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Editorial

It is a great honour and a privilege to welcome you to the inaugural edition of NeuroRegulation. NeuroRegulation represents an exciting new direction for the scientific and publishing arm of the International Society for Neurofeedback & Research. With the end of the Journal of Neurtherapy, an opportunity existed to totally re-evaluate the needs of members of the society and of the scientific community at large. To this end a decision was made to move from a traditional hard copy format to an electronic journal. This trend across all academic fields is now making the latest scientific advances in all fields available to everyone, not just those who are members of a society or have access to a tertiary education library. Electronic journals have also increased the speed at which knowledge is disseminated. In the past, the 'latest research' could actually be anything up to two years or more older before it was made available to the wider community. Electronic journals have seen this timeframe dramatically cut, which can only be good for any scientific endeavour. In relation to our field, this can only be seen as a good step. Clinicians are constantly looking for the latest knowledge and development in treatment options and an open access format will facilitate this. There are also many clinicians globally who do not have access to this information in hard copy. Now all they need is access to the internet to stay abreast of advances within the field.

One aspect of the journal that is quite novel for an open access journal, is there is no submission fee to authors. Most open access journal's charge the author to publish rather than the reader to access the journal. This is due to the generous sponsorship provided by the International Society for Neurofeedback & Research and Mount Mercy University who are hosting the journal. What this means is that there is an opportunity for many of our readers to publish without cost, something that has stopped many from publishing in the past. To this end I encourage our readership to consider publishing if they think they have an idea or data that will add to our body of knowledge. If you are a clinician and have had success with a certain protocol, then please consider a submission. Even if it is a single case study, it may be ideal for placement in the clinical corner section of the journal. Remember, many great advances in science start from a single observation or a decision to try something different.

In this edition of the journal we have a pair of strongly worded companion papers from Dr Pigott and Dr Cannon discussing two claims about the level of existing evidence for Neurofeedback treatment of Attention-Deficit/Hyperactivity Disorder. Do you agree with their position or do you disagree? I welcome letters to the editor on these issues and encourage debate over the claims from both sides of the argument. In our Special Features section also we have a third paper from Dr Krigbaum and Dr Wigton discussing whether modality matters in Neurofeedback. In addition to these papers we have three research papers. The first is reporting the results of a study directed at developing a sham condition for use in high-definition transcranial direct current stimulation research. The problem of developing

appropriate control conditions is very pertinent to new research as funding bodies are no longer happy to support simple wait list control trials and are asking for more sophisticated protocols that allow blinding to be carried out within the study. The second research paper by Dr Larson and colleagues investigated practitioner related factors that are associated with treatment adherence. This is a very important area of research and of particular interest to our readers in clinical practice. The final research paper by Dr Thornton and Dr Carmody investigated the Coordinated Allocation of Resource model using a recalling names of faces task. This interesting study identified specific changes in the EEG, which may have future relevance for enhancing cognitive performance in a number of clinical and subclinical populations. The final submission in the edition of the journal is a book review by Dr Lyle of the book 'Neurotherapy and Neurofeedback: Brain-based Treatment for Psychological and Behavioral Problem'.

Thank you to all of those authors who have submitted to the inaugural edition of NeuroRegulation and I look forward to future submissions to the journal.

Adam R. Clarke
Senior Editor

NeuroRegulation

Neurofeedback is the Best Available First-Line Treatment for ADHD: What is the Evidence for this Claim?

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Attention Deficit/Hyperactivity Disorder (ADHD) is a chronic syndrome characterized by deficits in executive functions and attentional processes. Persons diagnosed with ADHD have significant deficits in self-regulation evidenced by difficulty staying focused, controlling impulsive behaviors, and for many, restraining hyperactive motor activity. These symptoms typically create problems in academic, social, and familial contexts as well as in the planning and organization skills needed for daily functioning. Additionally, comorbid syndromes that can mimic the symptoms of ADHD and confound differential diagnosis are commonly present (e.g., anxiety, depression, learning disorders).

ADHD is the most frequently diagnosed pediatric disorder with 11% of American school-aged children (and nearly 20% of teenage boys) having been medically diagnosed with ADHD according to the latest report from the Centers for Disease Control (Schwarz & Cohen, 2013). Stimulant medication (SM) and behavior therapy (BT) are the two most widely accepted treatments for ADHD, with approximately 70% of those diagnosed prescribed medication (Schwarz, 2013). Although both interventions are considered to meet the highest standards for the evidence-based treatment of ADHD, and have been recognized as such by the American Academy of Child and Adolescent Psychiatry (AACAP) and Children and Adults with Attention-Deficit/Hyperactivity Disorder (CHADD), the leading ADHD advocacy group, the actual evidence is that these treatments fail to result in sustained benefit for the vast majority of children who receive them and, therefore, do not warrant being the first option for treating ADHD.

The Evidence Against Stimulant Medication and Behavior Therapy as First-Line Treatments

The evidence against SM and BT comes primarily from two large NIMH-funded ADHD studies that included long-term follow-up assessments. The first was the Multimodal Treatment of ADHD (MTA) Cooperative study, the gold standard study in ADHD treatment effectiveness research costing $21 million in taxpayer funding. The MTA trial was a cooperative study designed and overseen by America's foremost experts in SM and BT treatments for ADHD. This study randomly assigned 579 ADHD children to receive either systematic stimulant medication management (SSMM), multi-component BT, combined SSMM/BT, or simply an assessment and referral to community care (CC) in which the referred children/families may or may not have actually received treatment services (MTA Cooperative Group, 1999). The children that received SSMM, BT, or combined SSMM/BT were then referred to community professionals for ongoing care at the end of their 14 months of study-directed treatment. Follow-up assessments were then conducted at 10 months

(MTA Cooperative Group, 2004a, 2004b), 22 months (Jensen et al., 2007), and 4.83 and 6.83 years (Molina et al., 2009) after the end of study-directed treatment. The MTA authors took a "spare-no-expense" approach in designing each intervention to ensure that the children received optimal versions of the assigned care (Pigott et al., 2013). Table 1 describes each treatment condition. As detailed in Table 1, our cost estimate in today's dollars for the 14 months of SSMM is $5,310, $15,250 for BT, and $21,560 for the integrated SSMM/BT treatment.

The MTA trial was an open-label study and relied primarily on non-blinded parent and teacher rating scales to evaluate outcomes with these raters systematically involved in the delivery of BT, SSMM, and combined SSMM/BT treatments (but not CC), thereby biasing the reports of outcomes based on these measures when compared to CC (Hammond, 2011). The 14 months of BT failed to demonstrate better outcomes on the non-blinded measures than CC, and combined SSMM/BT failed to separate from SSMM alone. We were surprised to discover, though, that CC was actually found to be superior to BT on the blinded measure of ADHD classroom behaviors contrary to the widely reported equivalence in BT and CC outcomes (see Table 4; MTA Cooperative Group, 1999).

The lack of separation between SSMM and combined SSMM/BT on the non-blinded ratings presents difficulty in concluding that 14 months of BT in children's homes and classrooms provided any advantage over SSMM alone in treating ADHD. Furthermore, the fact that the children referred to the randomness of community/hodgepodge care improved substantially more on the blinded measure of ADHD than those who received BT adds new evidence to the conclusion that 14 months of "spare-no-expense" BT had only a small beneficial effect.

This conclusion regarding BT's relative ineffectiveness is supported further by Hodgson et al.'s (2012) meta-analytic finding that behavior modification, school-based behavior therapy, behaviorally-based parent training, and behavioral self-monitoring treatments each had ***negative effect sizes compared to the control group conditions*** prompting the authors to conclude that these four commonly-utilized BT treatments for ADHD "***cannot be deemed to be efficacious***." Similarly, Sonuga-Barke et al.'s 2013 meta-analysis published in the ***American Journal of Psychiatry*** found that BT had a non-significant effect size of only .02; demonstrating again that BT has, at best, only a minuscule benefit for the ADHD children receiving it. Consequently, BT should be disqualified as a first-line treatment based on both the MTA study findings and these two meta-analyses since BT is simply not reliably helpful no matter its components nor how optimally they are administered.

As for stimulant medication, while both SSMM and SSMM/BT separated from CC on the non-blinded parent and teacher ratings at the end of study-directed treatment, once again the blinded measures tell a different story. These blinded measures found equivalent improvements in ADHD and aggression/oppositional defiance disorder (ODD) behaviors for the CC group contrary to the widely reported superiority of SSMM and combined SSMM/BT (see Table 4; MTA Cooperative Group, 1999). The MTA authors' failure to elucidate this lack of separation on the blinded measures presents significant deficiencies in their conclusions drawn from the study. While the authors asserted the superiority of SSMM, stating in their main article's abstract that "study medication strategies were superior to community care treatments," the blinded assessments clearly do not support this claim.

To date, the findings from the blinded measures, and the implications thereof, have not been addressed by the authors, or to our knowledge, any article referencing this study. Instead, it is commonly cited that SSMM is superior to the randomness of community/hodgepodge care

referencing the MTA study to support this claim. For instance, the AACAP's ADHD Treatment Guideline states, "Children in the MTA who were treated in the community with care as usual from whomever they chose or to whom they had access received lower doses of stimulants with less frequent monitoring and ***had less optimal results***" (emphasis added; AACAP, 2011). Yet this AACAP claim was only true for the biased non-blinded ratings in which both parents and teachers were deeply involved for 14 months in the delivery of SSMM care (see Table 1).

