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Resting-state EEG gamma activity in children with Attention-Deficit/Hyperactivity Disorder

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ABSTRACT

Objective: Children with Attention-Deficit/Hyperactivity Disorder (AD/HD) have well-described abnormalities in the four traditional EEG bands. However, to date the gamma band has not been widely investigated. This study investigated resting-state EEG in children with AD/HD and matched controls, with a particular focus on gamma activity.
Method: Forty children with AD/HD, and 40 age- and sex-matched controls, participated. EEG was recorded from 19 sites during an eyes-closed resting condition and Fourier transformed to provide estimates for absolute and relative power in the delta, theta, alpha, beta and gamma bands.
Results: Children with AD/HD had elevated levels of absolute delta and theta power, and decreased levels of absolute beta and gamma power, compared to controls. With relative power measures, children with AD/HD showed enhanced delta and theta activity, with reduced alpha, beta and gamma activity. Inattention scores on the Conners' Parent Rating Scale were negatively correlated with absolute gamma.
Conclusions: These patients demonstrate the typical EEG profile in the eyes-closed resting state, over the delta, theta, alpha and beta bands, associated with AD/HD. In addition, compared with controls, they demonstrate reduced absolute and relative gamma activity. These differences appear to contribute importantly to their dysfunctional stimulus processing, and impact their behavioural outcomes.
Significance: This resting-state study extends the well-established fast-wave EEG deficits in children with AD/HD to the gamma band, and links that to increased inattention, which is of special importance in understanding their cognitive-processing problems.

1. Introduction

One of the most common psychiatric disorders treated by child and adolescent psychiatrists in the US is Attention-Deficit/Hyperactivity Disorder (AD/HD) (Cantwell, 1996), with the prevalence rate in children estimated at 3–6% (Pelham et al., 1992; Lindgren et al., 1990). AD/HD is characterized by hyperactivity, impulsivity and inattention, which may change in pattern from preschool to adulthood (APA, 1994). AD/HD occurs in both males and females, with a population sex ratio of approximately 3:1 males:females (Gaub and Carlson, 1997). The disorder interferes with many areas of normal development and functioning, and if untreated, may predispose a child to psychiatric and social problems in later life.

EEG studies of children with AD/HD have typically found increased theta activity (Satterfield et al., 1972; Mann et al., 1992; Janzen et al., 1995; Clarke et al., 1998, 2001a,b,c), occurring primarily in the frontal regions (Chabot and Serfontein, 1996; Lazzaro et al., 1998), increased posterior delta (Matousek et al., 1984; Clarke et al., 1998, 2001a,b) and decreased alpha and beta activity (Dykman et al., 1982; Callaway et al., 1983), also most apparent in the posterior regions (Clarke et al., 1998, 2001a,b; Lazzaro et al., 1998); see Barry et al. (2003) for a review. The bulk of these data have been obtained from the resting eyes-closed condition, which provides the best estimate of spontaneous EEG activity (Barry et al., 2007). It is increasingly recognised that spontaneous EEG is the substrate of brain activity underlying cognition and behaviour (e.g., Barry, 2009), and the consistency of such group differences may eventually help our understanding of the symptoms of the disorder.

Gamma activity lies above these traditional EEG frequency bands, and is commonly considered to range between approximately 30 and 80 Hz. Because of its association with cognitive functioning (Engel et al., 2001; Fell et al., 2003), recent EEG research has seen an increased focus on activity in the gamma band. In a study of young children (<3 years), Benasich et al. (2008) re-

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ported that children with better language and cognitive skills, and better attention and inhibition control, had higher levels of frontal gamma power in the eyes-open resting state. Links with composite IQ scores did not reach significance, and the authors suggested that developmental lag may be reflected in reduced gamma.

