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Cortical activity patterns in ADHD during arousal, activation and sustained attention

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Abstract

Objective—The goal of the present study is to test whether there are Attention-Deficit Hyperactivity Disorder (ADHD)-related differences in brain electrical activity patterns across arousal, activation and vigilance states.

Method—The sample consists of 80 adults (38 with ADHD and 42 non-ADHD controls) who were recruited for a family study on the genetics of ADHD. Patterns of cortical activity were measured using electroencephalography (EEG) during baseline and sustained attention conditions and compared according to ADHD diagnostic status. Cortical activity was examined separately for the first, middle, and last 5-minutes of the sustained attention task to assess whether patterns differed over time and according the ADHD status.

Results—In frontal and parietal regions, patterns of activation in the alpha (8-10 Hertz) range differed according to ADHD status, indicating increased cortical arousal among ADHD subjects. Beta power (13 -14 and 17-18-hertz) also differed between ADHD and controls, indicating increased cortical activation is associated with ADHD. Behavioral performance on the sustained attention task did not differ significantly by diagnosis. Significant differences in EEG correlates of cognitive performance emerged by ADHD diagnosis and were primarily in frontal regions. Brain activation patterns recorded during the sustained attention task suggest that the ADHD group exhibited significantly increased cortical activation at the end of the task when compared to controls.

Conclusions—Adults with ADHD may have different neural organization primarily in frontal regions which results in the need for continually high levels of cortical activation to maintain sustained attention.

Introduction

Attention-Deficit Hyperactivity Disorder (ADHD) (APA, 1994) is a common childhood psychiatric disorder that affects an estimated 5-10% of the population (Spencer, Biederman, & Mick, 2007). Numerous longitudinal studies have documented the persistence of the disorder into adolescence and adulthood for an estimated 74% (range 69-79%) of children with ADHD (Spencer et al., 2007), although there is notable symptom decline into adulthood. Poor long-

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term outcome for individuals with ADHD is typically seen in higher rates of incarceration, divorce, substance abuse, and school failure (Barkley, Smith, Fischer, & Navia, 2006).

Theories regarding the etiology of ADHD have long incorporated the concepts of arousal, activation, and alertness as basic mechanisms in ADHD. Over the past 25 years, several investigators have suggested that ADHD is associated with a hypoaroused brain state, which may be responsible for gross motor hyperactivity as well as slow and variable response patterns observed on sustained attention tasks. One of the earliest to put forth testable hypotheses was the Optimal Stimulation Theory (Zentall & Zentall, 1983), which postulates that ADHD symptoms such as restlessness and hyperactivity represent a functional set of responses to a chronic state of underarousal. More recent theories have generally included a top-down executive control network located in the frontal cortico-striatal pathway that regulates the level of arousal or alertness. For example, Sergeant (Sergeant, 2000; Sergeant, 2005) suggested that there are three cognitive energetic pools associated with information processing: effort, arousal and activation and that ADHD may be related to insufficient regulation of brain states rather than failures in executive functions such as inhibition. Similarly, Halperin & Schulz's (Halperin & Schulz, 2006) model of ADHD implicates early damage to several subcortical regions, including the locus coeruleus and reticular formation, that mediate arousal and alerting as possible etiologic factors in ADHD. Finally, Sikstrom & Soderlund (Sikstrom & Soderlund, 2007) postulate that low tonic levels of dopamine result in cortical hypoarousal, which in turn makes phasic dopamine hyper-responsive to environmental cues.

While the theoretical separation of arousal and activation has been discussed, these terms are often used interchangeably and very little empiric study has been conducted on whether these brain states are discernable using neurophysiologic measures. Given the longevity and popularity of using these brain states as causative mechanisms and the shift to etiologic models of ADHD that incorporate resting brain states (i.e., Default-mode brain network), more clarification of these terms is needed, particularly with regard to physiologic processes and their relationship to cognitive functions in ADHD.