Table 1

Descriptions of the MTA Cooperative Study Treatments

Systematic Medication Management	Children had an initial 28-day, double-blind, daily switch titration of methylphenidate, using 5 randomly ordered repeats each of placebo, 5mg, 10 mg, and 15 or 20 mg at breakfast and lunch with a half afternoon dose. Expert clinicians blindly reviewed graphs of daily-administered parent and teacher ratings of the child's responses to each of the three doses and placebo and by consensus selected his/her best dose. The agreed-on dose (if not placebo) became the child's initial dose. This procedure was followed to optimize symptom reduction while minimizing adverse side effects for each child. For children not obtaining an adequate response during titration, the pharmacotherapist performed non-blinded trials of 3 or more additional medications, and evaluated the effectiveness of each of these trials based again on parent and teacher ratings of the child's responses to same. The pharmacotherapist met monthly for a half-hour office visit with parents to review concerns, evaluate progress, and recommend readings. The pharmacotherapist communicated monthly by phone with the child's teachers and readjusted medications if the child was not doing well. **Cost Estimate:** Selection of optimal dose $1,000 13 half-hour office visits x $120 per visit = $1,560 13 teacher phone calls x $50 per call = $650 14 months of medication x $150 per month = $2,100 **Total Cost Estimate:** $1,000+$1,560+$650+$2,100 = **$5,310.00**
Multi-Component Behavior Therapy	**Parent Training:** Parents attended 27 group and 8 individual sessions for parent training. **Cost Estimate:** 27 group sessions x $70 per group = $1,890 8 individual sessions x $140 per session = $1,120 **Child-Focused Treatment:** Children attended an 8-week, 5-days-per-week, 9-hours per-day summer camp providing intensive behavioral interventions supervised by the same teacher-consultants who performed parent training and teacher consultation. Behavioral interventions were delivered in group-based recreational settings, and included a point system tied to specific rewards, time out, social reinforcement, modeling, group problem-solving, sports skills, and social skills training. The summer program included classrooms for individualized academic skills practice and reinforcement of appropriate behavior. **Cost Estimate:** $500 per week x 8 weeks = $4,000 **School-Based Treatment:** School-based treatment had 2 components: 10 to 16 sessions of biweekly teacher consultation focused on behavior management and 12 weeks (60 school days) of a part-time, behaviorally trained, paraprofessional aide working directly with the child. The aides had been counselors in the summer camp, and the program continued in the fall, to help generalize treatment gains made in the camp into the classroom. Throughout the school year, a daily report card linked home and school. The daily report card was a 1-page teacher-completed checklist of the child's successes on specific preselected behaviors, and was brought home daily by the child to be reinforced by the parent with home-based rewards. **Cost Estimate:** 16 teacher consultation sessions x $140 per session = $2,240 60 days of in-school aide x $100 per day = $6,000 **Total Cost Estimate for BT:** $1,890+$1,120+$4,000+$2,240+$6,000 = **$15,250.00**

Combined SSMM and BT	Combined SSMM/BT treatment provided all of the treatment components outlined above in an integrated manner. Information was regularly shared between the behavioral psychologist/teacher-consultant and pharmacotherapist. Manualized guidelines determined if and when an adjustment in one treatment should be made versus first intervening with the other. **Cost Estimate:** Information sharing and ongoing psychologist/pharmacotherapist consultations $1,000.00 **Total Cost Estimate:** $1,000+$5,310+$15,250 = **$21,560.00**
Additional Tx	The groups were authorized up to 8 additional sessions as needed. At the end of study-directed treatment, children/families were referred to CC.
Referral to CC	Children/parents assigned to CC were provided an assessment report and list of CC providers. The parents may or may not have followed through with treatment referrals. Two-thirds of the CC children received ADHD medications from their own provider during at least part of the 14 months.

Note. (Adapted from Pigott et al., 2013)

Interestingly, the initial superiority of SSMM and SSMM/BT over CC on the non-blinded measures was cut in half at the 10-month follow-up assessment and disappeared entirely in all subsequent assessments. The most parsimonious interpretation for this dramatic loss of separation over time is that they were foretold by the absence of separation in the initial blinded assessments. In other words, as parents and teachers became "less proximal" to their roles in delivering the SSMM and SSMM/BT children's treatment due to the passage of time, their ratings became less biased, thereby eliminating the initial appearance of added benefit from these high-cost treatments.

Fourteen years after publishing the MTA study's initial findings, many of its authors confessed regrets for overselling the value of SSMM in a New York Times (NYT) article titled, "***ADHD Experts Re-evaluate Study's Zeal for Drugs***" (Schwarz, 2013). Unfortunately, these researchers' regrets focused on underselling the value of BT and combined SSMM/BT relative to SSMM alone, ***not their selective reporting of study outcomes***. Their NYT regrets are not supported by their own blinded measures, ***since comprehensive and optimally-administered BT did substantially worse than CC***, and neither SSMM alone nor when combined with BT separated from "refer-and-forget" care.

What the Cooperative should regret is not exploring in detail the negative findings from the blinded measures back in 1999. Instead, these negative findings were buried on the second page of a table, leaving them to languish in obscurity versus compelling the search for more effective treatments that might be different from these experts' preferred ones. Perhaps the best indicator of the dismal impact from the MTA study's high-cost treatments is the fact that during follow-up 10.4 to 12.3% of the BT, SSMM, and integrated SSMM/BT treated children had one or more psychiatric hospitalizations compared to only 8.3% for the CC group, and many of the CC children received little-to-no actual treatment for their ADHD; certainly not the 14 months of optimized high-cost treatments the other children received (Molina et al., 2009).

More troubling still, in the 22-month follow-up assessment it was found that "***medication use was a significant marker, not of beneficial outcome, but of deterioration***" (Jensen et al., 2007), and similarly, in the last follow-up assessment they found that SM use "***was associated with worse hyperactivity-impulsivity and ODD symptoms and CIS*** [Columbia Impairment Scale] ***impairment***" (emphasis added; Molina et al., 2009).

It is unclear if these correlations between SM and deteriorating outcomes were causal (i.e., while initially helpful, continued SM became iatrogenic overtime as children habituated to their performance enhancing effects and then continued SM worsened outcomes across multiple dimensions), as it may only reflect that those ADHD children doing worse were taking SM because they were worse. ***These findings do indicate, though, that continued SM provided at best only a marginal and depreciating benefit, and perhaps significant harm, to struggling children*****.** Regarding sustained effectiveness, even the authors acknowledge, "although the MTA data provided strong support for the acute reduction of symptoms with intensive medication management, these long-term follow-up data fail to provide support for long-term advantage of (continued) medication treatment beyond 2 years for the majority of children" (Molina et al., 2009). Left unexplored by Molina et al. is the likelihood of harm from ongoing SM treatment.

The second NIMH-funded trial is the Preschool Attention-Deficit/Hyperactivity Disorder Treatment Study (PATS). This study was a multisite, randomized trial evaluating the short-term efficacy of SM in preschoolers, aged 3.0 to 5.5 years, with ADHD (Combined or Predominantly Hyperactive/Impulsive Type) in the moderate-to-severe range. PATS enrolled 304 children and their caregivers, of which 261 completed the opening 10-weeks of parent training, 169 completed open-label lead-in of SM, 147completed the double-blind SM phase comparing various dosing levels of SM to placebo, and 140 enrolled in the open-label SM maintenance phase. The main finding from this stage of the study was that only 21% of the preschoolers achieved remission from ADHD on the best-dose SM while even 13% achieved remission on placebo (Greenhill et al., 2006).

In 2013, Riddle et al. followed up on 207 of the 261 preschoolers whose caregivers completed parent training, re-evaluating them at years 3 (mean age 7.4), 4 (8.3), and 6 (10.4). This study found that "***medication status during follow-up, on versus off, did not predict symptom severity***" and despite parent training and systematic SM at the study's outset, the authors concluded:

> ADHD in preschoolers is a relatively stable diagnosis over a 6-year period. The course is generally chronic, with high symptom severity and impairment, in very young children with moderate-to-severe ADHD, ***despite treatment with medication***. Development of more effective ADHD intervention strategies is needed for this age group.

Furthermore,

> In this 6-year follow-up study, almost 90% of clinic-referred preschoolers initially diagnosed with ***moderate-to-severe ADHD***, who mostly received parent training followed by controlled medication treatment, continued to be diagnosed with ADHD in to mid-to-late childhood. ***Across the sample, severity of symptoms, despite initial decline, remained primarily in the moderate-to-severe clinical range*** (emphases added; Riddle et al., 2013).

Interestingly, the PATS researchers also reported "***medication treatment in the original PATS predicted HIGHER ADHD symptom severity*** between follow-up years 3 and 6 in some, but not all, models;" ***raising again the issue identified in the MTA study of the likelihood of harm resulting from continued SM treatment***. More troubling still, by year 3 (age 7), an antipsychotic had been added to 8.3% of the preschoolers' medication regimen (and for 10.7%, a norepinephrine reuptake inhibitor), and by age 10, 12.9% were taking an antipsychotic (and for 8.6%, an SSRI), suggesting that stimulant medications act as gateway drugs to psychiatric drug cocktails for many ADHD children.

This repeated pattern in the MTA and PATS studies of the loss of efficacy in ADHD medications likely accounts for the dramatic increase in the prescribing of antipsychotics to children, as it mirrors the dramatic increased diagnosis of ADHD and prescribing of stimulants to them. In a 2012 article published in ***Archives of General Psychiatry***, Olfson et al. report that between 1993-1998 and 2005-2009, the rate of antipsychotics prescribed to children increased by over 750%. Their analysis found that disruptive behavior disorders (primarily ADHD) were the most common diagnoses in children that were prescribed an antipsychotic, accounting for 63% of such cases, and that in ***54.1%*** of the outpatient visits, ***whenever an antipsychotic was prescribed, there was also an ADHD medication prescribed to the same child***. A similar pattern of dramatic increased prescribing of various psychiatric medications to children/teenagers has occurred (Olfson et al., 2014), adding further evidence that stimulant medications act as gateway drugs to more psychiatric drugs in the often fruitless pursuit of a chemical cure for many ADHD children whose parents initially choose this course of care.

It is troubling to read NIMH's conclusions drawn from the PATS study. The press release accurately notes that after six years there was high symptom severity and impairment for these children with 89% still meeting the diagnostic criteria for ADHD regardless of whether they were on or off medication during follow-up. Despite the clear implications from these findings, and those from the MTA study, for the need to dedicate research dollars into investigating alternatives to ADHD medications, the press release's "***What's Next***" section states, "In an effort to improve outcomes for these children, more research is needed on the effects of ADHD medications on preschoolers over the long term, ***as well as the effects of combining different medications***" (emphasis added, NIMH website). It is unclear if this press release just reflects the overzealous musings of an NIMH public relations' employee or is reflective of NIMH's leadership. Either way, it is clearly an inappropriate use of taxpayers' money to experiment on the effects of powerful psychiatric drug cocktails on preschoolers' developing brains in search of a chemical cure, as such proposed research is ethically dubious and would likely result in far more harm than good.

Stimulant medications have clear short-term effectiveness in treating ADHD for many children, which is why they are tested for, and banned, as performance enhancing drugs in most professional, national, and international sporting events. Similar to most psychiatric medications, even when initially helpful, stimulant medications commonly lose efficacy over time due to habituation and for many become deleterious. In 2009, NIMH Director Dr. Thomas Insel noted, "***The unfortunate reality is that current medications help too few people to get better and very few people to get well***" (Insel, 2009). Dr. Insel's cogent observation certainly applies to the use of stimulants to treat ADHD. When the documented adverse effects of stimulants on ADHD children's growth, neural functioning, and cardiovascular system (Graham et al., 2011) are combined with their lack of demonstrated long-term efficacy and gateway effect to other psychiatric drugs, stimulant medications must be displaced from their current status as the first-line treatment for ADHD.

The Evidence for Neurofeedback as First-Line Treatment for ADHD

Neurofeedback (NFB) is a form of BT with more than 50 years of basic and applied research combining real-time feedback of brain activity with the scientifically-established principles of operant conditioning to teach trainees how to self-regulate targeted aspects of brain functioning. As such, NFB is uniquely suited to treat ADHD children provided that 1) the child's symptoms are functionally related to the targeted brain activity, and 2) the child learns to self-regulate this activity.

