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SCIENTIFIC INVESTIGATIONS

Autistic Traits Are Associated With Decreased Activity of Fast Sleep Spindles During Adolescence

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Study Objectives: Autistic traits present a continuum from mild symptoms to severe disorder and have been associated with a high prevalence of sleep problems. Sleep spindles have a key function in sleep maintenance and in brain plasticity. Previous studies have found decreased spindle activity in clinical autism. Here we examine the associations between the entire range of autistic traits and sleep spindle activity in a nonclinical community cohort of adolescents.

Methods: Our cohort is based on 172 adolescents born in 1998 (58.7% girls, mean age = 16.9 years, standard deviation = 0.1), who filled in the adult autism-spectrum quotient (AQ), consisting of total score, and social interaction and attention to details subscales. Participants underwent an ambulatory overnight sleep electroencephalography. Sleep spindles (amplitude, duration, density, and intensity) were automatically detected from stage N2 sleep, and divided to slow and fast spindles.

Results: Higher AQ total sum and social interaction sum associated with lower fast spindle amplitude and intensity ($P < .04$). No associations were observed for attention to details.

Conclusions: Our findings indicate that a higher level of autistic traits in the nonclinical range among generally healthy adolescents associate with similar alterations in sleep spindle activity as observed in many neuropsychiatric conditions, indicating lower sleep-related brain plasticity. This indicates that sleep microstructures form a continuum that follows self-reported symptoms of autism.

Keywords: adolescence, AQ, autism, EEG, polysomnography, sleep spindle

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BRIEF SUMMARY

Current Knowledge/Study Rationale: Sleep maintenance problems as well as imbalanced and lower level of synaptic connectivity are often associated with elevated autistic traits. Whether this reflects in changes in sleep spindle activity, which has a key function in sleep maintenance and synaptic plasticity, is essential. Accordingly, we examined the association between autistic traits and sleep spindle activity among healthy adolescents.

Study Impact: Our findings indicate that a higher level of autistic traits during adolescence associate with lower sleep spindle activity. Alterations in sleep microstructures and sleep-related brain plasticity are not restricted to diagnosed neuropsychiatric conditions.