A series of studies have recently laid the foundation for examining the physiologic and neural correlates of arousal and activation in both normal children and adults. During eyes-closed resting condition, arousal level (as measured by electrodermal skin conductance level [SCL]) was negatively correlated with brain electrical activity in the 8-12 Hz range or alpha frequency band, which appears to be a robust indicator of arousal across development (Barry, Clarke, Johnstone, Magee, & Rushby, 2007; Barry et al., 2004). Opening the eyes was associated with a higher SCL and a concomitant reduction in mean activity across all frequency bands (delta, theta, alpha, beta), with the change in SCL being significantly correlated to global decrease in alpha power. Based on topography shifts in the EEG, the authors suggest that focal decreases in delta and theta activity as well as frontal increase in beta activity are EEG markers of activation (Barry et al., 2007). Based on these results, arousal was defined as the "current energetic level of the organism" and activation as a "separable tonic measure reflecting the task-related mobilization of energy needed to perform a task" (Vaez Mousavi, Barry, Rushby, & Clarke, 2007).

With this new data on cortical activity associated with arousal and activation, the stage is set for further examination of whether people with ADHD have different brain activity patterns in frontal and parietal regions across arousal, activation and vigilance states. The use of several different recording conditions allows examination of diagnostic group differences by condition (e.g., eyes closed [EC], eyes open [EO], continuous performance test [CPT]) as well as across conditions (going from EC to EO=arousal, EO to CPT=activation). Power in the alpha frequency range (8-12 Hz) is of particular interest given its strong relationship with arousal. Previous studies on EEG patterns in ADHD have identified theta and beta activity as significant

markers (Chabot & Serfontein, 1996; Clarke, Barry, McCarthy, Selikowitz, & Brown, 2002) and they also seem to be important indicators of activation. Therefore, we hypothesize that ADHD individuals will have lower resting state arousal (i.e., higher alpha activity) but require greater activation (i.e., decreased theta activity and increased beta activity) in order to engage in the sustained attention task. In order to assess the functional significance of the changes in cortical activity, we will test the correlations between EEG measures and behavioral performance on a sustained attention task.

A second hypothesis is that the pattern of cortical activation in ADHD subjects will be different from controls as attention is sustained over a long (~15 minutes) interval of time. Therefore, we will compare EEG-measured brain activity recorded during the sustained attention task for the first, middle and last 5 minutes of the CPT for those with and without ADHD.

Method

Participants

The sample consists of 80 adults (38 with ADHD and 42 non-ADHD controls) who were recruited as part of a family study on the genetics of ADHD Genetics. In order to participate in the study, families were required to have at least one child with ADHD. After receiving verbal and written explanations of study requirements, all participants provided written informed consent/assent approved by the UCLA Institutional Review Board. Demographics are presented in table 1.

Procedures

All members of the families underwent extensive assessment including diagnostic interviews, cognitive testing and EEG recording. Only data pertaining to EEG recording and cognitive tasks presented during EEG recording will be reported at this time. Participants were interviewed directly using the Schedule for Affective Disorders and Schizophrenia (SADS-LAR)(Fyer, Endicott, Mannuzza, & Klein, 1995) supplemented with the Behavioral Disorders supplement from the Schedule for Affective Disorders and Schizophrenia for School-Age Children (KSADS-PL)(Kaufman et al., 1997) to screen for the presence of ADHD and other psychiatric disorders. All interviews were conducted by clinical psychologists or highly trained interviewers with extensive experience in psychiatric diagnoses. 'Best estimate' diagnoses were determined after individual review of diagnoses, symptoms, and impairment level by senior clinicians (JJM, JTM). Inter-rater reliabilities were computed with a mean weighted kappa of 0.84 across all diagnoses with a greater than 5% occurrence in the sample. Participants who received a lifetime (defined as past and/or current diagnosis of ADHD) were placed in the ADHD group and those who did not qualify for a lifetime diagnosis of ADHD were placed in the Control group, but were allowed to have other psychiatric diagnoses (McGough et al., 2005). Subjects were excluded from participation if they were positive for any of the following: neurological disorder, head injury resulting in concussion, lifetime diagnoses of schizophrenia or autism, or estimated Full Scale IQ < 70. Subjects on stimulant medication were asked to discontinue use for 24 hours prior to their visit.

