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Randomized Control Study of Neurofeedback With College Students With ADHD

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RANDOMIZED CONTROL STUDY OF NEUROFEEDBACK WITH COLLEGE STUDENTS WITH ADHD

by

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DEDICATION

This work is dedicated to George DeOrdio, who taught me the meaning of hard work and perseverance, and who continues to inspire me today.

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ABSTRACT

Neurodevelopmental disorders such as ADHD represent a major national problem. There are increasing numbers of students in schools requiring special education services as a result of ADHD, and each of these students costs the U.S. education system approximately $5,000 per year (Robb et al., 2011). There are additional societal costs associated with the disorder, and ADHD can be debilitating for individuals with the disorder and their families (i.e., Barkley & Murphy, 2010; Ginsberg, et al., 2013). The most common treatments are stimulant medication and behavioral training (i.e., Pelham & Fabiano, 2008), but recently neurofeedback (EEG biofeedback) has been receiving a lot of press. Both the American Academy of Pediatrics and the American Academy of Child and Adolescent Psychiatry have endorsed neurofeedback as a viable option for the treatment of ADHD (AAP, 2012; Lofthouse, et al., 2012). Methods: The current study is a randomized controlled study investigating the effects of LORETA neurofeedback on a college population with ADHD. The study used a pre-test, multiple post-test design with delayed treatment to provide stronger evidence of its effectiveness. Both qEEG and behavioral data were collected to determine if there were changes in brain activity, and if these changes were evident on popular measures of cognitive ability (i.e., Woodcock-Johnson III) and attention (CPT-II). Results: The results indicated that following LORETA neurofeedback treatment, participants exhibited significant changes in z-score qEEG coherence within the prefrontal cortex. These changes were also related to changes in performance on a verbal working memory measure, which approached significance.

Finally, the results suggested that 25 sessions of LORETA NF are needed to affect meaningful change.

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CHAPTER 1

INTRODUCTION

Neurodevelopmental disorders such as attention deficit hyperactivity disorder (ADHD) represent a major national problem. First, there is a high prevalence of these disorders in schools. In fact, according to the US Census Bureau's American Community Survey (ACS, 2006; Brault, 2008), an estimated 6.3% of children ages 5-15 have a disability, which amounts to 2.8 million children in the United States. Of those, many are children who are diagnosed with ADHD, and receive special education services under the Individuals with Disabilities Education Improvement Act (IDEIA 2004). There are increasing numbers of students in schools requiring special education services as a result of this disorder, and each student diagnosed with ADHD costs the U.S. education system an average of approximately $5,000 per year versus students without, who cost, on average, approximately $300 each (Robb, Sibley, Pelham, Foster, Molina, Gnagy, et al., 2011). Additionally, some studies have shown a relationship between ADHD and societal costs such as those related to criminality and accidents (Bernfort, Nordfeldt, & Persson, 2008; Ginsberg, Långström, Larsson, & Lichtenstein, 2013; Matza, Paramore, & Prasad, 2005). In fact, the annual societal cost of an individual with ADHD is close to $15,000 (Pelham, Foster, & Robb, 2007). In addition to financial and societal concerns, ADHD can be debilitating for individuals with the disorder. While behavioral therapies and stimulant medications have historically demonstrated success for the treatment of ADHD, both treatments have also demonstrated differential effectiveness in terms of identifying

responders and non-responders (i.e., Elliott, et al., 2014; Lauth, Minsel, & Koch, 2015). Finally, drug therapies can be risky, particularly considering the impacts of long-term use (i.e., Wang, et al., 2013), suggesting a need for more and better treatment options.

Diagnosis and Impairment

According to the current *Diagnostic and Statistical Manual of Mental Disorders* (DSM-V; APA, 2013), the key feature of ADHD is a persistent pattern of behavior (i.e., inattention, hyperactivity and/or impulsivity) that is developmentally inappropriate. Historically there are three prominent theories of ADHD—Barkley's Behavioral Inhibition Model, the ADHD/I-ADHD/C dichotomy, and more recently, a working memory model—that attempt to explain the underlying causal mechanisms of the disorder (Barkley, 2003; Kofler, et al., 2010; Milich, et al., 2001; Raiker et al., 2012). Regardless of the theory to which one subscribes, all three include an emphasis on cognitive processing as a primary concern of the disorder. As cognition is an important facet of everyday life, it is not surprising that individuals with ADHD experience impairment in many areas. Indeed, the DSM-V criteria require clinically significant impairment in daily functioning (e.g., social, academic, occupational functioning) across two or more settings.

One way in which this manifests is in social functioning. Children and adolescents with ADHD often experience social isolation, in that they are often rejected by their peers (McConaughy, Volpe, Antshel, Gordon, & Eiraldi, 2011). Research has also demonstrated that children with ADHD tend to have lower academic achievement performance than their non-ADHD peers (Frazier, Youngstrom, Glutting, and Watkins, 2007; McConaughy, et al., 2011). Furthermore, studies have demonstrated that the

difficulties associated with ADHD continue into adulthood (Barkley, Murphy, & Fischer, 2010; Buitelaar, Kan, & Asherson, 2011). For instance, two meta-analyses found that individuals with ADHD tend to have lower rated self-esteem, lower educational outcomes, lower occupational status and job performance ratings, as well as less job stability than their non-ADHD peers. Additionally, individuals with ADHD consistently have higher medical billing costs, and are at elevated risk for developing comorbid psychiatric disorders, most notably substance use disorders (Bernfort, et al., 2008; Matza, et al., 2005).

As previous research has shown, although the characteristics of the disorder may change as an individual ages, ADHD can still impact an individual's daily life into adulthood (Barkley & Murphy, 2010; Barkley, Murphy, & Fischer, 2010; de Graaf et al., 2008; Halmoy, Fasmer, Gillberg, & Haavik, 2009; Painter, Prevatt, & Welles, 2008). In fact, symptoms of ADHD are estimated to affect five to eight percent of the general population across the lifespan (Goldstein, 2011), and two to eight percent of college students (Fleming & McMahon, 2012). As ADHD is often diagnosed in childhood, the majority of the literature base is focused on children. Although recently there has been a shift towards researching ADHD in adults, there is still a lack of well-established research for college students with the disorder (Fleming & McMahon, 2012). Adolescence and emerging adulthood are times of great personal growth and identity development, which often times is accompanied by experimentation (Schlegel, 2012). As such, risk-taking behaviors are often studied in adolescent and college populations. Moreover, research has demonstrated a link between risk taking behaviors and executive functions in this young-adult population (Pharo, Sim, Graham, Gross, & Hayne, 2011;

Steinberg, 2007). Executive dysfunction, specifically, impulsivity, is also one of the core cognitive deficits associated with ADHD. Thus, if non-clinical populations of college students demonstrate an affinity towards risky behaviors, and individuals with ADHD are at increased risk for engaging in risky behaviors, college students with ADHD are at even higher risk. Specifically, inattention and poor impulse control may be highly problematic for college students with ADHD, who endure long lectures, must organize their time and effort to study, and are exposed to a wide variety of risky situations, such as those associated with drinking and sexual activity (Barkley et al., 2002; Weyandt & DuPaul,2008).

Current Treatment

Like many mental health disorders, there is no cure for ADHD. Currently, the two most widely accepted treatments for ADHD are stimulant medication and behavioral modification, or some combination thereof (National Dissemination Center for Children with Disabilities [NICHCY], 2011; National Institute of Mental Health [NIMH], 2008). In 2008, Pelham and Fabiano reviewed 46 studies that evaluated a variety of behavioral evidence-based interventions (EBI) for ADHD, concluding that behavioral parent training, behavioral classroom management, and behavioral peer interventions (i.e., social skills training) are well-established EBIs for ADHD. Additionally, the National Registry of Evidence-based Programs and Practices lists four empirically supported (behavior-based) treatment programs for ADHD; however, none of these interventions have been researched with a college population.

ADHD and Neuroimaging

Furthermore, disorders such as ADHD are neurological in nature. In fact, several studies utilizing positron emission tomography (PET), magnetic resonance imaging (MRI), and functional magnetic resonance imaging (*f*MRI) have demonstrated structural and/ or functional brain differences in individuals with ADHD (i.e., Castellanos, Giedd, Marsh, Hamburger, Vaituzis, & Dickstein, 1996; Fonseca, Tedrus, Moraes, Machado, Almeida, & Oliveira, 2008; Koehler, Lauer, Schreppel, Jacob, Heine, Boreatti-Hümmer, et al., 2009; Monastra, Lubar, Linden, VanDeusen, Green, Wing, et al., 1999; Semrud-Clikeman, Steingard, Filipek, Biederman, Bejjen, Renshaw, 2000; and Vaiyda, Bunge, Dudukovic, Zalecki, Elliott, & Gabrieli, 2005).

One such PET study examined dopamine transporter (DAT) dysregulation in adults with ADHD (Spencer et al., 2007). The sample consisted of 47 adults (21 clinical, 26 control), and the final analyses were corrected for age, as the non-clinical group was significantly younger than the clinical group. After correcting for age, the results of the study suggested that DAT binding was 15% greater in the right caudate for the ADHD group than the control group
($t = 7.7$, $df = 45$, $p = .008$). Additionally, given that sex can moderate DAT binding, the authors reanalyzed the data simultaneously controlling for both age and sex. The results suggested an even larger effect in the right caudate, with 17% greater DAT binding in males ($t = 6.9$, $df = 24$, $p = .02$) and 22% in females ($t = 7.3$, $df = 21$, $p = .02$).

Similarly, a meta-analysis of 21 MRI studies of children (ages 9-14) with ADHD, found a number of structural differences (Valera, Faraone, Murray, & Seidman, 2007). Across these 21 studies, the most frequently assessed variable was total cerebral volume,

which was measured in 8 studies, and demonstrated global volumetric reduction across studies. Indeed, there were 9 regions of interest—including total cerebral volume —that demonstrated significant change between ADHD and control subjects across 3 or more studies, and another 6 areas, which demonstrated significant differences between the clinical group and controls in at least 2 studies. The areas found to be impacted the most included: total cerebral volume, the corpus callosum, caudate, and cerebellum, as well as the prefrontal cortex, frontal lobes, and deep frontal white matter. Notably, the right caudate, which was implicated in the PET study described above, was found to demonstrate significant standardized mean differences in 6 of the 21 studies.

Another more recent review article (Friedman & Rapoport, 2015) supports numerous structural differences between individuals with ADHD and controls. Similar to the 2007 meta-analysis, this study reports volumetric loss in the right striatum as a key feature of ADHD. In addition, the authors cite other studies, which have shown significant volume loss in the prefrontal cortex (PFC), parieto-temporal areas, basal ganglia, and cerebellum (i.e., Nakao, et al., 2011; Valera, et al., 2007). Lastly, the authors cite atypical brain development, which affects attention, cognitive control, and working memory processes. This is consistent with the previous meta-analytic study, suggesting that structural changes are particularly notable in the cerebellum, PFC, and right hemisphere for individuals with ADHD.

Finally, a recent meta-analysis was conducted, comparing 55 *f*MRI studies of individuals with ADHD (Cortese, Chabernaud, Proal, Di Martino, Milham, & Castellanos, 2012). Of these, 39 studies focused on children, and the other 16 examined adults with ADHD. The authors used activation likelihood estimation for the meta-

analysis. Results indicated that children with ADHD exhibited bilateral hypoactivation in the frontal regions and putamen, as well as the right parietal and temporal regions, and hyperactivation in the right angular gyrus, middle occipital gyrus, posterior cingulate cortex, and midcingulate cortex. Results of the adult analyses indicated hypoactivation in the middle frontal gyrus, right central sulcus, and precentral gyrus, and hyperactivation in the right angular gyrus and middle occipital gyrus. This suggests that both regional hypoactivation and hyperactivation persist into adulthood for individuals with ADHD. At minimum, frontal hypoactivation and hyperactivation of the right angular gyrus and middle occipital gyrus seem to exist in both child and adult clinical populations when compared to non-ADHD peers.

ADHD and QEEG

Overwhelmingly, PET, MRI, and *f*MRI research has suggested the existence of both structural and functional differences in the brains of individuals with ADHD. Studies examining differences between ADHD and non-ADHD populations using EEG have paralleled these results, thus demonstrating the utility of EEG and/or quantitative EEG (qEEG) in diagnostic clinical evaluations for ADHD. For example, Fonseca and colleagues (2008) demonstrated differences in electroencephalographic activity between children with ADHD and age-matched controls during an eyes closed resting state. Data was recorded from 15 electrode sites in this study, and the authors examined absolute and relative power across the frequency bands. First, the ADHD group exhibited greater absolute power in delta and theta bands across the brain. Second, this group exhibited greater absolute power in beta around the midline (i.e., C3, F4, C4 F0, C0, and P0). Third, the ADHD group exhibited smaller relative power in the alpha 1 and beta bands at

certain electrode sites (i.e., O1, F4; T6 respectively). Overall, the study found that qEEG provided 83.3% sensitivity and 83.3% specificity in the diagnosis of ADHD. Another study examined EEG differences in adults with ADHD (Koehler, et al., 2009). Koehler and colleagues (2009) recorded data from 21 electrodes, and also examined absolute power densities. In this study, the ADHD sample exhibited increased absolute power in the alpha and theta bands, with no differences in beta. This suggests that the patterns of activation, while still abnormal, may change as individuals age.

A 2015 study (Snyder, et al., 2015) examined the integration of EEG markers (i.e., theta/beta ratio) with clinical judgment in the diagnosis of ADHD. This study was a triple-blinded prospective study. 275 children and adolescents with attention and behavioral problems were evaluated at 13 sites. Each of these sites had a qualified clinician who completed differential diagnosis evaluations. A separate multidisciplinary team comprising a psychiatrist, psychologist, and neurodevelopmental pediatrician completed an independent consensus evaluation. Finally, separate teams collected EEG data at each site. Clinicians identified 209/275 subjects as having ADHD. The multidisciplinary team identified 93 less. However, 85 of these 93 also exhibited EEG characteristics of ADHD (i.e., lower theta/beta ratio). Overall, the results of the study indicated that the integration of EEG markers with clinical judgment could significantly improve diagnostic accuracy by 61 to 88%.

