstrated good psychometric properties [42]. In the current study, Pearson correlations between component scores and total score ranged from 0.26 (sleep medication) to 0.69 (habitual sleep efficiency).

2.5. Statistical analyses

Spearman correlations were used to examine the associations and possible multicollinearity between autistic traits and symptoms of attention-deficit/hyperactivity, depression and anxiety. Binary logistic regression was then used to explore the associations between autistic traits and both actigraphy-assessed weekday sleep and subjective sleep quality in adjusted models. Separate models were built for each sleep outcome. Due to the focus being on the more maladaptive lower or higher end of the distributions, each sleep parameter was dichotomized. Insufficient or disrupted sleep was determined as follows: the 10th percentile was used to identify adolescents with short total sleep time (≤6:00, hh: mm) [43]. The 90th percentile was used to classify adolescents as having long wake after sleep onset (>1:14, hh: mm) or late sleep midpoint (>5:18, hh: mm). Theoretical cut-offs based on previous work were applied for long sleep latency (>00:30, hh: mm) and for poor sleep efficiency (<85%) [29,44]. Adolescents scoring >5 on the PSQI were classified as having poor subjective sleep quality. Given the known sex bias related to autistic traits [33], interaction effects between autistic traits and sex were tested. In the first phase, all models were adjusted for parental education, sex, and BMI. In the second

phase, measures of attention-deficit/hyperactivity, depression and anxiety were all included in the models to control for comorbid psychiatric symptoms. The results of logistic regression analyses are reported as odds ratios (OR) with 95% confidence intervals (CI). Indirect effects of autistic traits on sleep outcome variables through anxiety were tested using the bias-corrected bootstrap method (5000 samples) with 95% confidence intervals (PROCESS v2.16 macro for SPSS; [45,46]). Following this approach, we considered indirect effects significant if the confidence interval did not contain zero. Separate mediation analyses were conducted for all sleep outcome variables, and all models included sex, parental education, BMI, depression and ADHD symptoms as covariates.

All statistical analyses were performed with SPSS 24.0 for MAC, and the significance level was set at $\alpha < 0.05$ (two-tailed tests) in all analyses.

3. Results

3.1. Sample characteristics

Table 1 presents the demographic information, sleep parameter and psychiatric symptom characteristics of the participants stratified by sex and for the total sample. Autistic trait scores were significantly higher for boys than for girls ($p = 0.003$), whereas girls scored higher on anxiety ($p = 0.008$). Two adolescents (both boys) scored at or above the clinical cut-off score of 32 on Autism Spectrum Quotient as determined by Baron-Cohen et al., [33].

No significant differences were observed in depression or attention-deficit/hyperactivity symptom scores or in age, BMI or parental education between boys and girls. Regarding sleep parameters, boys had significantly shorter weekday total sleep time ($p = 0.011$), longer weekday sleep latency ($p = 0.006$), longer weekday wake after sleep onset ($p = 0.028$) and poorer weekday sleep efficiency ($p < 0.001$) when compared to girls.

A similar trend in differences between boys and girls was observed for weekend sleep data. Boys had significantly shorter weekend total sleep time ($p = 0.022$), longer weekend sleep latency ($p < 0.001$), longer weekend wake after sleep onset ($p = 0.021$), poorer weekend sleep efficiency ($p < 0.001$) and later sleep midpoint ($p = 0.009$) when compared to girls. Adolescents' average total sleep time on weekends was 49 min longer and average weekend sleep midpoint 1 h 38 min later than on weekdays.

3.2. Correlations between autistic traits and psychiatric symptoms

To examine the associations and possible multicollinearity among the predictor variables, Spearman correlation analysis between continuous autistic traits and psychiatric symptoms was performed (Table 2). Significant but relatively low positive correlations were found between autistic traits and psychiatric symptoms as measured by Beck Anxiety Inventory (BAI), Beck Depression Inventory (BDI) and Adult ADHD Self-Report Scale (ASRS).

Furthermore, significant but moderate inter-correlations were found between anxiety, depression and attention-deficit/hyperactivity symptoms. Given that all correlations were either low or moderate, we can assume that there is no significant multicollinearity and thus the coefficients of logistic regression analyses can be interpreted individually.

