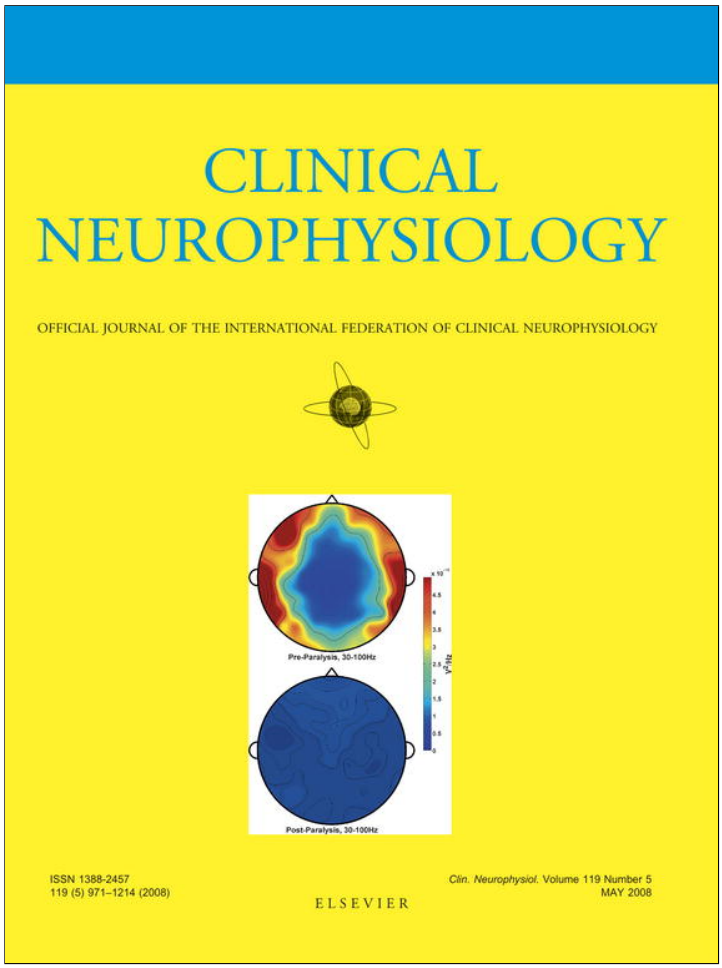

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EEG power and coherence in autistic spectrum disorder

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Abstract

Objective: Autistic spectrum disorder (ASD) has been defined as a neurodevelopmental disorder with associated deficits in executive function, language, emotional, and social function. ASD has been associated with pathophysiology in cerebral organization. The current study investigated quantitative EEG findings in twenty children diagnosed with autistic disorders as compared to 20 controls matched for gender, age and IQ.
Methods: The EEG was recorded during an eyes-closed resting condition and topographical differences in cerebral functioning were examined using estimates of absolute, relative, and total power, as well as intrahemispheric and interhemispheric coherences.
Results: There were group differences in power, intrahemispheric and interhemispheric coherences. Findings included excessive theta, primarily in right posterior regions, in autistics. There was also a pattern of deficient delta over the frontal cortex and excessive midline beta. More significantly, there was a pattern of underconnectivity in autistics compared to controls. This included decreased intrahemispheric delta and theta coherences across short to medium and long inter-electrode distances. Interhemispherically, delta and theta coherences were low across the frontal region. Delta, theta and alpha hypocoherence was also evident over the temporal regions. Lastly, there were low delta, theta and beta coherence measurements across posterior regions.
Conclusions: These results suggest dysfunctional integration of frontal and posterior brain regions in autistics along with a pattern of neural underconnectivity. This is consistent with other EEG, MRI and fMRI research suggesting that neural connectivity anomalies are a major deficit leading to autistic symptomatology.
Significance: This paper reports the largest integrated study of EEG power and coherence during a resting state in children suffering autism spectrum disorder.

Keywords: Autism; Children; EEG; Coherence; Diagnosis

1. Introduction

Autistic spectrum disorder (ASD) has been defined as a neurodevelopmental disorder with associated deficits in executive function, language, emotional, and social function (Rapin and Dunn, 2003; Belmonte et al., 2004; Hill, 2004; McAlonan et al., 2005). Increasing rates of prevalence have been reported for ASD. According to Blaxill (2004), the rates of ASD were reported to be <3 per 10,000 children in the 1970s and rose to >30 per 10,000 in the 1990s. The Centers for Disease Control and Prevention (CDC, 2006) summarized data from several studies on the prevalence rates for ASD ranging from 1 in 500 to 1 in 166, making it the sixth most common disability classification in the United States. In fact, their most recent report (CDC, 2007) suggests a prevalence of 1 in 150. The dramatic rise in the numbers of children classified as ASD highlights the need for further research to be conducted into this population.

