QEEG Characteristic and Spectral Analysis in Adults Diagnosed with Autism Spectrum Disorder

Carline Ouattara

Snr: 2010058; Anr: 700100

Supervisor: Dr. Geert van Boxtel

Independent assessor: Dr. Thijs van Laarhoven

July 6, 2021

Abstract

This study aims to investigate the differences in QEEG spectral analysis of adults diagnosed with ASD to a clinical control group. It intends to add to the research into using QEEG as a possible diagnostic tool for ASD. In total a sample of 78 participants, with 38 in the ASD group and 40 in the control group, were recruited and used to analyze, by using spectral analysis, the differences in frequency bands (alpha, theta, delta, and beta) at three regions in the brain (frontal, temporal, and occipital). The results showed no differences in the absolute power of delta in the frontal, temporal and occipital region of the brain. And, no differences between the two groups' absolute power of theta in the frontal, temporal, and occipital were found. Furthermore, did the results conclude no differences between the absolute power of alpha and beta between the ASD group and the control group in the temporal and occipital region. In spite of the current study's non-significant results, it still adds substantially to the research on QEEG in ASD. The importance of finding out more about the possible differences in frequencies in order to use QEEG as a possible diagnostic technique is still relevant. Especially the difficulties in the diagnostic process of adults with ASD would benefit greatly from an added biological and objective tool. Therefore, further research is needed to gain more insight into the relationship between ASD and absolute power of frequency bands.

Keywords: autism spectrum disorder, quantitative electroencephalography, spectral analysis, diagnostic process

QEEG Characteristic and Spectral Analysis in Adults Diagnosed with Autism Spectrum Disorder

Autism spectrum disorder (ASD), often also referred to as autism, is a term for a cluster of complex neurodevelopmental disorders. These are characterized by different degrees of social interaction, verbal and nonverbal communication impairments, restricted range of interests, and repetitive behavior according to the diagnostic criteria 299.0 in the American Psychiatric Association's Diagnostic and Statistical Manual, Fifth Edition (APA, 2013). Social interaction and communication impairments often include difficulties in holding a conversation, such as decreased sharing of personal interests and emotions. Furthermore, individuals with ASD can present difficulties in reading social cues, such as eye contact and facial expressions. This can lead to a deficit in the forming and maintaining of meaningful personal relationships. People with ASD frequently require a predictable routine and adherence to structure in their day-to-day life. Commonly, they have an extreme interest in activities that are unusual in their age-related peer group or, concerning children, that they play with toys in an uncommon way, such as lining up toy cars instead of driving them. Repetitive behavior like hand-flapping, rocking their body, using odd patterns in speaking, or speaking in citations from their favorite show are additional symptoms people with ASD often display. Also, an intense or extreme sensory experience of their surrounding is a common symptom in ASD. This can be expressed by, for example, the indifference to pain or temperature and a fascination with lights and movements. However, this can also go hand in hand with being more easily overwhelmed by loud noises and harsh lights than their neurotypically developed peers (APA, 2013)

The effects and severity of symptoms of ASD present themselves differently in each individual. (APA, 2013). Symptoms can range from mild to very severe, can change over time, but persist life-long and can lead to a poor outcome during adulthood (APA, 2013).

Usually, symptoms appear during early childhood, between the ages two and three. Nevertheless, in some cases, children appear to develop normally until toddlerhood whereon after they either stop acquiring new skills or lose already gained ones. Furthermore, has the DSM 5 newly recognized the possibility, that adolescence and adults can get diagnosed later in life as well, as there is a possibility that there are people whose impairments and symptoms will only become fully visible when "social communication demands exceed limited capacities" (APA, 2013). Thus, many clinicians and psychologist are trying to assess and diagnosis this "lost generation" of adults that previously missed the age and chance to get diagnosed (Lai et al. 2015).

Multiple factors pose challenges to the diagnosis of ASD in adulthoods. As it is a neurodevelopment disorder, to be able to get a diagnosis the patient must prove, that the symptoms already existed during childhood. Thus, one of the first challenges for the therapist or clinician is gathering enough information about the developmental history of the patient. However, parents or other caregivers may not be available, and /or the information given may not be reliable due to the time that has passed between the childhood and the assessment (Fusar-Poli et al. 2017). Furthermore, adults having lived undiagnosed for some time might have developed coping strategies, which can mask possible core symptoms the patient in fact has (Fusar-Poli et al. 2017). Especially adults with higher intelligence and cognitive abilities could have acquired the strategies. All of this makes it more challenging for the health care worker to diagnose the patients correctly. Another important point is, that adults with a higher intelligence and coping strategies ay recognize their impairments only well into adolescence or adulthood (DSM-5 2013). Other reasons why adults with ASD receive no or very late diagnosis may be that they get misdiagnosed. Multiple other disorders overlap with symptoms of ASD, such as different personality disorder, anxiety disorders and other neurodevelopmental disorders, such as attention deficit-hyperactive disorder (Lai et al 2019). Furthermore, there is a large comorbidity with anxiety disorders, depressive disorders and

schizophrenia in the ASD population compared to the neurotypical population (Lai et al. 2019).

Quantitative Electroencephalography (QEEG)

Quite recently neuroscientists and psychologists have begun to examine the connections of neural correlations to the symptoms and behaviors of patients with ASD (Rippon et al., 2007). Several different neuroimaging techniques have been looked at to use for the identification and characterization of central biological organizing principles for the dysfunction in ASD. Measures such as functional magnetic resonance imaging and electroencephalography (EEG) have been used to distinguish individuals with ASD from the neurotypical population (Murias et al. 2007). It has been hypothesized that people with ASD possibly differ from their neurotypical peers by different patterns of EEG activity and functional connection among different brain regions (Murias et al. 2007; Jokiranta et al., 2014). Furthermore, has QEEG been investigated as a possible diagnosing technique for the different subtypes of ASD and as an option to use in interventions and possible treatment option through the usage of neurofeedback (Gurau, Bosl, & Newton 2017).

