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EEG development in Attention Deficit Hyperactivity Disorder: From child to adult

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HIGHLIGHTS

- Maturation of the ADHD EEG profile is evident from child to adult.
- Significant normalisation of ADHD child EEG occurs by adulthood.
- Enhanced theta remains in ADHD throughout maturation.

ABSTRACT

Objective: Attention Deficit Hyperactivity Disorder (ADHD) is one of the most common psychiatric disorders found in children. While an extensive literature has documented the EEG in this clinical population, few studies have investigated EEG throughout the lifespan in ADHD. This study aimed to investigate EEG maturational changes, in subjects with ADHD combined type, that spanned from childhood into adulthood.
Method: Twenty five male adults with ADHD were assessed between the ages of 8–12 years and again as adults. At both ages, an EEG was recorded during an eyes-closed resting period, and power estimates were calculated for relative delta, theta, alpha and beta.
Results: At the childhood assessment, the ADHD subjects had elevated posterior delta. Relative theta was elevated, with diminished alpha activity across all sites. Significant maturational changes were observed, with reductions in the delta and theta bands, and increases in the alpha and beta bands across all electrodes. In adulthood, relative to controls, diminished frontal delta and elevated global theta activity were apparent.
Conclusions: Substantial developmental changes occurred in the EEG of these subjects. These results identify important issues when using EEG as part of the diagnosis for ADHD.
Significance: This study is the first to explore EEG changes from childhood to adulthood over an 11 year period in the same subjects with ADHD.

1. Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is a common psychiatric condition affecting approximately 5% of children and is found in 2.5% of the adult population (APA, 2013). The symptoms in childhood include hyperactivity, impulsivity and inattention, but these symptoms change greatly across the lifespan, with hyperactivity and impulsivity decreasing with age, although inattention seems to remain elevated (Biederman et al., 2000). One model of ADHD that still holds currency today is the maturational lag model, which was first hypothesised by Kinsbourne (1973). Kinsbourne noted that children with gross brain damage exhibited abnormalities on neurological examinations. However, when compared to younger children, many of the exhibited behaviours would not be considered clinically meaningful. This was seen to hold for overt behaviours, as well as attentional factors. From these observations, Minimal Brain Dysfunction (now known as ADHD) was seen as an immaturity of the brain, and the child was expected to show a reduction in symptoms with age, as the brain matured. In most cases, this outcome did in fact

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occur, as the focus of the diagnosis was primarily on the overt behavioural components of the disorder.

Previous research, that compared the EEG from children diagnosed with ADHD to non-ADHD control subjects, has identified a consistent EEG profile of greater theta activity, which is generally maximal in frontal electrode sites (Clarke et al., 2001b, 2001d, 2002a, 2002b, 2002c, 2002d, 2003a, 2003b, 2006, 2007, 2008, 2011; Hobbs et al., 2007). Greater delta activity in posterior regions (Clarke et al., 1998, 2001a, 2001c) is also often present, as is a reduction in activity in both the alpha and beta bands (Clarke et al., 1998, 2001a, 2001c, 2006, 2007, 2008, Loo et al., 2009; Nazari et al., 2011; Woltering et al., 2012; Dupuy et al., 2013). Several studies have interpreted their EEG findings as representing a maturational lag in the development of the central nervous system (CNS) (Mann et al., 1992; Clarke et al., 2001b, 2001c, 2001d, 2002c, 2011). These results have been interpreted based on findings within the normal population, where with increasing age, slow wave activity in the delta and theta bands reduce, and fast wave activity in the alpha and beta activity becomes dominant (John et al., 1980; Benninger et al., 1984; Gasser et al., 1988a, 1988b; Clarke et al., 2001a). Thus, younger children have more slow wave activity than older children.

As up to 75% of children with ADHD will continue to meet criteria as adults (Sibley et al., 2017), the problem for this model is whether a person can have a maturational lag that is still present in adulthood, or whether the problem represents a dysfunction that should be considered more pervasive. At present there are relatively few studies of EEG maturational changes in people with ADHD.

