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SPECIAL ISSUE

Parietal Foci for Attention Deficit/Hyperactivity Disorder: Targets for LORETA Neurofeedback with Outcomes

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Attention Deficit/Hyperactivity Disorder (ADHD) continues to present challenges to researchers and clinicians across disciplines. Many of the arguments and disagreements at hand may originate from the box, or polemic camps, we have created, rather than the disorder itself. With this in mind, this article presents a novel approach for neurofeedback training, neuropsychometric evaluation, and outcome reporting specific to ADHD. In short, for ADHD to be treated effectively and empirically, outcomes must show learning (acquisition) has taken place, the electroencephalogram (EEG) or EEG current source density has changed, and psychometric data correspondingly has improved. It is time for novel approaches to take form as the toll ADHD exacts on the individual across the lifespan continues to progress. The author proposes a protocol for assessment, using a LORETA-based quantitative EEG to identify a network of brain structures on the cortical surface and below, with abnormal activation and LORETA-guided neurofeedback training to normalize activity in this network.

Introduction

Attention Deficit/Hyperactivity Disorder (ADHD) is a chronic syndrome presenting deficits in executive functions (self-regulation) and attentional processes. The condition is characterized by difficulty with staying focused (inattention), difficulty controlling behavior, and hyperactivity. These symptoms can create problems in academic, social and familial contexts, as well as planning and organization skills needed for daily functioning (American Psychiatric Association [APA], 2000).

The Centers for Disease Control and Prevention (CDC) projected that as many as 9.5% of U.S. children are affected by this disorder (CDC, 2014). The CDC describes ADHD as a syndrome for which there is no single test that can be used to diagnose accurately. Additionally, comorbid syndromes are often present, which can mimic the symptoms of ADHD and confound the differential diagnosis (e.g., anxiety, depression, learning disorders). A consensus on its origins and the best neuropsychological measures for more accurate differential remain elusive. Specific combinations of neurometric and psychometric measures may improve our ability to differentiate the syndrome more accurately, as well as facilitate understanding the biobehavioral substrates across the spectrum of ADHD.

I have carefully evaluated ADHD and its clinical presentation and found it would be useful, if not mandatory, to utilize measures of attention and executive functions (self-regulation) in the assessment of ADHD, as well as an outcome criterion for neurofeedback training or pharmacologic intervention. Self-regulation and executive function describe the same process and are not two independent processes (Cannon et al., 2014) and as such can be considered one primary dysfunction across clinical populations, with emphasis in ADHD specifically. This is perhaps the most fundamental step in improving the differential for ADHD since numerous experts have associated these two components as primary core features of ADHD deficits without relative measurements being predictive or specific to ADHD (Mirsky & Duncan, 2001), nor showing associative patterns with brain imaging data. Yet, most research and clinical procedures tend to rely on parent or teacher rating scales as a primary diagnostic indicator (APA, 2000), which potentially contributes to both inaccurate diagnostics and poor research outcomes.

Barkley (1997) intuitively defined ADHD as a disorder of self-regulation that was reinforced by deficits in executive functions. This definition holds merit, with the exception that the potential neural correlates of executive functions, or self-regulation, have not been described in terms of any clear neurological mechanism. Research data examining the neural differences distinguishing ADHD

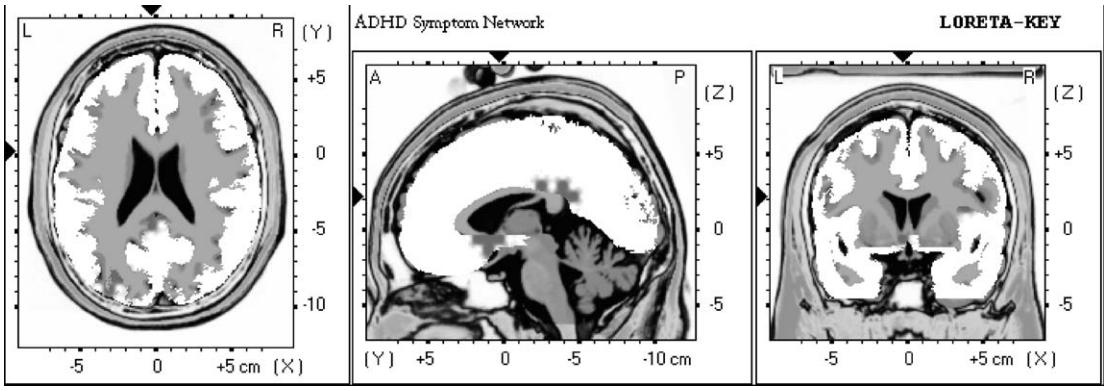

Figure 1. Attention Deficit/Hyperactivity Disorder Symptom Network (ADHD-SN). From left to right are horizontal, sagittal, and coronal views of the brain. The first image (left) shows the precuneus (Brodmann Area [BA] 19) and posterior cingulate (BA 30/29). The sagittal image shows BA 29 (posterior cingulate), BA 23 (mid-cingulate) and BA 25 (orbital frontal cortex). The coronal image (right) shows BA 25 and BA 13 (insular cortex).