Finally, an alternative qEEG methodology—wavelet synchronization—has been proposed as a new approach to diagnosing ADHD using EEG (Ahmadlou & Adeli, 2010). In this study, the authors used nonlinear modeling to identify functional connectivity deficits in a sample of children ages 7-12 (n = 47 ADHD, n = 7 control).

Similar to the results of the fMRI studies described above, this suggests that the distinction between ADHD and non-ADHD populations extends beyond structural differences and into differences in function, specifically in terms of brain connectivity. Specifically, the results of the study indicated that O2 and P4 theta, as well as T5 delta exhibited significant differences in connectivity between the groups, suggesting deficits in visual and auditory processing as well as data integration. This is consistent with the results of a 2012 study (Ahmadlou & Adeli, & Adeli, 2012) in which the authors found that differences in left-hemisphere connectivity, within the delta range, could differentiate between ADHD and non-ADHD individuals. This methodology has also been used in the diagnosis of epilepsy and seizure disorders (Faust, Acharya, Adeli, & Adeli, 2015) demonstrating again that qEEG is a useful diagnostic tool for a number of neurologically-based psychological disorders, which are summarized in Table 1.1.

QEEG as Treatment for ADHD

One of the greatest benefits of qEEG is that it provides not only a means of identifying disorders but it can also be extended for use in treatment. One area of research, neurofeedback (NF), does just that. In fact, NF has shown great promise in treating neurodevelopmental conditions, because it purportedly directly impacts brain functioning. Some researchers claim it is based on the scientific foundation of operant learning, where behavior is increased or decreased based on the consequences of behavior (e.g., Sterman, 2000, Thatcher, 2000) while others claim it is a form of self-regulation training (e.g., Decker, Roberts, & Green, 2014; Johnston, et al., 2010). Regardless of the theoretical underpinnings to which one subscribes., NF uses electroencephalography (EEG) to monitor cortical activity by placing small electrodes on

the scalp, via a fabric cap, or geodesic net. These electrodes detect very small electrical currents that are then amplified and recorded with the use of a computer. Like other forms of biofeedback, the subject is then provided with feedback (i.e., visual and/or auditory stimuli) contingent upon the brain activity detected. Low-resolution brain electromagnetic tomography (LORETA) is a more advanced form of NF that extends surface EEG NF. It works by using data from 19 (or more) electrodes to localize cortical and subcortical current densities.

For example, the dorsal and ventral attention networks, as well as the default mode network (Janssen et al., 2015; McCarthy, et al., 2014), have been implicated in ADHD. Each of these networks involves multiple areas of the brain. For example, the dorsal attention network includes the intraparietal sulcus and frontal eye fields (i.e., Brodmann Area [BA] 8), whereas the ventral attention system is made up of the ventral frontal cortex (i.e., BA 44, 45, 47) and the temporoparietal junction (Vossel, Geng, & Fick, 2014). The Default Network, on the other hand, is a much more vast and diffuse network, which comprises Brodmann Areas 8, 9, 10, 21, 23, 24, 28, 29, 30, 31, 32, 36, 39, and 40 (Buckner, et al., 2008; Thatcher, North, & Biver, 2014). Although these areas are oft associated with ADHD, research has demonstrated that other areas of the brain are also related to attentional difficulties (i.e., BA 10, 11, 22, 23, and 24; Gitelman et al., 1999). In looking at all of the possible brain regions, which could be impacted by ADHD, it becomes increasing important to examine the needs of each individual.

The use of LORETA enables the user to identify both dorsal (i.e., cortical) and ventral (i.e., subcortical) Brodmann areas that are exhibiting atypical patterns of activation, based on a normative sample, which allows for targeted and individualized

training protocols. One program, Neuroguide, compares individuals to a normative sample of 625 participants (ages birth – 82 years). The Neuroguide software (through which LORETA is available) also exports data on both raw EEG and Z-score metrics, such that areas of atypical activation (i.e., those with $Z \geq 2$) can be trained towards typical activation (i.e. $Z = 0$). Overall, the power of LORETA to generate inferences about, and to train subcortical areas, has the potential to greatly extend the scope and efficacy of NF.

Neurofeedback in Clinical Practice

Neurofeedback is a biofeedback technique that facilitates self-awareness and behavioral control by making the electrical activity of a person's brain activity observable on a computer screen. Although a relatively new technique, many research studies have supported the efficacy of NF for the treatment of children with neurodevelopmental disabilities in learning or attention (i.e., Arns, de Ridder, Strehl, Breteler, & Coenen, 2009; Beauregard & Levesque, 2006; Breteler, Arns, Peters, Giepmans, & Verhoeven, 2010; Gevensleben, Holl, Albrecht, Schlamp, Kratz, Studer, et al., 2010; Gevensleben, Holl, Albrecht, Vogel, Schlamp, Kratz, et al., 2009; Levesque, Beauregard, & Mensour, 2006; Lofthouse, Arnold, Hersch, Hurt, & DeBeus, 2012; Logemann, et al., 2010; Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Walker, 2010).

For example, three recent studies have supported the use of NF for the treatment of ADHD in both children and adults. The first examined changes across three groups of children (ages 6-18) with ADHD (Duric, Assmus, Gundersen, & Elgen, 2012). In this study, the authors compared three groups of children—those receiving NF only (n = 23), those receiving only methylphenidate (n = 29), and those receiving a combination of both

treatments (n = 24). The NF training in this study was operationalized as thirty 40-minute sessions, thrice weekly. The sessions consisted of 5 minutes of baseline (i.e., alpha training), followed by 30 minutes of beta/ theta NF training, and finally another 5-minute baseline at the end. Results indicated significant symptom reduction for all three treatment groups, based on parent report. Additionally, although significant differences were not found between the treatment groups, it is notable that the NF-only group exhibited more than twice the pre-test post-test change in attention. Overall, the authors concluded that NF treatment is as effective as methylphenidate treatment in the reduction of ADHD symptoms in children, based on parent-report.

A second study examined the activation of the Default Mode Network (DMN) in 12 children (age 9-15) with ADHD (Russell-Chapin, et al., 2013). All of the children were taking stimulant medication throughout the duration of the study, and participants were randomly assigned to receive NF treatment or no treatment [in addition to their medication regime]. The treatment group received 40 sessions of NF training over the course of 92 days. Results indicated that NF treatment resulted in both a reduction of clinical symptoms as a well as a consolidation (i.e., appropriate activation) of the DMN. The DMN was more consolidated in the treatment group than the control group, suggesting the NF treatment impacted the consolidation beyond what time alone would cause.

A third study, examined the effects of NF treatment of a group of 18 children (M_{age} = 13.6 years) with ADHD (Hillard et al., 2013). In this study, 12 of the participants (66.67%) were taking stimulant medication throughout the duration of the study. Participants each completed 12 weekly sessions, consisting of 25 minutes of NF training

each, which consisted of a “Focus/Alertness" protocol through the program Peak Achievement Trainer®. ANOVA results indicated main effects both between (i.e. session 1 to session 12) and within sessions (i.e., from minute 1 to minute 25). Specifically, the theta/low beta and theta/alpha ratios decreased significantly from pretest to posttest as well as from the start of a session to the end of that same session. Additionally, these changes generalized to participant’s performance on commonly used behavioral measures (i.e., IVA+, Aberrant Behavior Checklist [ABC]) from pretest to posttest. Participants’ performance on the IVA+ indicated significant changes on 10 metrics, including both visual and auditory attention metrics. Finally, parent report on the ABC showed a significant decrease in behavior problems (e.g., hyperactivity) from pre-test (15.28 ± 3.24) to post-test (10.83 ± 2.44); $t(17) = 3.189$, $P = .005$.

On the other hand, there are have been some studies, which have not supported the efficacy of NF for the treatment of ADHD. One such study (Ogrim & Hestad, 2013) was a randomized pilot study of 32 medication-naïve children (ages 7-16) with ADHD. Sixteen children received 30 sessions of 45 minute NF training (over 7-11 months), and the other group received either methylphenidate or dextroamphetamine. Both behavioral data (i.e., parent/teacher rating scales) and EEG data was collected. The results of the study indicated that neither treatment exhibited significant changes in qEEG or ERP. Additionally based on parent/ teacher ratings, the medication group exhibited significant symptom reduction while the NF group did not.

Similarly, Vollebregt and colleagues (2013) completed a double-blind placebo-controlled study of 41 children (ages 8-15) with ADHD using individualized NF protocols. The results of this study indicated mixed results. First, no group differences

were found on several neurocognitive measures (e.g., digit span, sustained visual attention). Second, although participants all demonstrated significant improvement on at least one metric, they all also demonstrated deterioration on at least one measure. As such, the study did not support the use of NF as a treatment for ADHD.

Although there is some conflicting evidence, most studies generally support the use of neurofeedback in the treatment of ADHD. Furthermore, previous research has suggested that NF is effective for treating a variety of neuropsychiatric disorders, such as, anxiety disorders (Moradi, Pouladi, Pishva, Rezaei, Torshabi, & Mehrjerdi, 2011) including obsessive-compulsive disorder (Sürmeli, & Ertem, 2011); depression (Baehr, Rosenfeld, & Baehr, 2001; Choi, Chi, Chung, Kim, Ahn, & Kim, 2011); autism (Jarusiewicz, 2002; Kouijzer, de Moor, Gerrits, Buitelaar, & van Schie, 2009); and schizophrenia (Sürmeli, Ertem, Eralp, & Kos, 2011). However, many of the previously published studies have methodological limitations that prevent a clear understanding of the efficacy of the technique (Loo & Barkley, 2005). More recently, Meisel and colleagues (2013) completed a randomized control trial comparing the long-term effects of NF versus stimulant medication. The study included 23 children who were randomly assigned to either a methylphenidate pharmacological intervention, or 40 sessions of NF, twice per week for approximately 35-minute sessions. Data was collected at pre-test and post-test, as well as a 3-month and 6-month follow-up after completion of the study. Results suggested that the NF group exhibited a significant reduction in symptoms (i.e., hyperactivity, impulsivity, inattention), a significant reduction in functional impairment, and a significant improvement in academic performance (i.e., writing, math), at the 3- and 6-month follow-ups. Additionally, this group exhibited and a significant

improvement in oppositional defiant (OD) behaviors at the 3–month follow-up, though not at 6 months post-treatment. The methylphenidate group exhibited similar core symptom reduction at 3- and 6-month follow-ups in addition to a reduction in functional impairment and OD symptoms at 6-months. In directly comparing the two groups, the authors found no significant differences between the groups; however, the results were confounded as many of the NF group participants (N = 8) began a medication regime prior to the 6-month follow-up. This suggests that in spite of the vast improvements in recent years, there is still a great deal of research needed in this area.

LORETA Neurofeedback

Several studies have examined more sophisticated approaches to using NF. One approach is LORETA, which can provide more localized targeting of brain regions in comparison to surface EEG. One study found that LORETA NF appeared to strengthen connectivity, and improve functioning in a nonclinical population (Cannon, et al., 2009). Another study, examined the utility of LORETA NF with an ADHD population (Koberda, et al., 2014). This study, an in-depth case study, demonstrated the impact of LORETA NF on both qEEG (i.e., reduction of excessive beta) and behavioral data (i.e., computerized neurocognitive assessment) metrics.

However, few NF studies using LORETA under randomized control conditions have been completed, and none to date have used a delayed treatment design. A delayed treatment design can provide stronger evidence in support of the effectiveness of a given intervention if both groups demonstrate change in the expected direction. Additionally, a delayed treatment design is often considered more ethical than a waitlist design, particularly when an effective treatment is being withheld. In fact, because of the great

potential of NF as a therapeutic option, in 2011, the American Academy of Pediatrics (AAP) listed NF as promising but in need of more research. However, in 2012, the AAP elevated NF to a "Level 1-Best Support" for intervention for attention and hyperactivity behaviors. Each year the AAP releases a report of evidence-based interventions (EBIs) for a variety of disorders in childhood and adolescence. The level designations were adapted from the American Psychological Association's (APA) Task Force on Promotion and Dissemination of Psychological Procedures. A Level-1 designation means that a given intervention has shown efficacy in at least two randomized trials by at least two different research teams.

Although NF has been recently gaining support as an empirically based intervention, more research is needed, particularly in terms of LORETA NF. Previous research (i.e., that reviewed by the American Academy of Pediatrics) has focused on surface NF, which has been demonstrated to be effective after several sessions (i.e., 60 sessions; Koberda, et al. 2014).

The current study, on the other hand, was one of the first investigations of LORETA neurofeedback using a randomized control research design with a placebo (or sham) condition for the treatment of learning and attention problems. As described above, LORETA neurofeedback is a more sophisticated NF technique intended to generate inferences about sub-cortical structures, with the goal of training these areas. It is notable that while LORETA can target subcortical structures, it is still largely based on Brodmann areas, which are by definition, cortical regions. However, by targeting these subcortical structures, it should allow for more targeted, and thus faster and/or more effective results (Simkin, Thatcher, & Lubar, 2014; Wigton & Krigbaum, 2014).

The Current Study

The current study aimed to overcome the methodological limitations of past research (see Loo & Barkley, 2005), and to extend this research with a delayed treatment design in order to provide a better understanding of the effectiveness of LORETA NF. Specifically, the delayed treatment design provided a unique opportunity to evaluate the dose-response rate of NF LORETA NF. Previous research suggests 20-50 sessions are needed to demonstrate change (Arns et al., 2009; Holtmann, et al., 2009, Holtmann, et al., 2014). LORETA NF may produce results in less time because it enables the clinician to target select areas of impairment, which are consistent with a specific set of symptoms and related brain regions (Simkin, Thatcher, & Lubar, 2014). Furthermore, with its focus on ADHD in college students, the study will contribute to the literature on the disorder with this population.