Satterfield et al. (1984) undertook a 4 year longitudinal study into maturational changes in children with hyperactivity. The results showed that the maturation of the EEG was greater in younger children, slowing as they became older. The rate of EEG maturation in the hyperactive group lagged behind that found in controls. From these results, Satterfield et al. concluded that hyperactivity was not caused by a maturational lag. Using a cross sectional design, Bresnahan, Anderson and Barry (1999) evaluated the EEG differences in ADHD subjects in childhood, adolescence, and as adults. Results identified that beta activity reduced with age, but theta activity remained elevated into adulthood. These results were interpreted as suggesting that beta activity and hyperactivity are associated, as is theta activity and impulsivity. Poil et al. (2014) investigated EEG maturational changes in children, adolescents and adults with ADHD using a cross sectional design. The ADHD subjects showed a normal pattern of development in their EEG, with slow wave activity reducing and faster wave activity increasing with age.

To date, no longitudinal ADHD studies have been performed that evaluate EEG changes from childhood into adulthood. This study aimed to evaluate maturation of the EEG of subjects with long term ADHD. We expect that long term repeated assessments from the same subjects will allow greater understanding of maturational processes in EEG development in this disorder and help determine the validity of the maturational lag model of ADHD.

2. Method

Seventy five males participated in this study. As this was an initial investigation, long-term patients of RM and MS were targeted for follow-up, as this increased the likelihood of contact, and also increased the probability that they would meet criteria for ADHD as adults. These subjects had previously been included in studies as children (Clarke et al., 2001a, 2001b, 2001c). All potential subjects were assessed as ADHD, combined type as children (DSM-IV; APA, 1994). Only combined type subjects were selected for this study as the maturational lag model was initially developed in children who met various versions of this diagnosis. Initially, attempts were made to contact 52 former patients. Forty five could be contacted, and of these, 40 agreed to participate in an assessment. However, due to distance constraints, EEGs were performed on only 37 subjects. Twenty five of the final 37 subjects met the diagnostic criteria for ADHD, Combined or Inattentive type in adulthood, according to the DSM-5 (APA, 2013). The mean time between assessments was 11.1 (SD 0.9) years. No subjects were still being treated for ADHD at the adult assessment. Twenty five boys and twenty five male adults were used as control subjects. The control groups were drawn from the local community at the time of the assessments, and were age-matched to the ADHD subjects at both assessments; they differed between assessments. Subjects were reimbursed for expenses incurred as part of this study.

In childhood, subjects were assessed by two mental health professionals, and were only included in the initial studies if they had never been medicated for ADHD prior to that assessment. Diagnosis used information from a clinical interview, school reports, their medical history, and behavioural observations conducted on the day of testing. The childhood exclusion criteria consisted of no history of central nervous system damage or disease, a diagnosis of a disorder of conduct, anxiety or depression, Autism Spectrum Disorder or Tourette's. All subjects also had an IQ assessment (WISC-III; Wechsler, 1992) and a reading assessment (Neale Analysis of Reading; Neale, 1999). Subjects were excluded if their IQ was less than 85.

Child controls were assessed on the same criteria as the clinical group, except reading ability had to be age appropriate, and they did not meet criteria for a psychiatric illness of childhood.

The adult assessment for ADHD was based on DSM-5 criteria (APA, 2013). Assessments included a clinical interview, an IQ test (WAIS-III), an assessment of reading age equivalence (Woodcock Reading Mastery Tests – Revised), an assessment for depression (Centre for Epidemiologic Studies Depression Scale; CES-D), and their general mental health status (General Health Questionnaire; GHQ). The Conners' Adult ADHD rating scale (CAARS; Conner et al., 1999) was used as part of this assessment to assess ADHD symptoms. A T-Score above 65 on the hyperactive-impulsive, inattentive, or total subscales was used for inclusion in this study. Adult control subjects were assessed using the same instruments as those used in the ADHD subjects, and had to score below the clinical range on all instruments.

2.1. Procedure

This study was approved by the University of Wollongong Human Research Ethics committee. Written consent was given by a parent at the child assessment and from the participants at the adult assessment.