Despite the obvious relevance of such observations to the AD/HD field, there is a paucity of research exploring gamma in this disorder. Yordanova et al. (2002) examined data from eight groups of control subjects ($N = 114$ in total) aged from 9 to 16 years. They also reported spontaneous gamma activity in an eyes-closed resting state, but this used RMS amplitude values derived from only 20 s of EEG data, filtered to 31–63 Hz. They noted a frontal distribution, with no developmental trends. With so little EEG data, substantially below what is normally considered acceptable, it is possible that these results reflect a lack of statistical power rather than a genuine lack of developmental change over this age range. While there are a few studies on gamma activity in AD/HD during cognitive processing (e.g., Yordanova et al., 2001), there is little information on spontaneous gamma activity in the eyes-closed resting state. Yordanova et al. (2001) did include a report on spontaneous RMS gamma amplitudes in an eyes-closed condition, but these were also derived from only 20 s of data, and from only 14 children with AD/HD. No gamma differences were obtained between the control and AD/HD groups, but it is possible that the restricted EEG sample and small *N* of that study reduced the power to exclude the null result. Also, that study closely matched patients and controls on IQ and various other variables, resulting in their inclusion of only 14 patients from the original sample of 540. Such a sample, although well-controlled on these variables, is unrepresentative of the disorder, and may be considered as a statistical outlier group as far as AD/HD is concerned. It is certainly of little direct relevance to the clinician faced with children with AD/HD of several types, and often with lower IQ.

Hence the aim of this study was to investigate the nature of resting-state EEG differences in the gamma band between children with and without AD/HD, using a larger and more-representative patient group, and more extensive EEG samples, than previous research. Based on the observations of Benasich et al. (2008) noted above, we expect that the AD/HD group will demonstrate reduced gamma activity. In order to place our study in the context of the wider AD/HD EEG literature summarised above, we also analysed the usual delta, theta, alpha and beta bands.

2. Method

2.1. Subjects

Two groups of 40 subjects, with 29 boys and 11 girls in each group (approximating the 3:1 population sex ratio), participated in this study. The groups consisted of an AD/HD group and a control group. All children were between the ages of 8 and 12 years, with a full-scale WISC-III (Wechsler, 1992) IQ score of 85 or higher. Only 3 girls in each group were aged >10 years, so the impact of pubertal differences in this study is likely to be small. All subjects in each group were individually matched on age, using 1 year age bands to control for maturational changes in the EEG.

The AD/HD group was drawn from new patients referred to a paediatric practice for a developmental assessment. These subjects had not been assessed previously, had no history of medication use for the disorder, and were tested before being prescribed any medication. The control group consisted of children from local schools and community groups.

Inclusion in the AD/HD group was based on a clinical assessment by a paediatrician and a psychologist; children were included only where both agreed on the diagnosis. DSM-IV criteria were used and children were included in the AD/HD group if they met the full diagnostic criteria for AD/HD. The clinical assessment incorporated information from as many sources as were available. These included a history given by a parent or guardian, school reports for a minimum of the past 12 months, scores on the Conners' Parent Rating Scale – Revised (L) (Conners, 1997), reports from any other health professionals, and behavioural observations during the assessment. Children were excluded from the AD/HD group if they had a history of a problematic prenatal, perinatal or neonatal period, a disorder of consciousness, a head injury with cerebral symptoms, a history of central nervous system diseases, convulsions or a history of convulsive disorders, paroxysmal headache or tics.

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. Assessment for inclusion as a control was based on a clinical interview with a parent or guardian similar to that of the AD/HD subjects, utilising the same sources of information.

Children were excluded from both groups if spike wave activity was present in the EEG. No data from these children have been reported previously.

2.2. Procedure

The parent(s)/guardian(s) of all participants gave their written informed consent prior to the release of any clinical records or testing, and all children gave their written assent. Ethics approval for this study was obtained from the combined Illawarra Area Health/University of Wollongong Human Research Ethics Committee. All AD/HD participants were assessed and received standard medical care at the paediatric practice of RM and MS. All subjects were tested in a single session lasting approximately 2.5 h. This included a psychometric assessment followed by an EEG assessment.