patients from normal controls are extensive, however; several important studies have reported white matter volume deficits in left parieto-occipital regions, as well as overall volume reductions in the precuneus in ADHD patients (Carmona et al., 2005). One important neurological finding in ADHD is the compromised functional integrity of the default network (DMN) of the brain and its interactions with other identified networks (Cannon, Kerson & Hampshire, 2012; Cannon, Kerson, Hampshire, & Garner, 2012; Fassbender et al., 2009; Liddle et al., 2011; Uddin et al., 2008; Zang et al., 2007). The interactions among the cingulate, medial prefrontal areas, and the parietal lobes have been identified as alternative loci for the manifestation of integrative dysfunctions associated with ADHD (Castellanos et al., 2008). It is also to be noted that the cerebellum and regional locations of the corpus callosum (splenium and isthmus) contribute to the symptoms of ADHD (Overmeyer et al., 2000; Steere & Arnsten, 1995). One of the more important issues is the symptom of hyperactivity and movement, and the prospective involvement of dopamine in mesolimbic and cerebellar pathways (Retz, Rosler, Supprian, Retz-Junginger, & Thome, 2003; Volkow et al., 2009; Volkow et al., 2010).

Alpha activity has been shown to be differentially modulated and anticorrelated with midfrontal theta activity (Mazaheri et al., 2010) in normal children but not in those with ADHD. As a result of numerous research studies and clinical cases, I propose that a specific network will play a role in the symptoms of ADHD, as well as their reduction or remission. Figure 1 shows this hypothesized ADHD symptom network (ADHD-SN) with voxels and coordinates shown in Table 1. This network in pretraining quantitative electroencephalogram analyses using the Neuroguide Lifespan database (Applied Neuroscience) can show a three Standard Deviation (*SD*) deficit in alpha in posterior regions, while showing up to a two *SD* increase in the anterior location (Cannon, 2012). Similarly, theta activity can reach three *SD*s in posterior regions. I do not assume that this network operates in a singular capacity; rather its location and interactions with other regions and important structures present a novel, yet functionally sound potential for two reasons. First, the posterior cingulate is highly involved in language and learning processes, which are the two fundamental components of self-regulation (Cannon et al., 2014; Locke, Angevine, & Yakovlev, 1964; Westlye, Walhovd, Bjornerud, Due-Tonnessen, & Fjell, 2009; Zhou et al., 2008). Second, the location and proximity to thalamus, brain stem, and cerebellar pathways are of particular interest, as well as connections with limbic structures.

LORETA-Based Protocol for Assessment and Treatment of ADHD

Low-resolution brain electromagnetic tomography (LORETA) is a method of probabilistic source estimation of electroencephalogram (EEG) signals in a standardized brain atlas space utilizing a restricted inverse solution (Pascual-Marqui, Esslen, Kochi, & Lehmann, 2002; Pascual-Marqui et al., 1999; Pascual-Marqui, Michel, & Lehmann, 1994). Modeling of neural activity by using measurements of the voltage potential at various locations on the scalp to estimate current sources inside the brain that best fits the data under study is a well-established methodology (De Munck, Van Dijk, & Spekreijse, 1988; Hallez, et al., 2007). LORETA and sLORETA have been used to examine EEG sources in depression (Pizzagalli, Oakes, & Davidson, 2003), epilepsy (Zumsteg, Wennberg, Treyer, Buck, & Wieser, 2005) and to evaluate state dependent changes associated with differential task specific Default Mode Network (DMN) activity (Cannon, 2012). LORETA has been adapted

Table 1. Regions of interest for this study data				
BA	**Hemisphere**	**Coordinates**	**Label**	**Voxels**
19	L	−31, −81, 22	Precuneus	3
13	R	39, −4, 8	Insula	6
23	R	4, −32, 29	Cingulate gyrus	5
25	L	−3, 10, −13	Subcallosal gyrus	6
29/30/31	L	−3, −46, 22	Posterior cingulate	6

Note. From left to right are the Brodmann Areas (BAs), hemispheres, low-resolution brain electromagnetic tomography *x*-, *y*-, and *z*-coordinates, neuroanatomical labels, and number of voxels in the regions of interest.

to provide real-time feedback to participants in order to facilitate operant conditioning of the current source density (CSD) in specific frequency ranges at a specific region of training (ROT) within Talairach three-dimensional (3-D) space (Congedo, Lubar, & Joffe, 2004). Through feedback the user can then change the CSD at the ROT to influence improvements in cognitive, attentional, and affective processes. The effects of LORETA neurofeedback have been demonstrated over the past few years with increasing use (Cannon & Lubar, 2011; Cannon et al., 2006; Cannon, Congedo, Lubar, & Hutchens, 2009; Cannon et al., 2007; Cannon, Lubar, Sokhadze & Baldwin, 2008; Liechti, et al., 2012).