In the 1960s, neuroscientists demonstrated that decreases in the motor activity of cats was associated with increased 12–16 Hz neuronal electrical activity in the sensorimotor cortex, an activity pattern Professor Sterman named the sensorimotor rhythm (SMR). Sterman and his colleagues found that when hungry cats were fed droplets of milk contingent upon the increase in SMR activity, the cats "became very alert" and displayed "an almost intense cessation of movement" (Sterman & Wyrwicka, 1967)—the essential behavioral deficits found in children with ADHD. In the 1970s, using a scientifically rigorous within-subject reversal design with blinded raters to treat four ADHD boys, Lubar and Shouse published the first controlled studies demonstrating a specific effect for NFB in reducing the core symptoms of ADHD (Lubar & Shouse, 1976; Shouse & Lubar, 1979). They found that when the ADHD boys were reinforced for increasing SMR, their hyperactive and distractible/inattentive symptoms significantly decreased, and these treatment gains were reversed when the boys were reinforced for decreasing SMR.

Building on the foundation provided by Professors Sterman, Lubar, and Shouse, NFB's evidence-base has grown to more than 60 published studies that find it effective in treating ADHD's core symptoms. The vast majority of these studies used either standardized EEG frequency-based protocols such as SMR training and increasing the theta/beta ratio (TBR) or slow cortical potential (SCP) training based on research demonstrating that trainees can learn to self-regulate the amplitude of a negative shift in slow-wave activity in anticipation of an expected event such as waiting for a timed test to start. A 2009 meta-analysis found NFB using these standardized protocols is efficacious and specific with large effect sizes (ES) for inattention and impulsivity and medium ES for hyperactivity (Arns et al., 2009). In 2012, the organization that maintains the American Academy of Pediatrics' ranking of evidence for psychosocial treatments awarded NFB the highest level of scientific support for treating ADHD (PracticeWise, 2012). More recently, Arns et al. (2014) published a meta-analysis of randomized trials comparing the standardized NFB protocols to semi-active (e.g., EMG biofeedback) and active (e.g., computerized cognitive training) treatment control group conditions. This analysis found that NFB demonstrated specificity and at least a medium ES in treating ADHD's core symptoms compared to these semi/fully active treatments.

Table 2 is a detailed review of 16 controlled studies published since 2000 that evaluated NFB's effectiveness in treating the core symptoms of ADHD. Summarizing across these studies (combined N = 828), our review found that, in comparison to control group conditions, NFB resulted in significant improvements in:

- Parent-rated core symptoms of ADHD (15 studies);
- Teacher-rated core symptoms of ADHD (12 studies);
- Computerized continuous performance tests of core ADHD symptoms (8 studies);
- Neuropsychological measures of response inhibition, reaction time, and concentration (4 studies); and

- Neurophysiologic measures of improvement relevant to ADHD, including the QEEG Attention Index (1 study), Event-Related Potentials (P300) during continuous performance testing (1 study), and activation of regions in the brain related to attention and executive functioning using fMRI (1 study).

Table 2

Controlled Neurofeedback Studies in Treating ADHD

Study	Subjects/Design	Key Findings
Carmondy et al., 2001	**16 children** ages 8-10, 8 with and 8 without ADHD. Children were randomly assigned to 2 groups of 4 matched pairs. The 1st group (4 with & 4 without ADHD) received 36 - 48 NFB training sessions at school. The 2nd group served as a wait-list control group. All children were unmedicated. **Outcome measures** included teacher-completed ADDES and the TOVA. All measures were administered before NFB training, at the midpoint, and after training.	**1)** Only the children with ADHD that were trained with NFB had significantly reduced hyperactivity/ impulsivity as assessed by the TOVA. **2)** Significant TOVA improvements occurred on the Commission Errors ($p < .01$) and Anticipatory Scores ($p < .03$) Scales. **3)** Due to study design, TOVA results cannot be attributed to maturation, time of year, repeated testing, or the training setting/experience. **4)** Teachers' ratings on the ADDES Inattention scale were significantly ($p < .002$) improved for the NFB group.
Monastra, Monastra, & George, 2002 Long-term follow-up study described in Monastra, 2005	**100 ADHD children and adolescents** ages 6-19 who demonstrated cortical EEG slowing from a central site. 51 subjects received an average of 43 NFB sessions, 49 did not. All patients received stimulant medication & academic support at school (IEP/504 plan with school accommodations), and their parents received a 10-week parenting program. **Outcome measures** were the Home & School versions of the ADDES, the TOVA, parenting style, and QEEG Attention Index. All pretreatment measures were administered when patients were unmedicated. Post-treatment measures were administered 1 year later while medicated, 1 week after off medication, and 3 years after the initial evaluation.	**1) Only NFB training resulted in significant improvements on behavioral, TOVA, and QEEG Attention Index measures when medications were withdrawn.** **2)** On the ADDES, parent & teacher ratings revealed significant ($p < .001$) improvements in hyperactive/impulsive & inattentive behaviors post-training, 1-week after medications were withdrawn. **3)** Post NFB training, all TOVA scales were improved to the unimpaired range when measured 1 week after medication withdrawal. **4)** Post NFB training, the QEEG Attention Index dropped into the normal range when measured 1 week after medication withdrawal. **5) 3-year follow-up after initial evaluation revealed that the NFB group alone sustained gains on all measures while unmedicated, and 80% of the NFB group had reduced their medications by 50% or more.** **6**) **None of the children who did not receive NFB had been able to reduce their dosage of stimulant medication in the follow-up assessment, and 85% had *increased* their dosage.**

Fuchs et al., 2003	**34 ADHD children** ages 8-12 were assigned based on parental preference to NFB (*n* = 22) or stimulant medication (*n* = 12). NFB consisted of 30 60-min sessions with sessions administered 3x's per week. The NFB protocol was either theta/beta or SMR training dependent the child's subtype of ADHD. The doses for the medication group were adjusted during study based on need and ranged between 10 and 60 mg/day. **Outcome measures** were the TOVA, Attention Endurance Test, parent & teacher rated CBRS, and the WISC.	**1)** Both groups showed significant improvement in each of the outcome measures with no significant differences between groups. **2)** The authors conclude ***"These findings suggest that neurofeedback was efficient in improving some of the behavioral concomitants of ADHD in children whose parents favored a nonpharmacological treatment****"*
Heinrich et al., 2004	**22 ADHD children** ages 7-13 were assigned to NFB (*n* = 13) and a wait-list control group (*n* = 9). The NFB children received 25 SCP training sessions over the course of 3 weeks. Starting at week 2, the NFB children were instructed to practice their strategies at home. **Outcome measures** were the parent rated FBB-HKS, CPT, and event-related potential (P300) during CPT.	**1)** SCP training resulted in significant reductions in core ADHD symptoms as rated by parents. **2**) SCP training resulted in significant improvements in the more objective laboratory measures compared to those children in the wait-list control group. **3**) The authors concluded that "***this study provides first evidence for both positive behavioral and specific neurophysiological effects of SCP training in children with ADHD***."
Rossiter, 2004	**62 ADHD children and adults** ages 7-55 were matched to NFB (*n* =31) or stimulant medication (*n* = 31) based on patient or parent preference. Patients were matched by (in order) age, sum of 4 baseline TOVA scores, IQ, gender, and ADHD subtype. The medication patients were titrated based on TOVA results and maintained on the dose that maximized TOVA scores. The NFB patients received either 40 sessions in office or 60 at home over 3-3.5 months. **Outcome measures** were the TOVA for both groups and for the NFB group only either a child or adult ADHD rating scale.	**1)** Both the NFB and stimulant medication groups had similar significant improvements in attention, impulsivity, and processing speed on the TOVA with no significant differences between groups. **2**) The NFB group demonstrated statistically and clinically significant improvement on behavioral measures (Behavior Assessment System for Children, ES = 1.16, and Brown Attention Deficit Disorder Scales, ES = 1.59). **3)** The author concluded that "***confidence interval and nonequivalence null hypothesis testing confirmed that the neurofeedback program produced patient outcomes equivalent to those obtained with stimulant drugs***."

deBeus, 2006; deBeuss & Kaiser, 2011	**53 ADHD children** ages 7-11 were randomly assigned in a cross-over design to first receive either 20 30-minute theta/beta NFB sessions or 20 sham NFB sessions. After these sessions, the children who had received active NFB received 20 sham sessions & those who had received sham NFB received 20 sessions of theta/beta NFB. Children were assessed after each block of 20 sessions. **Outcome measures** included the IVA, parent-rated CPRS, and teacher-rated CTRS.	**1)** NFB was superior to sham feedback on the IVA's response control and attention scales, on the CPRS's inattentive scale, and the CTRS's inattentive & hyperactive-impulsive scales. **2)** Of the 42 children who completed all 40 sessions, 31 were classified as NFB-learners because their theta/beta EEG ratio improved in the desired direction by one-half a standard deviation or more following active NFB and 11 were classified as NFB non-learners. **3) NFB-learners were superior to non-learners on the IVA's response control and attention scales and the CTRS's inattentive, hyperactive-impulsive, and ADHD total score scales.**
Levesque et al., 2006	**20 ADHD children** ages 8-12 were randomly assigned on a 3:1 ratio basis. The 15 NFB children received 40 sessions of theta/beta training while 5 children were waitlisted. **Outcome measures** included pre/post changes in fMRI, Digit Span subtest of the WISC, IVA, CPRS Inattention and hyperactivity scales, Counting Stroop and Go/No-Go Tasks.	**1) On the fMRI, NFB resulted in significant activation of the right anterior cingulate cortex (ACC), right ventrolateral prefrontal cortex, right dorsal ACC, left caudate nucleus, and left substantia nigra, whereas no significant changes were seen in the control group.** **2)** NFB was superior on each of the other outcome measures. **3)** The authors concluded that NFB "***has the capacity to functionally normalize the brain systems mediating selective attention and response inhibition***."
Strehl et al., 2006	**25 ADHD children** ages 8-13 received 30 SCP NFB sessions lasting 60 minutes in 3 phases of 10 sessions each. Transfer trials without SCP feedback were intermixed with feedback trials to foster generalization of treatment effects. In addition to the NFB sessions, in the third phase children practiced their SCP self-regulation strategy during homework. **Outcome measures** included parent and teacher ratings of ADHD symptoms (DSM questionnaire for ADHD; Eyberg Child Behavior Inventory; CPRS, and CTRS), IQ (WISC), and a computerized measure of attention.	**1**) Children with ADHD can learn to regulate slow negative cortical potentials. **2**) Children's ability to successfully produce SCP shifts in trials without feedback had better clinical outcomes than those children who were less successful. **3**) Parents and teachers reported significant behavioral and cognitive improvements for the children following SCP training. **4)** After SCP training, significant improvements in attention and performance IQ score were also observed. **5**) The positive changes in parent and teachers ratings, attention, and IQ continued when reassessed 6 months after SCP treatment ended. **While this is was not a controlled study, it was included because of its report of 6-month follow-up results and correlating the children's improvement in learning to regulate SCP and to having better clinical outcomes.**