The EEG was recorded for 5 min in an eyes-closed resting condition, while subjects were seated on a reclining chair. Electrode placement was in accordance with the international 10–20 system, using an electrode cap produced by Electrocap International. The activity in 19 derivations was divided into nine regions by averaging in each region. 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). A linked ear reference was used, with a cap ground between Fpz and Fz. Impedance levels were less than 5 kOhm.

The EEG was recorded using a Lexicor NRS-24 with a sampling rate of 256 Hz and gain of 32,000, with a low frequency filter set at 0.5 Hz, a high frequency filter set at 70 Hz, and a notch filter at 50 Hz, and data were stored for off line analysis. The Lexicor data were converted for analysis using Neuroscan software (version 4.3). The data were filtered using a low pass filter at 48 Hz (zero-phase shift, down 24 dB) and segmented into 2 s epochs. Every epoch was visually appraised to reject ocular (>50 μV) and other artefact; no child contributed <24 artefact-free epochs. Accepted epochs were DC-zeroed and their Fast Fourier Transforms (FFTs) were averaged to obtain power estimates in five frequency bands: Delta (1.5–3.5 Hz), theta (4–7.5 Hz), alpha (8–13 Hz), beta (13.5–25 Hz) and gamma, for both absolute and relative power, as well as the total power of the EEG in these bands. The 30–80 Hz gamma range includes the usual mains frequency of 50 or 60 Hz, commonly removed in EEG recording by a notch filter at that frequency, and this needs to be considered in choosing a practical frequency range. A gamma peak around 40 Hz has been found to be dominant throughout childhood (Takano and Ogawa, 1998),

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and hence we defined our gamma band using a bandwidth of 35–45 Hz, avoiding mains-frequency problems.

2.3. Statistical analysis

Because the absolute data of some bands were skewed, all absolute measures were log-transformed before 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 (9 levels), and group (2 levels).

Within region, two orthogonal three-level repeated-measures factors, and their interactions, were examined. The first of these was a sagittal factor, within which planned contrasts compared the frontal (F) region with the posterior (P) region, and their mean (F/P) with the central (C) region. The second factor was laterality, within which similar planned contrasts compared activity in the left (L) and right (R) hemispheres, and their mean (L/R) with the midline (M) region. These planned contrasts allow optimal clarification of topography, with complete specification of which region(s) is(are) dominant. Within group, a between-subjects factor, diagnosis (2 levels), was examined comparing the AD/HD (A) group to the control (C) group.

To check that the gamma results were not unduly influenced by muscle artefact in the temporal regions, absolute and relative gamma were reanalysed (with a similar sagittal analysis) using data only from the 3 midline sites.

Unless otherwise indicated, all F values reported have (1, 78) degrees of freedom. Such 1 df contrasts preclude the problems of non-sphericity of the variance–covariance matrix often found with repeated physiological measures, thus avoiding the need for their control using Greenhouse-Geisser type epsilon adjustments. 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 α is required (Tabachnick and Fidell, 1989). Only significant between-group effects and interactions are reported here for space reasons.

Pearson correlations were examined in both groups between gamma and IQ, and in the patient group between gamma and Conners' scores on the DSM Inattentive, DSM Hyperactive-Impulsive, and DSM Total scales. These analyses were treated as exploratory, and alpha was not adjusted for multiple tests.

3. Results

In the patient group, 25 children were diagnosed with AD/HD of the Combined type, and 15 had AD/HD of the Inattentive type; their Conners' T-scores are shown in Table 1. The groups did not differ significantly on mean age (controls: 10.2 years, c.f. AD/HD: 10.0 years; $F < 1$). Although both groups were in the normal range, mean IQ was significantly lower in the AD/HD group (95.7) than controls (105.6; $F = 16.63$, $p < .001$).