This section introduces a protocol using a LORETA-based quantitative EEG to assess the baseline activation in the ADHD-SN, in a given patient presenting for treatment. LORETA utilizes the cortical findings of an EEG, and statistically produces a 3-D localization of areas contributing to maladaptive patterns and correlated symptoms. Once the cortical sources are evaluated and linked to symptoms contributing to the disorder, a LORETA-guided neurofeedback (LNFB) treatment can begin normalizing activity in targeted areas of the brain.

This clinical work was fashioned with two primary theories. Firstly, functional specialization within cortical networks should show some association with post-training psychometric scores. Second, both EEG CSD levels in a trained frequency and functional correlations among specific network regions should also change posttraining. The CSD in LORETA can be thought of as the length of a vector in 3-D space (the square root of the sum of the squares at three CSD moments on the *x*-, *y*-, and *z*-planes), with only the length of the vector under consideration (Congedo, 2003; Congedo et al., 2004; Pascual-Marqui et al., 1994).

One of the more interesting points for this LNFB procedure for ADHD is the focus on the alpha frequency domain. Alpha is shown to be associated with integrative functions, self-regulation, attention, and self-referential activity (Klimesch, Doppelmayr, Russegger, Pachinger, & Schwaiger, 1998; Klimesch et al., 2001; Knyazev, Slobodskoj-Plusnin, Bocharov, & Pylkova, 2011). In the traditional neurofeedback approach, alpha has not been considered an option for diagnostic procedures and training targets for ADHD (Cannon et. al., 2014; Cannon, Kerson, & Hampshire, 2012; Cannon et al., 2012). This may be as result of improvements in technology, research, and neuroelectrical imaging techniques. Research has suggested an important interaction between alpha power in parietal regions and theta power in fronto-central locations (Gordon, Palmer, & Cooper, 2010; Hale et al., 2009; Koehler et al., 2009; Lansbergen, Arns, van Dongen-Boomsma, Spronk, & Buitelaar, 2010). Additionally, the parietal lobes have been implicated in attention, intention, and priority (Bisley & Goldberg, 2010), and alpha power has also been found to show some degree of association with white matter volumes (Valdes-Hernandez et al., 2009) implicated in ADHD. After review of a large body of data concerning ADHD, simple logic dictated that alpha in the left parietal area (precuneus) would be an ideal target for LNFB procedures.

Methods

The standardized procedures for LNFB training in the precuneus can be found elsewhere (Cannon et al., 2014). This study consisted of eight clinical patients, five males and three females, with prior diagnosis of ADHD with a mean age of 14.26 (range 7–17 years; $SD = 3.5$). It should be noted that three of the participants had comorbid diagnoses consisting of anxiety, tic disorder, and auditory processing disorder. All participants and parents read, signed, and agreed to informed consent for the clinical training and were apprised of all risks and benefits. This clinical study was conducted in accordance with standard