Additionally, EEG is a non-invasive neuroimaging procedure used to support the diagnosis of brain-related disorders and symptoms such as Alzheimer's Disease, epilepsy, and the effects of traumatic brain injury. Electrodes are put at the scalp and record the synchronized activity of the neurons in the respectable tissue underneath (Kilmesch et al., 2007). Often, EEG recordings are either performed at rest, in both eyes-open and eyes-closed conditions, or during the performance of a specific task. Following the analysis, normative or control data is needed to compare the output and give meaning to the functional information obtained. (Billeci et al., 2013)

Quantitative Electroencephalography (QEEG) is gaining interest as a technique used to study neurodevelopmental disorders and is increasingly used in studies, especially for ASD. QEEG is defined as ". . . the mathematical processing of digitally recorded EEG in

order to highlight specific waveform components, transform the EEG into a format or domain that elucidates relevant information, or associate numerical results . . .” (Nuwer, 1997). Thus, using statistical algorithms raw EEG data is transformed, into, usually, five frequency bands. Namely, delta (0.5–3 Hz), theta (3–7 Hz), alpha (8–13 Hz), beta (14– 36 Hz) and gamma (36–44 Hz) (Steriade et al., 1990).

Spectral analysis is the most common QEEG method used to analyze and interpret EEG signals. The continuous ranges of frequency are broken down into defined bands, from which the signal distribution is then evaluated. Both the absolute and the relative spectral power can be computed for each frequency band at each sensor (Gurau et al., 2017). Absolute EEG power is the amount of energy in μV^2, whereas relative EEG power is the percentage of total power within the frequency bands.

Each frequency band activity seems to be associated with certain cortical functions, but no consensus has yet been reached on these (Knyazev et al., 2007). Alpha waves are mainly generated during states of alertness. Also, beta and alpha rhythms are thought to be related to attention and voluntary-controlled visual behavior. While alpha is mostly generated in the occipital region, beta is most evident in the frontal region. Lower frequency rhythms, such as delta and theta are frequently used during the execution of automatic brain functions. Delta rhythms are mostly activated during motivation activity, whereas theta rhythms are related to working memory and the recognition of emotions (Knyazev et al., 2007; Aftanas et al., 2001).

Alpha rhythms are especially interesting to the study of ASD as they are hypothesized to represent the level of cortical excitability. During resting states, alpha waves are the dominant EEG signal to be found and they have been linked to attention, perceptual processing, and semantic memory. Higher resting alpha power is assumed to indicate cortical deactivation or inactivity (Kilmesch et al., 2007; Knyazev, 2007). Thus, individual differences in resting-state alpha power can show a person’s capacity for selective inhibition of irrelevant

network regions (Klimesch et al., 2007). Additionally, lower alpha resting power has been linked to greater neuronal excitability (Neuper & Pfurtscheller, 2001). Other evidence suggests that higher alpha resting power could show a high state of preparedness to perform demanding tasks. Behavioral evidence supports this, as higher resting alpha power is associated with successful response inhibition and improved performance on cognitive tasks, such as mental manipulation and target recognition (Klimesch et al., 2003; Mathewson et al., 2012).

Previous findings in spectral analysis differences

Multiple studies have reported significant differences in frequency bands, especially for alpha rhythms, for different disorders, such as schizophrenia and cognitive impairment (Alfimova & Uvarova, 2008; Babiloni et al., 2007). However, there is a distinct lack of knowledge on the neural activity, function, and connection in the adult ASD population.

Elhabashy et al. (2015) showed that children with ASD had a significant difference in EEG power compared to the control group. Furthermore, they were able to show that the frontal regions in children with ASD had a greater absolute power of delta and theta frequency bands compared to the control group. The central regions showed a reduction in absolute alpha and beta frequency power compared to the control group. Additionally, the authors found significant differences in relative power between the ASD group and the control group. The right central regions in the ASD group showed significantly greater relative theta band power compared to the control group. However, there was an overall reduction of relative EEG power of the alpha and beta frequency bands (Elhabashy et al., 2015).

Partly consistent with these results are the findings of Sheikani et al. (2012). Their study analyzed the QEEG data of 17 children diagnosed with ASD and 11 children in the control group between the ages of eleven and six. The study's aim was to evaluate if there is a significant difference in the QEEG data, which subsequently could be used to help diagnose

ASD in children. All the children in the ASD group were diagnosed after the DSM-IV-TR criteria by two child and adolescent psychologist. Results showed that children with ASD had significantly lower values at multiple electrodes in the alpha frequency band of the left hemisphere compared to the control group. Additionally, the results showed differences in beta rhythms and gamma rhythms between the control group and the ASD group. According to Sheikani et al. (2012) alpha reflects the coordination of wider areas of the brain and beta shows an integration role in neighboring areas of the brain. From this they concluded, that the alpha and beta differences showed that the abnormalities in ASD were likely to be related to the coordination of broader brain areas (Sheikani et al., 2012).

Similarly, the QEEG study of Coben et al. (2008) examined differences between ASD children and a control group. A group of 20 children between the ages of six and eleven diagnosed with ASD was compared to a control group. Their results showed an increase in relative theta power in ASD children, especially in the right posterior region. In addition, a reduction in absolute beta power over the right hemisphere and an increased beta power at the midline was found in the ASD group. The authors concluded that the results of heightened theta power suggest that this seems to be an area of abnormal functioning in children with ASD (Coben et al., 2008).

Furthermore, Lushcekina et al. (2014) found significant differences in spectral power, comparing children diagnosed with early childhood autism (mean age = 6 years) to neurotypically developing children (mean age = 6 years old). Contradictory to the findings of Coben et al. (2008) the results showed, that children diagnosed with ASD had lower levels of spectral power in the theta rhythm (4-7.5 Hz) and that the gamma rhythm (45-65 Hz) was overall higher in spectral power, compared to the control group. The control children showed an increase in the theta rhythm in the right hemisphere and an increase in gamma rhythm in the left hemisphere, during the cognitive task condition. These increases did not happen, during the same condition, in the children with ASD. Thus, it was concluded that the

differences between the two groups are the result of different neurophysiological mechanisms children with ASD use to successfully perform cognitive task (Lushcekina et al., 2014).

QEEG studies using children with ASD as subjects have shown significant differences in absolute and relative power, asymmetry, and coherence in EEG data compared to neurotypically developed children in various different brain regions (Coben et al., 2008; Elhabashy et.al., 2015; Lushcekina et al., 2014; Sheikani et al., 2012). Even though ASD is a neurodevelopment disorder that stays with the patients' life long and can have severe effects on their lives there is, however, still a lack of focus on the adult population, which can be observed in the previously published research. Nevertheless, the currently available studies that focused their research on the adult ASD population have shown a distinct lack of consistencies in their results.

The study by Mathewson et al. (2012) reported an overall greater alpha in all of the regions of the brain in their experimental group of 15 patients (age range= 18-51 years) diagnosed with the ASD subtype Asperger syndrome, compared to their control group (n=16; age range =22-47 years). In contrast to that, Tani et al. (2004) found a non-significant trend in decreased delta power and increased theta power in their ASD group of 20 adults, diagnosed with the ASD subtype Asperger syndrome that were compared to a clinically healthy control group in a QEEG study. Daoust et al. (2004) used both children and adults in their study and, found higher absolute power of theta in the left frontal region in the relaxed eyes open condition.