Cognitive measures

An estimate of intelligence functioning was derived from a two-subtest estimate (Vocabulary and Block Design) from the Wechsler Adult Intelligence Scale-3 (Wechsler, 1997). During the EEG recording, the Conners' Continuous Performance Test (CPT)(Conners, 2000) was administered as a measure of sustained attention. This widely used measure is a 14-minute computerized task during which subjects are asked to press the space bar when any letter except the target letter "X" appears.

Electrophysiologic methods

EEG recording was carried out using 40 Ag/AgCl surface electrodes that were embedded in an electrode cap in an extended International 10/20 location system (ElectroCap, Eaton, Ohio) and was referenced to linked ears. Impedance was below 10 kOhms and EEG signal was recorded using MANSCAN (Sam Technology, San Francisco, CA) hardware and software. EEG data were sampled at a rate of 256 samples per second. Eye movements were monitored by electrodes placed on the outer canthus of each eye for horizontal movements and by electrodes above the eye for vertical eye movements. EEG recording for all subjects consisted of 2 baseline conditions (eyes open [EO] and eyes closed [EC]) and a cognitive activation condition, the CPT. Continuous EEG data was reviewed off-line by a technician experienced in EEG and all segments containing eye, head movement or muscle artifact were removed from further analysis. Using a Fast Fourier Transform (FFT), EEG power (μV^2) was estimated for each 1-second epoch, averaged for each condition and exported in 1-Hz bins from 1-20 Hz. All data were log transformed (ln) and used in subsequent analyses. EEG technicians were blind to ADHD diagnostic status.

Data analytic plan

Statistical analyses were run in the Statistical Package for the Social Sciences 15.0 (SPSS) and SAS 9.1. To correct for multiple comparisons, we employed a False Discovery Rate (FDR) (Benjamini & Hochberg, 1995) to maintain the family-wise experimental error at $p .05$. Proc Mult-test was used to generate FDR-adjusted p -values, which are presented in addition to raw p -values; the threshold for statistical significance was set at $p \leq .05$. Our primary hypotheses involve the power in the theta (4-7 Hz), alpha (8-12 Hz) and beta (12-20 Hz) frequency bands. While the grouping of these frequencies into the particular bands has occurred historically, some researchers have advocated abandoning fixed frequency bands (Klimesch, 1999), particularly in disorders such as ADHD (Martijns et al., 2008). Therefore, we examined activation at each of the 1-Hz frequency intervals between 4-20 Hz for finer resolution of activation results. Activation patterns were compared with a repeated measures analysis of variance (ANOVA) with Greenhouse-Geiser correction when appropriate. Because we were most interested in frontal and parietal cortical activity to tap the fronto-parietal attention network and wanted to reduce the number of statistical comparisons, we grouped electrodes in the Frontal (F3,F4,Fz) and Parietal (P3,P4,Pz) regions and used the mean activity in these areas for analyses. Recording condition (EC, EO, CPT) was used as a within-subject variable and diagnostic status (ADHD, control) as a between-subjects variable. Separate 3×2 repeated measures ANOVAs were run for the frontal and parietal regions. Diagnostic group differences in CPT performance were tested using separate ANOVAs for each of the dependent variables. The association between CPT performance and EEG variables was assessed using a two-tailed Pearson Product Moment correlation. In order to limit the number of comparisons, only the EEG variables that exhibited significant interaction effects (EEG activity by diagnosis) were used in the correlation analysis. Significant differences in the strength of the association (i.e., size of the correlations) were tested using a Fisher z transformation. Temporal changes in cortical activation patterns were tested using repeated measures ANOVAs with a 3 (first, middle, last 5 minutes of EEG recorded during CPT) $\times 2$ (diagnostic status) design for both frontal and parietal regions.