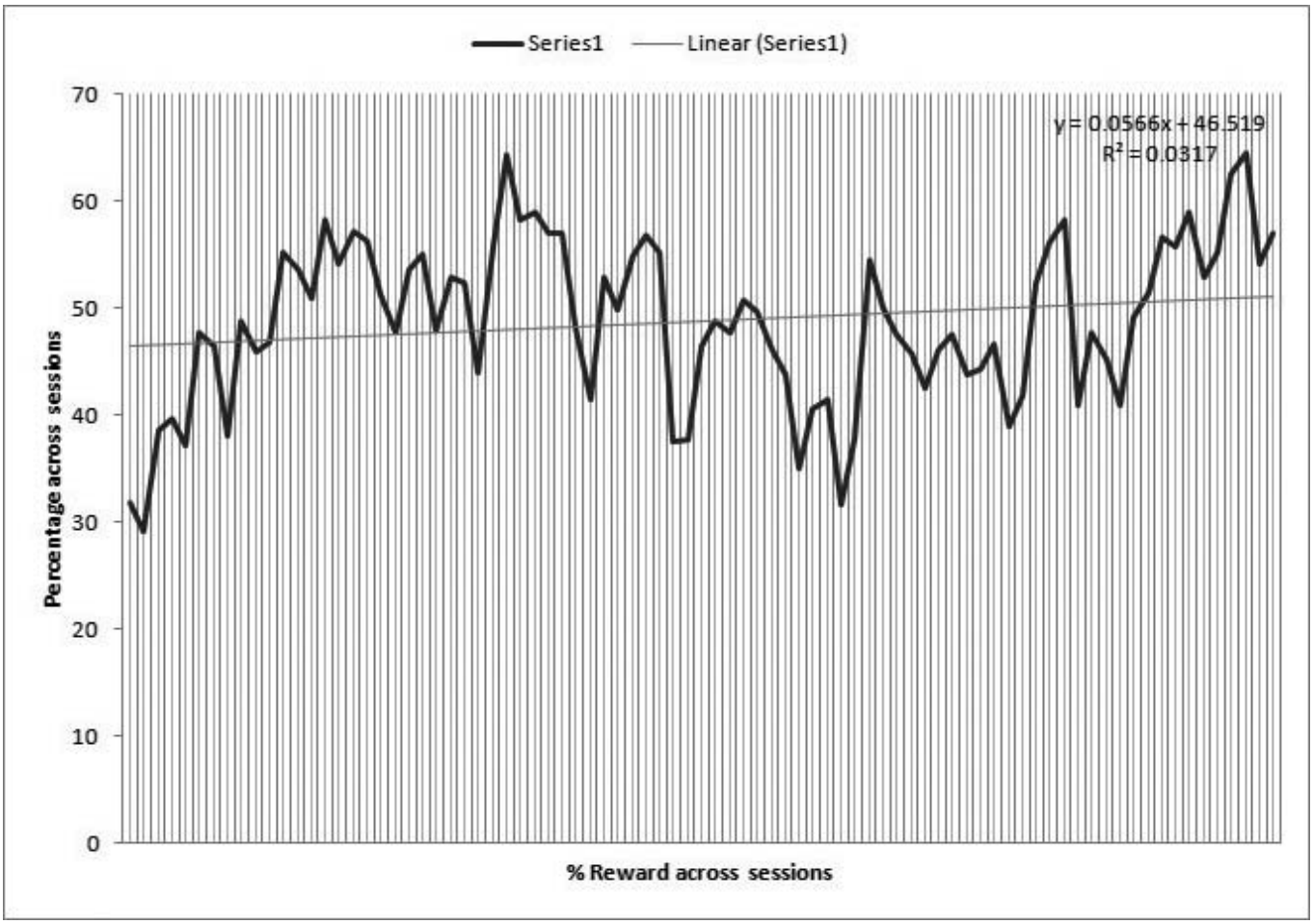

Figure 2. Mean percentage reward across sessions for the group of eight participants.

procedures used in prior Institutional Review Board (IRB) approvals and the Declaration of Helsinki (Cannon et al., 2009; Cannon et al., 2007). The LNFB training was conducted using the 19 leads of the standard international 10/20 system with linked ear reference with Deymed Truscan EEG System (Deymed Diagnostic, Payette, ID). The center of the three-voxel ROT was located at Talairach coordinates ($x = -31$, $y = -81$, $z = 22$) and the frequency trained was an extended alpha band (8–13 Hz). LNFB training sessions were composed of six 5-minute rounds and were conducted six times per week for 15 to 20 consecutive weekdays. During a preliminary session, shaping was induced to set thresholds such that each participant could meet the standard reward criteria (e.g., generate the desired response at a minimal rate), and participants were informed of the inhibitory and reward aspects of the training. Standardized thresholds were then set and maintained for each participant. The participants were provided visual and auditory feedback, and points were achieved when they were able to simultaneously increase alpha CSD (8–13 Hz) at the ROT, while minimizing electromyogram (EEG) (35–55 Hz) and electrooculogram (EOG) (1–3 Hz) in linear combinations of channels: EMG: T3, T4, T5, T6, O1, and O2; EOG: FP1, FP2, F3, F4, F7, and F8. These criteria had to be maintained for 0.75 seconds to achieve one point.