Purpose of this study

As previously shown the difficulty of ASD in adulthood is a problem, which can lead not only to misdiagnosis, but also to the complete missing of the diagnosis in ASD. An easy biological screening tool would go a long way in helping the clinicians and patients. A neurobiological measurement such as QEEG would provide a more objective diagnosis for ASD, than solely relying on self-reporting measures and interviews. Multiple studies have

already started analyzing the effectiveness of QEG in the diagnostic procedure for children with ASD, as the meta-analysis by Gurau et al. (2017) concluded that there is some indication for the usage of QEEG and more specifically, spectral analysis in the diagnostic procedure of ASD. However, there are still too many inconsistencies in the results and findings to fully rely on it.

Due to these large inconsistencies in the findings of previous studies on children and the lack of research into the adult population of individuals with ASD, this study intends to add to the research into using QEEG as a possible diagnostic tool for ASD. The aim of this study is to compare whether EEG power in a resting state would differ between adults diagnosed with ASD and a neurotypical control group. To extend previous research on this topic this study will compare an adult population diagnosed with ASD to a neurotypical adult control group and examine whether there is a difference in their absolute power values. More specifically, this study presents four hypotheses regarding those differences. The first is that the ASD group will differ from the control group in their absolute power of delta in the frontal, temporal, and occipital region of the brain. The second hypothesis, is that the ASD group will differ from the control group in their absolute power theta in the frontal, temporal, and occipital region. The third hypothesis is that the ASD group will differ from the control group in absolute power beta in the temporal region. The fourth and final hypothesis is that the ASD group will differ in absolute power of alpha in the temporal and occipital region of the brain.

Method

Participants

All subjects were current or previous patients at the *Neuropsychiatrisches Zentrum Hamburg-Altona* (NPZ) in Hamburg, Germany at the time of the study. EEG recordings were collected as part of their individual diagnostic processes. Decisive for the participation in the study was ASD diagnosis according to the International Classification of Diseases F84.0

(ICD-10, 2004) and the additional clinical evaluation by the treating psychotherapist, psychologist, or psychiatrist. Patients with known chronic medical, psychiatric, and/or neurological disorders and conditions, other than ASD, were excluded from the study. All participants were highly functional and ranged in the normal to high average of approximate intelligence and cognitive abilities.

The data from the non-clinical and medication free control group was collected from previously done EEG recordings of patients, who had been to the NPZ but had not received a diagnosis. Exclusion criteria were, similar to the ASD group, a history of head injury, chronic medical conditions, and/or neurological disorders or conditions. The distribution of sex was the same across both groups.

The Ethical Committee of the Neuropsychiatric Center Hamburg Altona NPZ GmbH in Hamburg, Germany, has approved this study under the registration number EK-2021-OUATTARA_01.

Measurements

Electroencephalography Recordings

All EEG recordings for the ASD group were obtained in one 20-minute-long session, during which the patients were sitting in a reclining chair in a dimmed and noise-controlled room. EEG traces were recorded during two conditions, resting eyes-closed and resting eyes-open. They were instructed to stay still and to keep their mouths slightly opened. Depending on the condition, participants were instructed to keep their eyes either opened or closed. At least one experienced technician was present during the recording, who instructed the patients during the conditions and reminded them to keep still in case of movements or talking. The EEG data was recorded using Neurosoft Neuron-Spectrum-4/P (Neurosoft, Ivanovo, Russia) with a sampling rate of 256 Hz. Electrodes were placed according to the international 10-20 system from 19 standard scalp locations, including, *Fp1, Fp2, F3, F4, F7, F8, T3, T4, C3, C4,*

P3, P4, T5, T6, O1, O2, Fz, C7, and Pz (Jasper H.H., 1958). Two reference electrodes, A1 and A2, were placed on both earlobes.

The raw EEG data was preprocessed in multiple steps, done both manually and statistically. First, artefacts, such as swallowing, movements, talking, and coughing, were identified, by trained technicians, and subsequently removed. Furthermore, settings were set to low-cutoff filter of 0.5 Hz, high-frequency filter of 70 Hz, and a notch filter at 50 Hz. Epochs of artefact free data were chosen for each subject and used for analysis.

Quantitative Electroencephalography spectral analysis

Fourier power spectral analysis was used to determine the magnitude of each frequency band in microvolt, after which the data was then transformed into three frequency bands. Namely, delta (0.5–3 Hz), theta (3–7 Hz), alpha (8–13 Hz), and beta (13-30 Hz). The absolute power ($\mu V^2/Hz$) was computed for each frequency band.

The electrodes power was averaged, after which they were grouped into five regions representing parts of the topography of the brain. Those regions were, frontal (Fp1 and Fp2), left frontal (Fp1 and F7), right frontal (Fp2 and F8), temporal (T5 and T6), and occipital (O1 and O2). Additionally, a log-transformation (10*log10(power)) was applied to the data.

To determine the number of participants needed to possibly achieve a significant result previous to the testing, a priori power analysis was conducted using G*power 3 (Faul et al., 2007) to test the differences between the ASD group and the neurotypical control group, using a repeated measure of variance analysis (ANOVA), achieving a medium effect size (f= 0.25) and an alpha of 0.05. The results showed that, to achieve a power of .95 a total number of 72 participants is required which results in two equal size groups of $n = 36$.

To compare the differences in sociodemographic characteristics between the ASD group and the control group, numerical data was expressed as mean and SD and qualitative data were expressed as frequency and percentage. Following, a chi-square-test was used to examine the relation between categorical variables and an independent sample t-test was

conducted for the continuous variables. It was expected that the values for spectral power will have near normal distributions, thus parametric statistics could be used. Next, an independent t-test was used to compare the individual electrodes absolute power between the ASD group and the control group. The single electrodes were the dependent variables, while diagnosis (ASD, control group) was the independent variable. Differences in absolute power for each frequency band, in the different regions, between the two groups were assessed in using repeated measure ANOVA with diagnosis as the between-subject factor and the regions as the within-subject factor. The existence of significant differences in spectral characteristics between the SD group and control group was accepted at a significance of $p < 0.05$. In all cases, age was controlled for and used as a covariate. Furthermore, were outliers and values of zero were counted as missing variables.