Results

Clinical characteristics

The ADHD and Control groups did not differ in terms of age, sex, educational level, ethnic background or IQ (see Table 1). The ADHD group had significantly higher rates of mood, other disruptive behaviors, and substance abuse disorders when compared to controls.

Patterns of cortical activation across baseline and cognitive activation conditions

Presented in Figure 1 are the power spectra for the ADHD and control groups across the EO, EC and CPT conditions. The repeated-measures ANOVAs indicated significant main effects of condition for all frequency bins (4-20 Hz) across conditions ($F(2,104)=3.6-69.0$), p -values range from $<.05$ to $<.0001$), which suggests that there are widespread cortical changes when going from EC to EO to CPT. Significant interaction effects of condition and ADHD diagnostic status emerged in the lower alpha range (8-10 Hz) in frontal ($F(2,104)=5.9$, $p=.008$, FDR-adjusted $p=.01$) and parietal ($F(2,104)=5.0$, $p=.01$, FDR adjusted $p=.03$) regions. Significant effects consistently emerged for the 8-9-Hz and 9-10-Hz frequency bins, therefore, we grouped them together for all analyses. Results of the analyses indicate that the ADHD group exhibited greater attenuation of 8-10 Hz power than controls suggesting increased cortical activation in the ADHD group. In addition, the ADHD group exhibited reduced power in the 8-10Hz range in the frontal (but not the parietal) region (Frontal: $F(1,52)=6.1$, $p=.02$, FDR adjusted $p=.05$; Parietal: $F(1,52)=1.2$, $p=.27$, FDR adjusted $p=.41$). This group difference was due primarily to significant differences in alpha power during the EC ($F(1,52)=6.3$, $p=.01$, FDR adjusted $p=.03$) and CPT conditions ($F(1,52)=7.1$, $p=.01$, FDR adjusted $p=.03$) where the ADHD group displayed significantly reduced alpha power. The group difference in alpha power during the EO conditions was not statistically significant ($F(1,52)=2.5$, $p=.11$, FDR adjusted $p=.17$). Because alpha activity is inversely related to cortical arousal, these results suggest that the ADHD group exhibited greater cortical arousal during the eyes closed condition.

In the beta range, another interaction effect emerged according to ADHD status in the 17-18 Hz range indicating that controls show a slight attenuation in frontal beta activity during the EO and CPT conditions that ADHD group does not (Frontal: $F(2,104)=3.91$, $p=.03$, FDR adjusted $p=.05$). In the parietal region, another effect of diagnostic status on cortical activation was found in the 13-14 Hz range (Parietal: $F(2,104)=3.6$, $p=.04$; FDR adjusted $p=.07$). This suggests that controls again had greater attenuation of beta power during EO and CPT conditions than the ADHD group. While ADHD adults exhibited slightly increased beta activity compared to controls, the between-group difference (i.e., main effect of diagnosis) was not statistically significant (Frontal 17-18Hz: $F(1,52)<1$, n.s.; Parietal 13-14Hz: $F(1,52)<1$, n.s.). Beta activity is thought to represent cortical activation and suggests that the ADHD group exhibited greater cortical activation than the controls during the CPT condition.

Behavioral performance on the CPT was compared across diagnostic groups and is presented in Table 2. Overall, CPT performance between ADHD and controls did not differ significantly, with the exception of beta response style ($F(1,77)=8.34$, $p=.005$). Beta in this context refers to a person's response style or approach to responding on the task. Low beta values indicate that a person has a more impulsive response style that emphasizes commission errors over omission errors. High beta values indicate a more cautious response style that emphasizes errors of omission over errors of commission. Controls had significantly higher scores on beta when compared to the ADHD group, suggesting that controls were more cautious or careful in responding, while the ADHD group had a more impulsive response style.