The major objective of this study was to test the effects of individualized LORETA NF in college students who experience difficulties as a result of ADHD. This was evaluated through three specific aims:

1. To test the hypothesis that LORETA NF can change brain activity in a sample of college students with ADHD.
2. To test the hypothesis that changes in brain wave activity as a result of LORETA NF, correspond to changes on behavioral tests of cognitive abilities (i.e., WJ-III and CPT subtests);.
3. To test the hypothesis that LORETA NF demonstrates faster changes than traditional surface NF (i.e., changes occur prior to 20 sessions).

These specific aims were then assessed according to the following hypotheses, which are summarized in Figure 1.1:

- Treatment vs. Sham (sessions 1, 10, 25)
    - Hypothesis 1: Students in both conditions will show equivalent impairment at pretest as measured by behavioral measures of cognitive ability and baseline qEEG;
    - Hypothesis 2: Students in the NF condition will demonstrate greater change toward normality than students in the sham condition at session 10, as measured by:
        a) Greater qEEG change toward normality (i.e., Z = 0);
        b) Better performance on widely used behavioral measures of cognitive ability;
    - Hypothesis 3: Students in the sham condition, after session 10, will begin to demonstrate changes in brain activity and cognitive performance similar to that of students in the NF condition at session 25, as measured by:
        a) Both groups exhibit qEEG change toward normality (i.e., Z = 0);
        b) Both groups exhibit improved performance on widely used behavioral measures of cognitive ability;

- Dose-Response Relationship (sessions 15, 20, 25)
    - Hypothesis 4: Students in NF condition will demonstrate a continual pattern of change toward normality from sessions 15 to 25, as compared to students in the sham condition at session 10 (i.e., maximum placebo effect).

Table 1.1 Studies in which EEG/qEEG metrics linked to diagnosis of other disorders

Disorder	Studies
Alzheimer's Disease	• Adeli, Ghosh-Dastidar, & Dadmehr (2008) • Gawel, Zalewska, Szmidt-Sałkowska, & Kowalsi (2009) • Herrmann & Demiralp (2005)
Antisocial Personality Disorder	• Calzada-Reyes, Alvarez-Amador, Galán-García, Valdés-Sosa (2012)
Autism	• Ahmadlou & Adeli (2014) • Cantor & Chabot (2009) • Sheikhani, Behnam, Mohammadi, Noroozian, & Mohammadi (2012)
Epilepsy/ Seizure Disorders	• Leach, Stephen, Salveta & Brodie (2006) • McGonigal, Oto, Russell, Greene & Duncan (2002) • Croona, Kihlgren, Lundberg, Eeg-Olofsson & Edebol- Eeg-Olofsson (1999) • Faust, Acharya, Adeli, & Adeli (2015) • Mormann, Lehnertz, David & Elger (2000)
Learning Disabilities	• Cantor & Chabot (2009) • Rocha, Massad, Thomaz, & da Rocha (2014)
Mood Disorders	• Begić, et al. (2011) • Koek, et al. (1999)
Schizophrenia	• Boutros, et al. (2008) • Knyazeva, et al. (2008)
Traumatic Brain Injury	• Bozorg et al. (2010) • Duff (2004) • Roberts, Englund, & Scherr (2011) • Ronne-Engstrom & Winkler (2006) • Thatcher et al. (2001) • Thatcher, et al. (1989)

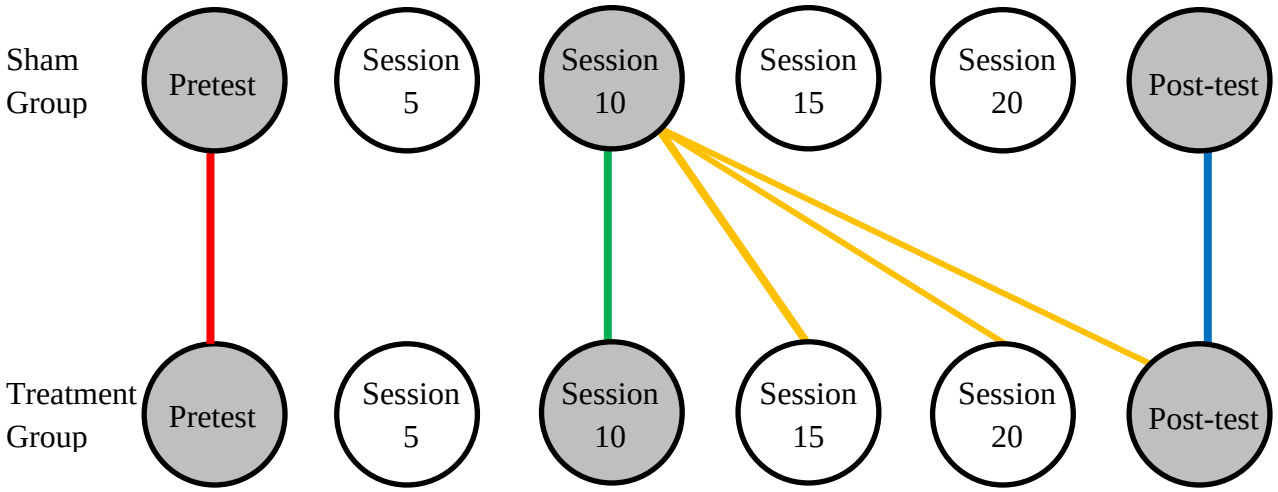

Figure 1.1 Study Hypotheses. This figure illustrates the four study hypotheses. Red = hypothesis 1 (equivalence at pretest), green = hypothesis 2 (group differences at session 10), blue = hypothesis 3 (group differences at post-test), orange = hypothesis 4 (dose-response relationship).

CHAPTER 2

METHODS

Participants

Participants for this study included 16 college undergraduates who were documented as having a diagnosis of ADHD. Participants were recruited through the university's participant pool, flyers posted around campus, newspaper advertising, and word-of-mouth. Each participant was randomly assigned to a treatment condition. Eight participants were randomly assigned to the sham (placebo) condition and eight participants were randomly assigned to the neurofeedback (NF) treatment condition. Participants received course credit for the initial screening process, and $125 for the completion of the sessions.

Inclusion/ exclusion criteria. In order to qualify for the study, participants were required to complete screening questionnaires documenting their diagnosis of ADHD. These included questions regarding the age at which they were diagnosed, the type of professional that made the diagnosis, possible comorbid conditions, and self-reported symptoms, as well as providing documentation regarding any stimulant medication they were currently prescribed. Additionally, they completed a baseline QEEG, and using the Neuroguide symptoms checklist, it was determined if there were matches between reported symptoms and QEEG abnormalities. Only those students who exhibited QEEG abnormalities consistent with ADHD continued in the study. As the study took place

during the academic year, it was not practical to ask students to discontinue their current treatment plan; however, an attempt was made to covary medication status.

Participant attrition. The use of college students allowed for easy access to follow-up with participants as well as to replace them as the need arose. Additionally, participants received course credit (or extra credit) for participation in research as well as remuneration in the hopes of retaining as many participants as possible. Using a college population and having multiple sources of compensation greatly increased the potential to successfully recruit participants and to complete the study within a reasonable time frame. However, twenty-six participants were enrolled in the study, of which 10 were replaced due to time constraints, personal matters, and/or poor attendance to reach the projected 16 participants.

Measures

Screening. Participants completed an online screener in order to determine initial eligibility. This screener included demographic information, questions regarding their diagnosis, and questions about past and current symptomatology. Demographic questions were included in order to assess pre-test group equivalency, and to evaluate possible covariates in later analyses. Participants were asked if they have a documented diagnosis of ADHD, which was confirmed by other assessments measuring symptom severity.

Symptom severity. In order to assess symptomatology, two published measures were adapted for use online. The first was Barkley's Current Symptoms Scale—Self-Report Form (BCSS; Barkley & Murphy, 2006), which provides a measure of self-reported ADHD symptoms. This scale has 36 items, which represent DSM-IV-TR diagnostic criteria for ADHD (APA, 2000). The first 18 items are alternating symptoms

of inattention and hyperactivity/impulsivity. The remaining 18 items assess settings of impairment, and comorbid symptoms of oppositional defiant disorder. Individuals were asked to respond on a Likert-type frequency scale, ranging from 0 (never/rarely) to 3 (very often).

Although psychometric evidence for the BCSS is reported infrequently, there have been a few studies to validate the measure with adults. For instance, the scale has been demonstrated to discriminate moderately well between ADHD and non-ADHD populations. Quinn (2003) found that the inattention symptoms had 75% sensitivity, and 61% specificity, while the hyperactivity/impulsivity symptoms had 69% sensitivity and 39% specificity. While these estimates are lower than desirable, it prompted additional research into the psychometric properties of the BCSS. Most recently, Ladner, Schulenberg, Smith, and Dunaway (2011) examined the reliability and validity of the scale with more than 600 university students. Ladner and colleagues reported moderately high internal consistency coefficients for both inattention (Cronbach's $\alpha = .88$) and hyperactivity/impulsivity (Cronbach's $\alpha = .82$). Additionally, Cronbach's $\alpha = .91$ for the entire scale. The study also investigated concurrent validity of the BCSS with the Conner's Adult ADHD Rating Scales-Self Report Long Form, and the Adult Attention Deficit Disorders Evaluation Scale. Results indicated that the BCSS correlated moderately to highly ($r = .58 - .87$) with both of these measures. Although ideally, measures would demonstrate consistently higher reliability and validity coefficients (i.e., above .7), this measure is based on the DSM-IV-TR criteria, thus making it clinically relevant.

The second scale, the Barrett Impulsiveness Scale (BIS; Patton, Stanford, & Barratt, 1995), is a 30 item, self-report measure, which provides an assessment of impulsivity. Now in its eleventh edition, the BIS has been used extensively in research and clinical practice for more than 50 years, and is arguably the gold standard for measuring symptoms of impulsivity (Malloy-Diniz, Fuentes, Leite, Correa, & Bechara, 2007; Stanford, Mathias, Dougherty, Lake, Anderson, & Patton, 2009). Factor analyses of the current edition of the BIS, identified six sub-traits of impulsivity—attention, cognitive complexity, cognitive instability, motor, perseverance, and self-control—that are subsumed under the three second order factors of attention, motor, and non-planning. Impulsivity is a symptom of ADHD, and as such was of relevance to the current study, particularly as the population of interest is college students. To complete the scale, individuals were asked to respond on a Likert-type frequency scale ranging from 1 (never/rarely) to 4 (almost always/always).

Stanford et al. (2009) provide the most recent and comprehensive evaluation of the psychometric properties of the BIS-11. In fact, the study included measures of reliability and validity for total scores, as well as for each of the first and second order factors. Internal consistency (Cronbach's α = .83) and test retest reliability (Spearman's Rho = .83) were acceptable for the entire scale; however, the reliability estimates were lower for the individual factors (Cronbach's α = .27 - .74, Spearman's Rho = .23 - .74). Intercorrelations among the subscales were also reported, ranging from r = .16 - .91. In addition to reliability, the authors evaluated the concurrent validity of the measure with four other impulsivity scales (e.g., Zuckerman Sensation Seeking Scale, Eysenck Impulsiveness Scale, Behavioral Inhibition/Activation Scales, and Behavioral Measures

of Impulsiveness). The BIS-11 significantly correlated (r = .10 - .63) with all but the last of these, though that study included a solely non-clinical population.

These measures were used to confirm diagnosis and to assess symptom severity, not to determine inclusion for the study. As neither of these measures were intended for diagnostic purposes, a cut-score was not used. Instead, scores were used on a continuum to indicate symptom severity for both groups. The original demographic survey and copies of both screening measures are included in Appendix B.

Outcome measures.

Quantitative electroencephalography. EEG and qEEG have demonstrated a high degree of both reliability and validity in the medical field, and more recently in psychology. There are several types of measurements within the context of qEEG, though only absolute power and coherence will be analyzed in this study. Absolute power refers to the amount of voltage recorded within each band (i.e., delta, theta, alpha, beta, gamma), and has been extensively researched, and demonstrated to be highly reliable (i.e., r ≥.9) for both split-half and test- retest reliability (Thatcher, 2010). Coherence on the other hand, refers to the communication between brain regions. Coherence has also been shown to be reliable (r ≥.8) across several studies (i.e., Corsi-Cabrera, Solís-Ortiz, Guevara, 1997; Corsi-Cabrera, Galindo-Vilchis, del-Río-Portilla, Arce, & Ramos-Loyo, 2007; Thatcher et al.,1986) with some studies reporting reliability coefficients as high as r = .95 (Fernández, Harmony, Rodríguez, Reyes, Marosi, & Bernal, 1993; Corsi-Cabrera et al., 2007). With regards to validity, less research has examined absolute power or coherence alone, but qEEG has been shown to have high sensitivity (as high as 96%) for correctly identifying individuals with post-concussion syndrome disorder (Duff, 2004).

Additionally, in 2001, Thatcher and colleagues also developed a discriminant function to classify traumatic brain injury patients based on symptom severity, which was validated based on its consistency with emergency department admission measures (e.g., Glasgow Coma Scale) and post-trauma neuropsychological testing results. For an in-depth review of the reliability, clinical utility, and validity literature, see Roberts (2012).

Working memory. Cognitive deficits, specifically in working memory, are common in individuals with ADHD (Alderson, Rapport, Hudec, Sarver, & Kofler, 2010; Kofler, Rapport, Bolden, Sarver, & Raiker, 2010; Martinussen, Hayden, Higg-Johnson, & Tannock, 2005; Rapport, Bolden, Kofler, Sarver, Raiker, & Alderson, 2009). As such, this study included three measures of short-term/ working memory. This study used the Woodcock-Johnson Tests of Cognitive Abilities, Third Edition (WJ III COG). The Woodcock-Johnson Tests are held in high esteem due to their performance in various reliability and validity analyses that have been conducted, both by the test developers and external researchers. For instance, the core subtests have median reliability coefficients of $r_{11} =$ .81 - .94 (McGrew, Schrank, & Woodcock, 2007). Additionally, according to Flanagan, Ortiz, & Alfonso (2007), the WJ tests are the most comprehensive of the mainstream intelligence batteries, because they include measures of all of the Cattell-Horn-Carroll (CHC) broad abilities. Furthermore, the WJ III COG has been shown to produce reliable and valid results across a variety of ages and cultures (Edwards & Oakland, 2006; McGrew, Schrank, & Woodcock, 2007, Taub & McGrew, 2004).