3.1. Absolute power

Fig. 1 shows the frequency distribution for each group, averaged across all sites. The top panel shows the usual low frequency range used here, where the AD/HD group shows enhanced delta and theta, a different alpha distribution, and reduced beta, compared with the control group. The bottom panel (note reduced power scale) shows the higher frequency range containing the present gamma band (35–45 Hz). The controls show a mean gamma peak at 38 Hz, which is largely missing in AD/HD. At the individual level, across-electrode mean gamma peaks could be identified in all subjects, although these were generally very small and very restricted in frequency range, particularly in AD/HD. The mean of these peak frequencies was 38.8 (SD 1.2) Hz in controls, and 38.4 (SD 1.5) Hz in AD/HD; these values were not significantly different. Beyond 47 Hz, the controls show some remnant of 50 Hz mains frequency leakage, but this is outside our gamma band.

The mean across-electrode absolute gamma power for each group is shown in Fig. 2 as a box and whisker plot, with each data point in the scatter representing one subject. There are two outliers in the control group, and one in the AD/HD group. With the log-transformed data used for the topographic MANOVA, there are no outliers.

Head maps indicating scalp topography of the two groups for each absolute power band are shown in the top panel of Fig. 3. Compared with controls, AD/HD children had elevated absolute delta (A > C: $F = 7.30$, $p < .01$), particularly in posterior compared with frontal regions in the midline (A > C × L/R < M × F < P: $F = 7.37$, $p < .01$). They also had elevated absolute theta (A > C:

Table 1
Scores of the AD/HD groups on the Conners' Rating Scales.

	N	DSM-IV inattentive	DSM-IV hyperactive-impulsive	DSM-IV total
AD/HD combined type	25	75.1 (11.4)	77.3 (8.8)	77.8 (7.8)
AD/HD inattentive type	15	68.2 (13.4)	53.8 (5.8)	62.4 (9.6)

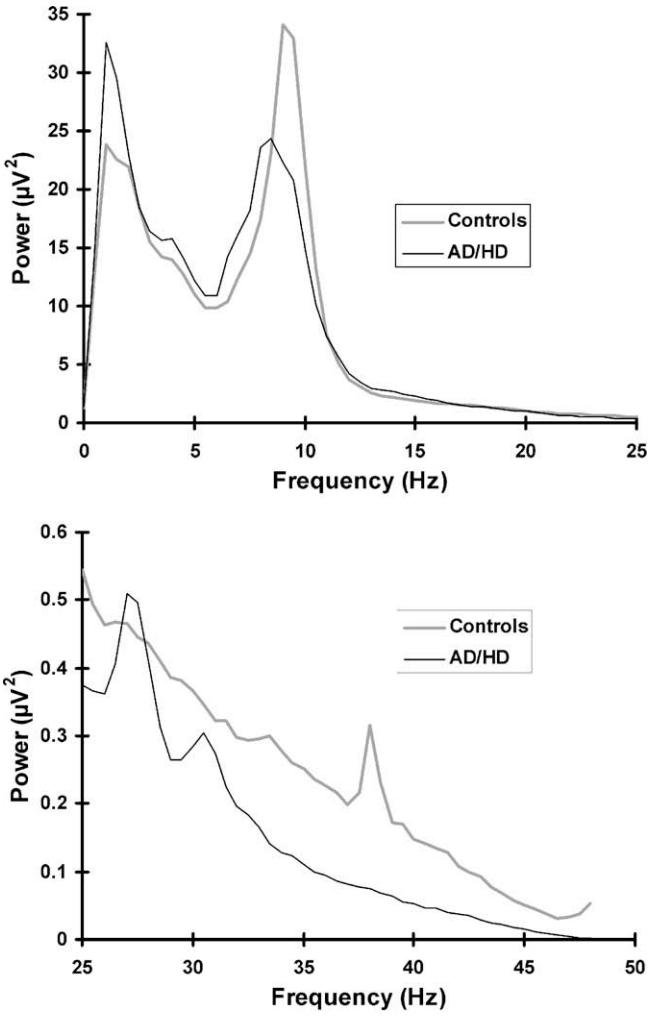

Fig. 1. Frequency distribution for each group, averaged across all sites. Top: the usual low frequency range reported; bottom: the higher frequency range containing the gamma band.