A review of research on EEG screening for ASD found that seizures were common among 20–30% of individuals

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with autism. Epileptiform abnormalities were found in 10.3–72.4% of patients and subclinical anomalies in 6.1–31% (Kagan-Kushnir et al., 2005). While such problems are important in the diagnostic and screening process, Deonna and Roulet (2006) concluded that there is no evidence that autism can be attributed to an epileptic disorder.

Only a few studies have investigated the EEG of children with ASD, and each of these has used different paradigms. Ogawa et al. (1982) examined sleep EEGs of 28 normal children and 21 children with autism. Findings indicated higher levels of alpha bilaterally and frontally, with no hemispheric lateralization, in children with autism. Ogawa et al. (1982) concluded that children with autism were less responsive to external stimuli and that they had fewer active internal regulatory mechanisms than the control group.

Dawson et al. (1982) investigated EEG measures of hemispheric activation during four cognitive tasks (rote verbal memory task, verbal categories, block design, and copying designs) in 10 males with autism (ranging in age from 9.1 to 34.0 years), and 10 normal individuals matched for gender, age, handedness and family patterns of handedness. Seven of the individuals with autism had atypical patterns of cerebral lateralization, involving right-hemisphere dominance for both verbal and spatial functions. The reversal in lateralization indicated a lack of left-hemisphere specialization for linguistic functions. These findings suggested selective impairment of the left cerebral hemisphere. The group with autism showed stronger right-hemisphere dominance during verbal tasks than during spatial tasks. Stroganova et al. (2007a,b) have also recently shown a pattern of abnormal lateralization in autistics. Specifically, they hypothesized a diminished capacity of the right temporal cortex in the generation of EEG rhythms.

Cantor et al. (1986) conducted computerized EEG analyses of 11 children with autism between the ages of 4 and 12 years, in contrast to three groups of children: (1) 88 normal children, (2) a matched group of 18 mentally handicapped children, and (3) a group of 13 mental-age-matched normal toddlers. The findings indicated that children with autism had significantly greater coherence between hemispheres in the beta band than mentally handicapped, normal children, or toddlers. Children in the Autistic group had higher coherence in the alpha band than those in the normal group, and less inter- and intrahemispheric asymmetry than participants in the normal or mentally handicapped group. Amplitude asymmetries were noted for autistic children in the posterior-temporal, central, and occipital regions, with greater amplitude in the left than right hemisphere. Based on these findings, the researchers concluded that autism may be characterized by a maturational lag in cerebral functioning and a lack of cerebral differentiation (Cantor et al., 1986). Murias et al. (2006) conducted analyses of high-density EEG recordings in an eyes-closed resting state in 18 adult autistics compared to normal controls. The findings included both excessive and reduced coherence in the Autistic group. The ASD group also exhibited higher theta and beta 1 power than the controls. However, this was a study of adults, not children, so the comparability of results to a pediatric population are limited.

These studies suggest that atypical structural and functional neurobiological patterns are associated with the multiple symptoms present in this disorder. However, none of these studies has used an eyes-closed resting condition, the most common paradigm used in the investigation of EEG abnormalities among children with behavioral disorders. Further research is needed to characterize these neurophysiological profiles. The current study was designed to extend previous research, by investigating power and coherence differences between ASD and control subjects during an eyes-closed resting condition.

2. Methods

2.1. Subjects

Two groups of 20 subjects between the ages of 6 and 11 years old, with 14 boys and 6 girls in each group, participated in this study. Twenty patients were consecutively seen in clinical practice with a diagnosis of autistic spectrum disorder or autism based on DSM-IV criteria (APA, 1994). The Control group was drawn from previously published normative data (Clarke et al., 2001a). Subjects in each group were individually matched on age, using 1-year age bands to control for maturational changes in the EEG and IQ. No subject was taking medication during the study period.

Inclusion in the Control group was based on: an uneventful prenatal, perinatal, and neonatal period; no disorders of consciousness, head injury with cerebral symptoms, history of central nervous system disease, obvious somatic diseases, convulsions, history of convulsive disorders, paroxysmal headache, enuresis or encopresis after the fourth birthday, tics, stuttering, pavor nocturnes, and conduct disorders. Children were excluded from the Control group if spike wave activity was present in the EEG.