Results

Demographic Characteristics

For the final analysis the data of 78 patients were used. The ASD group was comprised of 38 patients and the control group of 40. There were no significant differences in gender between the groups. However, the control group was significantly older compared to the ASD group (Table 1).

Table 1

Sociodemographic characteristics of the participants divided by diagnosis

	ASD group (n=38, 48%)	Control group (n=40, 52%)	P value
Age (mean (SD))	35,26 (10,3)	46,83 (19,7)	.002
Gender			.512
Male	18 (47%)	16 (40%)	
Female	20 (53%)	24 (60%)	

Note. SD = Standard Deviation.

Comparing absolute power over the different brain regions

Frequency distribution

For each frequency band (alpha, theta, delta, and beta) for both the control group (Figure 1) and the ASD group (Figure 2) a scalp map was constructed. The scale and colors represent the distribution of the absolute power of the frequency bands measured at the electrodes across all regions of the brain.

Figure 1

Frequency band distribution over the scalp in the control group

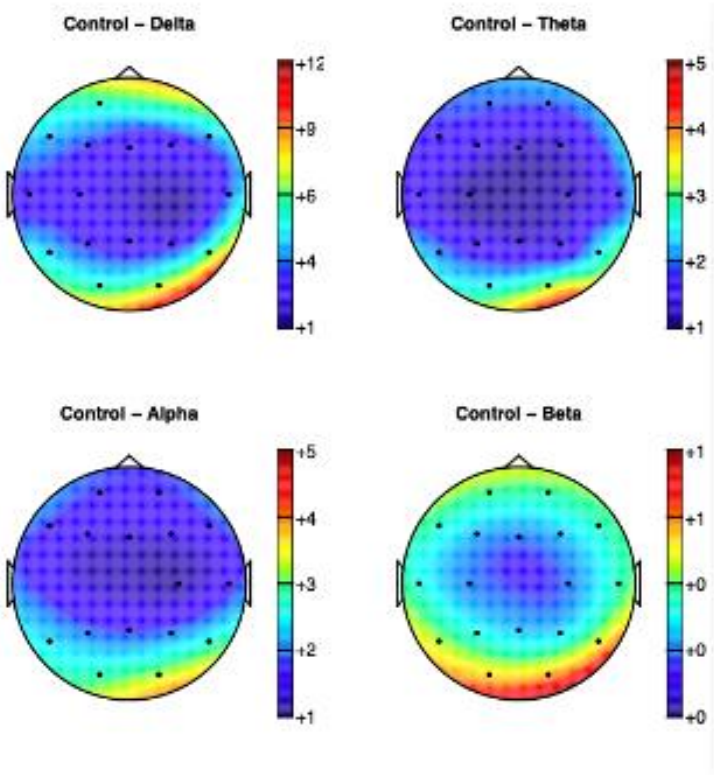
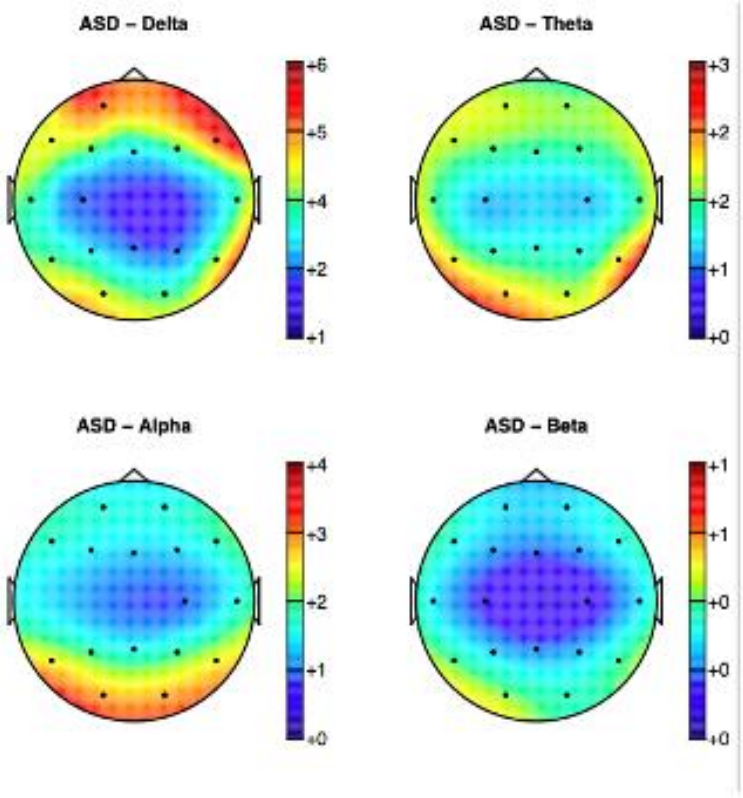


Figure 2

Frequency band distribution over the scalp in the ASD group



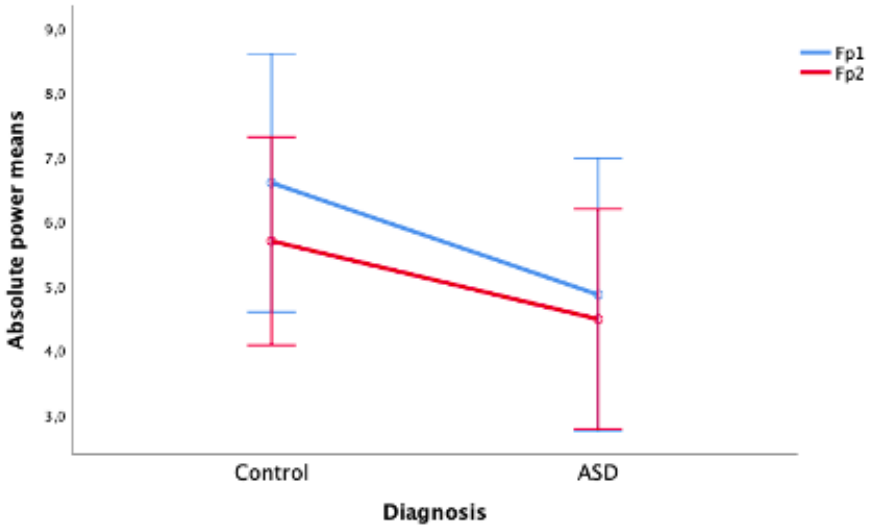
Differences in absolute delta power

The results showed no differences in the frontal absolute power between the control group and the ASD group F(1,73) = .18, *p* = .672, and no main effect for absolute power of delta F(1,73) = .001, *p* = .98 was found (Figure 3). Furthermore, there were no differences between the two groups regarding the left frontal absolute power F(1,73) = 2,01, *p* = .16 and the right frontal absolute power F(1,73) = 2.24, *p* = .139. No main effects were found for the left frontal absolute power of delta F(1,73) = .72, *p* = .399 and the right frontal absolute power of delta F(1,73) = .12, *p* = .729. Also, differences in the absolute power of delta between ASD and the control group were not found in the temporal F(1,73) = .14, *p* = .711 (Figure 4) and occipital region F(1,73) = 1.04, *p* = .312 (Figure 5). In addition, no main effects were found

for the temporal region F(1,73) =.18, p = .67 and the occipital regions absolute power of delta F(1,73) = . 86, p = .358.