As the construct of interest is working memory (*Gsm*), only scores from the numbers reversed, memory for words, and auditory working memory subtests were used. The median reliabilities (ages 2 – 90) for the three subtests are as follows: numbers

reversed, r_{11}= .87, memory for words, r_{11} = .80, and auditory working memory, r_{11} = .87. Furthermore, the authors provide reliabilities for these subtests for individuals' ages 18, (r_{11} = .87, .74, .86) 19 (r_{11} = .86, .80, .88), and 20 – 29 (r_{11} = .87, .78, .80) respectively. Additional information regarding the psychometric properties of the WJ III COG (i.e., intercorrelations, content and construct validity, and validity studies for specific subpopulations) is available in the Technical Manual (McGrew, Schrank, & Woodcock, 2007).

Attention. Another core executive function deficit associated with ADHD is sustained attention (Christakou, Murphy, Chantiluke, Cubillo, Smith, Giampietro, et al., 2012; Tillman, Bohlin, Sorensen, & Lundervold 2009). As such, a computerized sustained attention task was administered. The Conners' Continuous Performance Test, Second Edition (CPT-II) is one of the most widely used continuous performance assessments available. The administration of this test takes approximately 15 minutes, during which individuals were asked to discriminate between stimuli presented on the screen. Individuals were instructed to press the space bar when presented with target stimuli, and to suppress this behavior when a non-target stimulus was presented. The test provides a number of performance measures (i.e., reaction time, omission errors, commission errors), each of which have been independently evaluated for reliability and validity.

Psychometric data from the original standardization sample is provided in the *Technical Manual* (Conners, 2000). Split-half reliabilities for the three most commonly used metrics (i.e., reaction time, omission errors, commission errors) were *r* = .95, *r* = .94, and *r* = .83, respectively. Test-retest reliabilities were lower (*r* = .55, *r* = .65, and *r* =

.84), though still acceptable for many metrics. The author also conducted discriminant validity studies prior to initial publication, and found that the CPT significantly discriminated between ADHD, neurologically impaired, and non-clinical populations on all metrics. Finally, the manual reports the precision of the CPT in classifying ADHD versus non-clinical adults (sensitivity = 82%, specificity = 83%).

Equipment

Hewlett Packard laptops were used during data collection and analysis. Additional Dell desktop computers were used during the data analysis phase as well. The Brain Master Discovery 24E amplifier (Brainmaster Technologies, Inc., Bedford, OH; Discovery version 3.4) was used to record raw EEG data. The Brain Master Discovery 24E amplifier was selected as a result of its compatibility with Neuroguide (Applied Neuroscience, Inc., Largo, FL), which was used to collect the raw EEG data and to produce the quantitative EEGs (qEEG). Indeed, this amplifier has been used in a number of similar studies in conjunction with Neuroguide (i.e., Donaldson, et al., 2012; Luangboriboon, Tantayanon, & Wongsawat, 2013), and this combination of software was recommended in a recent textbook chapter entitled *Optimal Procedures in Z-Score Neurofeedback* (Lubar, 2014).

As described in Wigton & Krigbaum (2015), the Brain Master Discovery 24E amplifier has an EEG bandwidth of .43 to 80 Hz, and an A/D conversion of 24 bits. Additionally, while the amplifier has the capacity to sample at 1024 samples per second, the data rate to the computer is much slower (i.e., 256 samples per second). Furthermore, as described in Wigton & Krigbaum (2015), the Neuroguide acquisition module employs a high-pass filter at 0.5 Hz and a low-pass filter at 50Hz in order to filter out noise due to

other electronic devices in the laboratory (e.g., other computers, cell phones, building generators).

MATLAB 2007b (Mathworks, Inc.), Microsoft Office 2013, R (The R Foundation for Statistical Computing, 2004-2013), and IBM SPSS Statistics v. 22 were used for final data organization and analysis.

Research Design

This study employed a pre-test, multiple post-test, delayed treatment design with random assignment. The subjects were randomly assigned to one of two groups—a treatment and sham condition. At baseline, subjects completed a quantitative EEG (qEEG) in addition to a number of psychoeducational measures. These measures were completed again halfway through the study to examine whether the treatment group was making progress above that of the sham group. At the mid-point, the sham group began receiving the treatment series in order to provide a secondary assessment of the treatment. Both groups then completed post-test measures at the end of the study as well to again examine group differences.

Neurofeedback condition. Participants in the randomly assigned NF condition received LORETA Z score biofeedback of the default mode and attention networks following the first administration of the cognitive battery and the EEG baseline data collection. The baseline QEEG was used to identify Brodmann areas at a $Z \geq 2.0$ entry criteria that were consistent with a diagnosis of ADHD. In order to target both the default mode and attention networks within the constraints of the software, two symptoms were selected for all participants—attention difficulties and executive function deficits. This resulted in 24 possible areas to be trained, which covered key areas of both the default

mode network and the attention networks. Table 2.1 provides a list of the 24 possible Brodmann areas across hemispheres, and Table 2.2 provides a list of the matches by participant. Participants were asked to return to the lab to complete 25 LORETA Z score biofeedback sessions (each consisting of 20 minutes active training) over the course of the academic year. This number of sessions was selected based on the literature, suggesting that 20-50 sessions are needed for maintenance of the change (i.e., Fuchs et al., 2003). The goal of these sessions was to target the identified Brodmann areas. These Brodmann areas were then measured in real-time and the Z-tunes setting was used to train the selected brain regions toward Z = 0. Through Neuroguide, there are three options for Z-score neurofeedback training. These setting options include all-or-nothing, where the individual only receives feedback when 100% of the areas being trained meet the Z-score criteria during a set time period (i.e., window), average, where the average Z-score [computed across all areas trained] must meet the Z-score criteria to be rewarded, and Z-tunes, which is what was used in this study. The Z-tunes option is the default, and thus preferred, setting because it is a Gaussian Adaptive filter. This approach begins as all-or-nothing and adapts based on the individual's performance in order to prevent the reinforcement of extreme (i.e., outlier) scores. The feedback criterion was set to achieve > 60% rewards and adjusted toward smaller values as the subject progressed over sessions. The 60% threshold was based on the suggestion of Dr. Robert Thatcher, who created the Neuroguide software. The feedback signal was a multimedia display (i.e., a colored dot plus music) that faded when Z scores failed to meet criteria and played when Z scores meet the feedback criteria.

Sham control condition. Participants in this condition received a Sham (control) condition over the first 10 sessions. For this control, electrodes were attached and connected to the amplifier but the "playback" option was selected so that there was no relation between the NF EEG and the subject's EEG. Sham participants received feedback in the form of randomly selected baseline qEEGs from the other participants, rather than randomly generated noise, to better simulate feedback, and to ensure the single-blind nature of the study. After the first 10 sessions, they began to receive contingent feedback to see if they too began to show a learning curve and/or behavioral changes.

Procedure

Participants completed an online pre-screener providing demographic information and symptom severity. At the initial session, participants completed informed consent, and were given an opportunity to ask questions. They were administered the behavioral pretest measures (WJ subtests and CPT), which were counter-balanced to account for possible order effects. Participants were then fitted with a standard 19-channel Electro-Cap, which uses the international 10-20 system for electrode placement. Impedance was kept at or below 10KΩ for each of the electrodes, and below 5KΩ for most participants. Additionally, ground leads were placed on participants' ears, and impedance was kept at or below 5KΩ. Baseline recordings were taken for three minutes each while the participants' eyes were open and closed. If participants' baseline EEG recordings showed atypicalities consistent with ADHD (i.e., exhibited matches on the Symptoms Checklist), they were given the option to begin their first session of NF. Another eyes open baseline was recorded for three minutes at the end of the session.

At the start of each subsequent session, a baseline recording was taken for three minutes while the participants' eyes were open. Participants then completed four, five-minute sessions of (real or sham) neurofeedback, followed by a three-minute eyes open baseline. At the end of each session, participants were also asked to complete a brief subjective change index, indicating if they noticed changes in a number of areas (e.g., positive/ negative emotions, learning, attention, language) since beginning the study. After the 10th session, participants in the sham condition began receiving real NF, until the end of the study. At the tenth and final (25th) sessions, participants completed the post-test behavioral measures in addition to their 20 minutes of active treatment and EEG eyes-open baselines. An additional eyes-closed baseline was collected at the end of these sessions as well. A schedule of visits and the assessments given at each is included in Appendix A.

Table 2.1. Possible Symptom Checklist Matches

Brodmann Area	Brain Region
7	Parietal
8	Frontal
9	Dorsolateral Prefrontal Cortex
10	Prefrontal
11	Prefrontal
19	Occipital
23	Cingulate
24	Anterior Cingulate
33	Anterior Cingulate
45	Dorsolateral Prefrontal Cortex
46	Frontal
47	Frontal

Note. 12 areas across 2 hemispheres for 24 possible zones.

Table 2.2. Symptom Checklist Matches by Participant

Subject	BA Areas	Bands	Total Metrics
NF_005	8, 9, 24 ^{L}, 47^{L}	D	21
NF_006	10^{R}, 11^{R}, 47^{R}	D, T	9
NF_007	7, 8, 9, 19^{R}, 23, 24, 33, 45^{R}, 46^{R}	T, A, A1, A2, B1, B2	104
NF_008	7, 8, 9, 10^{L}, 11, 19, 23, 24, 45, 46, 47^{L}	A, A1, A2, B1, B2	123
NF_009	8^{R}, 9^{R}, 10^{R}, 11, 19^{R}, 45^{R}, 46^{R}, 47^{R}	D, T, A, A1, A2, B1, B2	128
NF_010	7, 8, 9, 10, 11, 19, 23, 24, 33, 45, 46, 47	D, T, A, A1, A2, B1	193
NF_011	8^{R}, 9, 10, 11, 19, 24, 45^{R}, 46, 47	D, T, A, A1, A2, B1, B2	146
NF_012	10^{R}, 11^{R}, 47^{R}	D	6
NF_013	10, 11, 46^{R}, 47^{R}	D	21
NF_016	8 ^{R}, 9 ^{R}, 10, 11, 45^{R}, 46, 47	D, T, B1	51
NF_019	7, 10 ^{L}, 11, 19, 23, 45^{L}, 47^{L}	D, T, B1, B2	46
NF_020	7, 8 ^{L}, 9 ^{L}, 19	T, A, A2	16
NF_023	7, 8, 9, 10, 11, 19, 23, 24, 33, 45, 46, 47	D, T, A, A1, A2	196
NF_024	7 ^{R}, 8, 9, 10, 11, 19, 23, 24, 33, 45, 46, 47	A, A2, B1, B2	91
NF_025	7 ^{R}, 8, 9, 10, 11, 19^{R}, 24, 33, 45, 46, 47	D, T, B1, B2	116
NF_026	8, 9 ^{L}, 23, 24, 33,	A, A2	66

Note. ^{L} = only left hemisphere. ^{R} = only right hemisphere.

CHAPTER 3

DATA ANALYSIS

QEEG Data Selection

Prior to conducting statistical analyses, previous research was consulted in order to complete more targeted analyses, and to preclude the need to correct for multiple comparisons. Prior research indicates that the prefrontal cortex (PFC) and inferior frontal gyrus (IFG) exhibit significant differences between ADHD and non-ADHD individuals (i.e., Friedman & Rapoport, 2015; Janssen, et al., 2015; Valera, et al., 2007). Similarly, the participants in this study exhibited atypical patterns of behavior in those areas. Specifically, all 16 participants received training in the PFC, and 14/16 participants received training in the IFG. As such, qEEG analyses were focused on these two areas.

Furthermore, the LORETA NF training was targeted via Brodmann Areas; however, the data was collected in reference to 19 electrode channels. As such, these regions of interest were related back to the electrode metric for ease of analysis based on previous research (Okamoto, et al., 2004; Thompson, Thompson, & Wenqing; 2013). The following electrodes were selected to account for the designated Brodmann Areas: PFC, including the dorsolateral prefrontal cortex and orbitofrontal cortex (i.e., BA 8, 9, 10) = FP1, FP2, F3, F4, Fz, and IFG (i.e., BA 45, 47) = F7 and F8.

QEEG Analyses

Prior to running analyses, all EEG data was visually inspected to select a minimum of ten seconds of artifact-free data within the first minute of each sample. Care

was taken to select data in two-second epochs whenever possible. This allowed for the use of the drowsiness and eye movement rejection options in Neuroguide, which helped to eliminate artifact from the data that followed recognizable patterns due to eye movement and/or drowsiness. Additionally, the automatic selection function was employed, which used the ten seconds of selected data as a model to automatically select similar data within the sample. This was done to ensure a minimum of 30-seconds of artifact-free data for each session. Next, power and coherence metrics were calculated via a fast-Fourier transformation. Neuroguide provided both raw scores and Z-scores (see Thatcher, 2011) for each. However, given that Neuroguide outputs tab-delimited-text (.tdt.) files, a simple Visual Basic Script was then used to transform these data files into Excel (.xls) files for ease of use. The data was then aggregated across subjects and sessions through MATLAB 2007b (Mathworks, Inc., 2007) and exported to Microsoft Excel, SPSS, and R (The R Foundation for Statistical Computing, 2004-2013) for final data analysis.