Drechsler et al., 2007	**30 ADHD children** ages 7-13 were randomized to NFB (*n* = 17) and a group for cognitive behavioral training CBT (*n* = 13). CBT groups had 15 90-min sessions. The NFB group had 30 45-minute SCP training sessions twice per day for 2 weeks, followed by a 5-week break, then 5 double sessions, once or twice per week for 3 weeks. Parents and children were taught how to practice generalizing SCP activation/deactivation to real life situations. **Outcome measures** included parent and teacher rated ADHD symptoms (FBB-HKS, CPRS, CTRS, BRIEF), neuropsychological measures for alertness, inhibitory control, selective attention, sustained attention, and switching attention using the TAP and subtest scores from TEA-ch. Learning cortical self-regulation was evaluated by computing the difference between activation during sessions 2 and 3 vs. 13 and 14.	**1)** NFB was superior to CBT in the parent and teacher ratings, particularly in the attention and cognition-related domains. **2**) Children in both groups showed similar improvement on the neuropsychological measures, however only about half of the NFB group learned to regulate cortical activation during the transfer condition without direct feedback. Behavioral improvements of this subgroup were moderately related to NFB training performance, whereas effective parental support better accounted for some advantages of NFB training compared to CBT group therapy according to parents' and teachers' ratings **3**) The authors concluded that "***there is a specific training effect of neurofeedback of slow cortical potentials due to enhanced cortical control. However, non-specific factors, such as parental support, may also contribute to the positive behavioral effects induced by the neurofeedback training***."
Leins et al., 2007 Gani et al., 2008 for 2-year follow-up	**38 ADHD children** ages 7-13 were matched by age, sex, IQ, dx, and medication status **and then randomly assigned** either theta/beta NFB (*n* = 19) or SCP NFB (*n* = 19). NFB training consisted of 30 60-minute sessions. For both groups, 23% of the NFB sessions were spent on transfer trials in which the subjects attempted to activate the targeted EEG via self-regulation only without real-time feedback and only learned if they were successful after the end of the transfer trial. Both groups also were taught ***transfer exercises*** to practice at home to use their self-regulation strategies for EEG activation in everyday life situations. Three booster sessions were also administered as part of the 6-month and 2-year follow-up assessments and used to calculate EEG self-regulation skills. **Outcome measures** included parent and teacher ratings of ADHD symptoms (DSM questionnaire for ADHD, Eyberg Child Behavior Inventory, CPRS, and CTRS), IQ (WISC), and for the SCP NFB group, SCP	**1**) Both NFB groups learned how to intentionally regulate cortical activity consistent with their training and significantly improved in attention and IQ. **2**) Parents and teachers reported significant behavioral and cognitive improvements for the children in both NFB groups. **3**) The NFB groups did not differ in behavioral or cognitive outcomes. **4**) **The clinical effects for both NFB groups remained stable six months after treatment termination**. **5**) In the 2-year follow-up, all improvements in behavior and attention that had been observed at previous assessments remained stable ***with further significant reductions in the number of reported problems and significant improvement in attention***. **6) EEG-self regulation skills were maintained for the children in both groups when reassessed 2 years after NFB treatment ended.** **7**) **In each NFB group, half of the children no longer met the criteria for ADHD,** and only 22% were talking medication for ADHD**.** **8**) The authors concluded that, "***neurofeedback appears to be an alternative or complement to traditional treatments. The stability of changes might be explained by normalizing of brain functions that are responsible for inhibitory control, impulsivity and hyperactivity***."

	amplitude during activation and deactivation tasks; and for the theta/beta group the theta/beta ratio during activation and deactivation tasks.	
Holtmann et al., 2009	**34 ADHD children**, ages 7 to 12, were randomly assigned on a 3:2 ratio basis to receive either 20 theta/beta NFB sessions (*n* = 20) or 20 sessions of Captain's Log (*n* = 14), a cognitive training software program. All children also received a 2-week intensive behavioral day clinic, weekly parent training, and 79% were on medication for their ADHD. **Outcome measures** included pre/post change on Stop-Signal test, a neurophysiologic measure of response inhibition (Go/NoGo-N2), and the parent-rated SNAP-IV.	**1) Only NFB resulted in normalization of a key neurophysiologic correlates of response inhibition.** **2)** Only NFB resulted in a significant reduction in impulsivity errors on the Stop-Signal test. **3)** There were no differential effects on parent ratings. **4)** The combination of both groups receiving intensive all-day behavior therapy and 79% of the children being on medication may have attenuated the ability to show differences between treatment groups on the parent ratings.
Gevensleben et al., 2009a, 2009b; Wangler et al., 2011; Gevensleben et al., 2010 for 6-month follow-up	**102 ADHD children**, ages 8 to 12, were randomly assigned on a 3:2 ratio basis to receive either 36 sessions of NFB or 36 sessions of Skillies, an award-winning German cognitive training software program. The 62 NFB children were further randomized to receive first either a block of 18 theta/beta training sessions OR 18 slow cortical potential (SCP) training sessions and to switch protocols for the second block of 18 NFB sessions. **Outcome measures** were German rating scales (FBB-HKS and FBB-SSV) blindly administered to teachers and parents at baseline, after 18, and after 36 sessions. Pre/Post changes in EEG were assessed along with 6-month follow-up data for the two-thirds of children who had not dropped out or started some other treatment.	**1)** Only NFB produced significant changes in EEG, and these changes were specific to each form of NFB training and furthermore, were associated with improvements on the ADHD rating scales. **2)** On the parent and teacher rating scales, improvements in the NFB group were superior to the Skillies group for reducing: • Overall ADHD symptoms ($p < .005$ & $p < .01$, both respectively) • Inattention ($p < .005$ & $p < .05$, both respectively) • Hyperactivity/Impulsivity ($p < .05$ & $p < .1$, both respectively) • Oppositional Behavior ($p < .05$, parent rating only) Delinquent & Physical Aggression ($p < .05$, parent rating only). **3)** No significant differences in effects were found between the two NFB protocols (theta/beta training & SCP training). **4) Overall, at the 6-month follow-up the NFB group continued their improvements compared to the Skillies group.** **5**) **Finally, as only 50% of the NFB group was classified as treatment responders, the authors concluded that "*though treatment effects appear to be limited, the results confirm the notion that NFB is a clinically efficacious module in the treatment of children with ADHD*."**

Bakhshayesh et al., 2011	**35 ADHD children**, ages 6 to 14, were randomly assigned to receive either 30 theta/beta NFB sessions (*n* = 18) or 30 sessions of electromyography (EMG) biofeedback (*n* = 17). Single-blinded RCT. **Outcome measures** included pre/post change on parent and teacher ratings using the FBB-HKS; CPT, the bp and d2 attention tests, and changes in the theta/beta ratio and EMG amplitude.	**1)** Training effectively reduced theta/beta ratios and EMG levels in the NF and BF groups, respectively. **2) Compared to EMG biofeedback, NFB significantly reduced inattention symptoms on the parent rating scale and reaction time and concentration on the neuropsychological measures.** **3)** While children in both groups made significant improvements on most measures thereby making it difficult with such a small *N* for NFB to separate from EMG biofeedback, in ALL 11 outcome measures (and subscales thereof), the level of improvement was greater for NFB, and a non-parametric binomial test would find this highly significant. **4)** Besides lowering muscular tension, EMG biofeedback teaches attention, which may further reduce the difference in outcomes.
Duric et al., 2012	**130 ADHD children and adolescents**, ages 6 to 18, were randomly assigned to receive either 1) NFB, 2) methylphenidate, or 2) combined NFB/medication. After randomization, 39 dropped out (36 immediately after randomization) 13 from the NFB group, 15 from the medication group, 11 from the combined group resulting in 91 completing the study; NFB (*n* = 30), methylphenidate (*n* = 31), and combined (*n* = 30). The NFB group received 30 40-minute theta/beta sessions 3 times per week for 10 weeks. **Outcome measures** were the inattention and hyperactivity subscales of the parent-rated CMADBD-P (& total score) with the post ADBD-P administered one week after the final NFB session for those in the NFB and combined groups.	**1**) The parents reported highly significant effects of the treatments in reducing the core symptoms of ADHD, but no significant differences between the treatment groups were observed. **2**) Although not significant, the NFB group showed more than double the pre–post change in attention compared with the other two treatments (3.1 vs. 1.1 and 1.5 for the means) and NFB's effect size was larger than the other two treatments on both the inattention and hyperactivity subscales and total score measures. **3**) The authors conclude that **"*NFB produced a significant improvement in the core symptoms of ADHD, which was equivalent to the effects produced by methylphenidate, based on parental reports. This supports the use of NFB as an alternative therapy for children and adolescents with ADHD*.**"**
Meisel et al., 2013	**23 ADHD children**, ages7 to 14, were randomly assigned to receive either 40 theta/beta NFB or methylphenidate. **Outcome measures** were behavioral rating scales completed by fathers, mothers, and teachers at baseline and post-treatment as well as 2 and 6-month follow-up academic performance.	**1**) In both groups, there were similar significant reductions in ADHD functional impairment as rated by parents and in primary ADHD symptoms by parents and teachers. **2) Significant academic performance improvements were only detected in the NFB group.** **3) NFB gains were maintained in both the 2 and 6-month follow-up assessment.**

Steiner et al., 2014a, 2014b	**104 ADHD children,** ages 7 to 11, were randomly to 40 sessions of NFB (*n* = 34), computerized cognitive training (CT; *n* = 34) or waitlist control (*n* = 36). **Outcome measures** were Conners 3-Parent, Conners 3-Teacher, BRIEF, Behavioral Observation of Students in Schools (BOSS), and dosing of stimulant medications by community physicians.	**1)** NFB children improved significantly more than both the CT and waitlist groups on the Conners 3-Parent, Conners 3-Teacher, and all BRIEF summary indices. **2)** NFB children improved significantly more than waitlist on the BOSS. **3)** CT children showed no improvement on any measure compared to the waitlist group. **4) The clear superiority of NFB over both the CT and waitlist conditions was then sustained in the 6-month follow-up assessment.** **5) NFB was the only group in which there were not significant increases in stimulant medication dosing at both the end of study-directed treatment and the 6-month follow-up assessment.**

Note. Behavior Rating Scales: ADDES = Attention Deficit Disorder Evaluation Scale; BRIEF = Behavior Rating Inventory for Executive Function; CBRS = Conners Behavior Rating Scale; CMADBD-P = Clinician's Manual for the Assessment of Disruptive Behavior Disorders – Rating Scale for Parents; CPRS = Conners Parent Rating Scale; CTRS = Conners Teacher Rating Scale; FBB-HKS = German Rating Scale for ADHD FBB-SSV = German Rating Scale for Oppositional Defiant/Conduct Disorders. Tests of Attention: CPT = Continuous Performance Test; IVA = Integrated Visual and Auditory continuous performance task; TOVA = Test of Variables of Attention; TAP = Test for Attentional Performance. Tests of Intelligence: WISC = Wechsler Intelligence Scale for Children

When assessed, NFB resulted in changes in EEG consistent with the NFB protocol that was trained (7 studies) and these changes in EEG self-regulation persisted when reassessed at 6 months (2 studies) and 2 years after treatment termination (1 study). Furthermore in four studies, the researchers correlated the extent of changes in subjects' EEG to ADHD symptom improvement. Similar to Lubar and Shouse (1976, 1979), in each of these studies, those subjects who were most successful in learning to self-regulate their EEG had the greatest improvement in ADHD symptoms ***providing additional strong evidence that changing the EEG is the mechanism of change in ADHD symptoms resulting from NFB treatment***. In follow-up studies, NFB resulted in significant improvement in core ADHD symptoms that were sustained when reassessed at six months (5 studies) and 2 years (2 studies) after treatment termination, and ***unlike stimulant medications, in no studies have there been any reported adverse effects from NFB***.