Relationship between cortical activation and behavioral performance

In order to assess the functional relationship of cortical activation patterns with CPT performance, Pearson correlations were run between spectral power and CPT performance in the whole sample. All of the correlations were modest (none larger than .23) and statistically non-significant. We then ran separate correlations for the control and ADHD groups; results are summarized in Table 3. Differential patterns of correlations emerged according to diagnostic group suggesting that cortical activation patterns are associated with different cognitive processes among ADHD and control groups. Within the control group, increased frontal and parietal power in the 8-10 Hz range was significantly associated with higher rate

of commission errors (frontal $r=.43$, $p<.01$; parietal $r=.45$, $p<.01$), faster reaction time (frontal $r=-.49$, $p<.005$; parietal $r=-.48$, $p<.005$) and higher scores on d-prime (frontal $r=.46$, $p<.01$; parietal $r=.45$, $p<.01$). These correlations suggest that increased alpha power is associated with a fast, impulsive response style and reduced stimulus discrimination among the controls. Only one performance variable (task variability) was significantly associated with cortical activity for the ADHD group, indicating that frontal alpha (8-10 Hz, $r=-.45$, $p<.01$), frontal beta (17-18 Hz, $r=-.52$, $p<.005$) and parietal beta power (13-14 Hz, $r=-.47$, $p<.01$) were associated with reduced task variability.

A Fisher r to z transformation was used to determine whether the strength of the correlations between cortical activation and CPT performance differed according to ADHD status. As seen in Table 3, several statistically significant differences in correlation emerged (as indicated by the boxes) according to diagnostic status, the majority of which (5 out of 7) were in the frontal region. This suggests that cortical activity in the frontal regions is significantly different among ADHD and control participants.

Patterns of cortical activation during sustained attention task across time

Presented in Figure 2 is the EEG power recorded during the first third, middle third, and last third of the CPT task (each section ~5 minutes) for the ADHD and control groups. Repeated measures ANOVAs indicate main effects of time for the 7-12 Hz range that trended toward significance in the frontal region ($F(1,62)=3.5-5.3$, $p=.03-.05$, FDR adjusted $p=.09$) and the 12-13 Hz range in the parietal region ($F(1,62)=4.2$, $p=.04$, FDR adjusted $p=.08$). This suggests that both groups showed decreases in alpha and beta power over time on the CPT task. Significant interaction effects (time by diagnosis) emerged in the 8-10 Hz frequency range in the frontal region ($F(1,62)=7.0$, $p=.01$, FDR adjusted $p=.05$) and trended toward significance in the 9-10 Hz frequency range in the parietal region ($F(1,62)=5.8$, $p=.02$, FDR adjusted $p=.07$). This suggests that control and ADHD groups exhibited significantly different patterns of cognitive activation across time with both groups exhibiting similar levels of activation in the initial 5-minutes of the task. The control group then exhibited a large increase in alpha power in the next 10 minutes, whereas the ADHD group maintained the same level of alpha power during the same time period. This suggests that the control group adapted to the sustained attention task as time went on and required less cortical activation, while the ADHD group maintained a high level of neural activation throughout the task.

Discussion

The current study examined patterns of cortical arousal and activation according to ADHD diagnostic status. The results presented here are consistent with previous brain imaging studies that suggest different patterns of cortical activation in ADHD and takes a step further in examining brain activity during arousal, activation and sustained attention. Contrary to previous hypotheses, adults with ADHD exhibit higher levels of cortical arousal than controls in resting states. When engaged in a sustained attention task, the adults with ADHD required increased cortical activation than controls, and this is particularly true as attention is sustained over longer periods of time. Examination of cortical activity across different baseline and cognitive activation conditions is a useful methodology for studying cortical arousal and activation that is not readily studied with other brain imaging technologies.