Figure 3

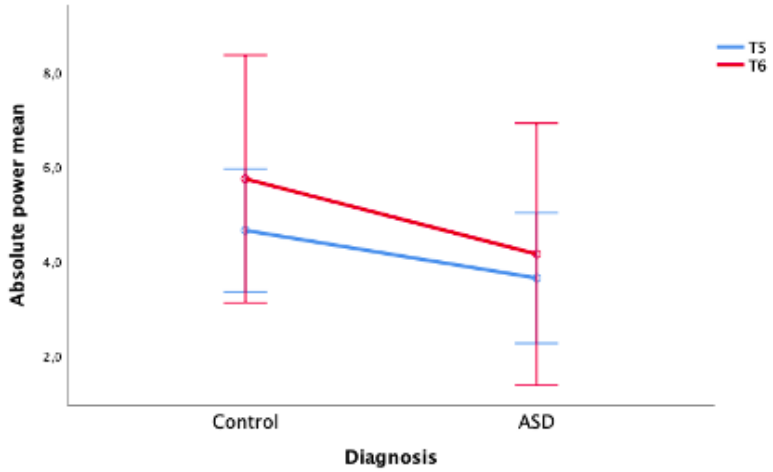
Differences in the mean absolute power of delta in the frontal region



Note. Error bars: 95% Confidence Interval.

Figure 4

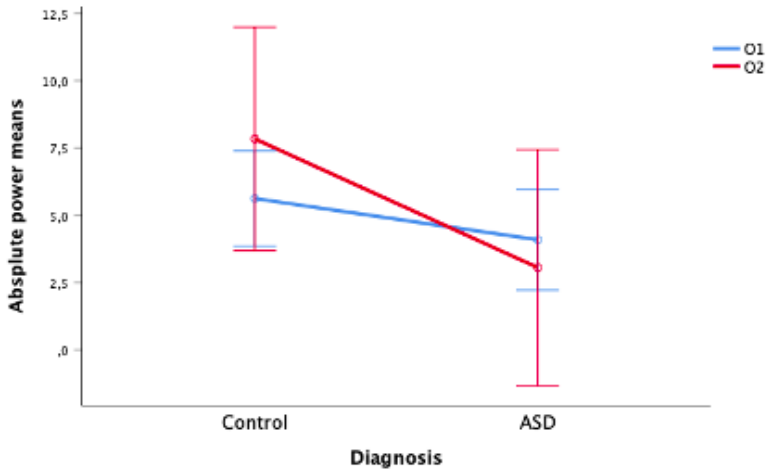
Differences in the mean absolute power of delta in the temporal region



Note. Error bars: 95% Confidence Interval.

Figure 5

Differences in the mean absolute power of delta in the occipital region



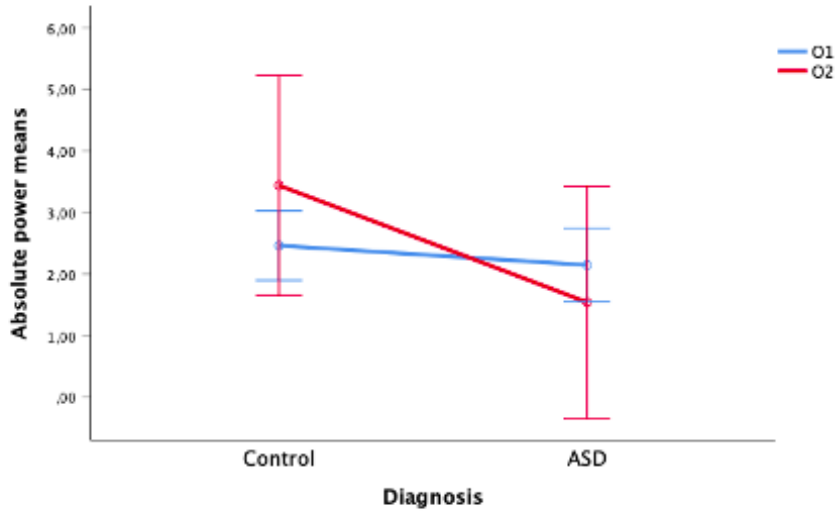
Note. Error bars: 95% Confidence Interval.

Differences in absolute power of theta

The results exhibited no interaction between the ASD group and the control group in the absolute power of theta in the occipital region $F(1,73) = 1.32$, $p = .254$, and no main effect for the occipital region $F(1,73) = 1.38$, $p = .244$ (Figure 6). Furthermore, no interaction between the absolute power of theta in the temporal region was found $F(1,73) = 07$, $p = .791$. Additionally, no main effect for the absolute power of theta in the occipital region was found $F(1,73) = .02$, $p = .888$ (Figure 7). The absolute power of theta in the frontal region showed no interaction with diagnosis $F(1,73) = .61$, $p = .437$, additionally, was there no main effect for the absolute power of theta in the frontal region $F(1,73) = .01$, $p = .922$ (Figure 8).

Figure 6

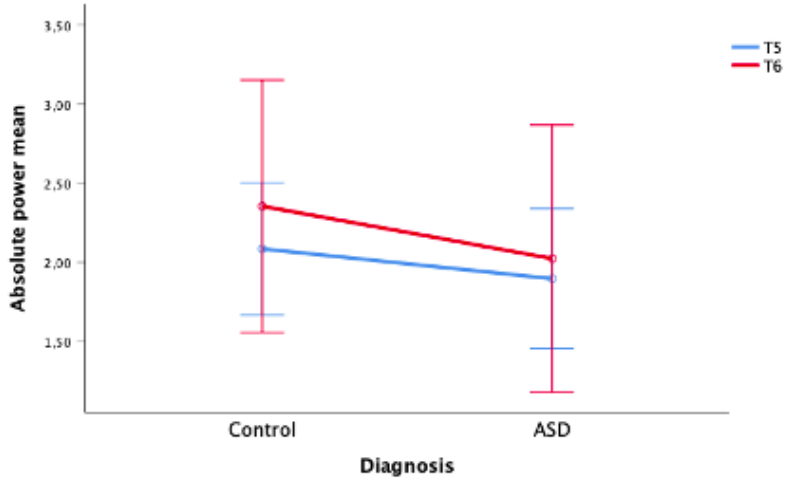
Differences in the mean absolute power of theta in the occipital region



Note. Error bars: 95% Confidence Interval.