Finally, in four studies (combined *N* = 249), NFB training resulted in improvements equivalent to those achieved by stimulant medication. While two of these studies relied on parental preference versus randomization to determine treatment group assignment, this reflects real-world practice and thereby strengthens the relevance of the results (Fuchs et al., 2003; Rossiter, 2004). The two most recent randomized trials (combined *N* = 153) found NFB equivalent to stimulant medication in treating ADHD's core symptoms (Duric et al., 2012), with Meisel et al. (2013) reporting sustained improvement for NFB in their 6-month follow-up assessment, and unlike stimulants, only the NFB group achieved significant improvements in academic performance.

Conclusion

It is time for professional societies, guideline committees, and healthcare payers to recognize NFB as the best available first-line treatment for ADHD. Given the current first-line treatments' poor real-world outcomes, with no evidence of sustained benefit even with

continued stimulant medication, often prescribed at increasing doses and/or combined with powerful new medications such as antipsychotics and antidepressants (Olfson et al., 2012, 2014; Riddle et al., 2013), ADHD children warrant wide-spread access to methodologically-sound NFB as it is the only treatment with credible evidence documenting sustained improvement in ADHD's core symptoms.

NFB is built on the scientifically-established twin pillars of operant conditioning to teach trainees how to self-regulate targeted aspects of brain functioning and neuroplasticity, which is the brain's ability to rapidly change and reorganize its neural pathways in response to new learning. To promote the advancement of empirically-based NFB, ADHD researchers and clinicians must: 1) demonstrate competence in operant conditioning (Sherlin et al., 2011) and consistently document the extent each NFB trainee learns to self-regulate the targeted brain activity (e.g., plot trainees' session-by-session learning curves), 2) learn to assess how other brain regions of interest are affected, and 3) most importantly, consistently document NFB's impact using relevant psychometric measures to assess the extent of change in each trainee's ADHD core symptoms and psychological functioning (Cannon et al., 2014; Cannon, in press). Such practices need to become the standard of care that all scientist/practitioner NFB clinicians adhere to.

Finally, operant conditioning is a formidable mechanism that has been employed using advanced neuroimaging methods including low-resolution electromagnetic brain tomography (LORETA) and functional magnetic resonance imaging (fMRI). It is noteworthy that researchers using these more advanced NFB technologies still adhere to the fundamental principles of operant conditioning and have shown corresponding positive psychometric outcomes when treating ADHD (Cannon, in press). Such close adherence to operant conditioning must be the case whether employing the established protocols of SMR, theta/beta, or SCP to treat ADHD or one of these neuroimaging-based NFB methods.

In sum, if we are to understand the basic mechanisms of neuronal self-regulation, learning, and their effects on ADHD's core symptoms, all NFB scientist/practitioners must adhere to the guidelines for NFB interventions as outlined at their conception by Professors Sterman, Lubar, and Shouse. Anything less creates more noise than clarity in our pursuit of a cure for ADHD. As the best currently available first-line treatment, ADHD children, their parents, and society-at-large, must learn to accept nothing less from us.

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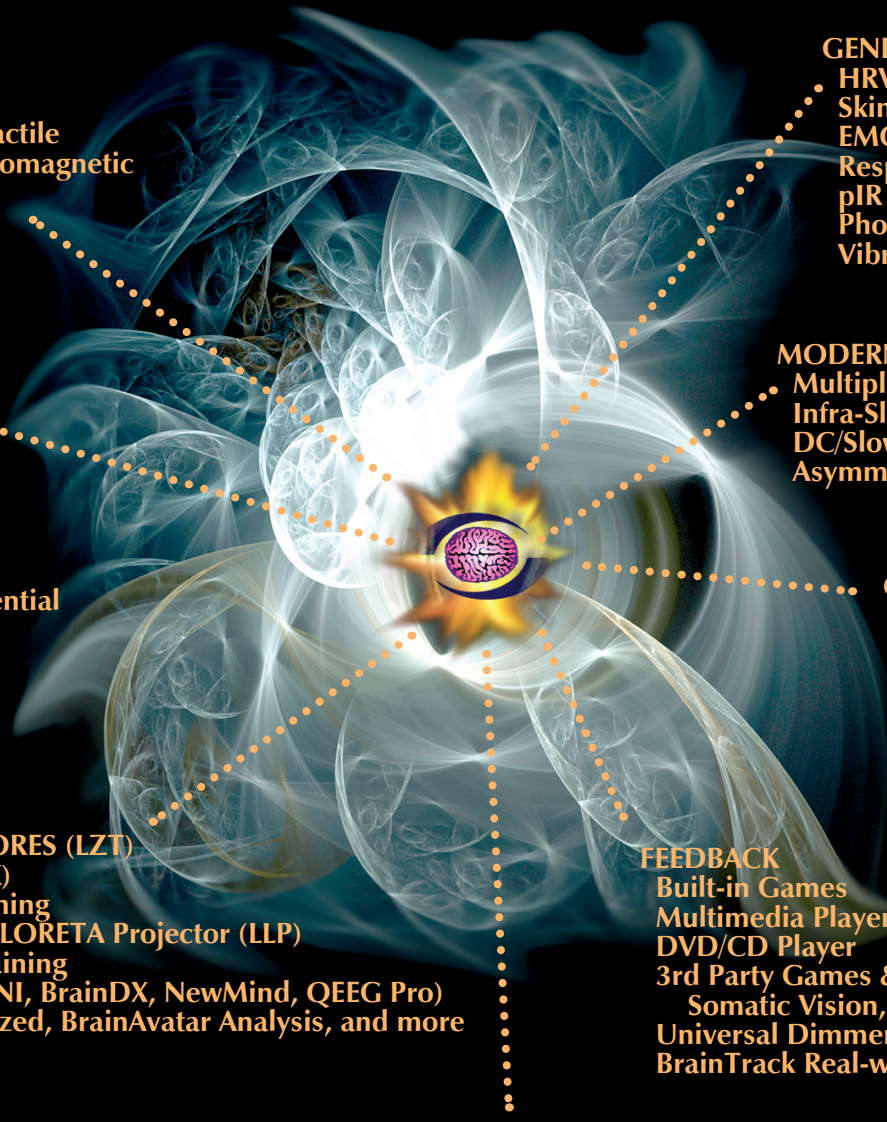

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NeuroRegulation

Neurofeedback Requires Better Evidence of Efficacy Before It Should Be Considered a Legitimate Treatment for ADHD: What is the Evidence for this Claim?

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Russell Barkley is the most prominent and longstanding critic of neurofeedback (NFB) treatment for ADHD (Barkley, 1992). In a 2005 review article coauthored with Sandra Loo, Loo/Barkley state that for NFB to be considered a "legitimate treatment" it must not only be found effective, but it also must be demonstrated in "studies that are scientifically rigorous" that:

- "Changing the EEG is the mechanism of change in ADHD symptoms;"
- The treatment effects must also "generalize to non-treatment settings" and "persist over time;" and furthermore,
- "Even with such demonstrations, it must also be shown that treatment is cost effective in managing the symptoms of ADHD relative to the prevailing empirically supported approaches" (Loo & Barkley, 2005).

What is Good for the Goose is Good for the Gander

Logically, if we accept Loo/Barkley's evidentiary standards for NFB, the same standards should be applied even-handedly to all psychological and pharmaceutical treatments for ADHD. Call it the "What is Good for the Goose is Good for the Gander" rule; a rule that is critical to minimize bias when evaluating the evidence of different treatments. By applying this rule, there are simply no psychosocial OR pharmaceutical treatments for any behavioral health disorder that meet the Loo/Barkley evidentiary standards.

Dr. Barkley violates his own "treatment legitimacy" standards. Barkley and many others have been strong proponents of stimulant medications to treat ADHD since the mid-1970s; but despite billions of dollars spent in "scientifically rigorous" research efforts over the past 40+ years, we still do not know what are the mechanisms of change from stimulants that account for the observed improvements in randomized trials typically lasting only 6 to 8 weeks.

In the Physician Desk Reference, every psychoactive medication has a statement similar to "presumably works by" or "is thought to…" when describing an FDA-approved drug's hypothesized "mechanism of change;" yet Loo/Barkley fail to hold stimulants to the same evidentiary standard they assert is necessary for NFB to meet before it can be considered a legitimate treatment. Take methylphenidate for example, the most commonly prescribed drug for ADHD:

> The mode of therapeutic action in humans is not completely understood, but methylphenidate ***presumably*** activates the brain stem arousal system and cortex to produce its stimulant effect. Methylphenidate ***is thought to*** block the reuptake of norepinephrine and dopamine into the presynaptic neuron and increase the release of these monoamines into the extraneuronal space. ***There is neither specific evidence that clearly establishes the mechanism whereby Methylin produces its mental and behavioral effects in children, nor conclusive evidence regarding how these effects relate to the condition of the central nervous system.*** [emphases added; FDA, 2013]

It is also well known that the effects of stimulant medications do not "persist over time" when treatment is stopped. Barkley himself emphasizes this point on his website. Furthermore, there are now three publically-funded studies finding no evidence that the benefits of stimulants "persist over time" even from continued stimulant medication treatment (Molina et al., 2009; Smith et al., 2010; Riddle et al., 2013) with substantial evidence suggesting that such continued treatment for many becomes iatrogenic overtime (Pigott & Cannon, 2014).

Regarding cost-effectiveness, medication-based treatment is expensive given the fact that in the attempts to sustain effectiveness, people have to take the medication(s) on an ongoing basis, and for many, at ever higher doses and/or with intermittent medication changes and new drug augmentation due to the habituation effects that commonly develop to the originally prescribed medication(s). This reality is seen in the Preschool Attention-Deficit/Hyperactivity Disorder Treatment (PATS) study (Riddle et al., 2013). By year 3, an antipsychotic had been added to 8.3% of the preschoolers' medication regimen (mean age: 7.4 years), and by year 6, 12.9% were taking an antipsychotic (mean age: 10.4 years). This loss of efficacy in ADHD medications accounts for the majority of the dramatic increase in prescribing antipsychotics to children. In a 2012 article, Olfson et al. report that between 1993-1998 and 2005-2009, the rate of antipsychotics prescribed to children increased by over 750%. Their analysis found that disruptive behavior disorders (primarily ADHD) were the most common diagnoses in children that were prescribed an antipsychotic accounting for 63% of such cases, and that in ***54.1% of the outpatient visits, whenever an antipsychotic was prescribed there was also an ADHD medication prescribed to the same child.***

The combination of open-ended treatment by medication(s), and the associated physician fees for overseeing the prescribing of these drugs, makes drug-centric treatment for ADHD very expensive with a poor cost-benefit return on investment as demonstrated by the MTA Cooperative study authors' own conclusion that they found no evidence to support the "long-term advantage of (continued) medication treatment beyond 2 years for the majority of children" (Molina et al., 2009); a conclusion identical to that found in the PATS and Australian studies (Riddle et al., 2013; Smith et al., 2010). The simple fact is that the available evidence from these large, taxpayer-funded studies indicates not only that the effects of stimulant medications do not "persist over time" after treatment is stopped, there is no evidence of a sustained benefit when stimulant drugs and new drug augmentations continue to be taken.