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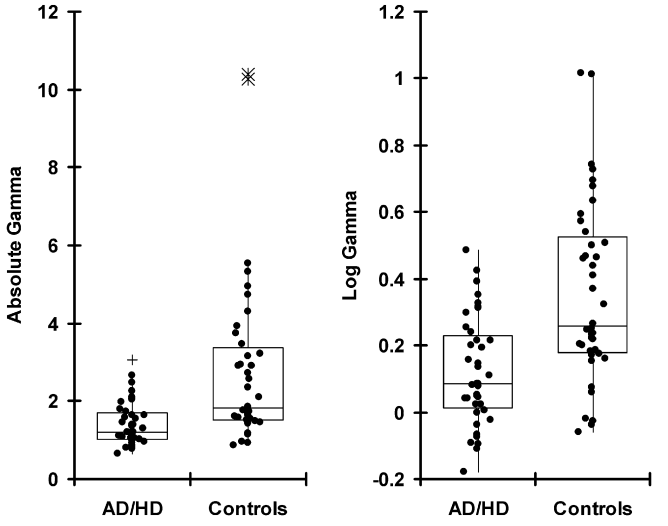

Fig. 2. Box and whisker plots for absolute gamma and log-transformed gamma in each group. Each point represents one subject's data. The centre line in each box represents the median value, with the top and bottom of the box at the upper and lower quartile values, respectively. The whiskers show the range of data within ±1.5 interquartile ranges (IQRs) from the median. There is one outlier apparent in the AD/HD group (>1.5 IQR) and two in the control group (> 3 IQR). With the log-transformed data, there are no outliers.

$F = 5.68$, $p < .05$), particularly in posterior compared with frontal regions (A > C × F < P: $F = 7.69$, $p < .01$). There were no significant group differences in absolute alpha power, suggesting that the reduced AD/HD higher alpha was partly cancelled by their enhanced lower alpha apparent in Fig. 1. Compared with controls, AD/HD children had reduced absolute beta (A < C: $F = 5.95$, $p < .05$).

Absolute gamma values at each site are shown in Table 2. The AD/HD group had reduced absolute gamma (A < C: $F = 16.95$, $p < .001$), particularly in posterior compared with frontal regions (A < C × F < P: $F = 11.53$, $p < .001$), and in the hemispheres compared with the midline (A < C × L/R > M: $F = 5.61$, $p < .05$). These last two effects interacted, with AD/HD children showing a greater posterior reduction in the hemispheres than midline (A < C × F < P × L/R > M: $F = 13.86$, $p < .001$); this reduction was larger in the left hemisphere (A < C × F < P × L > R: $F = 4.16$, $p < .05$). In total power across the bands, children with AD/HD showed elevated activity in the posterior hemispheres compared with the controls (A > C × F < P × L/R > M: $F = 4.66$, $p < .05$).

3.2. Relative power

Head maps showing scalp topography of the two groups for each relative power band are shown in the bottom panel of Fig. 3. Compared with controls, children with AD/HD had globally enhanced relative delta (A > C: $F = 4.36$, $p < .05$), and this increase