The most robust finding is that power in the lower alpha (8-10 Hz) range is attenuated in ADHD, suggesting that decreased alpha power may be an important neurophysiologic marker in adults with ADHD. While alpha activity is widespread throughout the cortex, source localization suggests it may reflect activity in the precuneus (Mantini et al, 2007; Michels et al, 2008), which is a locus recently suggested as playing a role in ADHD (Castellanos et al., 2007). Attenuation of 8-10 Hz alpha power is observed during a great variety of tasks, is globally

distributed, and most likely represents attentional task demands (Klimesch, Sauseng, & Hanslmayr, 2007). Reduced alpha power is also associated with expectancy and preparation of the visual cortex to processing incoming visual stimuli (Gomez, Vaquero, Lopez-Mendoza, Gonzalez-Rosa, & Vazquez-Marrufo, 2004) and inversely related to fMRI BOLD cortical response (Laufs et al., 2006). Collectively, these data suggest that attenuation of alpha power is associated with increased cortical arousal. Our finding of attenuated alpha power in adults with ADHD is contrary to our hypothesis of cortical hypoarousal, it is consistent with other studies that have found increased cortical activity (O'Gorman et al., 2008; Tian et al., 2008) in ADHD. This suggests that adults with ADHD may require more cortical arousal in order to comply with the directions of the experimental situation where they are asked to sit still in order to reduce muscle contamination in the EEG recording. In addition, the average estimated IQ for both the ADHD and controls groups is above average, which coupled with the essentially normal CPT results for the ADHD group suggest that they may have compensated particularly well for their own ADHD. Further research is needed to determine whether specific subgroups of adults with ADHD exhibit increased or decreased cortical arousal.

Although behavioral task performance did not differ according to ADHD diagnosis, cortical activation was associated with different cognitive functions in the ADHD and control groups. Differing functional relationships between cortical activation and behavioral performance were most marked in the frontal regions as opposed to the parietal regions. These results suggest that the neural organization, particularly in the frontal regions among adults with ADHD differs from non-ADHD adults. This alternative neural organization did not result in significantly worse behavioral performance, but does require increased cortical activation to sustain attention over a long period of time. While non-ADHD adults relied on cortical activation in frontal areas for successful performance in the beginning of a task, they adjusted to the task and were able to successfully perform the task without frontal involvement. On the other hand, adults with ADHD relied on frontal activation during the whole task in order to exhibit successful performance.

The results of this study differ from previous EEG studies of ADHD, many of which have found increased theta power among ADHD patients (Bresnahan, Anderson, & Barry, 1999; Chabot & Serfontein, 1996). While the majority of studies have compared children with and without ADHD, there are at least two studies that have examined adults. Both of these studies found significantly elevated theta power among adults with ADHD when compared to controls (Bresnahan et al., 1999; Bresnahan & Barry, 2002). This may be due to methodologic differences since previous studies have recorded brain activity during baseline (EO or EC) conditions and have not examined cognitive activation conditions. There has been one study that compared EEG measures recorded during cognitive activation in ADHD and control children, which found that ADHD children exhibited decreased alpha power during math calculations when compared to controls (Swartwood, Swartwood, Lubar, & Timmermann, 2003). The results of both studies suggest individuals with ADHD required increased cortical activation when compared to controls for task completion.

There are several limitations that should be considered along with the present results. Our sample of adults, both ADHD and control, are a community sample and may differ from samples ascertained through more clinical venues. Furthermore, both groups were ascertained through having at least one child with ADHD and may therefore differ from adults who do not have children with ADHD. As noted previously, our sample of adults with ADHD may not be representative of other adults with ADHD given their above average estimated IQ and well compensated performance on the CPT. In addition, we have performed a large number of statistical tests, which may increase the possibility of Type 1 (false positive) error. In order to address this possibility, we have calculated and reported p-values adjusted for the False Discovery Rate to control the family-wise experimental error rate. Further studies are needed

to assess generalization of these findings to other adults with and without ADHD and provide independent replication of results.

In conclusion, adults with ADHD exhibit increased levels of cortical arousal and activation. While performing a sustained attention task, adults with ADHD maintain consistently higher levels of cortical activation over a long period of time than non-ADHD adults in order to achieve the same level of behavioral task performance. This may be due to differing neural organization, particularly in frontal areas. Examining cortical activity during baseline and cognitive task performance is a powerful technique for studying arousal and activation patterns associated with psychiatric diagnoses.