2.2. Procedure

All subjects were tested in a single session. The EEG assessment was obtained during an eyes-closed resting condition while subjects were seated on a reclining chair. Autistic subjects were alerted by an experienced technician upon any signs of fatigue, sleep or reduced vigilance.

The EEG for the Autistic group was recorded using a Deymed TruScan 32 EEG System and NeuroRep QEEG software (Hudspeth, 1999) with a sampling rate of 256 Hz. The sensitivity was set at 70 μV/cm, low-frequency filter 0.1 Hz, high-frequency filter 100 Hz and 60-Hz notch filter. Common mode rejection ratio was 102 dB and isolation mode rejection ratio was 140 dB. A minimum of 75 s of artifact-free EEG was available for analysis. Using NxLink software, these data were downsampled to 100 Hz, from which 2.56-s epochs were Fourier transformed.

The EEG for the Control group was recorded and Fourier transformed by a Cadwell Spectrum 32, software version 4.22, using test type EEG, montage Q-EEG. The sensitivity was set at 150 μV/cm, low-frequency filter 0.53 Hz, high-frequency filter 70 Hz and 50-Hz notch filter. The sampling rate of the EEG was 200 Hz, which was downsampled to 100 Hz, and the Fourier transformation used 2.56-s epochs which were selected from continuous live trace. For both groups of data, the following procedures were employed. Thirty 2.56-s epochs were selected from the live trace and stored to floppy disk. Epoch rejection was based on both visual and computer selection. Computer reject levels were set using a template recorded at the beginning of the session and all subsequent epochs were compared to this. The EOG rejection was set at 50 μV. The technician also visually appraised every epoch and decided to accept or reject it. Twenty-four epochs (1 min) were selected for Fourier analysis. The software used to calculate power and coherence measures was the same as that used for the Autistic group, except that it was an earlier version which was built into the acquisition unit as an integrated package. Using calibration data provided by both EEG manufacturers, the data were then converted into microvolts. This process assures that adjustments are made for any differences in gain between the two systems (R. Eisenhart, personal communication, December 11, 2007). As such, the quantified results are comparable between both groups.

Electrode placement in both groups was based on the International 10–20 system (Jasper, 1958), using an Electrocap International recording cap. The 19 active EEG, linked ear reference and ground electrodes (9 mm disc) were made of tin. Impedance levels were set at less than 5 kOhm.

Absolute and relative power indices were computed for the delta (1.5–3.5 Hz), theta (3.5–7.5 Hz), alpha (7.5–12.5 Hz), and beta (12.5–25 Hz) frequency bands, as well as the total power of the EEG (1.5–25 Hz). The absolute and relative power indices from 19 derivations were averaged for nine regions to reduce the number of statistical comparisons. These regions were the left frontal (Fp1, F3, and F7), midline frontal (Fz), right frontal (Fp2, F4, and F8), left central (T3 and C3), midline central (Cz), right central (T4 and C4), left posterior (T5, P3, and O1), midline posterior (Pz), and right posterior (T6, P4, and O2).

Coherence indices were computed for eight intrahemispheric (F3–O1, F4–O2, Fp1–F3, Fp2–F4, T3–T5, T4–T6, C3–P3, and C4–P4) and eight interhemispheric (Fp1–Fp2, F7–F8, F3–F4, C3–C4, T3–T4, T5–T6, P3–P4, and O1–O2) electrode pairs.

2.3. Statistical analysis

For each band in absolute and relative power, and the total power of the EEG, an analysis of variance was performed examining the effects of region and group. Within region, two orthogonal three-level repeated-measures, and their interactions, were examined. The first of these was a sagittal factor, within which planned contrasts compared the frontal region with the posterior region, and their mean with the central region. The second factor was laterality, within which similar planned contrasts compared activity in the left and right hemispheres, and their mean with the midline regions. These planned contrasts allow optimal clarification of topography, with complete specification of which region/s was/were dominant.

A mixed-model analysis of variance was used to examine the effects of region and group upon coherences in each band. Prior to analysis, each coherence value was transformed using Fisher's z-transform. For the intrahemispheric coherences, the means within hemisphere were separately compared for (i) short/medium inter-electrode distances (left: Fp1–F3, T3–T5, and C3–P3 versus right: Fp2–F4, T4–T6, and C4–P4) and (ii) long inter-electrode distances (left F3–O1 versus right F4–O2), and within these analyses, laterality was examined. The interhemispheric coherences were separately examined within (iii) the frontal (Fp1–Fp2, F7–F8, and F3–F4), (iv) temporal (T3–T4 and T5–T6), and (v) central/parietal/occipital (C3–C4, P3–P4 and O1–O2) regions.