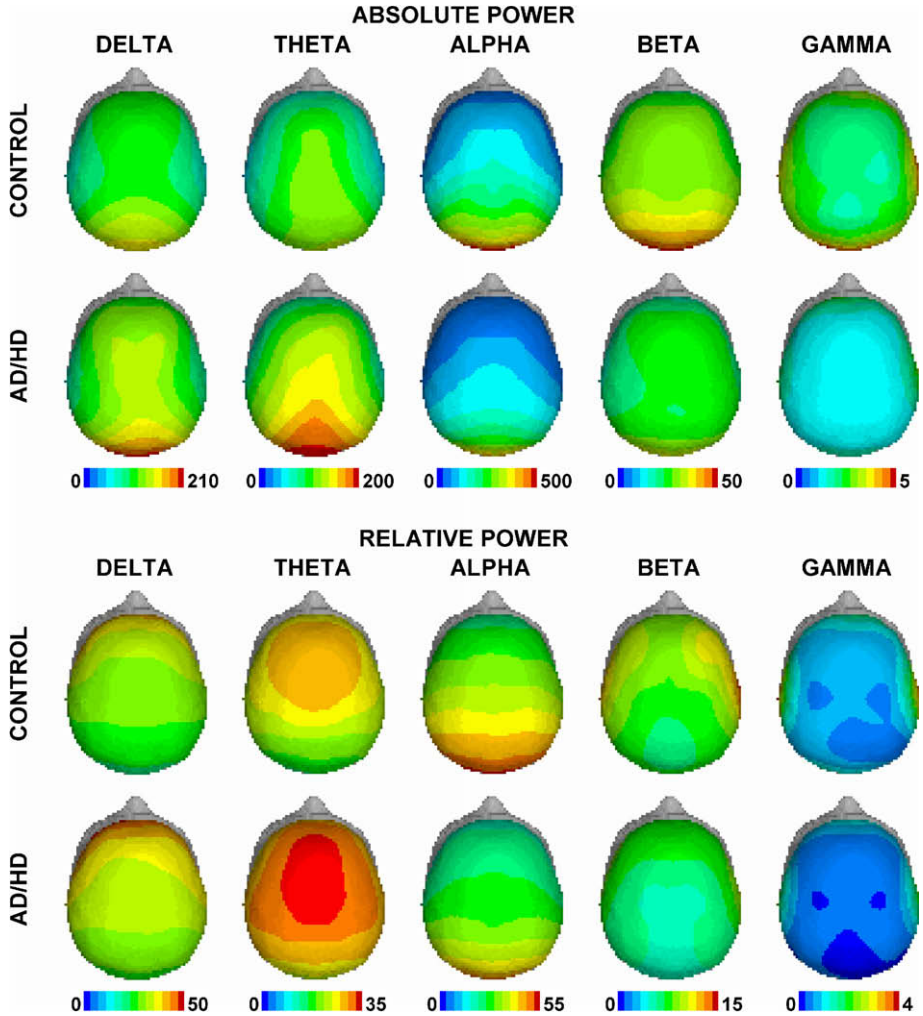

Fig. 3. Topographic maps for absolute and relative power for the AD/HD and control groups. Scales: absolute power in μV^2; relative power in%.

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Table 2
Means (standard deviations) for gamma activity at each electrode.

Electrode	Absolute Gamma		Relative Gamma	
	Control	AD/HD	Control	AD/HD
Fp1	2.44 (1.68)	1.85 (1.15)	1.08 (0.76)	0.89 (0.64)
Fp2	2.76 (2.28)	1.81 (1.26)	1.17 (0.96)	0.81 (0.56)
F3	1.61 (0.96)	1.13 (0.40)	0.58 (0.33)	0.39 (0.15)
F4	1.81 (1.18)	1.18 (0.14)	0.67 (0.49)	0.39 (0.14)
F7	2.65 (3.08)	1.46 (0.79)	1.32 (1.31)	0.82 (0.52)
F8	2.47 (2.26)	1.60 (1.00)	1.30 (1.11)	0.81 (0.58)
Fz	1.56 (0.83)	1.08 (0.37)	0.51 (0.27)	0.34 (0.12)
C3	1.85 (1.72)	1.07 (0.34)	0.60 (0.51)	0.33 (0.14)
C4	1.83 (1.56)	1.09 (0.39)	0.58 (0.41)	0.33 (0.13)
Cz	1.64 (0.94)	1.14 (0.38)	0.46 (0.29)	0.28 (0.10)
P3	1.96 (1.46)	1.01 (0.31)	0.49 (0.43)	0.25 (0.12)
P4	1.86 (1.30)	1.05 (0.38)	0.46 (0.37)	0.26 (0.12)
Pz	1.70 (1.07)	1.06 (0.34)	0.40 (0.32)	0.22 (0.10)
T3	3.32 (2.55)	2.08 (1.92)	2.78 (2.95)	1.67 (1.73)
T4	3.84 (3.59)	2.31 (2.08)	3.21 (3.58)	1.66 (2.40)
T5	2.51 (2.14)	1.14 (0.48)	0.88 (0.86)	0.39 (0.26)
T6	2.47 (1.79)	1.21 (0.45)	0.77 (0.72)	0.37 (0.21)
O1	4.76 (5.46)	1.30 (0.58)	0.96 (1.26)	0.24 (0.37)
O2	3.88 (4.08)	1.29 (0.49)	0.76 (1.16)	0.19 (0.11)