On the behavior therapy front, behaviorally-based parent training of the type developed by Barkley (1987) and classroom management strategies have not been subjected to rigorous controlled trials in which the specific aspects of the interventions were shown to be the mediating mechanisms of change, nor has it been shown that the observed changes generalized to other settings or persisted over time. ***In fact, we know that the effects of***

such behavioral strategies for ADHD do not generalize to other settings nor persist over time, as Barkley himself acknowledges on his website stating:

> Psychological treatments, such as behavior modification in the classroom and parent training in child behavior management methods, have been shown to produce ***short-term benefits*** in these settings. However, the improvements which they render are often limited to those settings in which treatment is occurring and ***do not generalize to other settings*** that are not included in the management program. ***Moreover, recent studies suggest, as with the medications discussed above, that the gains obtained during treatment may not last once treatment has been terminated.*** Thus, it appears that treatments for ADHD must often be combined and must be maintained over long periods of time so as to sustain the initial treatment effects. In this regard, ***ADHD should be viewed like any other chronic medical condition that requires ongoing treatment for its effective management but whose treatments do not rid the individual of the disorder***. [emphases added; Barkley website]

Neurofeedback Comes Closest to Meeting the Loo/Barkley Evidentiary Standards

The Loo/Barkley review article holds NFB to far higher evidentiary standards than are applied to the widely accepted treatments of stimulant medication and behavior therapy, thereby reflecting bias on their part and making it hard to take as credible their selective review of the NFB research. ***The irony here is that NFB comes far closer to meeting the Loo/Barkley evidentiary standards for the effective treatment of ADHD than either of these two widely accepted treatments***. Consider for example:

- The very first NFB studies by Lubar and Shouse demonstrated that "*changing the EEG is the mechanism of change in ADHD symptoms*" (Lubar & Shouse, 1976; Shouse & Lubar, 1979). Using a scientifically rigorous within-subject reversal design with blinded raters, these researchers found that when ADHD boys were reinforced for increasing their sensory motor rhythm (SMR), their hyperactive and distractible/inattentive symptoms significantly decreased and these treatment gains were reversed when the boys were reinforced for decreasing SMR.

- In 1995, Lubar et al. demonstrated that ADHD children who learned to decrease their theta/beta ratios through NFB training showed improvement on multiple outcome measures while non-learners did not improve. Furthermore, four new studies correlated the extent of changes in subjects' EEG to ADHD symptom improvement, and those subjects who were most successful in learning to self-regulate their EEG had the greatest improvement in ADHD symptoms, **thereby providing additional strong evidence that "*changing the EEG is the mechanism of change in ADHD symptoms*" resulting from NFB treatment**. Finally, there are now seven studies demonstrating that NFB resulted in protocol-specified "*changes in the EEG,*" and these improvements in EEG self-regulation persisted when reassessed at 6 months (2 studies) and 2 years (1 study) after treatment termination with associated sustained improvement in ADHD core symptoms (see Pigott & Cannon, 2014).

- In comparison to control group conditions, NFB has been shown to result in significant improvements in (a) parent-rated core symptoms of ADHD (15 studies); (b) teacher-rated core symptoms (12 studies); (c) computerized continuous performance tests of core symptoms (8 studies); (d) neuropsychological measures of response inhibition, reaction time, and concentration (4 studies); and (e) neurophysiologic measures of improvement relevant to ADHD including the QEEG Attention Index (1 study), Event-Related Potentials (P300) during continuous performance testing (1 study), and activation of regions in the brain related to attention and executive functioning when assessed using fMRI (1 study). These findings from numerous international research groups provide strong evidence that, unlike traditional behavior therapy, the gains from NFB treatment ***"generalize to non-treatment settings,"*** and this generalization effect is the result of subjects learning how to self-regulate their EEG (see Pigott & Cannon, 2014).

- In follow-up studies, NFB resulted in significant improvement in core ADHD symptoms that were sustained when reassessed at six months (5 studies) and 2 years (2 studies) after treatment termination, thereby providing strong evidence that, unlike stimulant medications and traditional behavior therapy, the gains from NFB treatment ***"persist over time"*** following treatment termination (see Pigott & Cannon, 2014).

In contrast to Dr. Barkley's acknowledgement of the limited effectiveness for what are widely deemed as "*legitimate treatments,*" those being open-ended medication(s) and behavioral management programs implemented across settings, such that **"***ADHD should be viewed like any other chronic medical condition that requires ongoing (combination) treatment(s) for its effective management but whose treatments do not rid the individual of the disorder*," the Gani et al. (2008) study found at the two-year follow-up assessment of NFB's effectiveness: (a) "***yet another significant reduction of number of*** (ADHD-related) ***problems and significant improvement in attention was observed****," (b) "****EEG self-regulation skills were preserved****," (c) "****half of the children no longer met ADHD criteria,***" and (d) only 22% were still taking medication for ADHD. These authors therefore concluded that, "***Neurofeedback appears to be an alternative or complement to traditional treatments. The stability of changes might be explained by normalizing of brain functions that are responsible for inhibitory control, impulsivity and hyperactivity***.***"***

Negative Findings from a Recent Meta-Analysis

Recently, the self-named European ADHD Guidelines Group conducted meta-analyses of randomized trials of six different non-pharmacological treatments using parent- and teacher-completed rating scales to compute each treatment's effect size (Sonuga-Barke et al., 2013a). This article found significant effect sizes for learning-based interventions using raters closest to the treatment setting (the so-called "most proximal assessment," parents for all home- and clinic-based interventions), but no significant effects using raters furthest from the treatment setting (the so-called "probably blinded assessment," typically teachers, except as the authors state, "If the intervention was implemented at school, teacher ratings were not considered probably blinded assessments"). The researchers concluded from these findings that "***better evidence for efficacy from blinded assessments is required for behavioral interventions, neurofeedback, cognitive training*** (CT), ***and restricted elimination diets before they can be supported as treatments for core ADHD symptoms***" [emphasis added].

While this group's approach was clever, their sweeping conclusions regarding NFB are not valid and instead demonstrate how meta-analyses are constrained by their methods and,

therefore, their conclusions should be similarly constrained. In this regard, it is important to know that NFB's treatment effect size narrowly missed reaching significance (p = .07) in the group's "probably blinded" meta-analysis, whereas CT (p = 0.34) and behavioral interventions (p = 0.92) did far worse, indicating that it would take only a slight shift in their methodology to find NFB efficacious, but not so for CT or behavioral interventions.

Garbage In, Garbage Out

There are several significant methodological errors in the group's meta-analyses that invalidate their conclusions. ***First***, the article's methods section states, "Participants (ages 3 to 18 years) had a diagnosis of ADHD of any subtype (DSM-defined ADHD or ICD-defined hyperkinetic disorder . . . ***OR met accepted criteria for clinical levels of symptoms on validated ADHD rating scales***" [emphasis added]. The authors fail to acknowledge that such allegedly validated ADHD rating scales have only class-IV evidence for diagnosing ADHD according to the American Academy of Neurology, and in a recent large blinded multi-site trial had exceptionally poor diagnostic sensitivity (range: 38–79%) and specificity (range: 13–61%) with an overall diagnostic accuracy similar to flipping a coin (range: 47–58%; see Snyder et al., 2008). Rigorous methods would have informed readers which of their included studies relied on such "***flip-a-coin***" rating scales to diagnosis ADHD—***versus the gold-standard of clinician diagnosis***—and then analyzed these low-quality studies separately to assess if they differentially affected their findings. The inclusion of such studies in their meta-analyses, an error the authors then compounded by not assessing for the low-quality studies' differential impact, is a prime example of the "***garbage-in-garbage-out***" critique of methodologically flawed meta-analyses.

Ignoring Neurofeedback's Use of Objective Outcomes

Second, to analyze outcomes, the authors relied on parent and teacher-completed ADHD rating scales that are subject to significant rater bias with poor inter-rater agreement and exceedingly high levels of false-positive and false-negative findings when used to assess ADHD (Snyder et al., 2008). Due to not having both parent- and teacher-completed rating scales, the group excluded from its "probably blinded" meta-analysis randomized NFB studies using more objective measures of ADHD including:

- Computerized continuous performance testing of core ADHD symptoms (Heinrich et al. 2004; Bakhshayesh et al., 2011; Levesque et al., 2006);
- Neuropsychological measures of core ADHD symptoms including response inhibition, reaction time, and concentration (Holtmann et al., 2009; Bakhshayesh et al., 2011; Levesque et al., 2006); and
- Neurophysiological measures of improvement relevant to ADHD such as Event-Related Potentials (P300) during continuous performance testing (Heinrich et al., 2004) and activation of regions in the brain related to attention and executive functioning assessed using fMRI (Levesque et al., 2006).

Unlike other treatments in the group's meta-analyses, the use of such objective measures is common in NFB studies. In general, randomized NFB studies blindly administered these more objective measures, and they consistently demonstrate NFB's superiority over the control group conditions. Unfortunately, the guideline group not only excluded these measures from its "probably blinded" meta-analysis, thereby significantly reducing the number of NFB studies with positive findings included in it, but also failed to inform journal

readers of the existence of these measures that are directly related to assessing the efficacy of ADHD treatments.

Protocol Violation #1

Third, one of the trials in the group's "probably blinded" analysis was Steiner et al.'s preliminary study (*N* = 41) comparing NFB, CT and waitlist groups (Steiner et al., 2011). In this study, the NFB and CT treatments were provided at school in a separate room during "team time" twice per week for 4 months while the waitlist subjects remained with their class. Many of the teachers, therefore, were not "probably blinded" about which students left their classes twice each week for four months to be treated, though the teachers did not know if these departing students were receiving NFB or CT. Despite this fact, the researchers chose to compare NFB to the waitlist group using the teacher ratings for their "probably blinded" analysis versus comparing NFB to CT; a comparison that was both blinded and more rigorous.

In a Letter to the Editor, Arns and Strehl (2013) noted how Sonuga-Barke et al. violated their published protocol, which stated they selected control conditions "in the following order: sham/placebo, attention/***ACTIVE CONTROL***, treatment as usual, waiting list" [emphasis added; Sonuga-Barke et al., 2013a]. According to this protocol, the guideline group should have compared NFB to CT, not waitlist, with CT as the active control group condition. ***Arns and Strehl then recalculated Sonuga-Barke et al.'s meta-analysis, and this effort found NFB as having a significant effect size in the "probably blinded" analysis***.