Within the diagnosis factor, the Autism group was compared to the Control group. As all these contrasts are planned, and there are no more of them than the degrees of freedom for effect, no Bonferroni-type adjustment to alpha is required (Tabachnick and Fidell, 1989). Only significant between-group effects and interactions are reported here for space reasons.

3. Results

There were no significant differences between the groups on age, gender, or IQ (Table 1).

3.1. Power difference between Autism and Control groups

The Autistic group had less absolute delta than controls ($F(1,38) = 5.79$, $p < .05$) (see Fig. 1 and Table 2). Topographically, this autistic difference was smaller in the central regions than the frontal and posterior regions ($F(1,38) = 5.81$, $p < .05$). This sagittal effect was larger in the left than right hemisphere ($F(1,38) = 7.77$, $p < .01$) and smallest at the midline ($F(1,38) = 4.78$, $p < .05$). Together these indicate that the largest reductions in absolute delta in autism are in the left frontal and posterior regions.

Table 1
Mean (SD) demographic data for the group with Autism and Control group

	Autism group	Control group
Mean age in months	107 (27.4)	110 (14.7)
Mean FSIQ	93 (16.8)	98 (15.4)
Gender		
Males	14	14
Females	6	6

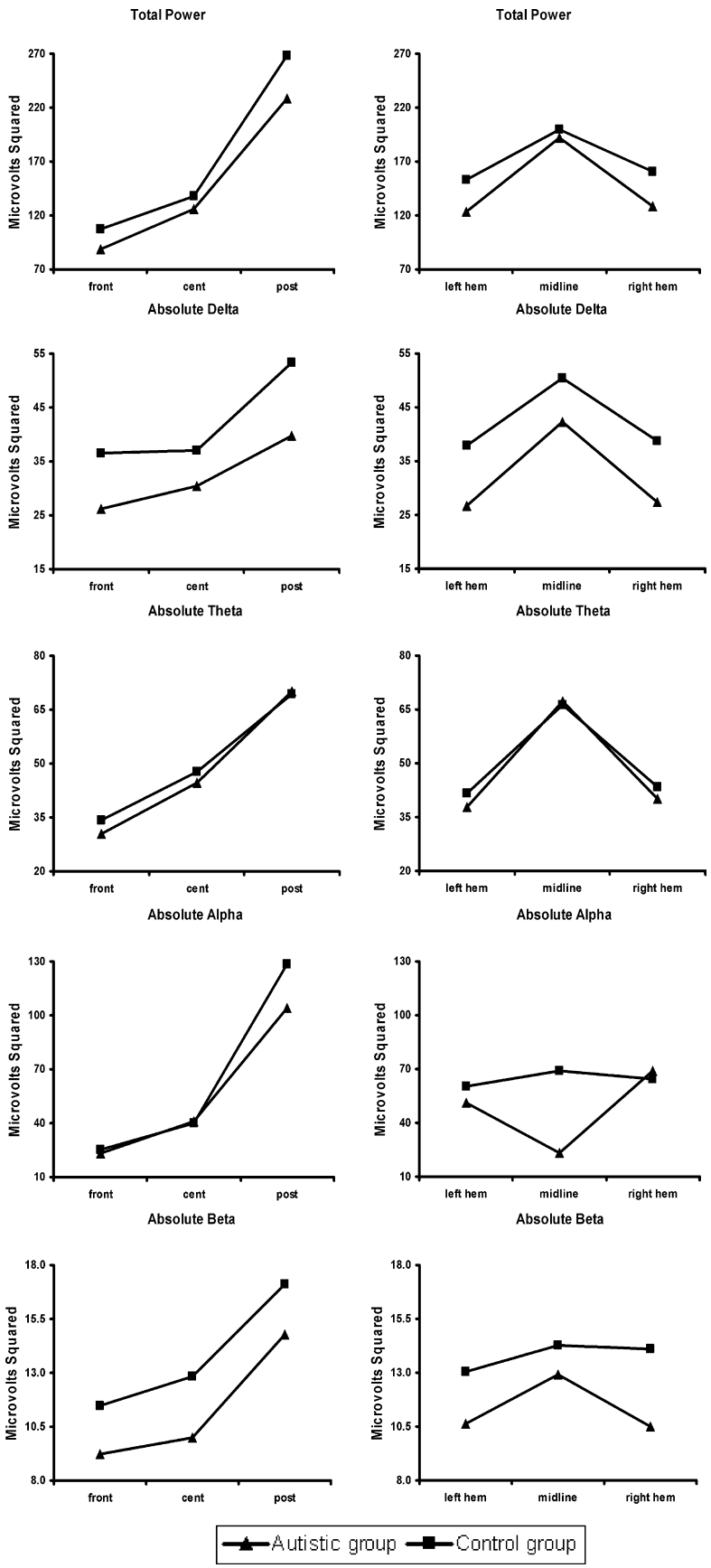

Fig. 1. Absolute band power in each group as a function of region in the sagittal plane (left) and lateral plane (right).