Assessment Measures

In order to assess learning effects I utilized paired comparisons to contrast the current source density levels for alpha at the ROT and all regions within the proposed ADHD-SN pre- and posttraining. Patients were administered the Delis-Kaplan Executive Function System (DKEFS) verbal fluency and color-word interference tasks (Delis, Kaplan, & Kramer, 2001) pre- and posttraining. This instrument assesses higher level cognitive functions (e.g., attention, language, and creativity) in both children and adult populations, as well as the Integrated Visual and Auditory Continuous Performance Test (IVA+). The IVA+ is an integrated 13-minute auditory and visual continuous

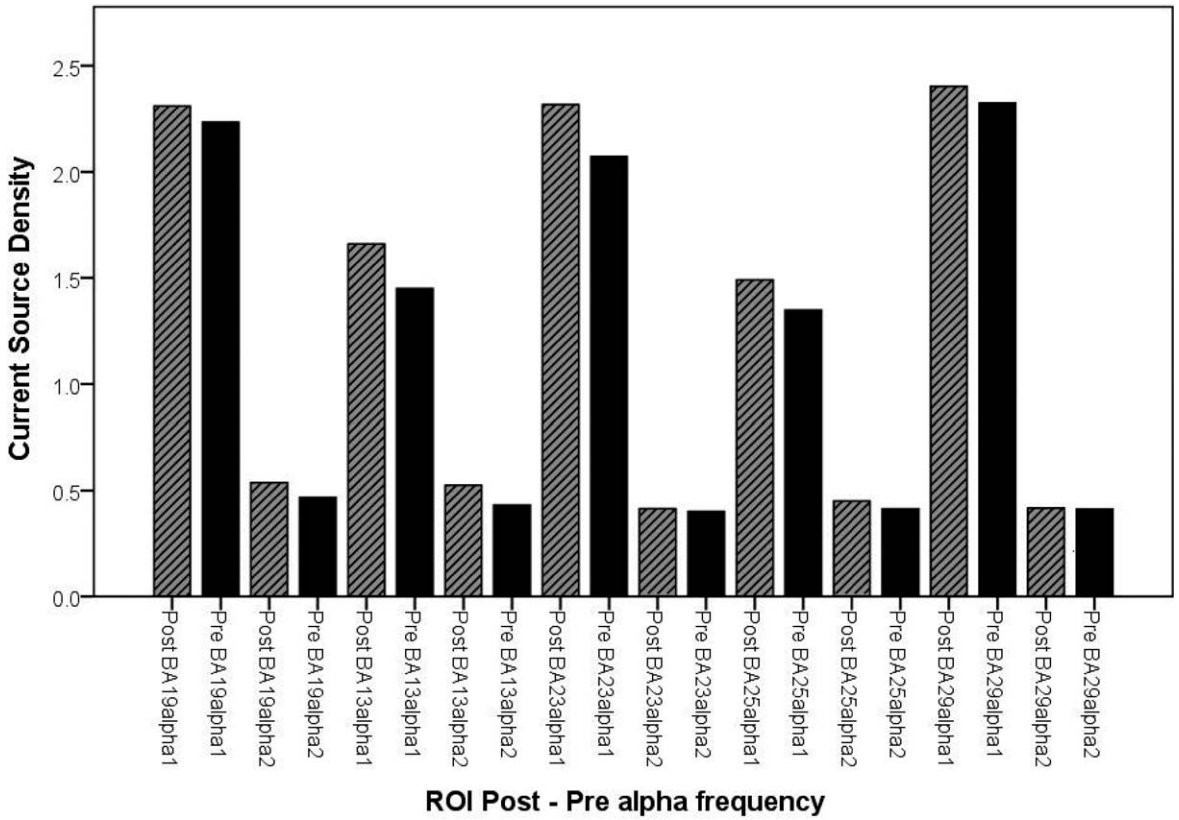

Figure 3. Post- > pre-alpha current source density at each of the five regions of interest for this study. Alpha is presented in two measures alpha 1 (8–10 Hz) and alpha 2 (10–12 Hz).

performance test designed to assess two major factors: response control and attention. The test task involves responding or inhibiting a response for a total of 500 trials (Sandford & Turner, 1994). The DKEFS and IVA+ were administered in a quiet office adjacent the EEG collection/neurofeedback training facility, prior to the initial baseline EEG and preliminary LNFB session. At the final session, the tests were given prior to the last LNFB session.

Posttraining Results

Alpha CSD. Figure 2 illustrates that participants were able to increase alpha in the desired direction; however, none of the regions of interest (ROI) differences posttraining reached significance. Interestingly, the greatest posttraining differences occur in right Broadmann Area (BA) 13 and BA 23 respectively (as seen in Figure 3).

DKEFS. One of the more significant findings for this study is the decrease in errors for the verbal fluency, $t(7) = 2.64$, $p = .033$, and color-word interference task, $t(7) = 2.72$, $p = .030$. The participants scored higher on posttesting for the DKEFS in all tasks: FAS, $t(7) = -2.77$, $p = .028$; CAT, $t(7) = -.530$, $p = .613$; SWC, $t(7) = -1.37$, $p = .213$; SWT, $t(7) = -2.14$, $p = .07$; Color, $t(7) = -2.47$, $p = .043$; Naming, $t(7) = -.704$, $p = .504$; Inhib, $t(7) = -1.46$, $p = .185$; Switch, $t(7) = -1.69$, $p = .133$.