3.3. Associations between autistic traits and sleep

Associations between autistic traits and sleep quality, quantity and timing were examined using binary logistic regression analysis. Table 3 shows the results from these analyses. Since no significant interaction effects were found between autistic traits and sex, analyses were not performed separately for each sex. Continuous autistic traits significantly predicted weekday total sleep time. As

Table 1
Descriptive statistics by sex and for the total sample.

	All (n = 157)	Girls (n = 89)	Boys (n = 68)	p value for sex difference
	Mean (SD) or N (%)	Mean (SD) or N (%)	Mean (SD) or N (%)	
Age, years	16.90 (0.12)	16.90 (0.13)	16.89 (0.11)	0.519[a]
BMI (kg/m^2)	22.07 (3.26)	22.3 (2.99)	21.77 (3.60)	0.102[b]
Parental education				0.805[c]
Secondary or lower	14 (8.90)	9 (10.10)	5 (7.40)	
Vocational	31 (19.7)	18 (20.20)	13 (19.10)	
University degree	112 (71.3)	62 (69.70)	50 (73.50)	
Weekday sleep measured by actigraphy				
Total sleep time (hh:mm)[d]	6:54 (00:46)	7:02 (00:42)	6:43 (00:50)	0.011[a]
Sleep latency (hh:mm)	00:16 (00:14)	00:14 (00:11)	00:20 (00:16)	0.006[b]
Sleep midpoint (hh:mm)[e]	3:49 (1:49)	3:41 (1:42)	3:59 (1:57)	0.117[b]
Wake after sleep onset (hh:mm)[f]	00:48 (00:21)	00:45 (00:15)	00:53 (00:26)	0.028[b]
Sleep efficiency (%)	86.18 (5.05)	87.51 (3.89)	84.44 (5.93)	<0.001[a]
Weekend sleep measured by actigraphy				
Total sleep time (hh:mm)	7:43 (1:10)	7:54 (1:11)	7:29 (1:06)	0.022[a]
Sleep latency (hh:mm)	00:15 (00:16)	00:10 (00:12)	00:21 (00:19)	<0.001[b]
Sleep midpoint (hh:mm)	5:27 (2:53)	5:14 (2:56)	5:43 (2:49)	0.009[b]
Wake after sleep onset (hh:mm)	00:55 (00:26)	00:50 (00:21)	1:01 (00:31)	0.021[b]
Sleep efficiency (%)	86.50 (6.08)	88.33 (4.63)	84.10 (6.91)	<0.001[a]
Autism spectrum quotient (AQ)	14.99 (5.72)	13.83 (5.02)	16.50 (6.24)	0.003[a]
Beck Anxiety Inventory (BAI)	5.28 (5.31)	6.08 (5.72)	4.24 (4.57)	0.008[b]
Adult ADHD self-report scale (ASRS)	23.80 (9.98)	25.34 (9.74)	21.78 (10.00)	0.027[a]
Beck Depression Inventory (BDI)	5.02 (6.86)	6.17 (8.10)	3.51 (4.39)	0.100[b]
Pittsburgh sleep quality index (PSQI)[g]	5.26 (2.16)	5.28 (2.20)	5.22 (2.12)	0.931[a]

[a] p-value calculated using the independent-samples t-test.
[b] p-value calculated using the non-parametric Mann–-Whitney U-test.
[c] p-value calculated using the Pearson's Chi-squared test.
[d] 10th percentile at 6:00, 25th percentile at 6:21, median at 6:55, 75th percentile at 7:25 and 90th percentile at 7:53.
[e] 10th percentile at 2:28, 25th percentile at 2:53, median at 3:19, 75th percentile at 4:09 and 90th percentile at 5:18.
[f] 10th percentile at 00:25, 25th percentile at 00:33, median 00:48, 75th percentile at 00:58 and 90th percentile at 1:14.
[g] Total sample n = 149, girls n = 85, boys n = 64.

Table 2
Correlations between autistic traits and psychiatric symptoms.