Table 2
EEG means (SD) for the Autistic and Control groups

Group	Absolute power (μV^2)					Relative power (%)			
	Total	Delta	Theta	Alpha	Beta	Delta	Theta	Alpha	Beta
Autistic	139.30 (45.09)	29.79 (11.87)	44.02 (24.11)	54.41 (30.31)	11.07 (5.00)	24.76 (5.32)	31.78 (9.06)	33.86 (12.37)	9.58 (3.15)
Control	168.06 (68.76)	41.08 (13.88)	46.88 (21.38)	66.44 (36.60)	13.78 (6.55)	28.78 (4.82)	29.30 (3.59)	32.15 (6.25)	9.43 (2.19)

As shown in Fig. 2, relative delta was reduced globally in autistic children compared with controls ($F(1,38) = 5.70$, $p < .05$). Topographically, the autistic reduction in the left compared with the right hemisphere was greater in the frontal than posterior regions ($F(1,38) = 6.04$, $p < .05$) and least in the central regions

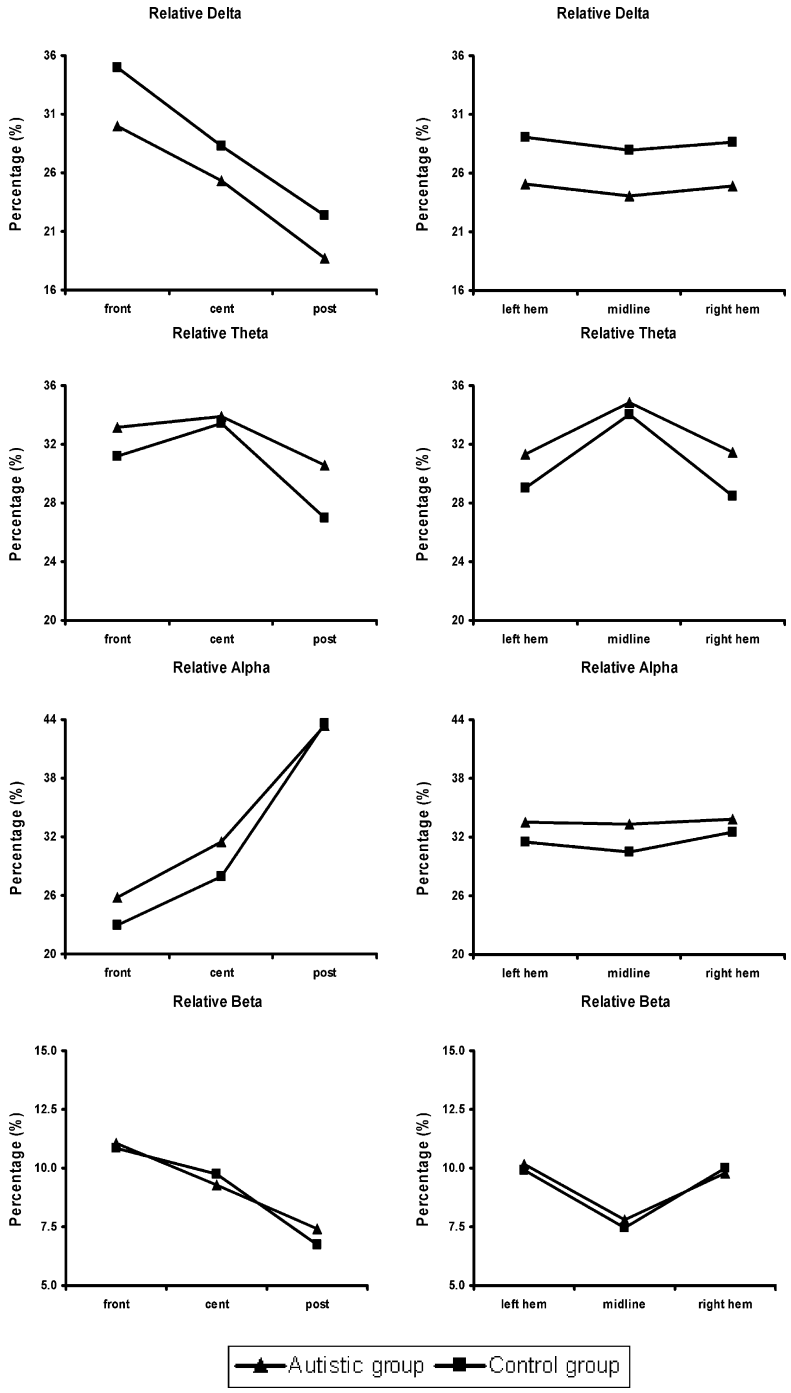

Fig. 2. Relative band power in each group as a function of region in the sagittal (left) and lateral (right) planes.

($F(1,38) = 4.95$, $p < .05$). Also, the autistic reduction in the midline compared with the hemispheres was largest at the vertex ($F(1,38) = 6.22$, $p < .05$). Together these indicate that the largest relative delta reductions associated with autism are in the left frontal and vertex regions.

Relative theta was greater in the Autistic group compared with the Control group at frontal and posterior regions than central regions ($F(1,38) = 4.18$, $p < .05$). The relative autistic increase in the right hemisphere was greater in the posterior region than the frontal region ($F(1,38) = 4.27$, $p < .05$). Together these indicate that the maximal group difference reflects the Autistic group's elevated power in the right posterior region.

In absolute beta (Fig. 1), a reduction in power in the Autistic group compared with the Control group was greater in the right than the left hemisphere ($F(1,38) = 4.78, p < .05$). In the comparison of the two hemispheres with the midline, the difference between groups was greater in the posterior than the frontal regions ($F(1,38) = 4.77, p < .05$). This was due to controls having reduced midline power compared to the two hemispheres, in the posterior regions, where the Autistic group had increased midline power.

There were no significant group differences for absolute theta, absolute or relative alpha, relative beta, or total power.

Table 3
Intrahemisperic and interhemispheric coherences for Autistic and Control groups