In their response letter, Sonuga-Barke et al. (2013b) claimed that they did not violate their protocol, stating that in Steiner et al. the "type of cognitive training was not considered a control condition but rather an optimized active ADHD treatment;" yet no such "optimized active" exclusion to their protocol is mentioned in the group's article. This rationale on Sonuga-Barke et al.'s part, therefore, appears more like a post hoc formulation to justify their protocol violation after it was discovered by Arns and Strehl than anything prespecified; otherwise, the "optimized active" exclusion would have been stated in their article.

Lack of Blinding to Study Outcomes during Consensus Decision-Making Process

Fourth, when selecting which control group to use for their "probably blinded" analysis, the group used a consensus decision-making approach with no attempt to keep group members blind to study outcomes prior to selection, a strategy that would have protected against members' biases for and against particular treatments. Similarly, the group used the same approach when deciding which measures to use for their "most proximal" and "probably blinded" meta-analyses. In response to our email inquiring about a second protocol violation we discovered, and specifically asking for evidence of the blinding of group members from knowledge of study outcomes in their decision-making processes, the group's leader, Joe Sergeant, wrote:

> With respect to what you and your colleagues refer to as a "protocol violation", this matter was dealt with a year ago in the pre-publication correspondence with colleagues Arns and Strehl. Our response to them was: Our meta-analytic approach compared the treatment effects for most proximal and probably blinded measures. ***It placed an onus on us to make judgments*** about what was and was not the best probably blinded measure in each trial. Some times this decision was very

> straightforward at other times it was less so. In these cases ***we had to use ALL AVAILABLE INFORMATION within the papers to make a judgment*** about what constituted probable blinding. Including the term probably in its title explicitly acknowledged the judgment that was required by the reviewers in choosing this class of outcomes. In the case of the Steiner (2011) paper, ***after much examination of the available information and discussion*** we decided that the teacher-ratings were probably blinded while the parent ratings were not. First the treatment took place outside the regular classrooms during the school's "team time" (so that classroom instruction was not affected). Second, no teachers were involved in the delivery of the interventions. ***Third, the post intervention ratings were made by different teachers than the ones that made the pre-treatment ratings due to teacher changes following treatment in the new school***. Fourth, in contrast, the researchers made no attempt to keep parents "blind" to the type of treatment. It seemed to us very likely that the parents knew which arm of the study the children were included in. Taking all these factors together our judgment was that the parent rating was probably not blinded while the teacher rating was. The statement in the paper "…if the intervention was implemented at school, teacher ratings were not considered probably blinded assessments…" referred essentially to classroom-based interventions involving teachers in their administration. However, the interventions in the Steiner et al. study, although they were delivered on school property, were not strictly speaking school-based intervention, in the sense of them being delivered by a teacher in the classroom [emphases added; Sergeant personal communication, 2014].

As is clear from Sergeant's response, group members were not blind to study outcomes when deciding either the group to use as NFB's control group comparator nor when assigning the parent and teacher ratings to their respective "most proximal" and "probably blinded" meta-analyses.

This lack of blinding to study outcomes is a significant methodological flaw, because the waitlist group in the Steiner et al. preliminary study had an unusually large effect size of .4 in the teacher ratings versus a negative effect size of .1 for CT (see Table 1). To our knowledge, a .4 effect size for a group of waitlisted ADHD children is unprecedented. It was unscientific for the group members to know these facts when deciding to use the teacher ratings of the waitlist group with its large effect size for their control group comparison in the "probably blinded' analysis versus the slight negative effect size for CT, and in so choosing violate their published protocol for guiding this decision.

Table 1: Effect Sizes in Steiner et al. Preliminary Study

	Neurofeedback	Cognitive Training	Waitlist
Aggregate of Parent Ratings	1.1	0.5	0.0
Teacher Ratings	0.2	-0.1	0.4

Protocol Violation #2

Fifth, group members knew when deciding which measures to use for their "most proximal" and "probably blinded" meta-analyses that the parents rated the NFB group an exceptionally large effect size of 1.1 and a 0.0 effect size for the waitlist group, while the teachers rated NFB as having only a modest effect size of .2 and, as previously noted, an unprecedented .4 for the waitlist group—an effect size twice as large as the teachers rated NFB. With full access to this knowledge, the guideline group then chose the parents' ratings of NFB to include in their "most proximal" meta-analysis and use the teachers' ratings for their "probably blinded" meta-analysis despite their protocol specifically stating, "***If the intervention was implemented at SCHOOL, teacher ratings WERE NOT considered probably blinded assessments***" [emphasis added].

As part of the group's rationale for this protocol violation, Sergeant writes, "The post intervention ratings were made by different teachers than the ones that made the pre-treatment ratings due to teacher changes following treatment in the new school . . . in contrast, the researchers made no attempt to keep parents "blind" to the type of treatment."

Sergeant's claim would have a thin modicum of merit if the 4-month-long NFB and CT interventions ***CONCLUDED*** at the end of the school year and then, at the start of the new year, new teachers blind to which children had been removed during team time for NFB and CT were the ones who rated all subjects. The post-intervention ratings would then have been provided by teachers who were blind to which ADHD children were in the waitlist versus intervention groups. Just the opposite, though, is what occurred in the Steiner et al. study. In the methods section, Steiner et al. states, "The preintervention questionnaires were filled out at the time of enrollment ***at the end of the previous school year***, and the postintervention questionnaires were filled out within 1 month ***after the intervention***" [emphases added, Steiner et al., 2011]. ***Similar to the group's response to the Arns and Strehl letter claiming an "optimized active treatment" exclusion to their published protocol, hereto the group's rationale appears like another post hoc formulation attempting to justify their second violation to a clearly stated protocol.***

Lack of Scientific Rigor

Finally, it was a significant abdication of scientific rigor that Sonuga-Barke et al. did not blind group members to study outcomes when deciding on (a) the assignment of control group conditions; and (b) which measures to use for the "most proximal/probably blinded" meta-analyses, as well as not ensuring the consistent adherence to their clearly stated protocols regarding these matters. ***This abdication of scientific rigor is especially the case here, since this was the whole point of Sonuga-Barke et al.'s 12 meta-analyses, which was to test the significance levels of the effect sizes for the "most proximal" and "probably blinded" assessments.***

As Sergeant states, "Sometimes this decision was very straightforward at other times it was less so." This is the reason why researchers are taught to follow rigorous methods to guard against their own biases during such decision-making processes; ***that is what makes it SCIENCE!***

Rigorous methods would have given group members just the methods sections of all studies meeting their selection criteria with author, title and journal names omitted. Then, each group member would independently make the control group, "most proximal," and "probably

blinded" assignments with inter-rater reliabilities calculated and reported for each of their 12 meta-analyses including a similarly blinded method for resolving disagreements in adherence with their prespecified protocols. Such practices form the basics of scientific methodology and are necessary to protect Sonuga-Barke et al.'s 12 meta-analyses from potential bias either for or against specific treatments by blinding study outcomes when making assignments.

Our email to Sergeant asked specifically for evidence that the group had followed a method to ensure the internal reliability of their meta-analyses by keeping members blind to study outcomes when making assignments. While not answering our question directly, Sergeant's email response states they followed a consensus approach to these decisions with group members using ***"ALL AVAILABLE INFORMATION within the papers to make a judgment***," apparently, even when these "consensus judgments" trumped their clearly stated protocols.

It is disappointing that a group of 21 researchers participated in such a flawed methodology with apparently none insisting on scientific rigor to protect their meta-analyses from researcher bias either for or against particular treatments. Instead, with full knowledge of the Steiner et al. results, out of the ***FOUR*** possible comparisons for their "probably blinded assessment" (i.e., parent NFB rating versus CT or waitlist; teacher NFB rating versus CT or waitlist), ***the researchers picked the ONLY comparison negative to NFB and in the process violated their TWO clearly stated protocols for guiding this decision*** (see Table 1).

These two protocol violations tipped the group's overall analysis to where NFB narrowly missed reaching significance in their "probably blinded" meta-analysis versus being found efficacious as NFB would have been if any of the other three possible comparisons had been used. The researchers then concluded that NFB requires "better evidence for efficacy from blinded assessments" before it can be supported as a treatment for ADHD. ***This is not science; it is researcher bias and incompetence masquerading as science***.

Fundamentally, science is sabotaged when sound methodology is not followed as occurred in this group's meta-analyses as the 21 Sonuga-Barke et al. authors took no apparent efforts to protect against the biases of group members in the study's conduct, they included low-quality studies without assessing for their differential impact, and they failed to follow their own published protocols. ***These significant errors consign this group's study to the growing library of other meta-analytic studies exemplary of the "garbage-in-garbage-out" phenomenon of poorly conducted meta-analyses that publish findings with sweeping conclusions unconstrained by their substandard methods***.

Steiner et al.'s Full Trial Results

The NFB results from Steiner et al.'s subsequent full trial (*N* = 104) are particularly impressive (Steiner et al., 2014a). The full trial found NFB resulted in superior improvements in ADHD's core symptoms compared to both the CT and waitlist groups in executive functioning, parent ratings, teacher ratings, and blinded classroom observations as well as being the only group in which there were not significant increases in stimulant medication dosing during the study (Steiner et al., 2014a). Finally, the clear superiority of NFB over both the CT and waitlist conditions was then sustained in the 6-month follow-up assessment including on the medication dosing measure (Steiner et al., 2014b).

Given the group's protocol violations in how they evaluated the Steiner et al. preliminary study, other significant methodological errors, and, more importantly, the results from Steiner's full trial with 6-month follow-up, the weight of the evidence overwhelmingly favors Arns et al.'s (2014) conclusion that standardized NFB treatment protocols have demonstrated efficacy and, at minimum, a medium effect size for ADHD's core symptoms in randomized trials using semi-active and active control groups.

Who is the European ADHD Guidelines Group?

It is worth noting that the self-named "European ADHD Guidelines Group" is a workgroup of the European Network for Hyperkinetic Disorders (ENHD), and the guideline group's effort was funded by five pharmaceutical companies. Neither entity has any apparent official "European" status other than being made up of European researchers, many of whom have extensive conflicts of interests (COIs) with pharmaceutical companies. For example, the Sonuga-Barke et al. authors had a cumulative total of 114 reported COIs with pharmaceutical companies in their disclosure statement.

It is unfortunate that this group of researchers with such extensive pharmaceutical COIs decided to conduct 12 meta-analyses on non-pharmacological treatments for ADHD without ensuring rigorous methodological controls of their own biases. These biases were blatant in their handling of the Steiner et al. preliminary study and are likely present elsewhere in their other analyses if carefully reviewed. ***Our view is that the extent of scientific error warrants the article being retracted in its current form***. It is even more unfortunate that this article was published in the ***American Journal of Psychiatry***, a high impact journal, and has been widely cited as finding that four non-pharmacological treatments require "better evidence for efficacy from blinded assessments . . . before they can be supported as treatments for core ADHD symptoms," ***a finding that is clearly not true for NFB and perhaps other non-pharmacological treatments as well***.