Figure 7

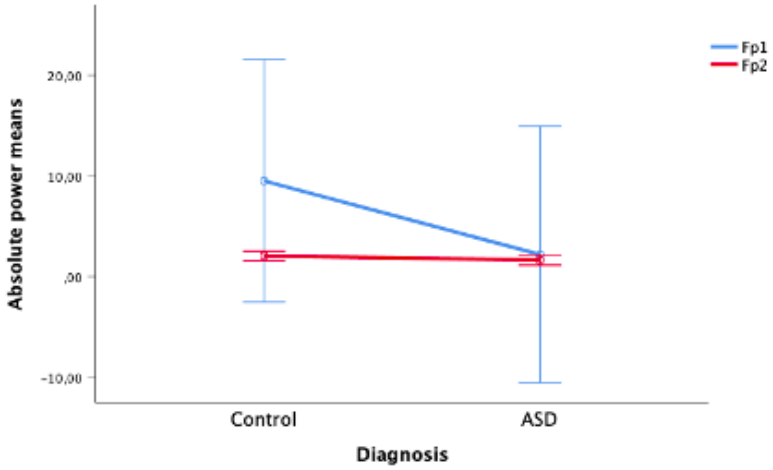
Differences in the mean absolute power of theta in the temporal region



Note. Error bars: 95% Confidence Interval.

Figure 8

Differences in the mean absolute power of theta in the frontal region



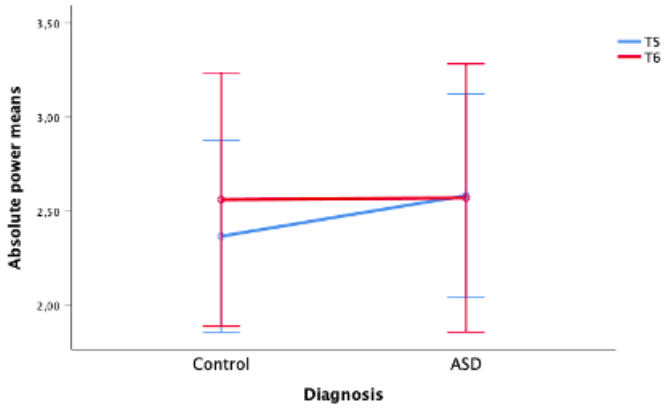
Note. Error bars: 95% Confidence Interval.

Differences in absolute power of alpha

No differences in absolute power of alpha were found between the two groups, neither for the temporal region F(1,73) = .51, p = .476 (Figure 9) nor the occipital region F(1,73) = .78, p = .381 (Figure 10). Also, no main effect for the absolute power of alpha in the occipital region was found F(1,73) = .81, p = .371, and neither was a main effect for the absolute power of alpha in the temporal lobe found F(1,73) = .04, p = .836.

Figure 9

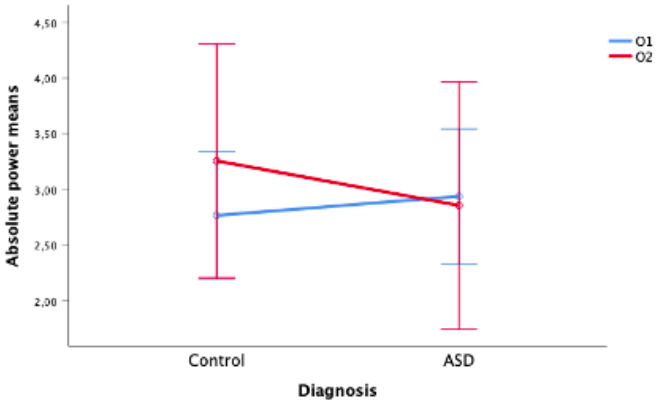
Differences in the mean absolute power of alpha in the temporal region



Note. Error bars: 95% Confidence Interval.

Figure 10

Differences in the mean absolute power of alpha in the occipital region



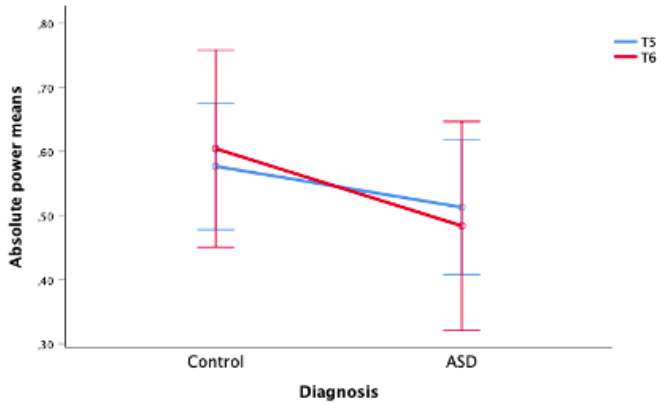
Note. Error bars: 95% Confidence Interval.

Differences in absolute power of beta

The results showed no differences between the ASD group and the control group in the absolute power of beta in the occipital region $F(1,71) = 1.14$, $p = .289$ and no main effect for the absolute power of beta was found $F(1,71) = .95$, $p = .333$ (Figure 11). Additionally, there were no differences found in the temporal regions' absolute power of beta, between the ASD group and the control group $F(1,71) = .26$, $p = .612$ and no main effect for the absolute power of beta was found in the temporal region $F(1,71) = .02$, $p = .882$ (Figure 12).

Figure 12

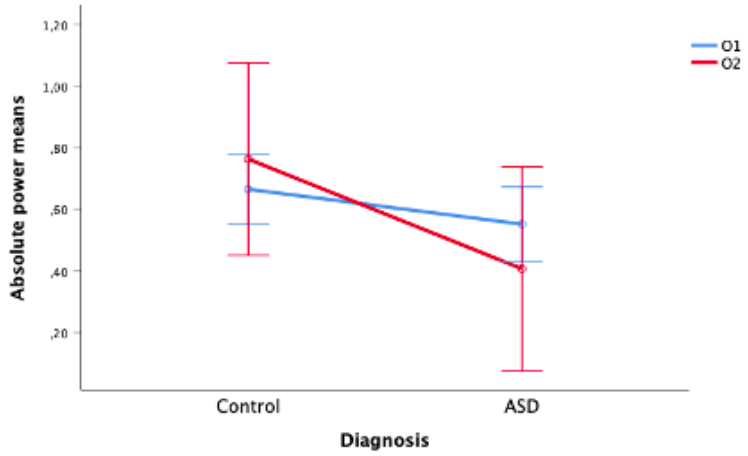
Differences in the mean absolute power of beta in the temporal region



Note. Error bars: 95% Confidence Interval.