	Delta	Theta	Alpha	Beta
Intrahemispheric coherences				
Short-medium				
L vs. R	–	–	–	–
Autistic vs. Controls	****	***	–	$p = .071$
Autistic vs. Controls X L vs. R	–	–	–	–
Long				
L vs. R	–	–	–	–
Autistic vs. Controls	****	***	–	–
Autistic vs. Controls X L vs. R	–	–	–	–
Interhemispheric coherences				
Frontal				
Autistic vs. Controls	***	*	–	–
Temporal				
Autistic vs. Controls	*	***	**	–
Central/parietal/occipital				
Autistic vs. Controls	*	*	$p = .064$	*

* $p < .05$.
** $p < .01$.
*** $p < .005$.
**** $p < .001$.

3.2. Coherence differences between Autism and Control groups

3.2.1. Intrahemispheric coherences

With short-medium inter-electrode distances, autistic subjects had reduced delta ($F(1,38) = 22.6$, $p < .001$) and theta ($F(1, 38) = 11.0$, $p < .005$) coherences (see Tables 3 and 4). With the long inter-electrode distances, coherences were also reduced in the Autistic group in the delta ($F(1,38) = 16.05$, $p < .001$) and theta ($F(1,38) = 9.87$, $p < .005$) bands.

3.2.2. Interhemispheric coherences

In the frontal regions, the Autistic group displayed lower interhemispheric coherences than controls in the delta ($F(1,38) = 9.00$, $p < .005$) and theta ($F(1,38) = 4.12$, $p < .05$) bands. In temporal regions, the Autistic group again showed lower coherences than controls in the delta ($F(1,38) = 5.37$, $p < .05$) and theta ($F(1,38) = 10.46$, $p < .005$) bands, but also had significantly reduced coherences in the alpha band ($F(1,38) = 7.46$, $p < .01$). In the central/parietal/occipital regions, interhemispheric coherences were again lower in the Autistic group than controls, reaching significance in the delta ($F(1,38) = 4.90$, $p < .05$), theta ($F(1,38) = 7.19$, $p < .05$) and beta ($F(1,38) = 4.51$, $p < .05$) bands.

4. Discussion

Previous research has found EEG power anomalies in autistics compared to controls (Cantor et al., 1986; Ogawa et al., 1982). While Ogawa et al. (1982) found elevated frontal alpha in autistics, Cantor et al. (1986) showed that autistic children had elevated power in frontotemporal regions, especially in the delta band. Murias et al. (2006) found autistic adults had greater relative theta (3–6 Hz) and beta1 (13–17 Hz) than controls. Only Cantor et al. (1986) investigated resting state power during an eye-closed condition in children.

