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Resting electroencephalogram in attention deficit hyperactivity disorder: Developmental course and diagnostic value

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ABSTRACT

This study investigated electroencephalographic (EEG) activity and its developmental course in attention deficit hyperactivity disorder (ADHD) throughout the lifespan, as well as the accuracy of EEG parameters in distinguishing ADHD patients from typically developing individuals. Three minutes eyes closed resting EEG was compared between 62 individuals with ADHD (36 children, 26 adults) and 55 typically developing individuals (30 children, 25 adults). EEG activity and maturation did not differ between individuals with ADHD and typically developing individuals. However, despite comparable developmental course between clinical groups, persistent elevated theta/beta ratio and reduced relative beta power were observed in the ADHD inattentive subtype compared to the ADHD combined subtype and controls across the lifespan. Therefore, a maturational deviation rather than a maturational delay may underlie a subgroup of ADHD. EEG based classification failed for ADHD but proved successful for age. These findings emphasize heterogeneity in ADHD throughout the lifespan and question clinical utility of conventional EEG approaches for diagnostic purposes in ADHD.

1. Introduction

Attention deficit hyperactivity disorder (ADHD) is a common neurobehavioral disorder with a childhood onset that often persists into adulthood (for a review, see Spencer et al., 2007) and is characterized by developmentally inappropriate symptoms of inattention and/or hyperactivity/ impulsivity, resulting in impairment in multiple life domains (APA, 2000). The worldwide prevalence of ADHD is estimated between 5% and 7% (Willcutt, 2012).

Much effort has been devoted to laying out clinical recommendations for diagnosing ADHD, which relies on the assessment of behavioral symptoms and functional impairment (Seixas et al., 2012). Still, more objective diagnostic procedures would provide a valuable supplement. To this end, brain-based approaches seem promising, since neuroimaging studies suggest that disturbances in brain structure and function are related to overt behavioral manifestations of ADHD (Williams et al., 2010).

In the last decades, resting state electroencephalographic (EEG) measures have been widely used to document underlying neurophysiological dysfunction in ADHD.

Although most EEG studies focus on children, there is a growing interest in adults with ADHD. The most robust findings in individuals with ADHD across the lifespan in eyes closed as well as eyes open resting state conditions are increased theta activity and/or an elevated proportion of slower to faster frequencies in the brain, as reflected in theta/beta ratio (TBR), particularly apparent at frontocentral sites (for a review, see Barry et al., 2003; e.g., Clarke et al., 2011b; Shi et al., 2012). This has also been confirmed by meta-analyses, reporting effect sizes between 0.58 and 1.31 for theta power and between 0.62 and 3.08 for TBR (Boutros et al., 2005; Snyder and Hall, 2006; Arns et al., 2013). However, a reducing discrepancy in TBR between youngsters with and without ADHD was found across publication years of studies (Arns et al., 2013). This corresponds with recent studies that could not invariably replicate theta or TBR discrepancies between ADHD and control groups, neither in children, nor in adults (Loo et al., 2009; van Dongen-Boomsma et al., 2010; Ogrim et al., 2012). Further, in a recent study, theta activity was observed to be enhanced in 60% and reduced in 40% of children with ADHD, highlighting the heterogeneity of spectral EEG in ADHD (Clarke et al., 2011a). Findings regarding beta activity appear even more mixed, with some studies reporting reduced beta power (Lazzaro et al., 1998; Loo et al., 2009) and others documenting no deviances (Bresnahan et al., 1999; Clarke et al., 2001b) or even a beta excess (Chabot and Serfontein, 1996; Clarke et al., 2001a) in ADHD.

Regarding the differentiation in EEG activity between ADHD predominantly inattentive type (ADHD-I) and ADHD combined type (ADHD-C), disparate findings have been reported. While in some studies individuals with ADHD-I have been documented to

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demonstrate an in-between pattern of abnormalities between ADHD-C and control subjects (Clarke et al., 1998, 2001b; Dupuy et al., 2011), in others no differences between subtypes (Monastra et al., 2001; Hermens et al., 2004; Loo et al., 2010) or greater deviances in ADHD-I compared to ADHD-C (Loo et al., 2010) have been described.