	Autistic traits	Anxiety symptoms	ADHD symptoms	Depression symptoms
Autistic traits	1			
Anxiety symptoms	0.29**	1		
ADHD symptoms	0.26**	0.60**	1	
Depression symptoms	0.34**	0.61**	0.54**	1

**p < 0.01.

Table 3
Results of the logistic regression analyses.

Predictor	Total sleep time ≤6:00			Sleep latency >0:30			Sleep midpoint >5:18			Wake after sleep onset >1:14			Sleep efficiency <85%			PSQI total score >5		
	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p
Model 1[a]																		
Autistic traits	1.11	1.02–1.22	0.018*	0.98	0.89–1.07	0.603	0.95	0.85–1.05	0.303	0.96	0.86–1.07	0.499	1.00	0.94–1.06	0.996	1.04	0.98–1.10	0.234
Model 2[b]																		
Autistic traits	1.14	1.03–1.26	0.009**	0.95	0.86–1.06	0.370	0.92	0.82–1.04	0.172	0.97	0.87–1.09	0.643	1.00	0.94–1.07	0.993	1.01	0.95–1.09	0.685

*p < 0.05; **p < 0.01.
[a] Adjusted for sex, parental education and BMI.
[b] Adjusted for the same variables as in model 1 + anxiety, ADHD and depression symptoms.

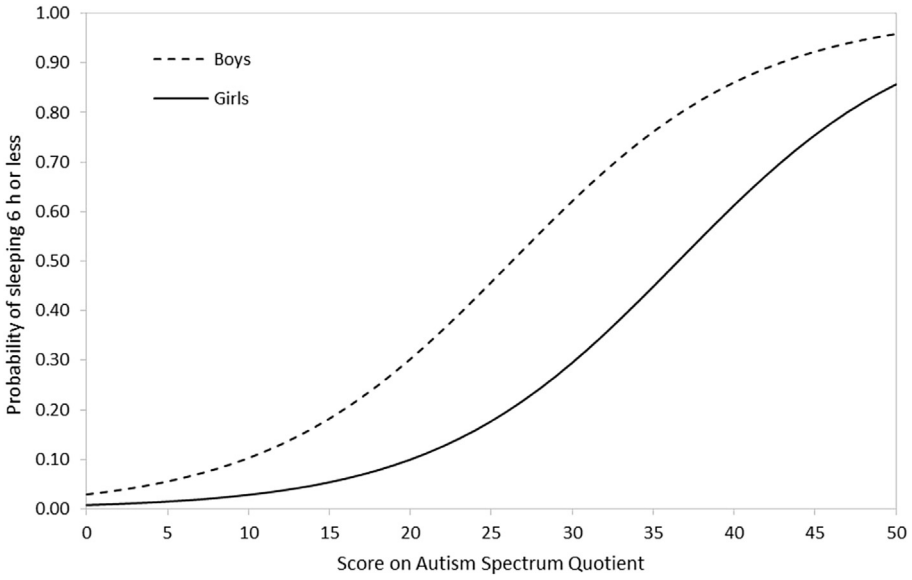

Fig. 1. Probability of short sleep duration (≤6 h) as a function of autistic trait score. In this example, adjustments for BMI, anxiety, depression and ADHD symptoms were based on mean values of the total sample and parental education was set at university degree.

illustrated in Fig. 1, having elevated autistic traits as measured by AQ (Autism Spectrum Quotient) increased the risk for short sleep duration.

One score increase on the autistic traits scale was associated with a higher odds ratio (OR) of 1.14 (95% CI: 1.03–1.26) for short total sleep time in a fully adjusted model controlling for both demographics and comorbid psychiatric symptoms. Sex had a statistically significant main effect (p = 0.032) on total sleep time.

Otherwise, no significant associations were found between autistic traits and other dichotomous sleep variables (sleep latency, sleep midpoint, wake after sleep onset, sleep efficiency and self-reported quality of sleep with PSQI). Finally, mediation analyses based on 5000 samples using bias-corrected 95% confidence intervals showed no evidence of mediation. Autistic traits had a non-significant indirect effect on total sleep time (b = −0.004; SE = 0.013; 95% CI: −0.038 to 0.018), sleep latency (b = 0.007; SE = 0.014; 95% CI: −0.015 to 0.044), sleep midpoint (b = −0.004; SE = 0.016; 95% CI: −0.050 to 0.019), WASO (b = −0.003; SE = 0.013; 95% CI: −0.037 to 0.018), sleep efficiency (b = −0.006; SE = 0.009; 95% CI: −0.027 to 0.008) and on self-reported quality of sleep (b = −0.010; SE = 0.009; 95% CI: −0.034 to 0.002) via anxiety, while controlling for covariates.