IVA+. The participants scored significantly better on the IVA+ post LNFB for all 6 six measures except the fine motor response quotient (FMRQ), $t(7) = 1.54$, $p = .167$; however, the change was in the desired direction. The remaining scores results are: full scale response quotient (FSRQ), $t(7) = 4.11$, $p = .005$, full scale attention quotient (FSAQ), $t(7) = 3.58$, $p = .009$, sustained auditory attention quotient (SAAQ), $t(7) = 3.86$, $p = .006$, and sustained visual attention quotient (SVAQ), $t(7) = 2.86$, $p = .024$. There was a significant reduction in the number of hyperactive events $t(7) = 4.54$, $p = .003$.

Neural Associative Pairing Procedures for ADHD

The regions within a proposed network of self-regulation, or ADHD-SN (lack of integrated self-regulation), were identified as a focus of study for this data, based on their association with the precuneus and studies showing activation across attention, cognitive, and self-regulatory tasks. I have often considered the cytoarchitecture of the cingulate gyrus and its role as a target for LORETA

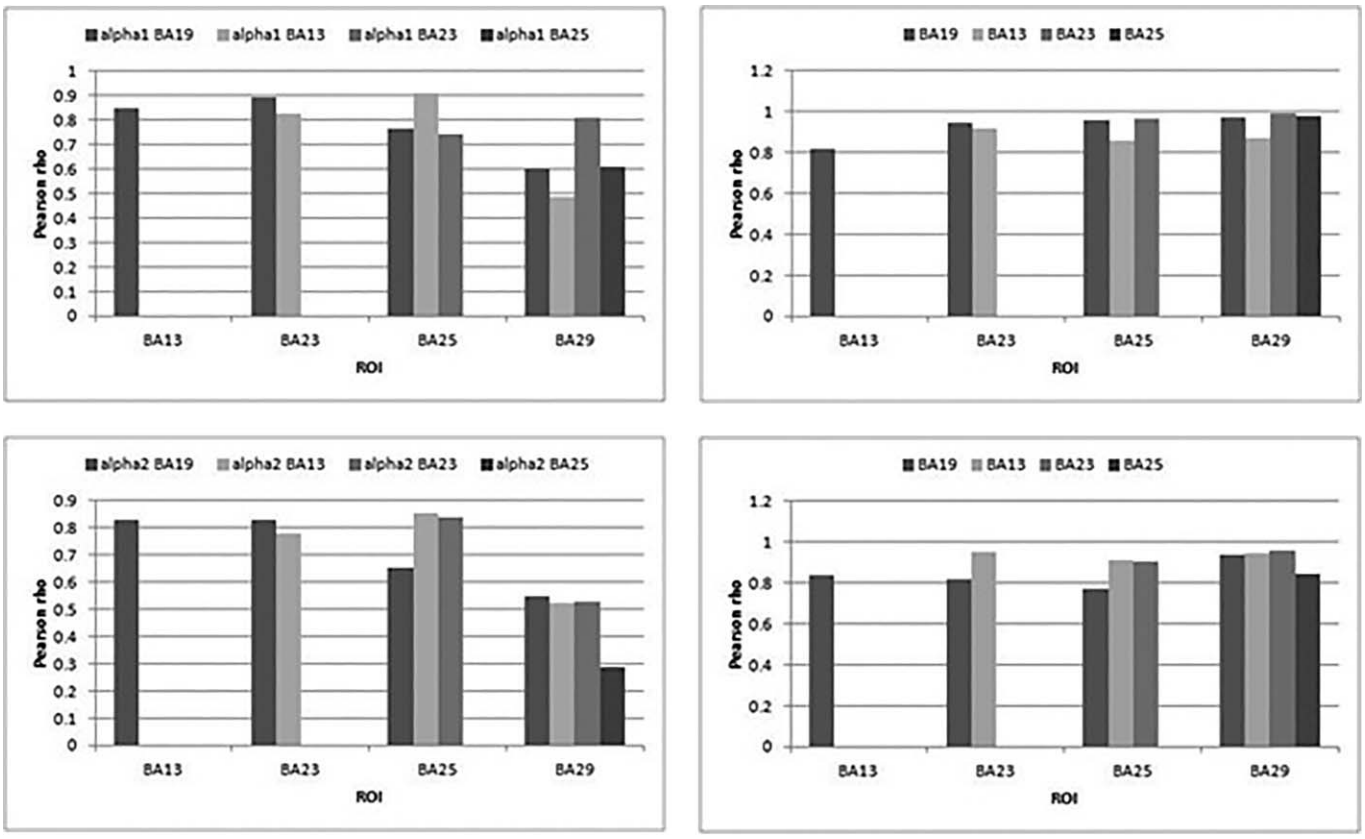

Figure 4. Pre- (left) and post- (right) correlation structure for Attention Deficit/Hyperactivity Disorder-Symptom Network in alpha 1 (top) and 2 (bottom) current source density (CSD) between the five regions of interest (ROIs) for this study. On the *x*-axis is the ROI, and Pearson rho on the *y*-axis. The correlation between ROI*alpha CSD is plotted within the graph.