Although theta and TBR are frequently reported to be aberrant in ADHD, their functional significance is not fully understood (Loo and Makeig, 2012). Traditionally, deviating resting state EEG in ADHD (e.g., theta and TBR) has been hypothesized as reflecting hypoarousal in the central nervous system (e.g., Mann et al., 1992; Monastra et al., 1999). However, this account has been challenged since recent studies failed to find an association between a well established measure of arousal (i.e., skin conductance level) and theta or TBR (Barry et al., 2009; Clarke et al., 2013). Other researchers have suggested that the deviances may indicate a maturational delay (e.g., Lazzaro et al., 1998) or developmental deviation (e.g., Hermens et al., 2004) in ADHD as with age EEG power is found to decrease for slow frequencies (e.g., theta) and to increase for faster frequency ranges (e.g., beta) (see Barriga-Paulino et al., 2011).

Still, research into the developmental course of EEG in ADHD is scarce. Some evidence has been found for a slower maturation of absolute beta (Satterfield et al., 1984) and theta (Satterfield et al., 1984; Clarke et al., 2001b) activity in children with ADHD compared to typically developing children. Comparing children with adolescents, a tendency of a smaller decrease in absolute theta power in ADHD relative to a control group has also been reported while no developmental differences were detected when those age groups as a whole were compared with adults (Liechti et al., 2013). Furthermore, along with maturation of EEG in the expected direction, a study documented absolute as well as relative theta activity and TBR to be continuously elevated in ADHD throughout the lifespan, while the amount of reduced relative beta activity in ADHD compared to typically developing individuals decreased with age (Bresnahan et al., 1999). Additionally, a recent longitudinal study revealed that different abnormalities in EEG in childhood preceded persistence and remission of ADHD in adulthood (Clarke et al., 2011b).

Corresponding to the frequently found abnormalities in TBR and theta power across a number of resting EEG studies, the potential of these EEG parameters as diagnostic markers has been proposed, yet remains equivocal. Across studies, sensitivity numbers of TBR in discriminating individuals with and without ADHD vary between 43.8% and 90%, while specificity percentages between 36.7% and 94% are reported (Monastra et al., 2001; Snyder et al., 2008; Liechti et al., 2013). The most recent studies documented an insufficient overall accuracy between 40.3% and 58% for TBR and between 46.8% and 63% for theta power in distinguishing children with ADHD from a control group (Ogrim et al., 2012; Liechti et al., 2013). Besides, increased theta appears to be a nonspecific phenomenon common to other disorders such as epilepsy, bipolar disorder, substance abuse, dementia, alcoholism and schizophrenia (Coutin-Churchman et al., 2003).

In the current study, eyes closed resting EEG was analyzed in children and adults with and without ADHD to gain further insights into the developmental course and diagnostic potential of resting EEG in ADHD (subtypes) throughout the lifespan. As most of the normative databases used in clinical practice to compare EEG activity are based on eyes closed resting EEG data, it is important to investigate eyes closed resting conditions in clinical populations (Clarke et al., 2008). If theta power and TBR are significant markers for ADHD across the lifespan, these EEG parameters were expected to be elevated in children as well as in adults with ADHD and consequently successful in distinguishing individuals with ADHD from typically developing individuals. Previous research addressing differences between inattentive and combined subtype or divergence in maturational changes between the ADHD and control group is scarce and revealed inconsistent results, which makes it difficult to formulate specific hypotheses.