2.1.1. Childhood assessment

This assessment took approximately 2.5 hours to complete. The EEG was recorded as the final procedure. Subjects were initially given a physical assessment by a paediatrician and a clinical history was taken using a semi-structured interview. Following this assessment, the psychometric testing was undertaken, which was subsequently followed by an EEG that consisted of an eye-closed resting condition and evoked potentials.

2.1.2. Adult assessment

Adult subjects were tested on one day, with the total testing taking approximately 4 h. This consisted of a clinical history that included past treatment for any psychiatric condition, their current housing, school and employment/unemployment history, criminal history, and legal or illegal drug use. The assessment of the ADHD

group was conducted a minimum of 10 years after the childhood assessment.

2.1.3. EEG assessment

Subjects were seated comfortably and their EEGs were recorded during an eyes-closed resting condition. All subjects were told to relax and stay as still as possible. When subjects lost concentration, or showed signs of drowsiness, the session was stopped for subjects to have a break before continuing.

An electrode cap with 19 tin electrodes was used in this study, and all active electrodes were referenced to linked-ears. For the data analysis, the activity in these sites were averaged, creating 9 regions of interest. These regions were right frontal (Fp2, F4, F8), right central (T4, C4), right posterior (T6, P4, O2), midline frontal (Fz), midline central (Cz), midline posterior (Pz), left frontal (Fp1, F3, F7), left central (T3, C3) and left posterior (T5, P3, O1). Reference and ground electrodes were tin disk electrodes. Impedances were set below 5 kOhm.

Childhood EEGs were analysed and recorded using the Cadwell Spectrum 32 system. The software was version 4.22, the Test used was EEG, and the Montage was Q-EEG. The gain used during recording was 12,800. Filter settings were: high frequency filter 70 Hz, low frequency filter 0.53 Hz, notch filter 50 Hz. The sampling rate was 200 Hz and 2.56 second epochs were used in the Fourier transformation.

Typically the recording session lasted about 15 min with 1 min of trace being selected for Fourier transformation, based on visual and automatically rejection. The EEG was Fourier transformed to provide estimates in 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) bands, with relative power estimates being calculated for each band by dividing absolute band power by the total power of the EEG.

For the adult assessment a Lexicor NRS-24 was used to record the EEG. This used a gain of 32,000 and a sampling rate of 256 Hz. The EEG was Fourier transformed using the NxLink software package. A minimum of one minute of EEG were selected by an experienced technician. The NxLink software used the same analysis package as was built into the Cadwell Spectrum 32. This software resampled the EEG to 100 Hz and epoched the data in 2.56-s epochs. This made the analyses of the childhood and adulthood data directly comparable.

2.2. Statistical analysis

Mixed MANOVAs were used to analyse the EEG data. Within these, group, age and region were examined within each frequency band. For each band, effects of regional difference were examined in two orthogonal three-level repeated-measures factors. Factor one was a sagittal factor, with planned contrasts comparing frontal (F) regions with posterior (P) regions, and the mean of these regions (F/P) with central (C) regions. Laterality was factor two. Planned contrasts compared the left (L) hemisphere with the right hemisphere (R), and the average of the two hemispheres (L/R) with midline (M) regions. These planned contrasts allowed complete examination of topographic effects. For the Group factor, orthogonal planned contrasts compared the control group with the ADHD group separately for both the child and adult assessments. A third repeated measures analysis was also performed to evaluate maturational process in the ADHD subjects.

Such F tests with 1 degree of freedom optimise exploration of site effects, as well as clarification of amplitude and topography differences between groups. They also avoid issues of variance-covariance matrix asymmetry in the data that would otherwise need Greenhouse-Geiser type adjustments (O'Brien and Kaiser, 1985). As all the contrasts were planned, and numbers of contrasts were less than the degrees of freedom for each effect, no adjustment of the α level was required (Tabachnick and Fidell, 2007). In the two ADHD versus control analyses all reported F values have (1, 48) degrees of freedom, and the assessment of maturational processes has (1, 24) degrees of freedom.

3. Results

3.1. Clinical data

Clinical data from each instrument is presented in Table 1. In childhood the ADHD subjects scored lower than controls on measures of IQ ($t = 2.371$, $p < .05$), and reading accuracy ($t = 5.409$, $p < .001$).