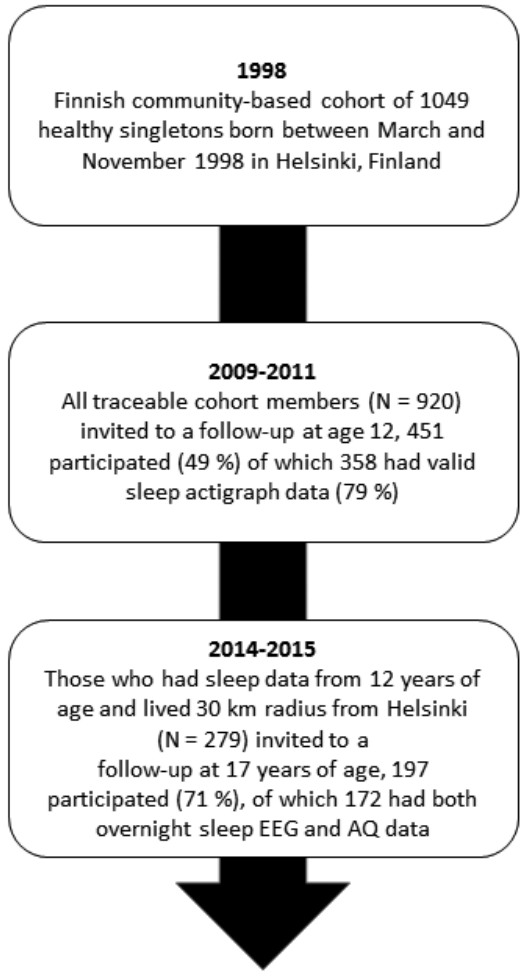
INTRODUCTION

Autistic traits consist of difficulties in social interaction and communication, restricted interests, and repetitive or restricted behavior. The estimate of the population prevalence of autism spectrum disorder (ASD) is approximately 1.5% in developed countries,[1] but autistic traits are considered to be continuously distributed in the population presenting a continuum from mild autistic traits to severe disorder.[2–5] Sleep problems commonly co-occur with ASD across all ages with a prevalence rate of 40% to 80%,[6] and a threefold risk as compared to normally developing children.[6,7] The most common sleep problems in autism include difficulties in falling asleep or maintaining sleep and parasomnias during non-rapid eye movement sleep.[7] In a large population-based longitudinal childhood cohort both autistic traits and clinical ASD were associated with sleep problems across childhood,[6] indicating that sleep problems are not exclusive to diagnosed ASD, but may co-occur even with autistic traits below clinical relevance. Furthermore, an imbalanced and lower level of synaptic connectivity has been associated with autism[8–11] and the genes influencing synaptic homeostasis have been suggested to affect both ASD and sleep.[12]

The sleep maintenance problems and imbalance in synaptic connectivity common in autism raise the question of whether there are any changes in sleep microstructure, such as in sleep spindles. Sleep spindles are brief bursts of rhythmic thalamocortical oscillations in sleep electroencephalography (EEG) at the sigma frequency range of 10 to 16 Hz and occur mainly during stage N2 sleep.[13,14] Their functional roles include maintenance of sleep,[15,16] memory consolidation during sleep,[14,17–22] and facilitation of adaptive plasticity in synaptic connections.[23–26]

Only two previous studies have examined the association between clinical ASD and sleep spindles.[27,28] The first study on 16 high-functioning adults with ASD found lower sleep spindle

Figure 1—Flowchart of the cohort.



AQ = adult autism-spectrum quotient, EEG = electroencephalography.

density in individuals with ASD as compared to controls.[27] The second study on 13 children with ASD without sleep complaints found a decreased amount of sleep spindles (lower spindle density) and lower sleep spindle duration in children with ASD as compared to 13 typically developing children.[28] Although the studies are small and restricted to clinical ASD, they raise the hypothesis whether the same phenomenon could be observed in the entire range of autistic traits in the general population.[6]

In this study, we pursue this hypothesis by examining the associations between self-reported autistic traits and sleep spindle activity among 172 adolescents from a community cohort. Furthermore, we broaden the methodological focus from sleep spindle density and duration used in previous studies[27,28] to sleep spindle amplitude and intensity. We also make a distinction between the slow and fast sleep spindles, which have different etiological backgrounds[29] as well as different temporal and topographic distributions.[29–31] We expected to find similar associations between elevated autistic traits and lowered spindle activity in our generally healthy cohort, than found in clinical samples.

METHODS

Participants

The study sample is derived from a Finnish community-based cohort of 1,049 healthy singletons born between March and November 1998 in Helsinki, Finland. The details of the cohort are described in more detail in previous reports.[32–35] As illustrated in **Figure 1**, we invited to the current study those cohort members who participated in the previous follow-up at age 12 years, had sleep measurement available from that follow-up, and lived within a 30-km radius from Helsinki (n = 279). Of them, 197 (70.6%) participated at the age of 17 years (mean age = 16.9 years, standard deviation = 0.1 years). The analytic sample comprised 172 adolescents (101 females and 71 males) who had both complete records of an overnight sleep EEG measurement and had completed the adult autism-spectrum quotient (AQ)[36] at the age of 17 years.

The analytic sample in this study did not differ significantly from the rest of the participants in the initial cohort (n = 877) regarding mother's age or body mass index (BMI) at birth, gestational age, birth weight, length at birth, or maternal alcohol or licorice consumption during pregnancy (all $P > .1$) in one-way analysis of variance. The analytic sample in this study did not differ from the rest who were invited to the follow-up but did not participate (n = 107) regarding mother's BMI at birth, gestational age, birth weight, length at birth, or maternal alcohol or licorice consumption during pregnancy (all $P \geq .05$). They did differ in mother's age at birth ($P = .04$), which was higher for the analytic sample in this study as compared with the rest of the invited participants. Three of the participants reported depression, one reported panic attacks, and one reported an eating disorder diagnosed by a doctor. None of the participants had any ASD or other psychiatric disorders diagnosed by a child or adolescent psychiatrist.