was greater in the hemispheres than the midline (A > C × L/R > M: $F = 5.83$, $p < .05$). They had increased relative theta (A > C: $F = 15.17$, $p < .001$), particularly in posterior regions (A > C × F < P: $F = 13.71$, $p < .001$), and in the midline (A > C × L/R < M: $F = 6.69$, $p < .05$). Children with AD/HD had globally reduced relative alpha (A < C: $F = 10.58$, $p < .005$), and relative beta (A < C: $F = 13.21$, $p < .001$), compared with controls. Relative gamma activity (see Table 2) was also globally reduced (A < C: $F = 13.07$, $p < .001$), particularly in the hemispheres (A < C × L/R > M: $F = 7.42$, $p < .01$).

3.3. Gamma at the midline sites

Absolute gamma was reduced in the AD/HD group (A < C: $F = 13.40$, $p < .001$); this was least at Cz (A < C × Cz < Fz/Pz: $F = 6.81$, $p < .05$). Relative gamma activity in the midline was globally reduced in children with AD/HD compared with control children (A < C: $F = 13.55$, $p < .001$).

3.4. Gamma and individual characteristics

Table 3 shows correlations between the individual characteristics of IQ and Conners' scores with across-scalp mean absolute and relative gamma. The only significant correlations were between absolute gamma and scores on the DSM Inattentive and DSM Total scales.

4. Discussion

The groups were well-matched on age, with IQs in the normal range, but the AD/HD group had a significantly-lower mean IQ level than the control group. However, our previous work (Clarke et al., 2001a, 2006, 2008a) has demonstrated that IQ differences do not contribute to observed group EEG differences in the lower bands, and this was confirmed here with correlations between IQ and absolute and relative gamma, in both the control and AD/HD groups (see Table 3). As mentioned in Section 1, Benasich et al. (2008) also failed to find a significant correlation between gamma and composite IQ. Thus we can relate EEG differences observed between the groups solely to the existence or otherwise of AD/HD.

EEG studies of children with AD/HD have typically found that these children have increased theta activity (Mann et al., 1992; Chabot and Serfontein, 1996; Clarke et al., 1998, 2001a,c), increased posterior delta (Clarke et al., 1998, 2001a,c), and decreased alpha and beta activity (Mann et al., 1992; Clarke et al., 1998, 2001a,c) compared to normal children. In the present study the AD/HD group had more absolute and relative delta and theta, less relative alpha, and less absolute and relative beta than the control group. This pattern of results is typical of the literature. The elevated absolute delta was dominant in the posterior midline, consistent with previous studies (Matousek et al., 1984; Clarke et al., 1998, 2001a,b). The elevation in absolute theta was dominant in the posterior, contrary to some reports of frontal elevations (Chabot and Serfontein, 1996; Lazzaro et al., 1998), but compatible with other observations (e.g., see Fig. 4 in Barry and Clarke, in press). The increase in relative delta was larger in the hemispheres than the midline. This has not been reported previously, although clear trends in this direction are apparent in other studies (e.g., Clarke et al., 2001d, 2008b). Relative theta was elevated more in posterior and midline regions. While the posterior enhancement is not common (most studies find only a global increase, as also found in the present results), increased midline theta activity is routinely found (Clarke et al., 1998, 2002a,b,c, 2006, 2007). These results indicate that the children with AD/HD in this study have an EEG profile in the lower four bands that is highly typical of this disorder.