2. Methods

2.1. Participants

Sixty-six children (30 without ADHD, 36 with ADHD) aged 7–14 years and 51 adults (25 without ADHD, 26 with ADHD) aged 18–55 years participated in the study (see Table 1). Participants were recruited through staff members, schools, advertisements, neurologists and self support groups for ADHD. Individuals with ADHD were previously diagnosed in a clinical setting according to DSM-IV criteria. Diagnosis was ascertained by a semi-structured clinical interview (behavioral module of Diagnostic Interview Schedule for Children IV for children, Schaffer et al., 2000; Diagnostisch Interview Voor ADHD bij Volwassenen 2.0 for adults, Kooij and Francken, 2010). The Disruptive Behavior Disorder Rating Scale (DBD, Pelham et al., 1992) was administered to furthermore evaluate ADHD symptoms in children. Presence of childhood ADHD in adults was retrospectively assessed with the Wender Utah Rating Scale (Ward et al., 1993), whereas ADHD manifestation through the lifespan was evaluated with the ADHD Rating Scale-IV (ADHD-RS, DuPaul et al., 1998). Following published diagnostic guidelines (Kooij et al., 2010), adults were required to exhibit at least four symptoms in the inattentive and/or the hyperactive/impulsive domain to meet criteria for ADHD during adulthood. Individuals with ADHD using stimulants were asked to refrain from medication 48 h before participation in the experiment. Exclusion criteria for all participants were history of brain related illness, neurological disorder, suspicion of autism spectrum disorder and estimated IQ below 80. Intelligence functioning was evaluated by an abbreviated Wechsler Intelligence Scale for Children-III (Wechsler, 1991; Grégoire, 2000) or Wechsler Adult Intelligence Scale-III (Wechsler, 1997; Ryan and Ward, 1999). Individuals reaching clinical scores on ADHD rating scales and on the Child Behavior Checklist (for children) or Adult Self Report (for adults) (Achenbach and Rescorla, 2001, 2003) were excluded from the control groups.

2.2. Procedure

The research protocol was approved by the local ethics committee and all participants or their parents signed an informed consent. The protocol involved recordings of resting EEG and execution of neuropsychological tasks. The present study focused on 3 min eyes closed resting EEG.

EEG data were obtained with an electrode cap employing 128 active Ag/AgCl electrodes (EasyCap Active, EasyCap GmbH) placed according to the 10/5 International System (Oostenveld and Praamstra, 2001). Signals were amplified with an open pass-band from DC to 100 Hz with a QuickAmp amplifier (Brain Products, Gilching, Germany) and digitized using Brain Vision Recorder software (version 1.10) with a sample rate of 500 Hz and an average reference derivation. The ground electrode was mounted within the cap at Fpz. Electro-oculogram was recorded with electrodes enclosed in the cap near the eyes and for adults, an additional electrode was placed below the right eye.

Analyses of EEG data were performed using Brain Vision Analyzer software (version 2.0.1). EEG data were filtered with a high pass filter of 0.5 Hz, a low pass filter of 50 Hz and a notch filter of 50 Hz. Data were segmented in 2 s epochs with 1 s overlap. Eye movement correction was conducted according to the Gratton and

Table 1
Sample characteristics.

	Control ($n=55$)	ADHD ($n=62$)
Children	$n=30$	$n=36$
Age in years	*M* 10.46 (SD 1.75)	*M* 10.11 (SD 1.82)
Estimated FSIQ (WISC)	*M* 108.10 (SD 11.55)	*M* 104.89 (SD 12.95)
Male/female	15/15	26/10
ADHD C/I	–	14/22
Stimulants (Yes/No)	–	20/16
Adults	$n=25$	$n=26$
Age in years	*M* 35.32 (SD 11.12)	*M* 33.76 (SD 10.17)
Estimated FSIQ (WAIS)	*M* 111.96 (SD 10.87)	*M* 109.96 (SD 12.16)
Male/female	14/11	12/14
ADHD C/I	–	15/11
Stimulants (Yes/No)	–	12/14

Note: FSIQ: Full Scale Intelligence Quotient, WISC: Wechsler Intelligence Scale for Children, *M*: mean, SD: standard deviation, ADHD: attention deficit hyperactivity disorder, C: combined subtype, I: inattentive subtype, WAIS: Wechsler Adult Intelligence Scale.

Coles algorithm (Gratton et al., 1983). Segments were rejected from further analyses if amplitudes exceeded ±100 μV for children and if absolute difference in amplitudes exceeded 120 μV for adults. Subsequently, spectral power was calculated using Fast Fourier Transform and the transformed data were averaged. For replication purposes (e.g., Ogrim et al., 2012; Liechti et al., 2013), absolute and relative power estimates were derived for theta (3.5–7.5 Hz) and beta (12.5–25 Hz) frequencies at frontal (Fz), central (Cz) and parietal (Pz) midline. Relative power was computed by dividing the average power in a frequency band by the total average power in the 1.5–25 Hz spectrum, multiplied by 100 (see also Dupuy et al., 2011). TBR was calculated by dividing power in the theta band by power in the beta band. A natural logarithmic transform was used to approach normal spreading of the data.