The Ethics Committee for Children and Adolescents' Diseases and Psychiatry at the Helsinki University Central Hospital approved the study protocol. All methods were performed in accordance with the relevant guidelines and regulations. All participants gave their written informed consent.

Sleep EEG Recording

As reported previously,[33] overnight polysomnography (PSG) sleep recordings were conducted in the homes of the participants with SOMNOscreen plus (SOMNOmedics GmbH, Germany). EEG was recorded with gold cup electrodes at six EEG locations (F3, F4, C3, C4, O1, and O2) and two channels for the mastoids (A1, A2) according to the standardized 10/20 system. The electro-oculogram (EOG) and the electromyogram (EMG) were measured by using disposable adhesive electrodes (Ambu Neuroline 715, Ambu A/S, Denmark), two locations for EOG and three locations for EMG. In addition, an online reference at Cz and a ground electrode in the middle of forehead were used. The sampling rate was 256 Hz (the hardware filters for SOMNOscreen plus are 0.2–35 Hz). All signals were digitally offline filtered with pass band of 0.5–40 Hz (Hamming windowed sinc zero-phase FIR filter, cutoff (−6 dB) 0.25 Hz and 44.3 Hz respectively) and re-referenced to the average signal of A1 and A2 electrodes. Sleep stages from PSG data were

scored manually with the DOMINO software (v2.7; SOMNOmedics GmbH) by three experienced researchers in 30-second epochs following the rules in The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications, Version 2.2 (2015).

Spindle Analysis

Spindles were computationally extracted with the method described by Ferrarelli et al.[37] The manually scored PSG signals were converted to EDF format in DOMINO software and then further analyzed for spindle detection by using functions of EEGlab 13.5.4b[38] running on Matlab R2015a (The Mathworks Inc., Natick, Massachusetts, United States). We extracted spindles from EEG signal during stage N2 sleep, with an impedance value equal to or lower than 10 kΩ during the corresponding 30-second epoch. The spindle analysis was conducted in two frequency bands (10–13 Hz, and 13–16 Hz) in order to differentiate respectively between the slow and fast spindles, which differ etiologically,[29] temporally, and in topographic distribution.[29–31] Before applying the spindle thresholding method, the preprocessed EEG data were further filtered using the aforementioned frequency bands separately. The threshold values for finding spindle peak amplitude in each channel were defined by the mean of the channel amplitude (μV) multiplied with 2 (lower) and 8 (higher) including all valid epochs (stage N2 sleep and impedance ≤ 10 kΩ). Thus, we used channel-wise threshold definitions, taking into account that signals may vary across the channels. Furthermore, restriction for the spindle duration was set to at least 250 ms in both directions from the peak maximum. Signal amplitude was required to stay under the lower threshold for 78.1 ms which is approximately the duration of one period of sine at 13 Hz. This was done in order to prevent false alarms in spindle detection.[33] Meeting these criteria, if the total duration of a spindle was 2000 ms or less, it was marked to be valid. The amplitude, spindle duration, intensity (spindle duration multiplied by spindle amplitude), and density (number of spindles per minute) were measured separately for central and frontal derivations and for fast and slow spindles.

Assessment of Autistic Traits

Autistic symptoms were self-reported with the 50-item AQ,[36] which has been previously used for studying autistic traits during late adolescence.[5] Each item is responded as 1 = "definitely agree"; 2 = "slightly agree"; 3 = "slightly disagree" and 4 = "definitely disagree." Answers "definitely agree" and "slightly agree" are scored as one indicating autistic tendencies for items 2, 4, 5, 6, 7, 9, 12, 13, 16, 18, 19, 20, 21, 22, 23, 26, 33, 35, 39, 41, 42, 43, 45, 46, and similarly was done for answers "slightly disagree" and "definitely disagree" for items 1, 3, 8, 10, 11, 14, 15, 17, 24, 25, 27, 28, 29, 30, 31, 32, 34, 36, 37, 38, 40, 44, 47, 48, 49, 50. The total AQ sum Cronbach alpha is .74. A previous study has shown that the AQ scores are continuously distributed in the general population during late adolescence.[5]