Group equivalency was evaluated at pretest through the use of independent samples t-tests on the LORETA absolute current density measures. Specifically, absolute power and coherence were examined. To minimize error due to individual differences, particularly those due to changes in brain development in young adulthood, the Z-score measures were selected for analysis. Additionally, given that the purpose of the study was to train atypical patterns of connectivity toward Z = 0, the Z-score metric was deemed the most appropriate.

To examine group differences across time, discriminant function analyses were run separately on the z-score absolute power and z-score coherence measures at 3

time points: at pretest, and following the completion of sessions 10, and 25. Additional discriminant function analyses were run to further investigate the findings, and to examine the dose-response relationship of LORETA NF.

Behavioral Data A Priori Power Analyses

Due to the small sample size, a priori power analyses were run through G*Power (Faul, et al., 2007; 2009) in order to determine the likelihood of finding significant ANOVA results. For the CPT, moderate to large effect sizes were used to estimate power, given that previous studies have found similarly high effects of neurofeedback on symptoms of inattention and impulsivity (i.e., Partial η^2 = .11 – .27) on similar tests (i.e., TOVA, IVA; Fuchs et al., 2003; Arns et al., 2009). The estimated effect sizes for the WJ subtests ranged from small (η^2= .0099) to large (η^2 = .2), as previous research into the efficacy of neurofeedback has tended to focus on measures of EEG (i.e., theta-beta ratios; Gevensleben et al., 2009), or ADHD symptomatology as measured by self, parent, or teacher report (e.g., BASC, Conners; Fuchs et al, 2003; Gevensleben et al., 2009) or continuous performance tasks (e.g., IVA, TOVA; Arns et al., 2009; Lubar et al., 1995). Additionally, previous studies that have examined changes in cognitive abilities, as measured by standardized measures such as the Wechsler or Woodcock Johnson Tests, have focused on full scale IQ, verbal/ perceptual abilities, or speed of processing, rather than on specifically measuring working memory performance. For all of the a priori analyses, alpha was set to .05, and the correlation among the repeated measures was set to .87, .80, and .87 and .55, .65, and .84 for the WJ III subtests and CPT measures respectively. These estimates were based on the test-retest reliabilities provided

in the technical manuals for the measures. Results of the power analyses are provided in the Table 3.1.

Given that previous research has suggested that neurofeedback has large effects on continuous performance tasks, it was anticipated that this study had sufficient power to detect similar effects. As previous research has not yet demonstrated the effects of NF training on the WJ measures of working memory, a range of effect sizes and power estimates were generated. As such, with such a small sample size, it was unlikely that small effects ($\eta^2 = .01$) would be detected; however, moderate ($\eta^2 = .06$) to large ($\eta^2 \geq .14$) effects should have been sufficiently powered.

Behavioral Data Analyses

Prior to conducting analyses, the data were examined for adherence to distributional assumptions. Additionally, the two groups were assessed for pre-treatment equivalence using an independent samples t-test.

The behavioral analyses for this project were two fold—focusing on the between group differences on the short-term/working memory measures (i.e., WJ III) as well as on CPT performance. As such, correlations were run to determine the need for univariate or multivariate analyses. Correlations were also run to see if a relationship existed between the total number of areas trained or time to completion, and performance on the behavioral measures across time. A factor analysis was also conducted to further examine the relationship amongst the working memory measures.

Next, in order to use a repeated measures multivariate analysis of variance (RM-MANOVA), several other assumptions were tested. One of the first assumptions that needed to be met was that the sample size (N = 16) needed to be larger than the

number of variables (p = 6) to be tested, which was met. However, in considering the data across the three time points, p = 18, and thus, the WJ and CPT analyses could not be run simultaneously. Furthermore, in testing some of the other assumptions, it became clear that multivariate analyses were not necessary for the CPT data, as the variables were not significantly related across time. As such, a repeated measures Multivariate Analysis of Variance (RM-MANOVA) was run for the WJ III subtests, and repeated measures Analyses of Variance (RM-ANOVAs) were run for each of the CPT measures separately. However, given that number of omission errors violated the assumption of homogeneity of variance at pretest, a nonparametric analysis was conducted to examine group differences on that CPT measure. Finally, post-hoc contrasts were run for measures approaching significance in the RM-ANOVA, in order to further examine the effect of NF treatment across time.

Table 3.1. A Priori Power Analyses

Test	Partial η^2 (Cohen's *d*)					
	.0099 (.2)	.039 (.4)	.083 (.6)	.138 (.8)	.2 (1.0)	.265 (1.2)
WJ-III numbers reversed (*r* = .87)	.35	.91	1.00	1.00	1.00	
WJ-III memory for words (*r* = .80)	.24	.75	.98	1.00	1.00	
WJ-III auditory working memory (*r* = .87)	.35	.91	.1.00	1.00	1.00	
CPT omission errors (*r* = .55)	-	-	.53	.78	.92	.98
CPT commission errors (*r* = .65)	-	-	.85	.98	1.00	1.00
CPT Hit RT (*r* = .84)	-	-	1.00	1.00	1.00	1.00

Note. Table cells are estimates of observed power

CHAPTER 4

RESULTS

QEEG Data

Correlations were run between time to completion, number of regions trained, and the total number of metrics trained with both the z-scored absolute power and z-scored coherence variables to determine if there was a need to control for any of these in subsequent analyses. Although a handful of metrics were significantly correlated, no patterns emerged across time, suggesting that neither time to completion nor the number of areas trained was significantly related to the effect of LORETA treatment. As such, these variables were not added as covariates to the analyses.

The results of the independent samples t-test indicated that the two groups were equivalent at pretest on all (i.e., 49) z-score absolute power current densities, and on 141 of the 147 z-score coherence current densities (see Tables 4.1 and 4.2). However, with 147 comparisons, a Bonferroni correction was applied, after which none of the differences on the coherence metrics remained significant. Additionally, the random assignment research design allows for the assumption of probabilistic equivalence, suggesting that any differences at pretest are by chance. Thus, the two groups were equivalent at pretest on all qEEG metrics.

Furthermore, while seven of the z-score absolute power and fourteen of the z-score coherence variables violated the assumption of homogeneity of variance, multivariate analyses tend to be robust to such violations, particularly with equal group

sizes. As such, the discriminant analyses were run as described above. Similarly, the results of the pretest discriminant function analysis (DFA) further indicated that there were no significant differences at pretest for z-score absolute power, $\chi^2(14) = 14.76$, $p \geq .05$, or coherence, $\chi^2(14) = 9.98$, $p \geq .05$.

Subsequent DFA indicated no significant group differences in z-score absolute power following session 10, $\chi^2(14) = 8.65$, $p \geq .05$, or at post-test, $\chi^2(14) = 19.23$, $p \geq .05$ (see Table 4.3). This suggests that LORETA NF training had no effect on z-score absolute power. However, although each of the participants received z-score training in absolute power, previous research has suggested that NF training is more likely to result in changes in sensorimotor rhythm or power ratios than in absolute power (i.e., von Carlowitz-Ghori, et al., 2015). Additionally, coherence has been demonstrated to be a better predictor of cognitive ability (Thatcher, North, & Biver, 2005; Thatcher & Lubar, 2009). Thus the z-score coherence analyses were of primary interest.

Similar to the results of the z-score absolute power analyses, no significant group differences were found at session 10 for z-score coherence, $\chi^2(14) = 21.28$, $p \geq .05$. However, following sessions 25, significant group differences were found, $\chi^2(14) = 23.73$, $p \leq .05$ (Table 4.4). This suggests that 10 sessions of LORETA NF was not sufficient to demonstrate change. As such, follow-up discriminant analyses were run to examine the dose-response relationship. Specifically, analyses were run to examine the differences between the maximum sham condition (i.e., session 10) and varying dosage strengths for the NF treatment condition—15, 20, and 25 sessions—in order to evaluate the number of sessions necessary to demonstrate this change. Additionally, since these were planned comparisons, a Bonferonni correction was not necessary.

The results of these planned comparisons suggest that 15 sessions, $\chi^2(14) = 21.18$, $p \geq .05$ and 20 sessions, $\chi^2(14) = 18.35$, $p \geq .05$ were not sufficient to demonstrate changes in coherence in the treatment group (see Table 4.5). Significant changes were again found in comparing the sham group at maximum placebo (i.e., session 10) to the NF group at maximum treatment (i.e.., session 25), $\chi^2(14) = 24.22$, $p \leq .05$. This further suggests that 25 sessions is the minimum number of sessions of LORETA NF needed to demonstrate meaningful change in coherence, as the treatment group demonstrated a significant difference following 25 sessions when compared to the sham group, both prior to receiving treatment (i.e., max sham condition) and after receiving 5, 10, and 15 session of NF treatment themselves (i.e., delayed treatment).

Furthermore, in exploring the standardized canonical coefficients resulting from these analyses, a pattern began to emerge. In comparing the two groups at post-test (i.e., following session 25), coherence between FP1 and FP2, and FP1-F3 differentiated the two groups across all hertz bands (i.e., delta, theta, alpha 1, alpha 2, beta 1, beta 2, and beta 3). In the follow-up DFA, the results were nearly identical (see Table 4.6). This suggests that 25 sessions of LORETA NF significantly affected the communication within the anterior prefrontal cortex (i.e., orbitofrontal cortex; Brodmann Area 10) with left lateralization within the dorsolateral prefrontal cortex (i.e., Brodmann Areas 8, 9).

Behavioral Data

The results of the independent samples t-test indicated that the two groups—sham and treatment—were equivalent at pretest on all WJ measures. However, the two groups were not equivalent on 2 of the 3 CPT measures—omission errors and hit reaction time (see Table 4.7). Indeed, the sham group performed significantly better than the treatment

group at pretest, in spite of randomized group assignment, which likely impacted the results. Additionally, the CPT omission errors variable violated the assumption of homogeneity of variance at pretest, resulting in a need for non-parametric tests to examine group differences.

Next, correlations were run to examine the relationship between the three short-term/working memory measures. The results of the initial analysis indicated that while the WJ III scores at all three time-points were significantly correlated, the coefficients among the measures were lower than anticipated. Indeed, the highest correlation in the sample was numbers reversed at time 1 and time 2 (r = .84) suggesting heterogeneity in the sample could impact the results. Thus, factor analyses were completed to further examine the relationship between these three variables, which purportedly measure the same construct (see Table 4.8). Results indicated at pre-test there was one working memory factor comprising the three subtests fairly equally, for both for the sham (eigenvalue = 2.56) and treatment (eigenvalue = 2.13) groups. The same was found at mid-point for both groups (i.e., sham eigenvalue = 2.33; treatment eigenvalue = 2.13), indicating no significant overall group differences after the completion of 10 sessions. However, at post-test, the results indicated a single working memory factor for the sham group (eigenvalue = 2.26), and two distinct factors—working memory (eigenvalue = 1.26) and short-term memory (eigenvalue = 1.02)—for the treatment group, suggesting a significant group difference in at least one of the subtests that makes up the composite. Additionally, the creation of a second factor for just the treatment group at time 3 suggests that this difference is driven by the memory for words subtest, which makes up the second factor.

These results were not supported by a repeated measures multivariate analysis of variance (MANOVA; Table 4.9), indicating no difference between the treatment and control groups on numbers reversed, auditory working memory, and memory for words over time, $F(6, 9) = 1.41$, $p = .31$, partial $\eta^2 = .49$. Univariate tests also indicated there was no treatment effect on cognitive performance, for numbers reversed, $F(2, 28) = 1.86$, $p = .18$, partial $\eta^2 = .12$, for auditory working memory $F(2, 28) = 1.16$, $p = .33$, partial $\eta^2 = .08$, or for memory for words, $F(2, 28) = .08$, $p = .92$, partial $\eta^2 = .006$.

Due to the differing results between the factor analysis and repeated measures MANOVA, a follow-up repeated-measures ANOVA (Table 4.10) was conducted for the memory for words subtest independently, using the "simple" contrast with the sham group as reference. The results indicated that group differences on this measure were in the expected direction, and approached significance, $F(1, 14) = 4.27$, $p = .058$, partial $\eta^2 = .23$. However, the study did not have sufficient power to find such an effect as evidenced by the observed power (.49) obtained through SPSS. The complete working memory analyses are included in Tables 4.8, 4.9, and 4.10.

Correlations were run for the CPT data as well, indicating that the three CPT variables were not significantly related across time. As such, group differences on the CPT measures were examined using individual repeated measures ANOVAs. There was no statistically significant difference between the treatment and control group over time on either of the error measures: number of omission errors, $F(2, 13) = .25$, $p = .78$, partial $\eta^2 = .04$ and number of commission errors, $F(2, 13) = .1.93$, $p = .19$, partial $\eta^2 = .23$. However, the results of the omission errors ANOVA were not interpretable due to violations of homogeneity of variance (Levene's test: $F(1, 14) = 6.31$, $p = .025$) and

sphericity (Mauchly's test: $\chi^2(2) = 11.35$, p =.003). As such Friedman's test was run to examine group differences in omission errors. The results of the nonparametric test (i.e., Friedman) were similar, indicating that there was not an effect of neurofeedback on the number of omission errors made by participants, $\chi^2(2) = 5.51$, asymmetric p =.064. The effect of NF on hit reaction time approached significance, $F(2, 13) = .3.71$, $p = .053$, partial $\eta^2 = .37$. However, post-hoc analysis using the "simple" contrast did not indicate a significant group difference over time, $F(1, 14) = 2.99$, $p = .11$ partial $\eta^2 = .18$, suggesting that there is not an effect of neurofeedback on hit reaction time on the CPT. Results are included in Tables 4.11 and 4.12.

Finally, no relationship was found between the number of areas trained and performance on the behavioral measures at any of the three time points. The highest correlation ($r = -.26$) found was for the number areas trained and memory for words performance at time 2. Additionally, only one behavioral metric (auditory working memory at time 2) was significantly correlated with time to completion ($r = .59$), though after applying a Bonferroni correction for multiple comparisons, it was no longer significant.