neurofeedback procedures across different psychopathologies. For example, depression, ADHD, anxiety, and response to treatment all have shown activation or the inverse in cingulate regions (Pizzagalli et al., 2003; Pizzagalli, Peccoralo, Davidson, & Cohen, 2006; Salvadore et al., 2010; Smith, Taylor, Brammer, Halari, & Rubia, 2008). The center voxels for the ROIs for this study data are shown in Table 1. Figure 4 shows the functional associations between these five ROIs pre- and posttraining for the alpha 1 (top) and alpha 2 (bottom) CSD levels. Changes in the correlations were examined utilizing Fisher's Z test. Significant differences were found in alpha 1 between BA 13/29, Z= 2.19, $p = .05$; BA 25/29, Z = 2.36, $p = .034$; and in alpha 2 between BA 13/29, Z = 1.86, $p = .09$; BA 23/29, Z= 2.13, $p = .06$. There were also significant changes in other frequency domains in these same ROIs, which will be published in another work.

Functional Relations among ROI, EEG CSD, and Outcomes

The correlations among ROI, EEG CSD, and functional outcomes are presented below by each functional measure. Only those functional associations that reached significance or that were very close to significant are presented. The DKEFS errors (not shown in graphical form) in verbal fluency decreased significantly posttraining, yet did not show significance with the ADHD-SN in any frequency. The color word errors showed functional association with alpha 1 at BA 25 with r = .70, $p = .052$ and beta at BA 13 with r = .76, $p = .030$. Table 2 shows the results for posttraining IVA+ scores and their associations with the ADHD-SN. Hyperactive events on the IVA+ posttraining show a significant negative relationships delta in all ADHD-SN ROIs. Theta shows positive associations with HE in BA 19, 13, and 29, while alpha 1 shows positive associations with all ADHD-SN ROIs, while also showing a negative association with the FSRCQ at BA 13. Table 3 shows the results for posttraining DKEFS scores and their correlations with EEG CSD in ADHD-SN ROIs. BA 23 is the only region to show positive association with the category fluency task. Likewise BA 19 shows significant positive associations with category switching task. The remaining color-word interference scores show both positive and

Table 2. Functional associations between frequency domain current source density levels and subscales of the Integrated Visual and Auditory Continuous Performance Test (IVA+) posttraining

IVA+	FSRCQ	FSAQ	SAAQ	SAVQ	HE
Delta					
BA 19					−.86 (.007)
BA 13					−.81 (.016)
BA 23					−.73 (.042)
BA 25					−.66 (.076)
BA 29					−.77 (.025)
Theta					
BA 19					.66 (.076)
BA 13					.69 (.056)
BA 23					
BA 25					
BA 29					.64 (.086)
Alpha 1					
BA 19					.69 (.056)
BA 13	−.67 (.070)				.78 (.022)
BA 23					.73 (.036)
BA 25					.68 (.060)
BA 29					.71 (.047)

Note. From top to bottom are the Brodmann Areas (BAs) for this study by frequency domain. From left to right: FSRCQ = Full Scale Response Control Quotient, FSAQ = Full Scale Attention Quotient, SAAQ = Sustained Auditory Attention Quotient, SVAQ = Sustained Visual Attention Quotient, HE = Hyperactive Events.

negative associations throughout the ADHD-SN in varying degree based on EEG frequency domain.

Alpha/Theta Relations within the ADHD SN

Table 4 shows the correlations between alpha CSD and theta CSD across the ADHD-SN post and pre-LNFB. There is evidence of a positive association between alpha 1 and theta across all network nodes posttraining that is different at pre-training. The alpha 2 range shows a significant negative association for all network nodes posttraining, which is different than pretraining with an emphasis on BA 13 and 25, respectively.