2.3. Statistical analyses

EEG activity across the lifespan in the ADHD and control group was compared by separate analyses of variance with repeated measures for absolute as well as relative theta and beta power and for TBR. Midline electrode position (Fz, Cz, Pz) was the within-subject factor and Clinical group (ADHD, non-ADHD) and Age group (child, adult) were between-subject factors. Effect sizes were determined by partial eta squared (η_p^2) and, according to guidelines (Cohen, 1988), are interpreted as small (<0.06), medium (0.06–0.14), or large (>0.14).

Receiver operation characteristic (ROC) curves were computed for determining the accuracy of logistic regression models of absolute as well as relative theta and beta power and TBR at the vertex in predicting clinical and age group membership. Cz was examined to warrant comparability with other publications, since most studies documenting on diagnostic validity of EEG parameters in ADHD have investigated EEG activity at the vertex (Monastra et al., 1999, 2001; Snyder et al., 2008; Ogrim et al., 2012; Liechti et al., 2013). First, binary logistic regression was performed to predict probabilities for each EEG parameter separately. Then, ROC curves were computed to determine the efficacy of the predicted probabilities in the respective binary outcomes of interest (i.e., age group and clinical diagnosis group). Accuracy is indicated by the area under the ROC curve, in which the reference line represents the null hypothesis (Ogrim et al., 2012).

All analyses were performed using the Statistical Package for the Social Sciences (SPSS) volume 20.

3. Results

3.1. EEG

Concerning clinical group effects, none of the EEG measures reached significance (see Table 2). Incorporation of sex as a covariate did not change the results significantly, neither did repeating analyses including only individuals expressing at least six symptoms in one or both domains of symptomatology in the adult ADHD group (i.e., 22 adults). Also, effect sizes were fairly similar in these additional analyses.

Regarding age group effects, children demonstrated higher absolute theta ($F_{(1,111)}=281.51$, $P=0.000$) and beta ($F_{(1,111)}=19.85$, $P=0.000$) power than adults. Regarding relative power, higher theta ($F_{(1,111)}=97.73$, $P=0.000$) as well as lower beta ($F_{(1,111)}=62.67$, $P=0.000$) activity were found in children compared to adults. Furthermore, a higher TBR ($F_{(1,111)}=169.44$, $P=0.000$) was observed in childhood relative to adulthood. Considering the scope of the present study, divergence between children and adults associated with electrode positions will not be elaborated on.

Since our results did not indicate differences between individuals with and without ADHD and literature points out that EEG deviances may differ across ADHD subtypes (e.g., Clarke et al., 1998), additional analyses of variance with repeated measures were performed where the within subject factor Clinical group was subdivided in non-ADHD (i.e., 55 individuals), ADHD-C (i.e., 29 individuals) and ADHD-I (i.e., 33 individuals).

Results regarding absolute theta and beta power as well as relative theta power did not change significantly, however main group effects appeared for relative beta power ($F_{(2,111)}=3.81$; $P=0.025$) and TBR ($F_{(2,111)}=6.06$; $P=0.003$). Bonferroni post hoc analyses indicated reduced relative beta power and enhanced TBR in individuals with ADHD-I compared to the other groups (see Fig. 1 and Table 3).

3.2. Classification analysis

ROC analyses indicated that EEG parameters were unsuccessful in distinguishing individuals with ADHD from typically developing individuals (accuracy rates varied between 49.2% and 54.8%). Additional analyses restricted to discrimination of individuals with ADHD-I did not substantially increase the accuracy (e.g., highest

Table 2
Repeated measures analyses of variance per EEG parameter: controls versus ADHD.