A population and ASD-based study has confirmed two subscales in the original AQ,[4] that were used in this study (correlation between subscales $r = .02$, $P = .8$). The first social interaction subscale encompasses the four original subscales of social skills, imagination, attention switching, and communication (Cronbach alpha = .76) and the second subscale encompasses the original attention to details subscale (Cronbach alpha = .62).

Potential Confounders

Sex was derived from the birth records and participant age was calculated on the day of sleep EEG recording. Maternal licorice consumption during pregnancy was self-reported shortly after delivery,[39] and categorized as zero or low (0–249 mg glycyrrhizin/wk) and moderate (250–500 mg glycyrrhizin/wk) or high consumption (≥ 500 mg glycyrrhizin/wk). Licorice consumption during pregnancy was included as a potential confounder as the previous follow-ups of this sample showed that high maternal licorice consumption during pregnancy increased the risk for cognitive impairment and psychiatric symptoms in 8-year-old[39] and 12-year-old children.[40] Sleep duration, wake after sleep onset (WASO), and sleep staging were derived from the sleep EEG recording explained previously.

Statistical Analysis

To study the initial associations between the studied variables for the selection of potential covariates, we used one-way analysis of variance for categorical variables and correlation analyses for continuous variables.

We used multiple linear regression analysis to study the associations between standardized sum scores for autistic traits and sleep spindle activity. Principal component analyses (PCA) with Varimax rotation were used to reduce the number of sleep spindle variables and potential type I error[29,41,42] reflecting amplitude, density, duration, and intensity at central and frontal derivation. PCA was performed based on standardized sleep spindle variables. PCA is the most common statistical method for reducing the number of interrelated variables while retaining the information of the original data by converting them to uncorrelated principal component variables.[43] Analyses were performed separately for slow and fast spindles. All regression analyses were adjusted for sex and stage N2 sleep duration. We used SPSS version 24.0 for all statistical analyses (IBM Corp, Armonk, New York, United States).

RESULTS

PCA for Sleep Spindle Variables

PCA using Varimax rotation produced one-factor solution for slow sleep spindle amplitude, fast sleep spindle amplitude, slow sleep spindle density, fast sleep spindle density, slow sleep spindle intensity, fast sleep spindle intensity, and fast sleep spindle duration. For slow sleep spindle duration, PCA using Varimax rotation produced a two-factor solution. The results from the PCA are outlined in **Table 1**.

Descriptive Statistics of General Sleep Characteristics by Sex and Initial Analyses Between Autistic Traits and Potential Covariates

As reported before,[33] PSG-based general sleep characteristics differed between sexes, with girls having longer sleep duration

Table 1—The results from the PCA using Varimax rotation for sleep spindle variables.

Factor Solution	Correlation Matrix Determinant	MSA	Variance Counted (%)	Component Loading C3 Channel	Component Loading C4 Channel	Component Loading F3 Channel	Component Loading F4 Channel
Slow sleep spindle amplitude	0.1	0.72	65.8	0.90	0.90	0.84	0.54
Fast sleep spindle amplitude	0.05	0.68	67.5	0.92	0.92	0.83	0.57
Slow sleep spindle density	0.4	0.61	52.2	0.76	0.86	0.64	0.61
Fast sleep spindle density	0.5	0.68	49.1	0.81	0.81	0.67	0.45
Slow sleep spindle duration	0.2	0.58					
Slow central sleep spindle duration			58.7	0.94	0.95		
Slow frontal sleep spindle duration			28.5			0.92	0.87
Fast sleep spindle duration	0.1	0.71	69.1	0.87	0.89	0.87	0.67
Slow sleep spindle intensity	0.1	0.71	68.7	0.89	0.90	0.84	0.66
Fast sleep spindle intensity	0.03	0.70	72.8	0.91	0.91	0.87	0.70

Kaiser-Meyer-Olkin MSA was weighted by all the channels for central and frontal derivations in PCA. MSA = measure of sampling adequacy, PCA = principal component analysis.