4. Discussion

4.1. Subclinical autistic traits and sleep

The purpose of this study was to examine the associations between autistic traits and actigraphy-based measurement of sleep quality, quantity, and timing in a non-clinical population of adolescents. As expected, elevated levels of autistic traits were significantly associated with shorter average weekly total sleep time.

Moreover, autistic traits remained as an independent predictor of short total sleep time when comorbid anxiety, depression and attention-deficit/hyperactivity symptoms were controlled for. We found no evidence for an association between autistic traits and sleep latency, sleep midpoint, WASO or sleep efficiency, which was against the hypotheses of this study. Similarly, no association between autistic traits and subjective reports of poor sleep quality was observed.

The current study is the first to examine the associations between autistic traits and objectively assessed sleep in a large community cohort sample. The only previous population-based study exploring the associations between autistic traits and sleep problems by Sivertsen et al., [5] found rather similar results as the current study. They [5] concluded that autistic traits independently predicted sleep problems when emotional and behavioral factors were taken into account. However, sleep problems were determined according to parent reports. In the current study, disturbed or insufficient sleep was determined by dichotomizing objectively measured sleep parameters and focusing on the more maladaptive high or low end of the sleep parameter distributions. In this study, autistic traits were only related to short total sleep time as an indication of sleep problems.

Previous actigraphy studies have suggested that high levels of autistic traits are significantly associated with shorter total sleep time [9,21]. Thus, the primary hypothesis of this study was partly supported. However, past results have been highly inconsistent and partly based on studies including children and adolescents with severe autistic traits and intellectual disabilities [21]. Indeed, Baker et al., [13] found no difference in total sleep time when comparing adolescents with high-functioning autism to typically developing adolescents. As opposed to the results in the current study, Baker et al., [13] did, however, find poor sleep efficiency and prolonged sleep latency among adolescents with high-functioning autism. Longer sleep latency among individuals with autism spectrum disorder (ASD) has been a frequent finding also in other actigraphy studies [9,11,12,21] but contradictory findings also exist [15].

Due to the focus of the previous studies being on clinical cases and controls, comparison of results should be taken with caution. The discrepancy between results could stem from the different approaches applied for studying the association between autistic traits and sleep. It is possible that subclinical variation of autistic traits in adolescence is related to both less severe and qualitatively different sleep characteristics when compared to adolescents exceeding the clinical criteria for autism spectrum disorders. In our study, only two adolescents scored at or above the threshold of 32 and the mean score of the total sample was 15 on average (possible range of scores is 0–50). Some previous studies have applied the top fifth percentile cut-off point in identifying elevated levels of autistic traits [6,7]. It is evident that a larger sample size would have been needed to be able to focus only on the highest end of the distribution.

Results of the current study should also be interpreted in the light of evidence from studies regarding the sleep quality and quantity of typically developing adolescents. In a large population-based study [29], short sleep duration, long sleep latency, and insomnia were found to be prevalent among adolescents aged between 16 and 19 years. The authors reported a mean total sleep time of 6:30 h on weekdays, mean sleep latency of 47 min and mean wake after sleep onset of 15 min. However, the estimates were based on self-reports. Hysing et al., [29] and other earlier studies [44] applied a threshold of >30 min indicating abnormally long wake after sleep onset. In our study, this cut-off point would have resulted in 80.9% of the adolescents being classified as having abnormal wake after sleep onset. Thus, long wake after sleep onset most likely reflects the typical sleep quality of adolescents in this sample. In the current study population, the theoretical cut-off of <85% for poor sleep efficiency also resulted in a relatively high proportion (36.3%) of adolescents being classified as poor sleepers. Also, poor subjective sleep quality was a common complaint among the adolescents (38.3% scoring >5 on PSQI). These findings generally support the high prevalence of disturbed and insufficient sleep among typically developing adolescents.