Table 4.1. Z-score Absolute Power Independent Samples T-Test at Pretest

	Sham	*Treatment*	*t-test*		*Levene's test*		*Mann-Whitney U test*
	M (SD)	*M (SD)*	*t (df = 14)*	*p*	*F*	*p*	*p*
D_FP1	-.83 (.64)	-.82 (.66)	-0.02	.99	0.09	0.78	
T_FP1	-.68 (.63)	-.73 (.53)	0.15	.88	0.43	0.52	
A 1_FP1	-.54 (.60)	-.63 (.66)	0.26	.80	0.04	0.85	
A 2_FP1	-.64 (.28)	-.59 (.80)	-0.17	.87	7.10	0.02*	.88
B 1_FP1	-.64 (.42)	-.41 (1.09)	-0.57	.58	5.36	0.04*	.65
B 2_FP1	-.59 (.73)	-.20 (1.27)	-0.75	.47	1.64	0.22	
B 3_FP1	-.16 (1.03)	.32 (1.35)	-0.80	.44	1.65	0.22	
D_FP2	-.92 (.61)	-.58 (.67)	-1.06	.31	0.23	0.64	
T_FP2	-.60 (.52)	-.52 (.51)	-0.30	.77	0.08	0.78	
A 1_FP2	-.53 (.57)	-.58 (.65)	0.17	.87	0.06	0.82	
A 2_FP2	-.62 (.32)	-.54 (.73)	-0.27	.79	4.72	0.05*	.96
B 1_FP2	-.63 (.58)	-.69 (.88)	0.14	.89	0.89	0.36	
B 2_FP2	-.60 (.81)	-.57 (.97)	-0.08	.94	0.001	0.97	
B 3_FP2	-.19 (.96)	-.29 (.95)	0.21	.84	0.004	0.95	
D_F3	-.92 (.64)	-.86 (.53)	-0.19	.85	0.68	0.43	
T_F3	-.42 (.75)	-.48 (.50)	0.18	.86	1.86	0.20	
A 1_F3	-.37 (.52)	-.52 (.62)	0.52	.61	0.10	0.76	
A 2_F3	-.50 (.26)	-.40 (.88)	-0.31	.76	4.22	0.06	
B 1_F3	-.41 (.37)	-.37 (1.05)	-0.10	.92	3.92	0.07	
B 2_F3	-.43 (.62)	-.35 (1.08)	-0.19	.85	1.68	0.22	
B 3_F3	-.06 (.88)	-.08 (.99)	0.03	.98	0.00	0.99	
D_F4	-.97 (.68)	-.73 (.70)	-0.70	.50	0.37	0.55	
T_F4	-.50 (.65)	-.48 (.47)	-0.08	.94	1.45	0.25	
A 1_F4	-.38 (.48)	-.51 (.65)	0.43	.67	0.11	0.75	
A 2_F4	-.48 (.31)	-.45 (.83)	-0.09	.93	4.55	0.05	
B 1_F4	-.36 (.42)	-.43 (1.04)	0.18	.86	3.19	0.10	
B 2_F4	-.40 (.57)	-.31 (1.16)	-0.19	.85	2.01	0.178	
B 3_F4	.07 (.98)	-.04 (1.16)	0.20	.84	0.04	0.85	
D_F7	-1.21 (.52)	-1.04 (.55)	-0.64	.53	0.05	0.82	
T_F7	-.94 (.76)	-1.02 (.46)	0.26	.80	1.92	0.19	
A 1_F7	-.58 (.64)	-.85 (.70)	0.80	.44	0.02	0.89	
A 2_F7	-.72 (.34)	-.80 (.86)	0.24	.82	4.98	0.04*	.33
B 1_F7	-.66 (.51)	-.44 (1.3)	-0.45	.66	4.79	0.05*	057
B 2_F7	-.51 (.64)	.21 (1.92)	-1.00	.33	5.11	0.04*	.80
B 3_F7	-.09 (.03)	.60 (2.12)	-0.83	.42	3.65	0.08	
D_F8	-1.07 (.68)	-.97 (.72)	-0.31	.77	0.12	0.73	
T_F8	-.76 (.50)	-.84 (.40)	0.33	.75	1.14	0.30	
A 1_F8	-.50 (.58)	-.79 (.51)	1.04	.31	0.54	0.47	
A 2_F8	-.69 (.38)	-.82 (.73)	0.46	.65	4.00	0.07	
B 1_F8	-.61 (.40)	-.77 (.75)	0.54	.59	2.29	0.15	
B 2_F8	-.63 (.65)	-.69 (.80)	0.15	.88	0.34	0.57	
B 3_F8	.08 (1.50)	-.35 (1.17)	0.64	.53	1.33	0.27	
D_Fz	-.96 (.65)	-.80 (.65)	-0.49	.63	0.26	0.62	
T_Fz	-.49 (.60)	-.44 (.54)	-0.17	.86	0.74	0.40	
A 1_Fz	-.36 (.47)	-.46 (.58)	0.39	.70	0.04	0.84	
A 2_Fz	-.46 (.26)	-.41 (.78)	-0.18	.86	5.71	0.03*	.80
B 1_Fz	-.37 (.40)	-.45 (.82)	0.26	.80	1.00	0.33	
B 2_Fz	-.41 (.70)	-.56 (.70)	0.41	.69	0.1	0.93	
B 3_Fz	.16 (1.40)	-.15 (1.03)	0.50	.63	1.58	0.23	

Note. $N = 8$ per group. *$p \leq .05$. D = delta, T = theta, A1 = alpha 1, A2 = alpha 2, B1 = beta 1, B2 = beta 2, B3= beta 3. Independent samples Mann-Whitney U test used to examine group equivalence for measures violating homogeneity of variance.

Table 4.2. Z-score Coherence Independent Samples T-Test at Pretest

	Sham	Treatment	t-test			Levene's test		Mann-Whitney U test
	M (*SD*)	*M* (*SD*)	*t*	*df*	*p*	*F*	*p*	
D_FP1_FP2	-.19 (1.48)	-.68 (1.32)	.69	14	.50	.02	.88	
T_FP1_FP2	-.03 (1.72)	-.21 (.64)	.28	14	.78	1.74	.21	
A 1_FP1_FP2	.28 (.79)	-.17 (.80)	1.13	14	.28	.12	.73	
A 2_FP1_FP2	.22 (.63)	-.24 (1.03)	1.06	14	.31	1.57	.23	
B 1_FP1_FP2	.21 (1.08)	-.33 (1.02)	1.02	14	.32	.003	.96	
B 2_FP1_FP2	.13 (1.14)	-.39 (1.18)	.90	14	.38	.26	.62	
B 3_FP1_FP2	.3 (1.25)	-.21 (1.29)	.38	14	.71	.03	.87	
D_FP1_F3	.25 (.86)	.02 (.58)	.62	14	.55	.54	.48	
T_FP1_F3	.52 (.92)	.20 (.39)	.91	14	.38	6.04	.03*	.72
A 1_FP1_F3	.21 (.87)	-.18 (.33)	1.18	14	.26	4.03	.07	
A 2_FP1_F3	.09 (.90)	-.45 (1.09)	1.09	14	.29	.77	.39	
B 1_FP1_F3	.30 (1.04)	-.76 (1.47)	1.65	14	.12	3.43	.09	
B 2_FP1_F3	.18 (1.33)	-1.07 (1.8)	1.58	14	.14	3.10	.10	
B 3_FP1_F3	.21 (1.08)	-.48 (1.27)	1.17	14	.26	.98	.34	
D_FP1_F4	.04 (1.59)	-.19 (.97)	.34	14	.74	.37	.55	
T_FP1_F4	.32 (1.43)	-.12 (.62)	.81	14	.43	7.91	.01*	.88
A 1_FP1_F4	.23 (.81)	-.260 (.37)	1.55	14	.14	8.34	.01*	.44
A 2_FP1_F4	.12 (.72)	-.22 (.79)	.90	14	.38	.62	.44	
B 1_FP1_F4	.16 (1.13)	-.68 (1.4)	1.33	14	.21	.82	.38	
B 2_FP1_F4	.20 (1.11)	-1.04 (1.71)	1.72	14	.11	3.43	.09	
B 3_FP1_F4	-.22 (1.21)	-.62 (1.48)	.59	14	.56	1.33	.27	
D_FP1_F7	-.05 (.95)	.03 (.49)	-.22	14	.83	2.70	.12	
T_FP1_F7	.16 (.59)	.02 (.32)	.62	14	.54	4.76	.05*	.65
A 1_FP1_F7	-.05 (.73)	-.35 (.44)	1.01	14	.33	1.35	.27	
A 2_FP1_F7	-.52 (0.5)	-0.550.56	.14	14	.89	.10	.76	
B 1_FP1_F7	-.17 (.81)	-.83 (1.39)	1.15	14	.27	.83	.38	
B 2_FP1_F7	-.24 (1.57)	-1.31 (1.42)	1.42	14	.18	.02	.88	
B 3_FP1_F7	-.12 (1.38)	-1.0 (1.46)	1.24	14	.24	.007	.94	
D_FP1_F8	.30 (1.25)	-.22 (.92)	.95	14	.36	.51	.49	
T_FP1_F8	.66 (1.19)	-.09 (.70)	1.55	14	.14	2.83	.12	
A 1_FP1_F8	.34 (.91)	-.29 (.740	1.51	14	.15	1.52	.24	
A 2_FP1_F8	.25 (.55)	-.18 (.71)	1.34	14	.20	.02	.88	
B 1_FP1_F8	.26 (1.06)	-.45 (.85)	1.49	14	.16	.69	.42	
B 2_FP1_F8	-.08 (1.04)	-.65 (1.14)	1.04	14	.32	.43	.52	
B 3_FP1_F8	-.53 (1.4)	-.70 (1.25)	.26	14	.80	.003	.96	
D_FP1_Fz	-.12 (1.99)	-.28 (0.75)	.21	14	.83	1.86	.19	
T_FP1_Fz	.46 (1.38)	.04 (.65)	.80	14	.45	5.56	.03*	.80
A 1_FP1_Fz	.24 (.90)	-.16 (.32)	1.19	14	.26	4.85	.05*	.23
A 2_FP1_Fz	.20 (.74)	-.16 (.92)	.87	14	.40	.35	.56	
B 1_FP1_Fz	.33 (1.06)	-.42 (1.06)	1.43	14	.18	.35	.56	
B 2_FP1_Fz	.28 (1.14)	-.82 (1.4)	1.71	14	.11	2.26	.16	
B 3_FP1_Fz	.06 (1.290	-.66 (1.46)	1.05	14	.31	.93	.35	
D_FP2_F3	0 (1.15)	-.71 (1.42)	1.09	14	.29	.46	.51	
T_FP2_F3	.46 (1.21)	-.18 (.61)	1.34	14	.20	1.79	.20	
A 1_FP2_F3	.27 (.81)	-.13 (.56)	1.14	14	.28	2.88	.11	
A 2_FP2_F3	.23 (.54)	-.21 (.71)	1.42	14	.18	1.42	.25	
B 1_FP2_F3	.42 (.70)	-.25 (.97)	1.59	14	.14	.79	.39	
B 2_FP2_F3	.24 (.95)	-.35 (1.38)	1.00	14	.34	1.28	.28	
B 3_FP2_F3	-.02 (1.13)	-.33 (1.34)	.51	14	.62	1.12	.31	
D_FP2_F4	.49 (1.28)	-.30 (1.5)	1.14	14	.27	.84	.38	
T_FP2_F4	.55 (1.28)	.24 (.56)	.63	14	.54	1.76	.21	
A 1_FP2_F4	.46 (.45)	.06 (.31)	2.02	14	.06	.57	.46	
A 2_FP2_F4	.34 (.52)	-.05 (.77)	1.20	14	.25	1.66	.22	
B 1_FP2_F4	.36 (.87)	-.31 (.26)	1.23	14	.24	1.88	.19	
B 2_FP2_F4	.17 (1.19)	-.35 (1.49)	.77	14	.45	.42	.53	