Clinical group				Age group		Electrode
	x age group	*x* electrode	*x* age group *x* electrode		*x* electrode	
(1,226) *F* (η_p^2)	(1,113) *F* (η_p^2)	(2,226) *F* (η_p^2)	(2,226) *F* (η_p^2)	(1,113) *F* (η_p^2)	(2,226) *F* (η_p^2)	(2,226) *F* (η_p^2)
aθ						
0.24 (0.002)	0.80 (0.007)	0.06 (0.001)	0.48 (0.004)	280.35*** (0.713)	0.44 (0.004)	0.03 (0.000)
rθ						
0.01 (0.000)	0.02 (0.000)	0.32 (0.003)	0.22 (0.002)	101.91*** (0.474)	0.33 (0.003)	96.56*** (0.461)
aβ						
0.33 (0.003)	0.05 (0.000)	0.09 (0.001)	0.92 (0.008)	19.22*** (0.145)	1.52 (0.013)	4.21* (0.036)
rβ						
1.71 (0.015)	0.57 (0.005)	0.13 (0.001)	1.25 (0.011)	65.16*** (0.366)	11.86*** (0.095)	19.75*** (0.149)
TBR						
1.38 (0.012)	0.51 (0.004)	0.22 (0.002)	0.37 (0.003)	173.10*** (0.605)	6.31** (0.053)	10.90*** (0.088)

Note: *aθ*: absolute theta power, *rθ*: relative theta power, *aβ*: absolute beta power, *rβ*: relative beta power, TBR: theta/beta ratio.

* $P<0.05$.
** $P<0.01$.
*** $P<0.001$.

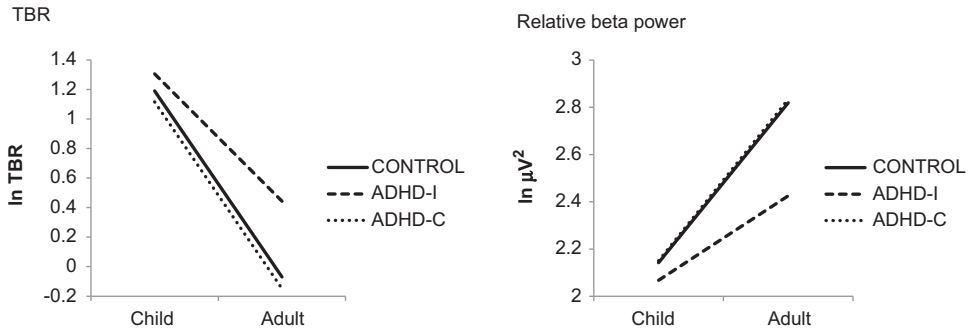

Fig. 1. Mean TBR and relative beta power at Cz for children and adults without ADHD, with ADHD-I and with ADHD-C.

Table 3
Repeated measures analyses of variance per EEG parameter with clinical groups non-ADHD versus ADHD-I versus ADHD-C.

Clinical group				Age group		Electrode
	x age group	*x* electrode	*x* age group *x* electrode		*x* electrode	
(1,222)	(1,111)	(2,222)	(2,222)	(1,111)	(2,222)	(2,222)
F	*F*	*F*	*F*	*F*	*F*	*F*
(η_p^2)	(η_p^2)	(η_p^2)	(η_p^2)	(η_p^2)	(η_p^2)	(η_p^2)
rβ						
3.81*	1.74	0.11	0.96	52.56***	8.76***	17.82***
(0.064)	(0.030)	(0.002)	(0.017)	(0.321)	(0.073)	(0.138)
TBR						
6.063**	1.98	0.39	0.43	151.42***	4.90**	9.68***
(0.098)	(0.034)	(0.007)	(0.008)	(0.577)	(0.042)	(0.080)

Note: *rβ*: relative beta power, TBR: theta/beta ratio.
* $P < 0.05$.
** $P < 0.01$.
*** $P < 0.001$.

accuracy rate was 64%). However, children and adults could be successfully discriminated based on absolute as well as relative theta power, relative beta power and TBR (accuracy rates varied between 89.8% and 96.5%). Absolute beta power was less effective in discriminating children from adults (see Fig. 2 and Table 4).

4. Discussion

In the current study, no EEG differences were found between the total ADHD group (i.e., comprising both ADHD-I and ADHD-C) and the healthy control group. Also, no divergence in the developmental course of EEG activity was found between those groups. However, further analyses detected an elevated TBR and reduced relative beta power across the lifespan in ADHD-I compared to ADHD-C and controls.