As adults, subjects were required to refrain from tobacco or caffeine for 4 hours prior to testing, and not to use prescription medications for ADHD or alcohol 24 hours prior to testing. All subjects indicated compliance with these instructions. During the assessment it was found that no subjects were being treated with any medication for ADHD.

All clinical subjects in this study met criteria for the Combined Type of ADHD as children. As adults, 19 subjects still met criteria for the Combined Type of ADHD and 6 subjects for the Inattentive Type. The mean t-scores for the CAARS were 64 for the Hyperactive-Impulsive subscale, 70 for the Inattentive subscale and 71 for the Total.

In adulthood the ADHD subjects scored significantly higher than controls on the CAARS Inattentive subscale ($t = 7.067$, $p < .001$), CAARS Hyperactive/Impulsive subscale ($t = 6.085$, $p < .001$), the CAARS Total ($t = 7.278$, $p < .001$), the CES-D ($t = 2.846$, $p < .01$) and reading ability ($F = 3.591$, $p < .01$).

The groups did not significantly differ for age in either the child or adult sample, or on the GHQ-60, or the measure of IQ in adults.

3.2. ADHD vs. controls in childhood

Across groups, we found relative delta was greatest in the frontal regions (F > P: $F = 121.69$, $p < .001$). Power at the frontal midline was significantly lower whereas central-posterior midline power was higher in comparison to the hemispheres (F < P × M > L/R: $F = 77.01$, $p < .001$; F/P < C × M > L/R: $F = 33.25$, $p < .001$). The ADHD group had significantly more posterior relative delta than controls (ADHD > Control × F < P: $F = 4.10$, $p = .020$; see Fig. 1 top row left side).

3.2.1. Theta

Across groups, relative theta was greatest fronto-centrally (F > P: $F = 31.53$, $p < .001$; F/P < C: $F = 57.44$, $p < .001$), and greater

Table 1
Mean (SD) ages and psychometric instrument scores for the ADHD and control groups in childhood and adulthood.

Mean (SD)	ADHD Group	Control Group
Childhood Assessment		
Age (Years)	10.31 (1.5)	10.04 (1.5)
Full Scale IQ (WISC-III)	105.2 (11.8)	113.4 (12.4)
Reading Accuracy (Neale)	8.7 (2.0)	11.7 (1.1)
Adulthood Assessment		
Age (Years)	21.69 (1.9)	21.0 (2.2)
CAARS E: Inattentive	70 (10.2)	49 (9.9)
CAARS F: Hyper-Impulsive	64 (11.7)	47 (7.3)
CAARS G: Total ADHD	71 (11.1)	49 (9.6)
Full Scale IQ (WAIS-III)	105.4 (15.0)	116 (8.6)
Depression (CESS-D)	13.5 (10.0)	7.0 (5.0)
Reading Ability (Woodcock)	16.2 (3.3)	18.5 (0.2)
General Mental Health (GHQ)	6.8 (8.2)	4.0 (4.0)