Discussion

The medial posterior regions of the cortex play a significant role in self-regulation and learning (Cannon et al., 2014). The neural substrates of ADHD remain unclear as does the best neurometric and psychometric data to facilitate a more rigorous and accurate differential. The current data presents a potential focal network (ADHD-SN) that is shown to increase in CSD relative to training one node within it. Functional connectivity also changes as a result of LNFB training and most interestingly, two psychometric measures that show promise in capturing the symptomatic essence of ADHD show positive and negative relationships with nodes within this network. In the most basic sense, if a treatment for ADHD is effective, improvements should be obvious in the primary domains shown to be deficient in ADHD. Similarly, networks of functional connectivity should change relative to the treatment modality and targets for intervention. Additionally as shown in Figure 1, patients were able to produce a positive linear trend in the percentage of time in the reward condition. The current network and outcomes meet these criteria and warrant further research.

LORETA neurofeedback immersive technique presents a unique mechanism to influence EEG CSD within the cortical landscape at posterior medial regions to facilitate

Table 3. Functional associations between frequency domain current source density levels and subscales of the Delis-Kaplan Executive Function System (DKEFS) posttraining

DKEFS	FAS	CAT	Switch	Color	Name	Inhib	Switch
Delta							
BA 19							.66 (.078)
BA 13							
BA 23		.62 (.097)					.75 (.031)
BA 25				.86 (.007)		.71 (.049)	
BA 29							.72 (.043)
Theta							
BA 19				−.81 (.014)		−.66 (.072)	
BA 13				−.77 (.027)			−.63 (.092)
BA 23				−.73 (.041)			
BA 25				−.88 (.004)		−.78 (.023)	
BA 29				−.74 (.038)			
Alpha 1							
BA 19				−.65 (.084)			−.67 (.071)
BA 13							−.83 (.010)
BA 23							−.83 (.011)
BA 25				−.73 (.041)			−.68 (.061)
BA 29							−.84 (.009)
Alpha 2							
BA 19			.63 (.096)			.65 (.049)	
BA 13							
BA 23							
BA 25							
BA 29							

Note. From top to bottom are the Brodmann Areas (BAs) for this study by frequency domain. From left to right: FAS = phonetic fluency, CAT = category fluency, Switch = switching, for the verbal fluency tasks followed by the color-word interference tasks color, naming, inhibition, and switching.

improvements across cognitive and behavioral domains. Several notable effects occurred as a result of LNFB in the precuneus, including improvements in performance on standardized psychometric instruments, increases in alpha CSD in all network nodes influenced by this training protocol, dramatic decrease in the number of errors, and hyperactive events and functional connectivity changes in alpha CSD between these nodes. The functional associations among ROI, EEG CSD, and outcome measure may provide important insight into the theta/beta ratio and alpha deficits or excesses that can be found in persons with ADHD as contrasted with normative samples. Hyperactive events show a positive association with theta and alpha 1 and a negative association with delta across all ROIs in this study. These are interesting findings that are different from the results for pretraining measures and will be published at a later date. Delta, theta, alpha 1, and alpha 2 show positive and negative associations with the DKEFS outcome measures posttraining. It is important to note that these correlations represent improvements in these scores as they correlate with network nodes, which are markedly different from pretraining results. This also adds support to the

Table 4. Post/Pre correlations between alpha current source density (CSD) at the region of interest and theta CSD with all regions in the Attention Deficit/Hyperactivity Disorder-System Network

BA 19	BA 19	BA 13	BA 23	BA 25	BA 29
Post					
alpha 1	0.7*	0.64	0.6	0.69*	0.64
alpha2	−0.79*	−0.7*	−0.74*	−0.76*	−0.77*
Pre					
alpha 1	0.339	0.51	0.42	0.6	0.33
alpha2	−0.72*	−0.62	−0.7*	−0.51	−0.72*

Note. BA = Brodmann Area. *Indicates the correlations are significant or near significance at the .05 level.

notion that there is an interdependence among EEG frequency domains that deserves further study, as can be seen in the analyses for theta and alpha between ADHD-SN ROIs.

This data provide but a first step in understanding the relations between attention and executive processes (self-regulation). There are limitations to the current data, notably the alpha range increases in the regions of interest did not reach statistical significance. However, the clinical and functional measures improved significantly.

Increased sample size would be desired in future studies, and may produce more significant results across all study measures. It would also be of interest to study the ADHD-SN identified in this work across neuroimaging modalities as well as assess the effects of stimulant medications on all measures presented in this study. Additionally, rating scale outcomes may be of interest and should correlate with these objective outcomes. Several of the participants had comorbid diagnoses and thus a "pure" sample of ADHD may provide more substantial insight into these neural mechanisms. This study was not a randomized controlled trial, yet all individuals completed a standardized protocol. We have conducted this same protocol in older nonclinical and clinical groups with similar results (Cannon et al., 2014). Thus the mechanisms utilized in this work clearly warrant further investigation across disciplines.

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