The current findings showed differences in EEG power compared to age-matched controls. Relative theta was greater in autistics, especially over the right posterior region. There was also a related reduction in absolute beta over the right hemisphere for the Autistic group, but an increase in midline beta power. Our findings of excess theta and beta are consistent with those of Murias et al. (2006).

Excesses of theta power over the right posterior region of the brain in autistics suggest that this is an area of abnormal functioning. While the exact role of theta activity in clinical populations is not fully understood, excess theta activity is commonly found in children with executive functioning and mental activity problems, including attention-deficit/hyperactivity disorder (Clarke et al., 1998), learning disabilities (Dykman et al., 1982) and mental retardation (Katada et al., 1981). Findings of posterior cortical dysfunction have also been reported in previous investigations of ASD. This has included deficits related to eye gaze (Senju et al., 2005), facial processing (Critchley et al., 2000), and social cognition (Pelphrey et al., 2004). Stroganova et al. (2007a,b) have also shown deficient visual processing in an event-related study in similar brain regions. Pierce

Table 4
Mean coherence level across subjects for each electrode pair (SD in brackets)

Group	Delta		Theta		Alpha		Beta	
	Control	Autistic	Control	Autistic	Control	Autistic	Control	Autistic
Fp1-Fp2	0.89 (0.35)	0.83 (0.19)	0.87 (0.22)	0.84 (0.19)	0.88 (0.22)	0.86 (0.29)	0.73 (0.16)	0.67 (0.16)
F7-F8	0.39 (0.17)	0.28 (0.12)	0.35 (0.15)	0.31 (0.11)	0.46 (0.19)	0.44 (0.17)	0.19 (0.09)	0.19 (0.12)
F3-F4	0.79 (0.16)	0.73 (0.14)	0.78 (0.11)	0.74 (0.12)	0.79 (0.22)	0.76 (0.22)	0.52 (0.12)	0.49 (0.12)
C3-C4	0.71 (0.15)	0.65 (0.13)	0.68 (0.11)	0.63 (0.10)	0.54 (0.23)	0.48 (0.20)	0.48 (0.12)	0.51 (0.14)
T3-T4	0.18 (0.10)	0.11 (0.08)	0.15 (0.07)	0.08 (0.08)	0.18 (0.13)	0.13 (0.08)	0.08 (0.04)	0.13 (0.08)
T5-T6	0.39 (0.18)	0.32 (0.11)	0.24 (0.12)	0.17 (0.09)	0.26 (0.14)	0.15 (0.14)	0.12 (0.06)	0.06 (0.06)
P3-P4	0.74 (0.20)	0.68 (0.15)	0.70 (0.18)	0.63 (0.11)	0.59 (0.24)	0.51 (0.17)	0.56 (0.16)	0.49 (0.10)
O1-O2	0.80 (0.21)	0.76 (0.35)	0.75 (0.23)	0.70 (0.20)	0.69 (0.31)	0.64 (0.24)	0.65 (0.23)	0.61 (0.18)
Fp1-F3	0.74 (0.21)	0.69 (0.12)	0.79 (0.16)	0.75 (0.12)	0.86 (0.21)	0.81 (0.31)	0.70 (0.13)	0.64 (0.19)
Fp2-F4	0.74 (0.14)	0.65 (0.20)	0.80 (0.14)	0.74 (0.25)	0.88 (0.22)	0.82 (0.32)	0.69 (0.13)	0.65 (0.21)
T3-T5	0.57 (0.16)	0.53 (0.15)	0.58 (0.15)	0.55 (0.13)	0.56 (0.18)	0.53 (0.22)	0.42 (0.17)	0.46 (0.11)
T4-T6	0.57 (0.12)	0.51 (0.17)	0.59 (0.13)	0.55 (0.14)	0.55 (0.20)	0.57 (0.25)	0.46 (0.13)	0.45 (0.14)
C3-P3	0.77 (0.16)	0.69 (0.20)	0.78 (0.15)	0.70 (0.20)	0.70 (0.22)	0.65 (0.27)	0.69 (0.13)	0.64 (0.12)
C4-P4	0.76 (0.14)	0.73 (0.16)	0.79 (0.11)	0.70 (0.21)	0.67 (0.22)	0.63 (0.30)	0.70 (0.11)	0.64 (0.15)
F3-O1	0.19 (0.10)	0.08 (0.05)	0.21 (0.11)	0.11 (0.08)	0.28 (0.13)	0.26 (0.40)	0.14 (0.07)	0.17 (0.43)
F4-O2	0.18 (0.11)	0.10 (0.07)	0.18 (0.10)	0.12 (0.05)	0.19 (0.13)	0.28 (0.52)	0.11 (0.08)	0.09 (0.12)

Data are shown for each frequency band for each group.

et al. (2001) have shown that autistics do not activate the fusiform gyrus during facial processing as normals do, suggesting that this may be due to a disordered system of neural connectivity.