B 3_FP2_F4	.02 (1.36)	-.28 (1.29)	.46	14	.66	.05	.83	
D_FP2_F7	-.20 (1.03)	-.90 (.95)	1.40	14	.18	.03	.88	
T_FP2_F7	-.12 (1.04)	-.62 (.46)	1.25	14	.23	4.99	.04*	.23
A 1_FP2_F7	-.03 (.65)	-.51 (.59)	1.55	14	.14	.39	.54	
A 2_FP2_F7	-.46 (.39)	-.48 (.52)	.09	14	.93	1.06	.32	
B 1_FP2_F7	-.25 (.86)	-.73 (.59)	1.28	14	.22	1.78	.20	
B 2_FP2_F7	-.17 (1.11)	-.81 (1.08)	1.19	14	.26	.09	.77	
B 3_FP2_F7	-.37 (1.29)	-.52 (.98)	.27	14	.79	1.23	.28	
D_FP2_F8	.72 (1.01)	-.09 (.95)	1.65	14	.12	.16	.69	
T_FP2_F8	.82 (.74)	0.470.32	1.20	14	.25	3.42	.09	
A 1_FP2_F8	.59 (.59)	.09 (.48)	1.84	14	.09	.12	.74	
A 2_FP2_F8	.47 (.33)	.07 (.66)	1.50	14	.16	1.66	.22	
B 1_FP2_F8	.44 (0.72)	-.13 (1.06)	1.27	14	.22	1.44	.25	
B 2_FP2_F8	.22 (.77)	-.15 (1.2)	.74	14	.47	3.58	.08	
B 3_FP2_F8	.06 (.76)	-.21 (1.24)	.52	14	.61	4.18	.06	
D_FP2_Fz	.19 (1.33)	-.79 (1.66)	1.31	14	.21	.99	.34	
T_FP2_Fz	.54 (1.54)	-.07 (.59)	1.05	14	.31	2.85	.11	
A 1_FP2_Fz	.40 (.71)	.06 (.36)	1.19	14	.25	3.63	.08	
A 2_FP2_Fz	.43 (.58)	-.04 (.62)	1.55	14	.14	.03	.87	
B 1_FP2_Fz	.46 (.70)	-0.12 .79	1.58	14	.14	.74	.41	
B 2_FP2_Fz	.25 (.82)	-.15 (1.13)	.82	14	.43	.77	.40	
B 3_FP2_Fz	.12 (1.04)	-.11 (1.05)	.45	14	.66	.05	.83	
D_F3_F4	.41 (.77)	.21 (.52)	.59	14	.56	1.00	.33	
T_F3_F4	.54 (.54)	.04 (.48)	1.92	14	.08	.84	.38	
A 1_F3_F4	.22 (.69)	-.09 (.53)	1.01	14	.33	2.65	.13	
A 2_F3_F4	.16 (.47)	-.13 (.75)	.92	14	.37	1.53	.24	
B 1_F3_F4	.21 (.63)	-.23 (1.03)	1.03	14	.32	.38	.55	
B 2_F3_F4	.39 (.69)	-.48 (1.76)	1.31	14	.21	1.41	.25	
B 3_F3_F4	-.17 (.89)	-.18 (1.09)	.008	14	.99	.07	.80	
D_F3_F7	.50 (.57)	.09 (.30)	1.84	14	.09	5.72	.03*	.20
T_F3_F7	.45 (.52)	.10 (.21)	1.77	14	.10	7.38	.017*	.16
A 1_F3_F7	.12 (.55)	-.49(.44)	2.46	14	.03*	.20	.66	
A 2_F3_F7	-.17 (.62)	-.53 (.61)	1.16	14	.27	.06	.81	
B 1_F3_F7	.28 (.60)	-.81 (1.38)	2.05	14	.06	6.38	.02*	.16
B 2_F3_F7	.10 (.99)	-1.53 (2.07)	2.00	14	.07	8.77	.01*	08
B 3_F3_F7	.09 (.98)	-.93 (1.61)	1.53	14	.15	11.03	.005*	.16
D_F3_F8	.21 (.90)	-.29 (.67)	1.27	14	.23	.66	.43	
T_F3_F8	.77 (.82)	-.19 (.71)	2.50	14	.03*	.41	.53	
A 1_F3_F8	.27 (.86)	-.32 (.77)	1.45	14	.17	.41	.53	
A 2_F3_F8	.21 (.46)	-.27 (.66)	1.70	14	.11	.42	.53	
B 1_F3_F8	.41 (.89)	-.39 (.65)	2.07	14	.06	3.08	.10	
B 2_F3_F8	.21 (.67)	-.63 (1.30)	1.62	14	.13	3.81	.07	
B 3_F3_F8	-.25 (1.16)	-.57 (1.21)	.53	14	.60	.33	.58	
D_F3_Fz	.56 (.44)	.54 (.28)	.13	14	.90	2.09	.17	
T_F3_Fz	.61 (.40)	.43 (.28)	1.07	14	.30	2.41	.14	
A 1_F3_Fz	.26 (.68)	.13 (.49)	.45	14	.66	.49	.50	
A 2_F3_Fz	.38 (.32)	.15 (.50)	1.09	14	.29	.55	.47	
B 1_F3_Fz	.52 (.22)	.24 (.49)	1.44	14	.17	1.27	.28	
B 2_F3_Fz	.57 (.32)	-.13 (1.24)	1.54	14	.15	2.55	.13	
B 3_F3_Fz	.30 (.71)	.07 (.81)	.61	14	.55	.18	.68	
D_F4_F7	.17 (.67)	-.54 (.70)	2.09	14	.06	.39	.54	
T_F4_F7	.18 (1.01)	-.56 (.59)	1.79	14	.10	2.21	.16	
A 1_F4_F7	.03 (.70)	-.58 (.63)	1.82	14	.09	.15	.71	
A 2_F4_F7	-.45 (.42)	-.55 (.71)	.36	14	.73	1.36	.26	
B 1_F4_F7	-.14 .(82)	-.94 (.70)	2.10	14	.05	.10	.75	
B 2_F4_F7	.04 (1.17)	-1.611.51	2.44	14	.03*	1.19	.29	
B 3_F4_F7	-.39 (1.03)	-.94 (1.25)	.95	14	.36	1.01	.33	
D_F4_F8	.61 (.74)	.40 (.37)	.70	14	.50	1.79	.20	
T_F4_F8	.83 (.63)	.39 (.44)	1.64	14	.12	.80	.39	
A 1_F4_F8	.54 (.65)	-.01 (.57)	1.78	14	.10	.04	.84	
A 2_F4_F8	.48 (.39)	-.12 (.74)	2.05	14	.06	5.86	.03*	.28
B 1_F4_F8	.46 (.65)	-.45 (1.24)	1.85	14	.09	2.77	.12	

B 2_F4_F8	.06 (.95)	-.60 (1.64)	.98	14	.34	2.18	.16	
B 3_F4_F8	-.04 (1.39)	-.51 (1.41)	.68	14	.51	.17	.69	
D_F4_Fz	.59 (.46)	.40 (.29)	1.01	14	.33	.11	.74	
T_F4_Fz	.62 (.38)	.25 (.32)	2.08	14	.06	.71	.41	
A 1_F4_Fz	.39 (.53)	.13 (.39)	1.10	14	.29	2.33	.15	
A 2_F4_Fz	.27 (.48)	-.06 (.64)	1.17	14	.26	.82	.38	
B 1_F4_Fz	.09 (1.1)	-.22 (.95)	.61	14	.55	.12	.75	
B 2_F4_Fz	-.18 (1.85)	-.58 (2.04)	.41	14	.69	.02	.89	
B 3_F4_Fz	-.42 (1.47)	-.33 (1.28)	-.13	14	.90	.04	.84	
D_F7_F8	.10 (.88)	-.94 (.89)	2.35	14	.03*	.08	.78	
T_F7_F8	.45 (1.01)	-.73 (.76)	2.65	14	.02*	.82	.38	
A 1_F7_F8	.08 (.72)	-.67 (.78)	1.98	14	.07	.03	.87	
A 2_F7_F8	-.37 (.38)	-.61 (.53)	1.05	14	.31	.37	.55	
B 1_F7_F8	.03 (.88)	-.70 (.36)	2.17	14	.05*	10.20	.007*	.16
B 2_F7_F8	-.19 (.98)	-.89 (.69)	1.64	14	.12	.53	.48	
B 3_F7_F8	-.26 (.65)	-.75 (.73)	1.43	14	.18	1.33	.27	
D_F7_Fz	.27 (.58)	-.43 (.62)	2.32	14	.04*	.04	.84	
T_F7_Fz	.27 (.96)	-.39 (.62)	1.63	14	.13	3.80	.07	
A 1_F7_Fz	-.03 (.72)	-.61 (.59)	1.77	14	.10	1.11	.31	
A 2_F7_Fz	-.50 (.53)	-.61 (.68)	.36	14	.73	.21	.66	
B 1_F7_Fz	-.06 (.74)	-.88 (.97)	1.91	14	.08	.87	.37	
B 2_F7_Fz	-.04 (1.2)	-1.74 (1.48)	2.52	14	.02*	1.56	.23	
B 3_F7_Fz	-.27 (.88)	-1.13 (1.45)	1.44	14	.17	4.52	.05	
D_F8_Fz	.37 (.81)	-.04 (.53)	1.20	14	.25	.87	.37	
T_F8_Fz	.81 (.92)	.02 (.54)	2.09	14	.06	3.83	.07	
A 1_F8_Fz	.39 (.87)	-.13 (.63)	1.37	14	.19	1.64	.22	
A 2_F8_Fz	.38 (.54)	-.26 (.66)	2.14	14	.05	.006	.94	
B 1_F8_Fz	.41 (.94)	-.37 (.65)	1.91	14	.08	1.72	.21	
B 2_F8_Fz	.03 (.73)	-.58 (1.14)	1.28	14	.22	2.04	.18	
B 3_F8_Fz	-.22 (.99)	-.49 (.97)	.55	14	.59	.17	.69	

Note. $N = 8$ per group. *$p \leq .05$. D = delta, T = theta, A1 = alpha 1, A2 = alpha 2, B1 = beta 1, B2 = beta 2, B3= beta 3. Independent samples Mann-Whitney U test used to examine group equivalence for measures violating homogeneity of variance.

Table 4.3. Z-score Absolute Power Discriminant Function Analyses

	Wilk's λ	χ^2 (*df*)	*p*
Pre-test	.12	14.76 (14)	.40
Session 10	.29	8.65 (14)	.85
Post-test	.06	19.23 (14)	.16

Note. *$p \leq .05$

Table 4.4. Z-score Coherence Discriminant Function Analyses

	Wilk's λ	χ^2 (*df*)	*P*
Pre-test	.24	9.98 (14)	.76
Session 10	.05	21.28 (14)	.10
Post-test	.03	23.73 (14)	.05*

Note. *$p \leq .05$

Table 4.5. Dose-Response Z-score Coherence Discriminant Function Analyses

	Wilk's λ	χ^2 (*df*)	*p*
Session 15	.05	21.18 (14)	.10
Session 20	.07	18.35 (14)	.19
Post-test	.03	24.22 (14)	.04*

Note. *$p \leq .05$

Table 4.6. Standardized Canonical Discriminant Function Coefficients

	Post-test Analysis	Dose Response Analysis
D_FP1_FP2	1.139	-5.872
T_FP1_FP2	4.788	2.709
A 1_FP1_FP2	-9.041	2.334
A 2_FP1_FP2	3.989	-10.508
B 1_FP1_FP2	-1.223	15.314
B 2_FP1_FP2	.306	-11.792
B 3_FP1_FP2	1.727	6.472
D_FP1_F3	.247	18.616
T_FP1_F3	-.227	-8.059
A 1_FP1_F3	2.453	-10.128
A 2_FP1_F3	2.041	7.238
B 1_FP1_F3	-.236	-17.204
B 2_FP1_F3	2.796	8.269
B 3_FP1_F3	-3.537	
A 1_FP1_F4		4.677

Note. D = delta, T = theta, A1 = alpha 1, A2 = alpha 2, B1 = beta 1, B2 = beta 2, B3= beta 3

Table 4.7. Independent Samples T-Test at Pretest for Behavioral Data

	Sham Condition *M* (*SD*)	Treatment Condition *M* (*SD*)	t-test t (df)	t-test p	Levene's test *F*	Levene's test p
Numbers Reversed	100.75 (12.08)	99.13 (15.39)	.24 (14)	.82	.27	.62
Auditory Working Memory	105.88 (13.44)	109.250 (12.75)	-.51 (14)	.61	.04	.85
Memory for Words	99.25 (9.74)	106.75 (12.14)	-1.36 (14)	.19	.73	.41
Omissions	43.85 (2.75)	51.1075 (6.23)	-3.02 (14)	.009*	6.31	.03*
Commissions	59.04 (10.73)	55.1462 (8.02)	.82 (14)	.43	1.90	.19
Hit Reaction Time	36.26 (6.86)	48.078 (9.19)	-2.92 (14)	.01*	.46	.51

Note. $N = 8$ per group. *$p \leq .05$

Table 4.8. Factor Analysis of Working Memory Measures

	Component 1	Component 2
Model 1a		
Numbers Reversed	.948	
Auditory Working Memory	.904	
Memory for Words	.921	
Model 1b		
Numbers Reversed	.927	
Auditory Working Memory	.809	
Memory for Words	.787	
Model 2a		
Numbers Reversed	880	
Auditory Working Memory	.926	
Memory for Words	.833	
Model 2b		
Numbers Reversed	.927	
Auditory Working Memory	.809	
Memory for Words	.787	
Model 3a		
Numbers Reversed	.899	
Auditory Working Memory	.860	
Memory for Words	.843	
Model 3b		
Numbers Reversed	.808	066
Auditory Working Memory	.717	-.451
Memory for Words	.299	.903

Note. Extraction Method: Principal Components Analysis.
Model 1a = sham group at pretest, Model 1b = NF group at pretest, Model 2a = sham group following session 10, Model 2b = NF group following session 10, Model 3a = sham group at post-test, Model 3b = NF group at post-test

Table 4.9. Repeated Measures MANOVA for Working Memory Measures

Effect		Wilk's λ	*F* (*df*)	*p*	Observed Power
Between Subjects					
	Intercept	.004	976.10 (3, 12)	.000**	
	Condition	.65	2.18 (3, 12)	.14	.42
Within Subjects					
	Time	.16	7.68 (6, 9)	.84	
	Time*Condition	.52	1.41 (6, 9)	.31	.31

Note. *$p \leq .05$ **$p \leq .01$ ***$p \leq .001$

Table 4.10. Post Hoc Analysis with Simple Contrast: Memory for Words

	Sums of Squares	*F* (*df*)	*p*	Observed Power
Contrast	240.25	4.27 (1, 14)	.058	.49
Error	788.39			

Note. Sham group was used as reference

Table 4.11. CPT Repeated Measures ANOVAs

	Wilk's λ	*F* (*df*)	*p*	Observed Power
Omission Errors^	.96	.25 (2, 13)	.78	.08
Commission Errors	.77	1.93 (2, 13)	.19	.33
Hit Reaction Time	.64	3.71 (2, 13)	.053	.57

Note.^ Uninterpretable due to violation of homogeneity of variance.
*$p \leq .05$

Table 4.12. Post Hoc Analysis with Simple Contrast: Hit Reaction Time

	Sums of Squares	*F* (*df*)	*p*	Observed Power
Contrast	281.01	2.99 (1, 14)	.11	.36
Error	1314.68			

Note. Sham group was used as reference

CHAPTER 5

DISCUSSION

It was hypothesized that the two groups would not exhibit significant differences at pre-test, the group differences would be greatest at the mid-treatment time point (i.e., session 10), prior to the control group receiving the treatment, and at post-test, the groups would begin to demonstrate similar scores again, due to the nature of the delayed treatment design.