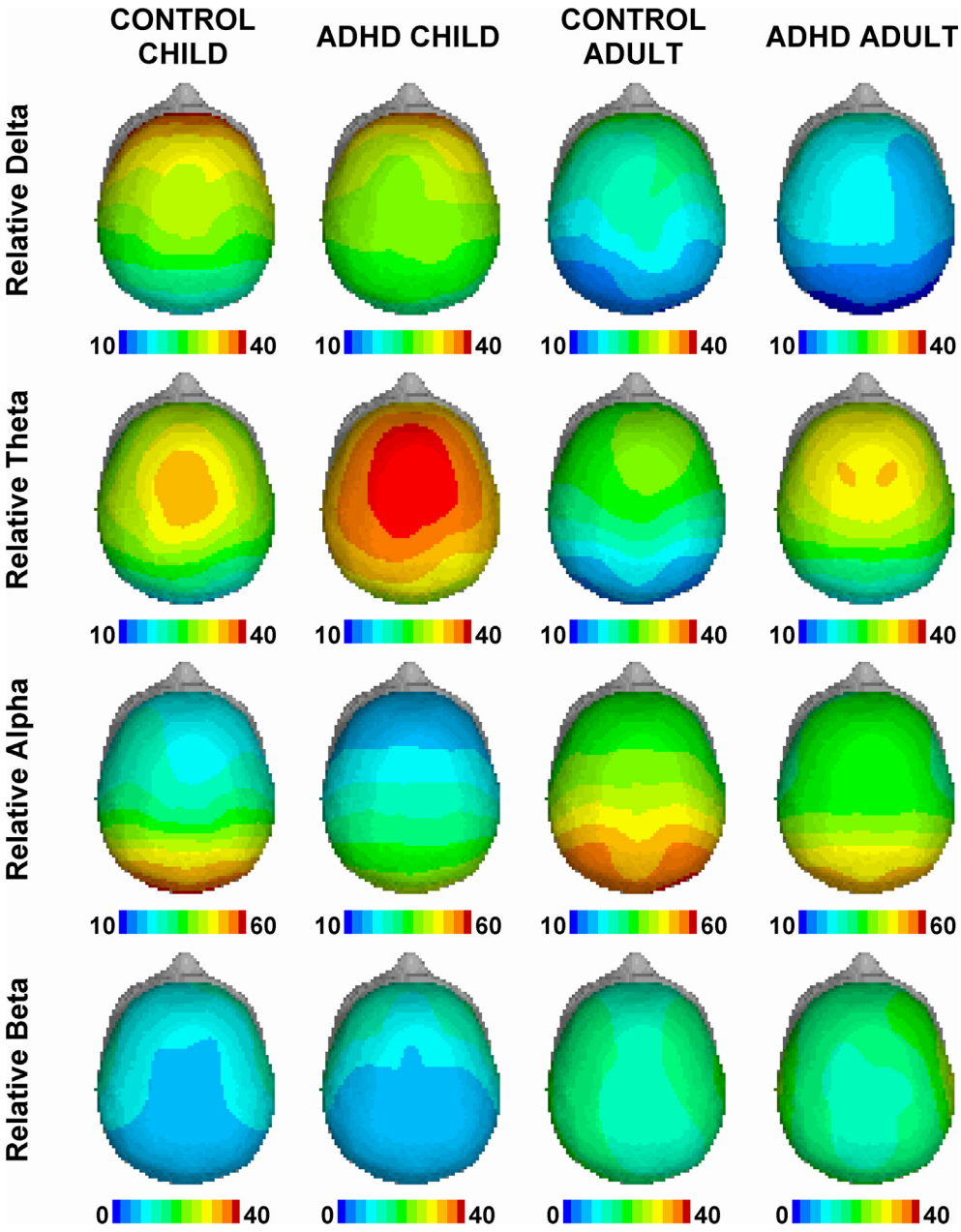

Fig. 1. Relative power as a function of scalp region for the control groups and ADHD groups in childhood and adulthood.

along the midline than in the hemispheres (M > L/R: $F = 163.81$, $p < .001$). Relative theta was significantly elevated across the entire scalp in the ADHD group compared to controls (Group Main Effect: $F = 10.81$, $p = .002$; Fig. 1 row 2 left side).

3.2.2. Alpha

Relative alpha was greatest in posterior regions across both groups (F < P: $F = 182.7$, $p < .001$; F/P > C: $F = 75.33$, $p < .001$). Midline alpha was significantly less than in the hemispheres (M < L/R: $F = 10.53$, $p = .002$). This reduction was greater in the posterior regions than frontally (F < P × M < L/R: $F = 32.51$, $p < .001$) and smallest at the vertex (F/P > C × M < L/R: $F = 10.86$, $p = .002$). The ADHD group had a global reduction in relative alpha compared to controls (Group Main Effect: $F = 4.23$, $p = .045$; Fig. 1 row 3 left side), with this group difference being maximal in the posterior regions (ADHD < Control × F < P: $F = 4.15$, $p = .047$; F/P > C: $F = 13.31$, $p = .001$).