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Figure 1.

Spectral power showing brain activity across conditions and by diagnosis. Lines indicate significant condition by diagnosis interaction effect; p-value is noted between the lines. Frequency bins are in 1 hertz (Hz) each. Alpha power is inversely correlated with cortical arousal, therefore the ADHD group exhibits greater cortical arousal. Beta power is positively correlated with cortical activation, thus the ADHD group demonstrates greater cortical activation in the eyes open and CPT (sustained attention) conditions. EC=Eyes closed, EO=Eyes Open, CPT=Continuous performance test.

Figure 2.

Alpha power (8-10 Hz) during sustained attention over time in frontal and parietal regions. Lines indicate significant condition by diagnosis interaction effect; p-value is noted between the lines. Note that alpha power is inversely related to cortical activation, thus the control group exhibits decreased cortical activation over time. CPT=Continuous performance task.

Table 1

Sample demographics and clinical characteristics

	Non-ADHD	ADHD	p-value
N=	42	38	
age in years	46 (5.4)	45 (6.0)	0.49
sex, % males	50%	53%	0.655
educational level	3.97 (1.4)	3.52 (1.4)	0.16
Est.Full Scale IQ	116 (12.6)	116 (14.3)	1
Other Lifetime Psychiatric Diagnoses			
Any mood dx	30%	63%	0.01
any anxiety dx	30%	53%	0.07
ODD/CD	0%	30%	>0.001
Substance abuse/dep	15%	41%	0.02
ADHD type-Lifetime			
Combined Type		55%	
Inattentive Type		37%	
Hyperactive-Imp Type		8%	

Note. ADHD=Attention Deficit Hyperactivity Disorder, IQ= Intelligence Quotient, dx=diagnosis, ODD=Oppositional Defiant, Disorder, CD=Conduct disorder

Table 2

Continuous Performance Test performance by ADHD diagnosis

	Control		ADHD		F	p-value
	Mean	SD	Mean	SD		
N=	42		37			
Omission Errors	53.14	12.49	53.47	13.69	0.01	0.92
Commission Errors	46.99	9.31	50.95	10.34	3.12	0.08
Hit RT	57.00	11.30	54.50	13.61	0.77	0.38
RT Variability	58.35	11.95	57.59	12.45	0.07	0.79
Task Variability	58.16	12.20	54.64	11.02	1.68	0.20
D-prime	47.65	9.59	50.93	9.41	2.29	0.13
Beta (response style)	56.51	13.11	49.41	7.72	8.34	0.005

Note. RT=Reaction Time

Table 3

Correlations between cortical activation and task performance

		Omission errors	Commission errors	Hit Reaction time	Response Variability	Task Variability	D-prime	Beta
Frontal:8-10Hz	Control	0.20	0.43*	-0.49**	-0.20	-0.04	0.46*	-0.24
	ADHD	0.02	-0.03	-0.03	-0.23	-0.45*	-0.03	-0.25
Frontal:17-18Hz	Control	0.04	0.18	-0.34	-0.14	0.05	0.36	-0.01
	ADHD	-0.09	-0.20	-0.04	-0.29	-0.52**	-0.13	-0.20
Parietal:8-10Hz	Control	0.15	0.45*	-0.48*	-0.05	0.14	0.45*	-0.46*
	ADHD	0.14	0.20	-0.17	-0.24	-0.29	0.18	-0.16
Parietal:13-14Hz	Control	0.15	0.19	-0.28	-0.07	0.08	0.32	-0.30
	ADHD	0.02	-0.02	-0.03	-0.31	-0.47**	0.02	-0.17

*
p ≤ .01**
p ≤ .005.

Correlations with cell borders denote significantly different correlations for ADHD and Control groups; single border p < .05, double border p < .01