The first hypothesis was generally supported, as the groups were equivalent at pretest for the z-score absolute power and z-score coherence metrics. Results from the behavioral analyses similarly indicated no significant group differences at pretest for 4 of the 6 behavioral measures. Additionally, although the sham group outperformed the treatment group at pretest, by chance, the scores on the omission errors measure were within the average range for both groups, further suggesting approximate group equivalence. The sham group's faster reaction time is also likely related to the number of commission errors, which was equivalent across groups. Finally, due to random assignment and equal group sizes, the study can assume probabilistic equivalence.

The second hypothesis was not supported by the either the qEEG or behavioral analyses as there were no significant group differences following 10 NF sessions. As such, the results of this study are consistent with the current literature base, suggesting that 10 sessions of training is not sufficient to demonstrate significant change. Interestingly, previous research has indicated that a large number of sessions (i.e., 20-50;

(Arns et al., 2009; Holtmann, et al., 2009, Holtmann, et al., 2014) are necessary for surface NF, though it is hypothesized that LORETA NF can demonstrate faster change (Simkin, Thatcher, & Lubar, 2014; Wigton & Krigbaum, 2014). However, the results of this study suggest that 10 sessions of LORETA NF is still insufficient to demonstrate meaningful change on either a qEEG metric, or on behavioral measures of cognitive ability.

The third hypothesis was that at post-test, the sham group would begin to demonstrate change in the same direction as the treatment group. This hypothesis was partially supported. First, the qEEG analyses demonstrated significant change between groups at session 25, for the z-score coherence metric. Additionally, while both groups improved on the behavioral measures over time, the groups began to demonstrate significant between-group differences at post-test, particularly on the verbal working memory measure—memory for words—which approached significance, but was underpowered. Furthermore, the factor analytic examination of the working memory data supports the existence of group differences on the memory for words subtest, due to the change in factor structure (i.e., emergence of a second factor for that subtest for only the treatment group, at posttest).

Further examination of the qEEG data, by comparing the maximum placebo effect (i.e., sham group at session 10) with the treatment group at differing points of treatment allowed for an approximation of a dose-response relationship (i.e., hypothesis 4). This post-hoc analysis further supported the finding that 25 sessions of LORETA NF was necessary to demonstrate significant change across time.

Finally, in examining the canonical coefficients from the discriminant analyses, coherence within the prefrontal cortex—bilaterally within the left and right orbitofrontal cortex (i.e., Brodmann Area 10) and between the left orbitofrontal cortex and left dorsolateral prefrontal cortex—was where the most drastic changes occurred. The prefrontal cortex is involved with cognitive control, and the secondary left lateralization suggests possible implications for language. Memory for words is a verbal working memory measure. The behavioral analyses approached significance, and though not statistically significant in this study, the changes were in the expected direction, and more importantly, the results were consistent with the qEEG changes in coherence.

Overall, results of this study suggest that 25 sessions of LORETA NF training is needed to demonstrate meaningful changes in a college-aged ADHD population, which is consistent with previous literature. Additionally, the consistency between the qEEG and behavioral data is also consistent with previous research, suggesting the importance of the left prefrontal cortex in the acquisition and maintenance of one's verbal working memory.

Limitations

This study has a number of limitations. First, and foremost, the small sample size is a major limitation. Although the a priori power analyses suggested that the study was sufficiently powered, these analyses were run for univariate models, not multivariate. As such the small sample size, and small effect sizes likely contributed to the lack of significant findings. However, the emergence of a second WJ factor, as well as the results of the post-hoc ANOVA contrast suggests that the results are in the expected direction, and thus likely just underpowered.

Some of the outcome measures were also not ideal for this population. Although the WJ-III Tests of Cognitive Abilities has excellent psychometric properties, it is better at demonstrating developmental change (i.e., within a school-age population). This was determined following the completion of phase 1 of the study, as many of the participants exhibited a ceiling effect (i.e., scored near the maximum score prior to post-test). As such, it is likely that the use of a different measure, one meant specifically for adults, could have provided a better measure of change over time for working memory.

Previous research has also suggested that individuals with ADHD are highly motivated by immediate, and personally salient rewards (Marco, et al., 2009). Subjective report from the participants indicated that the stimuli used in the study were boring. In fact, several participants requested to change their chosen stimuli part way through the study, suggesting that they were no longer interested in the stimuli, and thus less likely to maintain focus. The payment scale also seemed to be too far spread out. Future studies should consider more frequent, smaller payments (i.e., $5 at the end of every session). Although the larger sums seem like a greater reward, it is possible that this smaller, consistent reward would be more meaningful, and thus help to improve the outcome(s).

Finally, this began as an efficacy study, to evaluate the efficacy of LORETA NF for the treatment of ADHD in a college population. However, there was significant attrition, and participant's treatment compliance was inconsistent. For instance, participants were asked to complete 2-3 sessions weekly for a duration of approximately 3 months, yet the average time to completion was 5.34 months. Additionally, one participant took 11 months to complete the study, and another took 14 months, likely resulting in little to no treatment effect due to inconsistency. Furthermore, 2 participants

reported inconsistency in their regular stimulant medication regime, and 2 others changed their dosage and/or the prescribed stimulant during the study. Although attempts were made to control for this, it became too cumbersome with the sheer volume of variables. Finally, at the completion of the study 2 participants acknowledged that they recreationally used marijuana at some point during the study, and 1 participant attempted to quit smoking during the study, which could have further impacted the results. As such, what began as an efficacy study became more of an effectiveness study, demonstrating that a college campus is likely not the best environment for LORETA NF treatment. However, in spite of these challenges, the results were still within the expected direction, providing even stronger support for the use of this treatment within a clinical setting.

Implications

Although the group level behavioral analyses for this study were not statistically significant, the results are in the expected direction, and consistent with the qEEG coherence results. Additionally, the results are consistent with other recent studies in the field, suggesting that NF is an effective and appropriate intervention for individuals with ADHD. Furthermore, this study is one of very few investigating the impact of NF training with a college population, thus filling a much-needed gap in the literature. Finally, the results of this study provide support for the use of LORETA NF with a college-aged population with ADHD, and more importantly, demonstrate the need for 25 sessions of LORETA NF to truly affect change. Although there is hope for NF LORETA to enable faster change, the results of this study suggest that a large number of sessions are still needed even with this more sophisticated technique. However, 25 is still less than

the 30-50 sessions reported in other studies of surface NF, suggesting that perhaps LORETA NF is more efficient.

Future Directions

Given the significant limitations to this study, further investigation is needed to examine the efficacy and effectiveness of NF training for the treatment of ADHD in a college population. Future studies should include a larger sample size, and stricter inclusion criteria, specifically that participants must agree to a schedule of 2-3 weekly sessions prior to their first session. Additionally, future studies should examine more closely the dose-response relationship in order to more fully answer the question of whether LORETA NF is a faster and more efficient means of affecting change for young adults with ADHD. Lastly, given the heterogeneity of ADHD, specifically the vast differences in symptomatology and onset of ADHD-predominately inattentive type, future studies should examine the effects of LORETA NF training on the distinct subpopulations separately.

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APPENDIX A

SCHEDULE OF VISITS

Visit	Tasks
Prior to 1st visit: screening	Online screener (including demographics, assessment measures)
1st visit: baseline (pre-test)	Baseline EEG data collection, pretest measures, begin neurofeedback or sham condition
2nd – 10th visits	Continue neurofeedback or sham condition
10th visit (1st post-test)	Re-administer outcome measures
11th-25th visits	All subjects getting neurofeedback
25th visit: post-test data collection	Re-administer outcome measures

APPENDIX B

ONLINE DEMOGRAPHIC SCREENER SURVEY

OVERVIEW

What is "neurofeedback"?

Neurofeedback = EEG biofeedback = The process of changing brain functioning/ "moving" the brain toward a healthier state by using operant conditioning methods. This is similar to "rewarding" your brain waves for firing at a target rate...like training your brain.

Why do neurofeedback with students with ADHD?

We want to investigate whether neurofeedback is effective in changing functioning in brain areas associated with ADHD. For example, there is previous research to suggest large effect sizes for neurofeedback reducing impulsivity and hyperactivity in children with ADHD (Arns de Ridder et al., 2009). We want to build on this previous research.

What do I get out of this?

1. Up to 3 hours of SONA credit (for completion of screening measures and the first visit).
2. $125 in cash (for completion of all 25 sessions. The first 10 sessions must be completed within 4 weeks, ideally 2-3 sessions per week for the duration of the study).
3. You will be provided with a snapshot report of your brain activity over the sessions.

What will be required of you?

1. Complete a brief online screening to determine eligibility and you may be asked to provide written documentation, and/or complete additional questionnaires.

2. Visit the ACN lab for 25 total neurofeedback sessions ranging from 30 minutes to 2 hours each (the first and last sessions will be the longest)

a. Capping: each session, the research team will fit you with an EEG cap. This is not invasive, but does include application of saline gel to the scalp.

b. Fill out forms and answer questions about how you are feeling after receiving neurofeedback (each session)

c. At the first and last sessions, perform an attention task on the computer, and complete some measures of cognitive ability.

d. You must complete all 25 sessions. Sessions are made by appointment and if you are unable to make the appointment, you will need to give 24 hours' notice. (Two no-shows is grounds for dismissal without compensation.)

CLICK HERE IF YOU ARE INTERESTED IN SEEING IF YOU ARE ELIGIBLE...

ACN Lab Neurofeedback Study

Please complete all of the information below to the best of your ability. All information will be kept confidential.

---

* Required

First and Last Name*

Email Address*

Age in Years*

Do you have a medical diagnosis of ADHD?*

- Yes
- No

At what age (in years) were you diagnosed with ADHD?*

Who gave you this diagnosis?*

- General Practitioner/ Physician
- Psychiatrist
- Psychologist
- Don't Know
- Other:

Are you currently prescribed medication for ADHD?*

- Yes
- No

Please list all medications you currently take, including those for ADHD. Include dosage and frequency (e.g., daily, twice daily, etc.)*

Please list any other current diagnoses (e.g., depression, anxiety). If applicable, include the type of treatment. *

Are you willing and available to commit to 25 sessions of Neurofeedback?*

- Yes
- No

Submit

Current Symptoms Scale

For the following questions, please select the answer that best describes your behavior DURING THE PAST 6 MONTHS.

Fail to give close attention to details or make careless mistakes in my work*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Fidget with hands or feet or squirm in seat*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Have difficulty sustaining my attention in tasks or fun activities. *

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Leave my seat in situations in which seating is expected*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Don't listen when spoken to directly*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Feel restless*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Don't follow through on instructions and fail to finish work*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Have difficulty engaging in leisure activities or doing fun things quietly*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Have difficulty organizing tasks and activities*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Feel "on the go" or "driven by a motor"*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Avoid, dislike, or am reluctant to engage in work that requires sustained mental effort.*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Talk excessively*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Lose things necessary for tasks or activities*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Blurt out answers before questions have been completed*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Am easily distracted*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Have difficulty awaiting turn*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Am forgetful in daily activities*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Interrupt or intrude on others*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

How old were you when these problems with attention, impulsiveness or hyperactivity first began to occur?*

To what extent do the problems you may have checked above interfere with your ability to function in each of these areas of life activities?

In my home life with my immediate family*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

In my work or occupation*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

In my social interactions with others*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

In activities or dealings in the community*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

In any educational activities*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

In my dating or marital relationship*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

In my management of my money*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

In my driving of a motor vehicle*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

In my leisure or recreational activities*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

In my management of my daily responsibilities *

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Instructions: Again, please check the number next to each item that best describes your behavior DURING THE PAST 6 MONTHS**.**

Lose temper *

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Argue*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Actively defy or refuse to comply with requests or rules*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Deliberately annoy people*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Blame others for my mistakes or behavior*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Am touchy or easily annoyed by others*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Am angry or resentful*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Am spiteful or vindictive*

- 0) Never or Rarely
- 1) Sometimes
- 2) Often
- 3) Very Often

Impulsivity Scale

People differ in the ways they act and think in different situations. This is a test to measure some of the ways in which you act and think. Read each statement and check the box next to the appropriate response. Do no spend too much time on any statement. Answer quickly and honestly.

I plan tasks carefully*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I do things without thinking*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I make-up my mind quickly*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I am happy-go-lucky*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I don't "pay attention"*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I have "racing" thoughts*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I plan trips well ahead of time*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I am self-controlled*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I concentrate easily*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I save regularly*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I "squirm" at plays or lectures*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I am a careful thinker*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I plan for job security*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I say things without thinking*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I like to think about complex problems*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I change jobs*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I act "on impulse"*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I get easily bored when solving thought problems*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I act on the spur of the moment*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I am a steady thinker*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I change residences*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I buy things on impulse*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I can only think about one thing at a time*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I change hobbies*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I spend or change more than I earn*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I often have extraneous thoughts when thinking*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I am more interested in the present than the future*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I am restless at the theater or lectures*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I like puzzles*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

I am future oriented*

- 1) Rarely/Never
- 2) Occasionally
- 3) Often
- 4) Almost Always/Always

Thank You

Thank you for taking the time to complete our survey.

APPENDIX C

SUBJECTIVE CHANGE INDEX

Please indicate if you have noticed any changes in any of the following areas since starting this study. Use the following rating scale for each area. Place an **X** in the appropriate box that best represents of change in different areas.

Rating Scale

0 No Change
1 Maybe a little change
2 Some Change
3 Definitely some change
4 Definitely moderate amount of change
5 Definitely a large amount of change

	0	1	2	3	4	5
Positive Emotions						
Negative Emotions						
Attention Concentration						
Body Awareness						
Body Movement						
Visual Perception						
Auditory Perception						
Language						
Thinking						
Memory						
Anxiety						
Sadness						

In your opinion do you feel there was a relationship or connection between what you were thinking and the feedback you were receiving on the screen?

Please Circle your Answer:

0 Definitely Yes

1 Maybe

2 Probably Not

3 Definitely Not