Table 2—Descriptive statistics by sex.

	Females (n = 101)	Males (n = 71)	*P*
AQ total sum	13.9 ± 4.8	16.5 ± 6.0	.001
Social interaction subscale sum score	9.5 ± 4.2	12.6 ± 5.4	.00004
Attention to details subscale sum score	4.4 ± 2.1	3.9 ± 2.3	.2
Sleep duration (hh:mm)	7:50 ± 1:04	7:24 ± 1:14	.02
Wake after sleep onset (hh:mm)	0:32 ± 0:23	0:35 ± 0:26	.3
REM sleep duration (hh:mm)	1:42 ± 0:28	1:30 ± 0:31	.009
NREM sleep duration (hh:mm)	6:08 ± 0:48	5:54 ± 0:58	.09
Stage N1 sleep duration (hh:mm)	0:48 ± 0:21	0:53 ± 0:22	.1
Stage N2 sleep duration (hh:mm)	3:18 ± 0:38	2:58 ± 0:46	.003
Stage N3 sleep duration (hh:mm)	2:02 ± 0:27	2:03 ± 0:28	.9

Values presented as mean ± standard deviation. AQ = adult autism-spectrum quotient, NREM = non-rapid eye movement, REM = rapid eye movement.

(P = .02), REM sleep duration (P = .009), and stage N2 sleep duration (P = .003) than boys (**Table 2**).

As **Table 2** shows, boys had higher AQ total sum score and social interaction sum score than girls. Maternal licorice consumption during pregnancy did not correlate significantly with autistic traits ($P \geq .07$). There were no significant correlations between the sleep staging, sleep duration, or WASO minutes and AQ scores (for the correlations between general sleep characteristics and AQ scores $P \geq .07$). As **Table 3** shows, there were no differences between AQ total sum score subgroups and general sleep characteristics (all $P > .1$). Final models included sex, age, and stage N2 sleep duration as covariates, these having been associated with sleep spindle activity.[23]

Associations Between Autistic Traits and Sleep Spindle Activity

The associations from the regression analyses between autistic traits and sleep spindle activity are presented in **Table 4**. One standard deviation (SD) unit higher AQ total sum score was associated with 0.2 SD unit lower fast sleep spindle amplitude and 0.2 SD unit weaker fast sleep spindle intensity. Of the two AQ subscales, one SD unit higher social interaction subscale sum score was associated with 0.2 SD unit lower fast sleep spindle amplitude and 0.2 SD unit weaker fast sleep spindle intensity. No significant associations between sleep spindle activity and attention to details subscale were found (all $P \geq 0.1$).

DISCUSSION

Our study is the first to report the associations between nonclinical autistic traits and sleep spindle activity in an adolescent community cohort. Our results indicate that elevated autistic traits, especially those related to social interaction, are associated with lower fast sleep spindle amplitude and weaker fast sleep spindle intensity.

Similar to the two previous sleep spindle studies in ASD children (mean age 10 years)[28] and young adults (mean age 21 years),[27] the current results showed lower sleep spindle activity associated with elevated autistic traits in adolescence. Although our effect size in this nonclinical sample was small, it has to be noted that the 0.2 SD unit change is sleep spindles corresponded to only five-point differences (1 SD) in the AQ scores. However, according to a meta-analysis[44] the clinical cutoff of 35 points corresponds to approximately + 3 SD increase (18 absolute points in the scale) from the mean AQ score.

Table 3—Descriptive statistics by AQ subgroups.