Figure 11

Differences in the mean absolute power of beta in the occipital region



Note. Error bars: 95% Confidence Interval.

Discussion

The purpose of this study was to investigate possible QEEG differences between adults with ASD compared to a neurotypical control group. However, no significant differences between the two groups could be found.

More specifically, the first aim of this study was to see whether adults with ASD differ from the control group in their absolute power of delta in the frontal, temporal and occipital regions of the brain. The results showed that the absolute power of delta did not interact significantly with the variable diagnosis in either the frontal region, temporal region, or occipital region. Furthermore, the results showed no interaction between the absolute power of delta in the right frontal region and neither in the left frontal region. Thus, whether the patients had ASD or not did make a difference towards the absolute power values for delta in the frontal, temporal, or occipital lobe, which means that the first hypothesis was not supported by the results of this study. The results of the scalp maps showed that delta was most prominent in the frontal region, temporal, and occipital region for the ASD group, while it most occurred in the occipital region in the control group

Furthermore, the second aim of this study was to examine if adults with ASD differ from neurotypical peers in their absolute power values theta in either the frontal, temporal, or

occipital region. The results showed no significant interaction between the absolute power values and whether the patients had a diagnosis of ASD or not, in either of the three regions. In addition, the scalp maps examining the distribution of theta showed the highest concentration for the ASD group in the temporal and occipital region and the control group in the occipital region.

The third aim of this study was to look into the differences in the absolute power of alpha between the control group and the ASD group, in the temporal and occipital region. The results could not support the hypothesis, no differences between the two groups were found for any of the regions' absolute power of alpha. The results of the scalp maps showed the highest concentration of alpha in the temporal and occipital region for the ASD group and in the occipital region for the control group.

The fourth and last aim of this present study was to examine the differences between the absolute power of beta in the control group and the ASD group. It was hypothesized that ASD influenced the absolute power of beta, thus that a difference between the two groups would be found. However, the result portrayed no significant differences between the two groups' absolute power of beta values. According to the results of the scalp maps the Beta was mostly located in the temporal region for the ASD group and the temporal and occipital region for the control group.

The results showed no differences between the two groups' frequency bands in any of the regions examined, so whether the participants had ASD or not did make a difference regarding the absolute power of the frequency bands.

This study adds to the vast number of different results reported in previous QEEG studies, using spectral analysis, comparing participants with ASD to their neurotypical peers. Contrary to the results of this present study in previous studies differences in frequency bands between participants with ASD and a control group were found in any of the regions

examined in this study, which were the frontal region, the temporal region, and the occipital region.

This current study did not find any differences in the absolute power of delta between the experimental and control group. However, previously Elhabashy et al. (2015) were able to find increased values of the absolute power of delta in their ASD sample compared to their control group. In line with these results, the study Chan et al. (2007) reported a significantly higher absolute power of delta score. Compared were children diagnosed with ASD to a neurotypical control group. Additionally, Stroganov et al. (2007) reported higher absolute power of delta in the prefrontal region of the brain, in their ASD sample of children aged three to eight compared to an age-matched control group. Other studies, for example, the one by Dawson et al. (1995) found reduced absolute power of delta, in their ASD sample compared to the control group, in the frontal region. Whereas this study did not find any differences in the frontal regions' absolute power of delta values between the two groups. Congruent with the findings of this present study Tani et al. (2004) found no significant differences in delta power between the adult ASD group and the adult control group.

Furthermore, the result of this current study showed no differences in theta between the two groups examine, neither in the frontal region, the temporal region, nor the occipital region. However, previously it had been found that children with ASD had higher absolute power of theta values compared to neurotypical control groups (Daoust et al. 2004; Elhabashy et al. 2015). Other studies reported a lower absolute power of theta in the ASD sample compared to the control group (Cantor et al. 1986; Matlis et al. 2015). Looking more specifically at the brain regions' theta band values, Daoust et al. (2004) reported a higher absolute power of theta in the frontal region, whereas and Elhabashy et al. (2015) examined the absolute theta power for the whole scalp. Dawson et al. (1995), examined and found the decrease of theta in the frontal and temporal region of the brain. In line with the results of the current study, Chan et al. (2007) found no significant differences in the absolute power of

theta values. Compared were QEEG recordings of children diagnosed with ASD to those of neurotypically developed children.

Moreover, Chan et al. (2007) reported no significant differences in the beta frequencies between the children diagnosed with ASD and the control group, these results are in line with those of this present study. This study found no differences between the ASD group and the control group in their absolute power of beta. The results of the study by Dawson et al. (1995) support these findings, no significant differences in beta frequency were found between the children diagnosed with ASD and the control group. Contrary to the results of this present study Daoust et al. (2004) found lower absolute beta power in the ASD group compared to the control group.

Furthermore, results regarding the differences in absolute alpha power between ASD and control groups differ vastly. While Cantor et al. (1986), Dawson et al. (1995), and Matlis et al. (2015) reported lower-alpha frequencies in their ASD group compared to the control group. Chan et al. (2007) and Mathewson et al. (2012) found significantly higher absolute power values f alpha in the ASD group compared to the control group. Compared to the results of this current study, neither the higher nor the lower findings of alpha are in agreement with the results, as this study found no differences between the ASD groups' and the control groups' absolute power of alpha.

One fact that stands out is that most of the reported studies used child samples. This makes the comparability to the current study difficult. Spectral analysis is dependent on brain maturation. Thus, age is a critical point of interpretation, which makes comparing a large number of studies to the current one with adults the more difficult. An important point for the usage of spectral analysis is its easy usage and interpretation, and that it is possible to use it in multiple different conditions. However, this advantage is at the same time another factor that could influence the substantial inconsistencies in the findings of these studies. The different

types of experimental designs make generalization difficult, as spectral analysis is dependent on it (Gurau et al. 2017).

In addition, it is noteworthy that in this study the control group was significantly older than the ASD group. This could have influenced the results as previously mentioned, age has a possible impact on spectral analysis. Another limitation of this study is that it was not taken into account which type of ASD the patients had. Further, the study did not take into account if the participants were on medication or not. It is not uncommon for patients with ASD to take antiepileptic medication which can have a substantial impact on the results of the spectral analysis (Gurau et al. 2017).