In the present study there was a reduction in absolute delta across the entire scalp, which was maximal in left frontal and posterior regions and in relative delta in frontal regions and over the vertex. While our findings of lower delta over the frontal cortex of autistics are not consistent with those of Cantor et al. (1986), they do suggest difficulty in the functional integration of these regions of the brain. Prior research has supported the finding of frontal dysfunction by showing evidence of anomalies in neuronal integrity (Murphy et al., 2002), malformation of minicolumn microcircuitry (Courchesne and Pierce, 2005), frontal lobe enlargement, as well as atypical patterns of brain connectivity (Hill, 2004) in autism. These results show that frontal system abnormalities are one aspect of brain dysfunction in children with autism.

Atypical patterns of brain connectivity were also evident in the results for the coherence analyses. In this sample of autistic children, coherences were lower than in control subjects, suggesting a pattern of underconnectivity. Within the same hemisphere, delta and theta coherences were low for both short to medium and longer inter-electrode distances. Interhemispherically, delta and theta coherences were low across the frontal region. Delta, theta, and alpha hypocoherence was also evident over the temporal regions. Lastly, there were low delta, theta and beta coherence measurements across posterior regions. These differences appear to be global, with slow-wave coherences reduced in every analysis undertaken. Of note were the greater number of significant findings in the coherence versus power domains, and a greater level of significance for the majority of coherence findings. This suggests that neural organization and connectivity may be a primary dysfunction of the autistic brain. This is consistent with previous EEG research suggesting anomalous cerebral lateralization (Dawson et al., 1982), asymmetry (Ogawa et al., 1982) and coherence (Cantor et al., 1986; Murias et al., 2006). There are also inconsistencies in the EEG autism – coherence literature. Cantor et al. (1986) found evidence of elevated coherence compared to a comparison group, but with a small ($N = 11$) sample size. Murias et al. (2006) found a combination of higher and lower coherences in different regions of the brains of adult autistics. Our study is not comparable given their sample of adults only and differences in methodology, including referencing and coherence measurements. Future research in this area is needed to study the referencing dilemma and its impact on coherence measurements. Enhancing the regional resolution and EEG coherence measurements should also be a priority.

Consistent with our findings of reduced coherence, other researchers have also reported findings of atypical cortical connectivity patterns in individuals with autism (Rippon et al., 2007). Evidence of abnormal connectivity has been associated with social cognition deficits (Barnea-Goraly et al., 2004), frontal system dysfunction (Belmonte et al., 2004), and facial and sensory processing (Frith, 2003) impairments in autistics.

These findings of reduced EEG coherence are further bolstered by MRI and fMRI research showing similar findings. MRI studies have shown decreased size of the corpus callosum (Courchesne et al., 1993) in general and in posterior aspects specifically (Chung et al., 2004). Pertaining to frontal system connectivity, Courchesne and Pierce (2005) suggested that the frontal lobes of autistics were disorganized and inadequately selective, whereas connectivity between the frontal cortex and other systems were poorly synchronized and weakly responsive. fMRI connectivity studies have shown low frontal to parietal connectivity (Just et al., 2007) and

reduced anterior to posterior connectivity in general (Cherkassky et al., 2006). Lastly, Pierce et al. (2001) have shown that autistics do not activate the fusiform gyrus during facial processing as normals do, suggesting that this may be due to a disordered system of neural connectivity. Hughes (2007) summarized these findings and concluded that in autistics there is initial overgrowth of the white matter tracts in the first 2 years of life, often followed by arrested growth and aberrant connectivity patterns. Future work is clearly needed to further detail these connectivity patterns. The use of multiple technologies, including EEG, will be helpful in this regard.

The current study is unique in that it is the largest study conducted to identify abnormalities of EEG power and coherence in autism. There is clear evidence of frontal and parietal dysfunction in autistic children and a pervasive pattern of neural underconnectivity that warrants further and intensive investigation. Advances in the measurement of EEG coherence should be a goal for future investigations.

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