In the gamma band, the AD/HD children displayed reduced absolute and relative activity. Fig. 2 indicates the generality of this gamma reduction – it is obviously not due solely to extreme values in a few outliers in either the Control or AD/HD group. With absolute gamma, reductions were larger in the posterior and left hemisphere regions. With relative gamma, the reductions were larger in the hemispheres. Because muscle activity in temporal regions may contribute to apparent EEG gamma levels, we repeated the gamma analyses using only the 3 midline sites. These analyses confirmed the major results for both gamma measures, suggesting that these data represent genuine gamma band activity in AD/HD.

If gamma activity is important in cognitive and attentional processing, reduced spontaneous gamma activity in AD/HD may underlie many of the deficits associated with the disorder. Benasich et al.'s (2008) study with young children implied a direct link between reduced gamma and poor language/cognitive skills, attention and inhibition control, suggesting that developmental lag may be reflected in reduced gamma. The present results are directly compatible, showing significant negative correlations between absolute gamma and scores on the Conners' DSM Inattentive and DSM Total scales. The negative (but non-significant) correlations of gamma with scores on the Conners' DSM Hyperactive-Impulsive

Table 3
Pearson correlations for individual characteristics and gamma.

Group	Controls		AD/HD							
Measure	IQ		IQ		DSM inattentive		DSM hyperactive-impulsive		DSM total	
	r	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*
Absolute gamma	.21	.19	.03	.83	**−.35**	**.02**	−.22	.17	**−.37**	**.02**
Relative gamma	.12	.46	−.22	.17	−.09	.58	−.17	.30	−.16	.33

Significant values are shown in bold.

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scale supports the assertion that our "gamma" was not merely an artefact of muscular origin, as we would expect a positive correlation between hyperactivity and muscular artefact. There are some differences in the topography of the reductions noted by Benasich et al. (frontal) and those found here (posterior and hemispheric), but this may reflect differences between eyes-open (Benasich et al.) and our eyes-closed conditions.

This study examined gamma in AD/HD relative to an age- and gender-matched control group. The AD/HD group contained patients with the combined and inattentive types of the disorder, and future studies could profitably explore type differences. Previous work on the lower bands has found reliable differences relatable to the symptom differences (e.g., Clarke et al., 1998, 2001a), and similar finding can be expected in gamma. Also, although we used a gender balance compatible with recent reports in the literature, it would be useful for future studies to examine gamma in both controls and AD/HD groups in separate gender samples.

We note that Yordanova et al. (2001) reported greater evoked gamma activity in AD/HD in a cognitive-processing ERP study. The connections between resting gamma activity and evoked gamma activity are unclear (see Herrmann and Demiralp, 2005 for a review), but increased gamma activity in a task, compared with a lower resting baseline level, could indicate that children with AD/HD require greater gamma activation to attain and maintain adequate performance in the task. Such an interpretation would mesh in with a recent ERP study of adults with AD/HD, which reported evidence that patients used more-effortful processing to overcome early sensory-processing impairments (Barry et al., 2009). Further research on the gamma band in AD/HD, in both resting and active task situations, is clearly warranted.

From a wider clinical perspective, the emerging recognition that spontaneous EEG functions as the substrate of brain activity, underlying cognition and behaviour, suggests the value of increasing such EEG research in a range of disorders. In particular, gamma's involvement in the mechanism enabling transient associations of cortical ensembles underlying information processing (Fell et al., 2003) suggests that resting-state gamma anomalies may be of core relevance in a range of syndromes with dysfunctional attention and cognition. The review by Herrmann and Demiralp (2005) sketched fragmentary evidence suggesting that spontaneous gamma is elevated in epilepsy, hallucinations and schizophrenia with positive symptoms, and reduced in Alzheimer's, migraine, stroke and brain injury, and schizophrenia with negative symptoms. It will be important in future work to clarify which symptoms in these disorders, and in AD/HD, are specifically linked to gamma anomalies. Our linkage of inattention, a core symptom of AD/HD, to reduced gamma is a first step in this direction.

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