Despite this study's limitations and non-significant findings, it still adds substantially to the research on QEEG in ASD. The importance of finding more about the possible differences in frequencies to use as a possible diagnostic technique is still relevant. Especially the difficulties in the diagnostic process of adults with ASD would benefit from an added biological, and objective tool. Thus, for future research to gain more insight into the relationship between ASD and absolute power values a longitudinal study could be of great interest. Researching if the possible found differences in childhood persist or change during adolescence and adulthood could give important information about the QEEG characteristics of patients with ASD. Overall, QEEG is not yet applicable in clinical practice, as there is still too much inconsistency in the findings. However, the results to date and the simplicity of the method of the analysis suggest that it can become highly beneficial in the future. It may become an additional tool in the diagnostic procedure that will simplify the assessment, especially for adults.

References

Aftanas, L. I., Varlamov, A. A., Pavlov, S. V., Makhnev, V. P., & Reva, N. V. (2001). Affective picture processing: Event-related synchronization within individually defined human theta band is modulated by valence dimension. *Neuroscience Letters*, *303*(2), 115–118. https://doi.org/10.1016/S0304-3940(01)01703-7

Billeci, L., Sicca, F., Maharatna, K., Apicella, F., Narzisi, A., Campatelli, G., Calderoni, S., Pioggia, G., & Muratori, F. (2013). On the Application of Quantitative EEG for Characterizing Autistic Brain: A Systematic Review. *Frontiers in Human Neuroscience*, *7*. https://doi.org/10.3389/fnhum.2013.00442

Cantor DS, Thatcher RW, Hrybyk M, Kaye H. (1986)Computerized EEG analyses of autistic children. J Autism Dev Disord 16(2):169–87. doi:10.1007/ BF01531728

Chabot, R. J., & Serfontein, G. (1996). Quantitative electroencephalographic profiles of children with attention deficit disorder. *Biological Psychiatry*, *40*(10), 951–963. https://doi.org/10.1016/0006-3223(95)00576-5

Chan, A.S., Sze, S.L., & Cheung, M.C. Quantitative electroencephalographic pro- files for children with autistic spectrum disorder. Neuropsychology (2007) 21(1):74–81. doi:10.1037/0894-4105.21.1.74

Coben, R., Clarke, A. R., Hudspeth, W., & Barry, R. J. (2008). EEG power and coherence in autistic spectrum disorder. *Clinical Neurophysiology*, *119*(5), 1002–1009. https://doi.org/10.1016/j.clinph.2008.01.013

Daoust, A. M., Limoges, E., Bolduc, C., Mottron, L., & Godbout, R. (2004). EEG spectral analysis of wakefulness and REM sleep in high functioning autistic spectrum disorders. Clinical neurophysiology: official journal of the International Federation of Clinical Neurophysiology, 115(6), 1368–1373. https://doi.org/10.1016/j.clinph.2004.01.011

Dawson G, Klinger LG, Panagiotides H, Lewy A, Castelloe P. (1995) Subgroups of autistic children based on social behavior display distinct patterns of brain activity. J Abnorm Child Psychol 23(5):569–83. doi:10.1007/ BF01447662

Fusar-Poli, L., Brondino, N., Rocchetti, M., Panisi, C., Provenzani, U., Damiani, S., & Politi, P. (2017). Diagnosing ASD in Adults Without ID: Accuracy of the ADOS-2 and the ADI-R. Journal of autism and developmental disorders, 47(11), 3370–3379. https://doi.org/10.1007/s10803-017-3258-

Jokiranta, E., Sourander, A., Suominen, A., Timonen-Soivio, L., Brown, A. S., & Sillanpää, M. (2014). Epilepsy Among Children and Adolescents with Autism Spectrum Disorders: A Population-Based Study. *Journal of Autism and Developmental Disorders*, *44*(10), 2547–2557. https://doi.org/10.1007/s10803-014-2126-6

Lai, D. M.-C. (2015). *Identifying the lost generation of adults with autism spectrum conditions*. *2*, 15.

Lushchekina, E. A., Podreznaya, E. D., Lushchekin, V. S., & Strelets, V. B. (2012). A Comparative EEG Study in Normal and Autistic Children. *Neuroscience and Behavioral Physiology*, *42*(3), 236–243. https://doi.org/10.1007/s11055-012-9558-2

Mathewson, K. J., Jetha, M. K., Drmic, I. E., Bryson, S. E., Goldberg, J. O., & Schmidt, L. A. (2012). Regional EEG alpha power, coherence, and behavioral symptomatology in autism spectrum disorder. *Clinical Neurophysiology*, *123*(9), 1798–1809. https://doi.org/10.1016/j.clinph.2012.02.061

Matlis S, Boric K, Chu CJ, Kramer MA. (2015) Robust disruptions in electroenceph-alogram cortical oscillations and large-scale functional networks in autism. BMC Neurol (2015) 15:97. doi:10.1186/s12883-015-0391-4

Murias, M., Webb, S. J., Greenson, J., & Dawson, G. (2007). Resting State Cortical Connectivity Reflected in EEG Coherence in Individuals With Autism. *Biological Psychiatry*, *62*(3), 270–273. https://doi.org/10.1016/j.biopsych.2006.11.012

Pop-Jordanova, N., Zorcec, T., Demerdzieva, A., & Gucev, Z. (2010). QEEG characteristics and spectrum weighted frequency for children diagnosed as autistic spectrum disorder. *Nonlinear Biomedical Physics*, *4*(1), 4. https://doi.org/10.1186/1753-4631-4-4

Stroganova, T. A., Nygren, G., Tsetlin, M. M., Posikera, I. N., Gillberg, C., Elam, M., & Orekhova, E. V. (2007). Abnormal EEG lateralization in boys with autism. *Clinical Neurophysiology*, *118*(8), 1842–1854. https://doi.org/10.1016/j.clinph.2007.05.005

Tani, P., Lindberg, N., Nieminen-von Wendt, T., von Wendt, L., Virkkala, J., Appelberg, B., & Porkka-Heiskanen, T. (2004). Sleep in Young Adults with Asperger Syndrome. *Neuropsychobiology*, *50*(2), 147–152. https://doi.org/10.1159/000079106

World Health Organization. (2004). ICD-10: international statistical classification of diseases and related health problems: tenth revision, 2nd ed. World Health Organization. https://apps.who.int/iris/handle/10665/42980