Sleep Characteristics (hh:mm)	AQ Total Sum Score				*P*
	5 to 10 (n = 37)	> 10 to 15 (n = 65)	> 15 to 20 (n = 43)	> 20 to 37 (n = 27)	
Sleep duration	7:34 ± 1:10	7:44 ± 1:02	7:49 ± 1:08	7:19 ± 1:25	.3
Wake after sleep onset	0:31 ± 0:20	0:36 ± 0:28	0:33 ± 0:26	0:28 ± 0:17	.5
REM sleep duration	1:31 ± 0:29	1:40 ± 0:30	1:39 ± 0:28	1:34 ± 0:34	.5
NREM sleep duration	6:02 ± 0:51	6:05 ± 0:47	6:10 ± 0:54	5:46 ± 1:03	.3
Stage N1 sleep duration	0:52 ± 0:20	0:49 ± 0:19	0:53 ± 0:25	0:46 ± 0:26	.6
Stage N2 sleep duration	3:14 ± 0:43	3:12 ± 0:42	3:14 ± 0:39	2:51 ± 0:47	.1
Stage N3 sleep duration	1:56 ± 0:27	2:04 ± 0:25	2:02 ± 0:32	2:08 ± 0:27	.4

Values are presented as mean ± standard deviation. AQ = adult autism-spectrum quotient, NREM = non-rapid eye movement, REM = rapid eye movement.

Table 4—Results from regression analysis for spindle characteristics by autistic traits with sex and stage N2 sleep duration as covariates.

Spindle Characteristics	AQ Total Sum Score		Social Interaction Subscale Sum Score		Attention to Details Subscale Sum Score	
	B (95 % CI)	*P*	B (95 % CI)	*P*	B (95 % CI)	*P*
Amplitude (μV)						
Slow	−0.2 (−0.3 to 0.02)	.08	−0.1(−0.3 to 0.04)	.1	−0.09 (−0.3 to 0.09)	.3
Fast	−0.2 (−0.36 to −0.01)	.04	−0.2 (−0.4 to −0.04)	.01	0.05 (−0.1 to 0.2)	.6
Density (number of spindles per 60 seconds)						
Slow	0.02 (−0. to 0.2)	.8	0.02 (−0.2 to 0.2)	.8	0.001 (−0.2 to 0.2)	.9
Fast	0.03 (−0.1 to 0.2)	.7	−0.02 (−0.2 to 0.2)	.8	0.1 (−0.04 to 0.3)	.1
Duration (seconds)						
Slow in central	0.09 (−0.08 to 0.3)	.3	0.1 (−0.03 to 0.3)	.1	−0.1 (−0.3 to 0.06)	.2
Slow in frontal	−0.05 (−0.2 to 0.1)	.6	−0.07 (−0.2 to 0.1)	.5	0.04 (−0.2 to 0.2)	.7
Fast	−0.1 (−0.3 to 0.8)	.2	−0.1 (−0.3 to 0.03)	.1	0.06 (−0.1 to 0.3)	.5
Intensity (duration [seconds] multiplied by amplitude [μV])						
Slow	−0.1 (−0.3 to 0.05)	.2	−0.09 (−0.3 to 0.08)	.3	−0.1 (−0.3 to 0.08)	.3
Fast	−0.2 (−0.3 to −0.007)	.04	−0.2 (−0.4 to −0.04)	.01	0.06 (−0.1 to 0.2)	.5

AQ = adult autism-spectrum quotient, CI = confidence interval.

In addition to showing that the association is valid among the normal range or symptoms, the current study contributed to the literature by presenting a more fine-grained sleep spindle analyses than in previous studies,[27,28] including a differentiation between fast and slow spindles. We found that higher AQ total sum score and social interaction subscale sum score were associated with both lower sleep spindle amplitude and intensity, and especially at the fast spindle range. Fast spindles increase in their activity as compared to slow spindles during adolescence,[23] when synaptic pruning downsizes synapses for improved neural functioning.[45] Fast sleep spindles also show greater heritability than slow sleep spindles.[46] Considering the high heritability estimates of autism[47] and the suggested pleiotropic effects of genes influencing to synaptic homeostasis, sleep, and predisposition to autism,[12] it is possible that these same genes contribute to fast sleep spindle activity. We did not replicate the prior finding related to sleep spindle density or duration.[27,28]