Although our findings of comparable EEG profiles and developmental pathways of individuals with ADHD-C and typically developing controls disaccord with previous reports on theta and TBR deviances in ADHD (for a review, see Barry et al., 2003), they correspond with a few recent studies that also failed to replicate theta and TBR abnormalities in ADHD relative to typically developing individuals (e.g., van Dongen-Boomsma et al., 2010; Liechti et al., 2013). Furthermore, our results are in line with findings of a smaller difference in TBR between ADHD and normal controls in more recent publications compared to earlier studies (Arns et al., 2013). It is plausible that differences in inclusion and exclusion criteria for control subjects as well as ADHD patients have contributed to different findings across studies (Arns et al., 2013; Liechti et al., 2013). As for the current study, it seems unlikely that the predominance of boys in the child ADHD group contributed to the failure of finding EEG abnormalities in children with ADHD, since supplementary analyses controlling for sex did not change the results significantly. Besides, given that some studies documented more deviances in ADHD boys compared to girls (e.g., Clarke et al., 2001b), this would rather have resulted in detecting instead of overlooking abnormalities in our sample. Lowering the cut-off of symptomatology for incorporating adults with ADHD in our study did also not impede finding EEG differences between individuals with ADHD and typically developing individuals since additional analyses using a more restrictive symptom threshold for including adults did not affect the significance of findings. Moreover, this cannot explain the absence of differences between children with and without ADHD. The current ADHD sample comprised both individuals diagnosed with the ADHD combined as well as the inattentive subtype. This differs from studies that incorporated only individuals with ADHD combined subtype (e.g., van Dongen-Boomsma et al., 2010; Liechti et al., 2013) but accords with others that included individuals with different ADHD subtypes (e.g., Ogrim et al., 2012; Loo et al., 2010). Only a few studies directly compared the combined and inattentive subtype and presented mixed results, making comparability to existing literature difficult. Furthermore, exclusion and inclusion criteria regarding comorbid disorders in individuals with ADHD are different across studies and the influence of comorbidity on resting EEG findings in ADHD has not been investigated systematically. Also in the current study, comorbid problems were present in individuals with ADHD as indicated

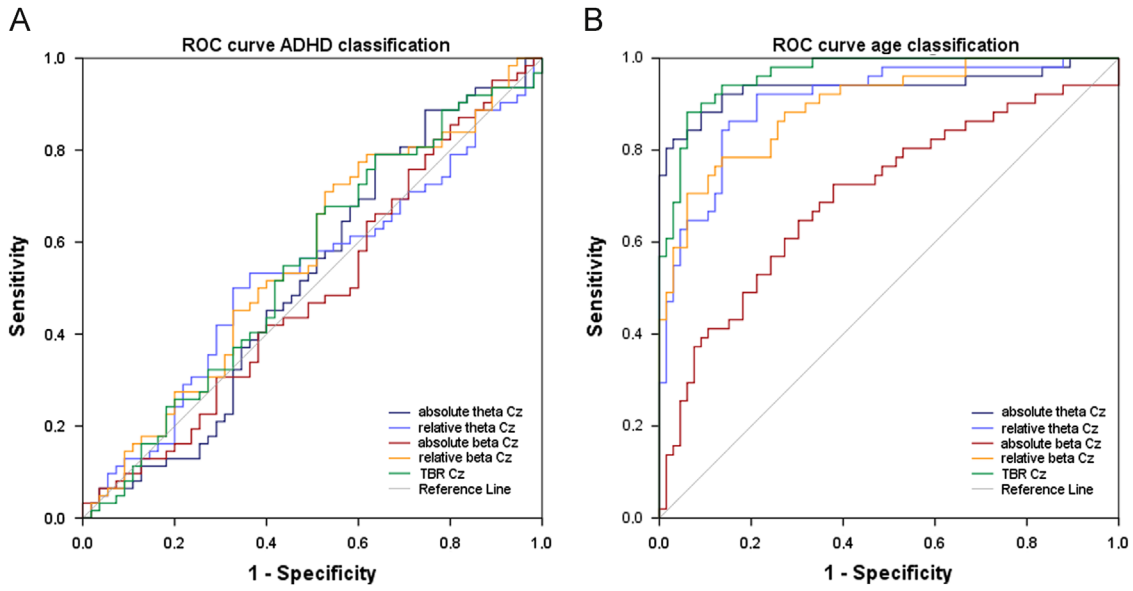

Fig. 2. ROC curves expressing accuracy of EEG parameters in ADHD (A) and age classification (B).

Table 4
ADHD and age classification based on EEG parameters.