3.2.3. Beta

Relative beta was frontally greater across groups (F > P: $F = 38.68$, $p < .001$). The midline had significantly less beta than the hemispheres (M < L/R: $F = 115.99$, $p < .001$). This midline reduction was greater frontally than posteriorly (F > P × M < L/R: $F = 13.42$, $p = .001$) with maximal reduction at the vertex (F/P < C × M < L/R: $F = 36.82$, $p < .001$). No group differences were found in relative beta (Fig. 1 row 4 left side).

3.3. Maturational changes in ADHD subjects

3.3.1. Delta

There was a significant global reduction in relative delta with increasing age in the ADHD subjects (Adult < Child: $F = 54.56$, $p < .001$; see Fig. 1 columns 2 and 4). The reduction was greatest in the frontal regions (Adult < Child × F > P: $F = 4.54$, $p = .043$; Adult < Child × F/P > C: $F = 4.85$, $p = .037$). In childhood the ADHD

group had greater relative delta in the right hemisphere but in adulthood, this changed to be greater in the left hemisphere (Adult < Child × R > L: $F = 4.47$, $p = .045$).

3.3.2. Theta

Relative theta in ADHD subjects significantly reduced across the scalp with age (Adult < Child: $F = 11.44$, $p = .002$; see Fig. 1 columns 2 and 4), with maximal change occurring in the posterior regions (Adult < Child × F < P: $F = 6.81$, $p = .015$). The midline enhancement of relative theta reduced with age (Adult < Child × M > L/R: $F = 5.04$, $p = .034$).

3.3.3. Alpha

Globally, relative alpha significantly increased with age in ADHD subjects (Adult > Child: $F = 12.97$, $p = .001$; see Fig. 1 columns 2 and 4) and this increase was smallest centrally (Adult > Child × F/P > C: $F = 10.35$, $p = .004$). Midline power changed from being less than in the hemispheres to being greater (Adult > Child × M > L/R: $F = 8.14$, $p = .009$).

3.3.4. Beta

Across the scalp, relative beta significantly increased with age in ADHD subjects (Adult > Child: $F = 39.20$, $p < .001$; see Fig. 1 columns 2 and 4). This change was greater posteriorly than frontally (Adult > Child × F < P: $F = 8.31$, $p = .008$) and maximal centrally (Adult > Child × F/P < C: $F = 6.13$, $p = .021$). In childhood the ADHD group had greater relative beta in the right hemisphere but in adulthood, power was greater in the left (Adult < Child × R > L: $F = 4.32$, $p = .048$). The midline reduction of relative beta became greater with age posteriorly (Adult > Child × F < P × M < L/R: $F = 15.33$, $p = .001$).

3.4. ADHD vs. controls in adulthood

3.4.1. Delta

Relative delta was maximal in the fronto-central regions across all groups (F > P: $F = 136.97$, $p < .001$; F/P < C: $F = 12.9$, $p = .001$). However, midline power was dominant in centro-posterior regions (F < P × M > L/R: $F = 69.53$, $p < .001$; F/P < C × M > L/R: $F = 14.8$, $p < .001$). In the ADHD group the frontal enhancement of relative delta was smaller than in the controls (ADHD < Control × F > P: $F = 4.23$, $p = .045$; see Fig. 1 row 1 right side). The ADHD group had relatively less power in the central hemispheres than the control group (ADHD < Control × F/P < C × M < L/R: $F = 4.94$, $p = .031$).

3.4.2. Theta

Relative theta was greatest in fronto-central regions across groups (F > P: $F = 130.73$, $p < .001$; F/P < C: $F = 31.28$, $p < .001$). At the midline, relative theta was enhanced compared to the hemispheres (M > L/R: $F = 70.85$, $p < .001$). ADHD subjects had a global elevation in relative theta compared to controls (Group Main Effect; $F = 6.70$, $p = .013$; see Fig. 1 row 2 right side).