To summarize thus far, this study provides preliminary support for the hypothesis that elevated levels of autistic traits increase the risk for short sleep duration among typically developing adolescents regardless of sex. It is well established that short total sleep time negatively affects adolescents' daytime functioning including increased daytime sleepiness [47,48]. The effects of short sleep duration on daytime behavior, especially increased irritability, have also been demonstrated in children and adolescents with autism spectrum disorder [4]. In this study population, 93.6% of the adolescents had weekly average sleep duration less than the recommended 8 h [56]. Although short sleep duration is a typical finding among adolescents [49], our results highlight the importance of studying the different possible causes of it further. Broadening the scope of research by taking into account the subclinical autistic traits, could shed light on the issue also beyond typical developmental paths.

4.2. Subclinical autistic traits, comorbid psychiatric symptoms, and sleep

The results of this study are in line with previous studies suggesting that elevated levels of autistic traits are related to a higher degree of comorbid psychiatric symptoms also in the general population [7,8,18,19,50]. Previous studies have also found a relationship between psychiatric symptoms and sleep among typically developing adolescents [20] as well as among individuals with ASD [2,3]. However, we did not find a relationship between comorbid psychiatric symptoms and sleep quantity, quality or timing. It is possible that this discrepancy in studies results partly from the use of different methods, (ie, objective versus subjective measurement of sleep quality and quantity). This view is supported by Moore et al., [51], who found that adolescents' self-assessed sleepiness but not actigraphy estimate of sleep duration or its variability is associated with higher levels of anxiety and depression. Moreover, it is known that actigraphs may overestimate sleep efficiency and total sleep time [21].

The possible mediating role of anxiety in the association between autistic traits and disturbed or insufficient sleep was explored in our study. Although autistic traits were associated with total sleep time and with anxiety symptoms, no mediation effect of anxiety was found. These results could reflect the fact that the direction of effects between sleep and co-occurring psychiatric symptoms is not yet fully understood. A study by May et al., [52] proposed that sleep problems predict anxiety and not vice versa. However, their relatively small study population comprised adolescents of younger age and the results were based on parent or self-reports.

4.3. Strengths and limitations of the study

The strengths of this study include a representative community sample of adolescents, objective measurement of sleep quality and quantity with actigraphy, a large sample size compared to recent previous actigraphy studies focusing on adolescents [13] and using a questionnaire (the AQ) designed to capture the full spectrum of autistic traits among individuals with normal IQ. Furthermore, comorbid psychiatric symptoms were assessed using self-report measures with good psychometric properties. However, possible limitations are also recognized. The

adolescents in this study wore actigraphs on average eight nights, which is regarded as sufficient to obtain reliable results [53]. Yet, only weekday sleep data (at least four consecutive nights) were included in the analyses. The results of this study, thus, can only be compared with studies providing separate weekday and weekend sleep data. Studying both weekend and weekday data would have produced a more comprehensive understanding of adolescents' sleep patterns and their possible associations with subclinical autistic traits. Additionally, weekday sleep variables are of great interest since the largest sleep deficits are observed during the school week [49]. It is also noteworthy that due to pauses in the use of the actigraph during the daytime, napping was not monitored in our study population. The present study sample was biased towards a higher representation of adolescents with highly educated parents when compared to the population in Finland [54]. This bias can affect the generalizability of the results.

4.4. Conclusions

In conclusion, this study provided preliminary evidence that elevated levels of autistic traits increase the risk for short sleep duration in a general adolescent population. However, this study did not show consistent evidence of the associations between elevated levels of autistic traits and more maladaptive sleep patterns and sleep quality among typically developing adolescents. In general, the findings of this study support the well-established view that inadequate and disturbed sleep is highly prevalent among adolescents and highlight the need to study the underlying mechanisms further. Given the preliminary connection between autistic traits and decreased sleep duration, assessment of subclinical autistic traits should be included in studies of adolescent sleep as a possible underlying mechanism affecting sleep.

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Conflict of interest

None declared.

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.2018.09.028.

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