Our results can be viewed in light of previous findings showing deficits in neurotransmitter pathways related to sleep spindle formation in autism. The abnormal function of gamma-aminobutyric acid (GABA)ergic neurons in thalamic reticular nucleus has been suggested to associate with autism[48] and they are also essential for the production of sleep spindles.[23] Also genetic variants for $GABA_A$ receptor are associated with higher risk for autism.[49]

A recent systematic review showed elevated prevalence rates of ASD at the diagnostic level and at the trait level in psychotic disorders.[50] We have previously shown in this cohort that schizotypal personality symptoms[51] and genetic variants for schizophrenia[52] associated with sleep spindle activity. In line with the finding between elevated autistic traits and lowered sleep spindle activity here, we found that elevated schizotypal personality symptoms were associated with lower sleep spindle activity.[51] However, the heightened genetic predisposition towards schizophrenia in nonschizophrenic adolescents was positively associated with sleep spindle activity.[52] If there are similar possible discrepancies with self-reported and genetic factors of ASD regarding sleep spindle activity, it remains to be seen.

Problems regarding maintenance of sleep seem to be intertwined in the symptomatology of autism.[6] Sleep spindles

have a key role in maintenance of sleep by blocking external stimuli processing during sleep[15,16]; thus, deficits in sleep spindle activity can explain why fragmentation of sleep is common among actual autistic patients.[7] In addition, sleep spindles function in facilitating adaptive plasticity in synaptic connections,[23–26] reflected in overnight memory consolidation processes.[14,17–22] Noteworthy, autism has previously been associated with lowered synaptic connectivity.[8–11] Because sleep spindles are a marker of synaptic plasticity, our findings suggest that even in the subclinical range of autistic traits, there might be sleep-related processes that contribute to the synaptic plasticity.

Moreover, our results can also reflect a general tendency of lower sleep spindle activity seen in many neurological or psychiatric conditions.[37,53–57] This may reflect common, rather that autism-specific, pathological processes of brain maturation and function in these disorders that are associated with altered sleep.[58]

Strengths and Limitations

The strengths of our study include methodological aspects. First, participants slept according to their normal schedule in their home environment, which should improve ecological validity as compared to sleeping in unfamiliar laboratory settings. Second, the homogenous age distribution of our sample increases the reliability of our study as spindles are rather age dependent and age differences in the sample may skew the results.[23] Third, control of the EEG data quality was good, with an impedance control at all channels. However, somnographic data were available only for 1 night. In order to increase data reliability, data recordings during 2 or more nights would be recommended. As a limitation, although the method used in this study by Ferrarelli et al.[37,54] is widely applied, there is still lack of cross-validation studies across different sleep spindle analysis methods.[29] We also acknowledge that the translation of entire range of AQ scores to autistic activity is not validated. However, most of the items in AQ refer to issues of inner states, preferences, feelings and thoughts, not directly to behavioral activity. Although some of these would be measurable "autistic activity," most of the items touch on issues that may not be observable from outside. Thus, validation of such states is not easily feasible, and reliance of self-reports may be the best way of addressing these subtle characteristics.

CONCLUSIONS

Our study is the first to report that elevated autistic traits are related to lower sleep spindle activity, specifically with regard to fast spindles during adolescence. Our results show that a higher level of autistic traits even in the nonclinical range is associated with similar alterations in sleep spindle activity at frontal areas as observed in many neuropsychiatric conditions. As sleep spindles influence the adaptive plasticity of synaptic connections, our findings highlight that even in the nonclinical range, elevated autistic traits are related to lowered sleep spindle activity that may contribute to the synaptic plasticity.

ABBREVIATIONS

AQ, adult autism-spectrum quotient
ASD, autism spectrum disorder
BMI, body mass index
EEG, electroencephalography
EMG, electromyogram
EOG, electro-oculogram
PCA, principal component analyses
PSG, polysomnography
WASO, wake after sleep onset

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