3.4.3. Alpha

Across groups, relative alpha was greatest posteriorly (F < P: $F = 139.72$, $p < .001$; F/P > C: $F = 55.06$, $p < .001$). There was a right posterior enhancement in power (F < P × L < R: $F = 4.79$, $p = .034$), and in the hemispheres alpha power was maximal in central-parieto regions (F < P × M < L/R: $F = 13.35$, $p = .001$; F/P < C × M < L/R: $F = 7.34$, $p = .009$). No significant group differences were found (Fig. 1 row 3 right side).

3.4.4. Beta

Across groups, relative beta was elevated in central regions (F/P < C: $F = 12.95$, $p = .001$). Right frontal beta was elevated (F > P × L < R: $F = 5.06$, $p = .029$). The midline had significantly less beta than the hemispheres (M < L/R: $F = 79.78$, $p < .001$). This reduction was greater in the posterior regions than the frontal regions (F < P × M < L/R: $F = 6.20$, $p = .016$) and maximal at the vertex (F/P < C × M < L/R: $F = 18.10$, $p < .001$). ADHD subjects had greater central relative beta than controls (ADHD > Control × F/P < C: $F = 4.43$, $p = .040$; see Fig. 1 row 4 right side). In the hemispheres, the ADHD group had a right greater than left difference in power and this was reversed in the control group (ADHD > Control × L < R: $F = 7.68$, $p = .008$). The reduction in midline power was smaller in controls than in ADHD subjects (ADHD > Control × M < L/R: $F = 5.49$, $p < .023$).

4. Discussion

The initial stage of this analysis compared the ADHD group as children with a child control group, to determine whether the childhood EEG profile of the clinical subjects was typical of the profile found in past research, and by so doing, allowing conclusions about the development of the EEG in these children that are generalisable to the greater population. As children, the ADHD group had less posterior delta; globally enhanced relative theta; and a global reduction in relative alpha activity, which, in line with past research, was greatest posteriorly. This EEG profile is largely consistent with the existing EEG literature for children with this disorder (Clarke et al., 1998, 2001a, 2001b, 2001d, 2002a, 2002b, 2002c, 2002d, 2003a, 2003b, 2006, 2007, 2008; Hobbs et al., 2007; Loo et al., 2009; Nazari et al., 2011; Woltering et al., 2012; Dupuy et al., 2013).

As it was not possible to retest the childhood control group as adults, a second adult control group was recruited for this study. This meant that it was not possible to directly compare maturational changes over time in ADHD and control subjects. However, the child and adult ADHD groups were compared to determine the nature of maturational changes in this population. The mean time between assessments was 11.1 year, which is the longest time between assessments that has currently been investigated. Delta activity globally reduced with age and this change was greatest in the frontal regions. Theta activity also demonstrated a global decrease with maximal change in the posterior regions. In both the alpha and beta bands, there was a global increase in relative power with age. Topographically beta activity changed the most centrally, followed by the posterior regions, then the frontal regions. Except for the frontal reduction in delta activity, these changes are nearly identical to the changes in EEG activity reported in normal populations, both in the direction of change in the EEG power (Gasser et al., 1988b) and in the topography of the change (Gasser et al., 1988a). From these results, while it is clearly evident that the EEG of these subjects as children is abnormal, there is a substantial amount of normal maturation occurring in the development of their EEG.

It has been suggested that the abnormal EEG in ADHD represents some form of undefined developmental deviation (Chabot and Serfontein, 1996; Clarke et al., 2001d; Buyck and Wiersema, 2014). This label implies that the EEG is abnormal or has developed in an abnormal way. The current data actually indicate that the EEG is developing in a normal way albeit from an abnormal starting point. As such, these data suggest that it is no longer accurate to consider the EEG anomalies in this population as representing a developmental deviation in CNS functioning, and the use of this label should be discontinued.

From the comparison of the adult ADHD and control subjects, our results generally indicate a reduction in the degree of the EEG abnormality evident in these subjects as children, although significant differences remain. The frontal reduction in delta activity remained, but the posterior elevation was no longer significant

in the adult EEGs. The global enhancement in theta activity across the scalp remained, but the magnitude of this was reduced. The significant results found in the alpha band as children were no longer significant as adults. In the beta band, no significant differences were found in the childhood EEGs, but a central elevation in beta activity was found in the adults, and the normal left > right power asymmetry was reversed in the ADHD group. Once again these results are generally consistent with past research in adults with ADHD which used an eyes-closed resting condition (Clarke et al., 2008; Hermens et al., 2004; Woltering et al., 2012), and consistent with past cross sectional studies which have found greater EEG abnormalities in children with ADHD than adults (Bresnahan et al., 1999; Liechti et al., 2013).