EEG parameter	AUC	*P*
ADHD (C+I) classification		
aθ	0.520	0.710
rθ	0.526	0.623
aβ	0.492	0.878
rβ	0.562	0.251
TBR	0.548	0.370
ADHD-I classification		
aθ	0.566	0.303
rθ	0.594	0.142
aβ	0.529	0.645
rβ	0.641	0.027
TBR	0.633	0.037
Age classification		
aθ	0.938	0.000
rθ	0.907	0.000
aβ	0.701	0.000
rβ	0.898	0.000
TBR	0.965	0.000

Note: AUC: area under the curve.

by elevated symptoms of anxiety/depression, learning disorders and disruptive behavior measured through interviews and questionnaires. Future studies with larger sample sizes are needed to evaluate the impact of comorbidity on resting EEG findings in ADHD. These factors, as well as the relatively high intelligence profile of our ADHD sample compared to other studies, may confound the comparability of our study to others and therefore impede generalizability of the results.

Few EEG studies have addressed the comparison of ADHD-I and ADHD-C, and yielded disparate findings. In some studies, both ADHD subtypes similarly deviated from a control group, while in others, more pronounced quantitative EEG differences in one subtype relative to the other and/or the control group were documented (Clarke et al., 1998, 2001b; Loo et al., 2010; Hermens et al., 2004; Dupuy et al., 2011). In this study, the observed larger EEG deviances in ADHD-I were not accounted for by a greater severity of inattention problems in the ADHD-I group compared to the ADHD-C group, as inattention scores as derived from the questionnaires did not differ between ADHD-I and ADHD-C. Also, ADHD subtypes did not differ on severity of comorbid problems as screened by the subscales of Achenbach questionnaires. Our findings may suggest that distinct types of inattention characterize differences between ADHD-I and ADHD-C, involving different neural circuits, rather than variations in symptom severity (Diamond, 2005; Milich et al., 2001). However, this statement cannot unequivocally be confirmed by our data. Moreover, this interpretation discords with a recent meta-analysis of 546 studies, that found minimal support for defining ADHD-I and ADHD-C as distinct disorders (Willcutt et al., 2012). In view of the literature that defined EEG based subgroups in ADHD through cluster analyses (Clarke et al., 2011a), our findings may be better explained as emphasizing heterogeneity of EEG in ADHD, confirming deviances in a subgroup of patients rather than in the whole ADHD group.

Since EEG deviances were found to persist throughout the lifespan, it seems questionable that a maturational delay may account for EEG discrepancies in a subgroup of individuals with ADHD. A developmental deviance may be a better interpretation of these results. However, findings should be seen as tentative since direct individual measurements of developmental changes are not provided in this cross sectional study. Ideally, longitudinal designs are applied in gaining more insights in the nature of the developmental course of EEG deviances in ADHD(-I).

Given the lack of consistent EEG abnormalities in the ADHD group, the accuracy rates reflecting failure in discriminating this group from the control group based on these parameters were not surprising. Our overall accuracy rates below 57% align with recent documents, that also demonstrated inadequacy of EEG based classification (Ogrim et al., 2012; Liechti et al., 2013) but contrast with earlier findings of accuracy rates up to 90% (Monastra et al., 2001; Snyder et al., 2008). Alternatively, accuracy in distinguishing children from adults attained 96.5% despite using parameters selected for deviances in ADHD. Since our findings regarding maturation of EEG activity are consistent with literature (e.g., Barriga-Paulino et al., 2011), we believe our data would have allowed reliable detection of a maturational lag, as has been suggested for ADHD.

In sum, our EEG findings confirm the view that ADHD is a heterogeneous disorder, which is also established in multiple pathway models of ADHD (e.g., Sonuga-Barke et al., 2010). Our results suggest that it is unlikely that ADHD is characterized by generalized EEG abnormalities and that the use of conventional

EEG practices as a diagnostic adds-on in ADHD has to be regarded as insufficient. However, since reduced relative beta power and elevated TBR were found in a subgroup of patients, it seems plausible that EEG measures may be of interest in guiding prognostic practices. That is, they may possibly be applied to distinguish subgroups that respond differently to various treatments. This has already been illustrated in research investigating the relationship between EEG parameters and treatment response to stimulant medication (Arns et al., 2008) and neurofeedback treatment (Arns et al., 2012).

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