One of our aims in this study was to evaluate changes in the EEG in terms of the maturational lag model of ADHD. This model views the abnormal levels of hyperactivity and inattention in terms of a delay in CNS development, such that if the behaviours of these children were compared to younger normal children, the behaviours would not seem so abnormal. Behaviourally, all subjects in the current study were diagnosed with ADHD Combined type as children. As adults, 19 subjects were reassessed as meeting criteria for the Combined type of ADHD and 6 with the Inattentive type. CAARS scores indicated that across subjects, mean scores on the Hyperactive/Impulsive subscale were just below the clinical threshold but mean scores on the Inattentive subscale were in the clinical range. Over this period of time there was also clear evidence of normalisation of the EEG, with the elevations in posterior delta and global theta activity reducing, and the global reduction in alpha failing to reach significance in adulthood. These results are supportive of the maturational lag model of ADHD and suggest that delayed maturation may be involved in at least some components of both the behavioural and EEG profiles of people with ADHD.

One of the influential models of ADHD has been the hypoarousal model (Barry et al., 2003). This was first proposed by Satterfield and Dawson (1971), who found that half their sample of children with hyperactivity had low skin conductance levels (SCL). This model was further developed by Lubar (1991), who hypothesised that the theta/beta ratio was associated with arousal. Subsequent research linking EEG activity and SCL found that it was in fact the alpha band and not the theta/beta ratio that is associated with arousal, with increased alpha levels indicating lower arousal in both ADHD and control subjects (Barry et al., 2009, 2012). The interesting paradox in ADHD is that while these children are hypoaroused (a factor that should increase alpha levels), they typically have reduced alpha levels compared to control subjects. This finding suggests that there are two factors impacting on the alpha band, the arousal factor increasing alpha levels, but another as yet unidentified factor that is reducing the recorded alpha levels in these children. In the current sample we found significant lower alpha levels in the child ADHD group, but as adults, the alpha levels were no longer significantly lower. This suggests maturation of one or both of the factors/mechanisms contributing to activity in the alpha band. Unfortunately SCL was not recorded in this study, so it is not possible to determine whether this is a change in the arousal mechanism or something else. Future research needs to evaluate arousal in adults with ADHD.

One area of ADHD, that has been increasingly researched over the past decade, is the use of EEG as a diagnostic tool. Those studies have primarily used power analysis in specific regions or the ratio of theta to beta activity as the primary diagnostic aid (Magee et al., 2005; Quintana et al., 2007; Snyder et al., 2008; Gonzalez et al., 2013). Overall, there is considerable variability in the results that have been obtained. From the current study, one fact that would have to be carefully accounted for is the degree of normal maturational change that is occurring in subjects with ADHD. If too wide an age range was used without precise matching of subjects or control for age effects, then simple maturational processes may have reduced the obtained sensitivity and specificity of EEG markers in past studies. Currently only one EEG-based test has FDA approval as a diagnostic aid in ADHD (USFDA, 2013), and future research in this area must give careful consideration to the age range of the subjects in their study design.

This study aimed to undertake an initial evaluation of maturation of the EEG of subjects with ADHD. As children, these subjects had more posterior and less frontal delta, globally greater relative theta, with globally lower relative alpha activity, with the group differences being maximal in posterior regions. With increasing age, significant normal maturation in the EEG was identified, which has implications for database development. As adults, the degree of EEG abnormality found in the clinical sample was less than in childhood. These results give support to the maturational lag model of ADHD, with at least some components of the EEG appearing to be associated with cortical immaturity. The normalisation of the alpha band also suggests that there could be changes in the arousal mechanisms in the subjects, but this needs further evaluation.

Declaration of Competing Interest

The Authors have no conflicts of interest to report.

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