Evidentiary Bias

Similar to Barkley, this network of researchers demonstrates significant evidentiary bias in how they assess the evidence-base of their preferred treatments for ADHD and neurofeedback. The ENHD group's website recommends psycho-education, parent training, school-based interventions and cognitive behavioral training as "evidence-based" treatments—***recommendations DIRECTLY counter to their OWN published meta-analyses in which these behavioral interventions performed the WORST out of the SIX non-pharmacological treatments they analyzed*** —while it lists NFB as an "area of controversy in the treatment of ADHD" and goes on to state that "more high quality, randomized controlled studies are needed to support neurofeedback training as a treatment for ADHD" (ENHD website).

It is unbecoming for a group of researchers to convey such false information to the public as to the relative evidence-base for different treatments. ***What's good for the goose is good for the gander***. Evidentiary standards should be evenly applied as is clearly not the case on the ENHD's website.

Perhaps it is only coincidental, but several of the more prominent members of the European research network receive royalties from behavioral intervention products for treating ADHD. For example, the conflict of interest section at the beginning of the article states, "Dr. Sonuga-Barke has been involved in the development, implementation, and trialing of the

New Forest Parenting Programme for preschool children with ADHD and has received royalties from sales of a New Forest Parent Training self-help manual" (Sonuga-Barke et al., 2013). Could it be that such conflicts of interest help explain why the network's website is so tolerant of evidentiary bias that favors their own group members' commercial interests but is directly counter to their own published findings?

Negative Findings in Recent Sham Trials

Three recent studies failed to find significant separation between "real" NFB and sham-feedback, raising doubts about the specificity of NFB effects. Two studies used non-standardized protocols and training procedures contrary to operant conditioning (see Arns et al., 2014 for detailed review) while the third randomized only 9 subjects, and the authors, therefore, did not evaluate for specific effects (Perreau-Linck et al., 2010).

The first study was an NIMH-funded feasibility trial that was ***explicitly designed NOT TO BE an efficacy study*** due to its small sample size (*N* = 39) and the study design randomizing treatment frequency. For blinding purposes, this study attempted to train decreased theta/alpha and increased SMR/beta using an automated procedure in which the EEG threshold necessary to play SonyPlayStation® videogames was reset every minute (up or ***DOWN***) based on subjects' immediately preceding EEG. The auto-threshold was set to ensure NFB subjects played videogames with full-control approximately 75 to 80% of the time during their sessions. When this NFB strategy failed to separate from sham-feedback, the authors noted that this may have been due to their inadequate NFB training protocol stating, "In fact, many NF experts feel that manually adjusted thresholds ***that remain fixed*** for periods of time work better than the fuzzy-logic moment-to-moment adjustments used in the CyberLearning technology used by us. ***Therefore, we do not have as much confidence in 30 treatments showing the maximal*** (NFB) ***effect***" [emphasis added; Arnold et al., 2013b].

The Lansbergen/van Dongen-Boomsma/Vollebregt Trilogy

The second trial involved an initial pilot study using similar auto-thresholding procedures as used by Arnold et al. (Lansbergen et al., 2011). The children assigned to NFB watched a movie 20 minutes per session for 30 sessions. Positive feedback was provided by both brightening the computer screen and presenting an auditory tone when the targeted brainwave frequencies remained above/below the targeted thresholds using a computerized auto-thresholding procedure that reset the threshold every "30 seconds so that the child was rewarded about 80% of the time (i.e., received positive feedback)" and "the amount of reward remained at about the same level across sessions and across groups. During training, children were instructed to try to self-regulate their brain activity by receiving positive feedback based on the real-time EEG signal . . . Training was conducted in an 'active focusing state' with eyes open" (Lansbergen et al., 2011).

When Lansbergen et al.'s NFB training protocol failed to separate from their sham feedback condition for the 14 pilot study subjects, the authors hypothesized that it might be due to their use of auto-thresholding and decided to instead have a therapist manually perform ***the same re-thresholding function*** for the additional 14 subjects randomized to NFB. In the first published article with these additional 14 NFB subjects, the authors state, "***Reward thresholds were manually adjusted so that the child was rewarded about 80% of the time*** (i.e., received positive feedback). ***Consequently, the amount of reward remained about at the same level across sessions and across groups"*** [emphases added; van

Dongen-Boomsma et al., 2013]. It is important to note that this is the same ***"about 80%"*** reward criteria maintained ***"at the same level across sessions and across groups"*** as in the pilot study's NFB training protocol except now NFB therapists were manually implementing it.

Two-Thirds Underpowered, Mid-Stream Changes and Selective Reporting

Despite changing their research design mid-stream (including adding undefined "active learning strategies . . . so that children could apply the learned strategies into daily life") and being ***two-thirds underpowered having randomized only 41 of the 120 intended subjects their own analysis had determined was necessary prior to starting the study***; the authors pooled their underpowered dataset and published two additional articles finding no specific NFB effects on behavioral and neurocognitive outcomes. The authors then claimed, "***The existing literature and this study fail to support any benefit of neurofeedback***" [emphasis added; Vollebregt et al., 2013). The Arnold and Perreau-Linck trials, along with the European guideline group's article, were a significant part of the "existing literature" used to support the authors' claim.

In their discussion section, Vollebregt et al. state, "***The most likely explanation*** why we did not find improvement of neurocognitive functioning after F-NF ***is that F-NF is not an effective treatment in ADHD***. This conclusion is in line with ***THREE*** recently published placebo-controlled F-NF studies reporting no superior effect on the core behavior symptoms of ADHD (Arnold et al., 2012; van Dongen-Boomsma et al., 2013; Lansbergen et al., 2011; Perreau-Linck et al., 2010)" [emphases added; Vollebregt et al., 2013]. ***Vollebregt et al. fail to acknowledge in this highly critical and misleading statement that***:

- Two of the four referenced studies were part of their own trial, and by the authors' own analysis, their study was two-thirds underpowered; to form even this underpowered dataset, the researchers pooled subjects after making two significant changes in their research design mid-way through the study;
- The Arnold et al. trial was explicitly a feasibility study, not an efficacy trial, and its authors specifically stated they did not have confidence in their NFB's results due to their study's minute-by-minute re-thresholding of the EEG reward criteria, contrary to the recommendations of, as they state, the "NF experts"; and
- The Perreau-Linck et al. study had only 9 subjects (7 completers), and the authors clearly state that "***the small sample size precludes from evaluating specific neurofeedback effects***" [emphasis added; Perreau-Linck et al., 2010].

Certainly, these facts from the referenced articles by others should have been acknowledged by Vollebregt et al. so that journal readers could then judge for themselves the extent to which the "existing research" supports the authors' claims.

Whose Neurofeedback Training Protocol did Lansbergen*/van Dongen-Boomsma/Vollebregt Follow?*

Another highly misleading assertion by Vollebregt et al. is when they state, "Thresholds were manually adjusted according to the expertise of the NF-therapist. ***NO specific guideline or protocol was followed***. ***This method was in line with the OBJECTIVE OF THIS STUDY to investigate the efficacy of F-NF as delivered in 'CARE AS USUAL', in which decisions about adjustments of the threshold are determined by the involved clinical***

NF-therapist. All of the NF-therapists were ***BCIA certified*** (Board Certification International Alliance)" [emphases added].

As made clear in van Dongen-Boomsma et al., instead of the automatically adjusted thresholds, the NFB therapists were to manually adjust them such that "the child was rewarded about 80% of the time." This decision was the ***RESEARCHERS' PROTOCOL ADAPTATION*** to maintain as best they could the same "about 80%" level of reward "across sessions and across groups" as they had in the pilot study with the NFB therapists using their software skills to continuously monitor each subjects' EEG training targets and then to the best of their ability adjust up or down the reward thresholds for each target so that "the child was rewarded about 80% of the time…across sessions." The NFB "therapists" were merely attempting to follow the ***RESEARCHERS' "about 80%" reward guideline/protocol.*** The NFB "therapists" did not use their NFB expertise to treat these children's ADHD; they used their knowledge of the NFB software to implement the researchers' protocol to the best of their ability, nothing more.

Vollebregt et al. add to their lack of scientific transparency by then claiming, "This method was in line with the ***OBJECTIVE OF THIS STUDY*** to investigate the efficacy of F-NF as delivered in '***CARE AS USUAL'***, in which decisions about adjustments of the threshold are determined by the involved clinical NF-therapist.**"** There is no evidence in the prior Lansbergen/van Dongen-Boomsma et al. articles for this claim on Vollebregt et al.'s part. This was certainly ***NOT*** **NFB "*CARE AS USUAL*"** treatment for ADHD or any other disorder. Yes, the NFB "therapists" utilized their NFB software expertise to implement to the best of their ability the researchers' "about 80%" reward guideline/protocol; but it was the researchers' protocol (including how the EEG training targets were selected in the first place) that the "therapists" were attempting to implement.

Vollebregt et al. then add further to the confusing and misleading presentation of their methods by stating, "All of the NF-therapists were ***BCIA certified***" as though this adds legitimacy to their claim that this was a study of NFB **"*CARE AS USUAL*,"** now with the added moniker, **"*as provided by BCIA certified therapists***." We do not doubt that the "therapists" were BCIA certified, but their BCIA training, certification, and NFB competence were not used in this study, only their expertise in using the NFB software in their attempts to adhere to the researchers' 80% reward protocol; ***NOTHING MORE!***

Vollebregt et al.'s disingenuous presentation misleads journal readers and the public-at-large into believing that theirs was a study of NFB "***CARE AS USUAL"*** for ADHD as provided by "***BCIA certified***" therapists and that their findings failed "***to support any benefit of neurofeedback***." Vollebregt et al. then repeat this false "***CARE AS USUAL***" claim three more times in their discussion section, thereby tarnishing further the BCIA certification accomplishment of those NFB therapists achieving this designation, discouraging other professionals from seeking BCIA Neurofeedback Board Certification, harming NFB therapists' professional reputation and ability to earn a living from their chosen profession, and most importantly, harming ADHD children and their families by discouraging them from seeking the best currently available first-line treatment for ADHD (Pigott & Cannon, 2014).

What "Active Learning Strategies" did the Researchers Use?

As stated previously, the van Dongen-Boomsma**/**Vollebregt et al. researchers added "active learning strategies" mid-stream during their study "so that children could apply the learned strategies into daily life," without describing what these "learning strategies" were.

Furthermore, they did not state what they did to ensure that these strategies were in fact learned; otherwise, how could the children then apply them outside the treatment setting "into daily life"?

It is only by reading the Lansbergen et al. pilot study's discussion section that we learn that "a third limitation of this study is the lack of actively practicing mental strategies to self-regulate brain activity (e.g., Gevensleben et al., 2009). In other words, EEG-neurofeedback might need ***explicit*** learning rather than ***implicit*** learning . . . Based on these results . . . ***we will assist children in developing and practicing active learning strategies to self-regulate brain activity*** (e.g., focusing one's attention) ***and promote the children to implement the acquired strategies in daily-life situations, aimed at optimizing the therapeutic effects of EEG-neurofeedback training*** (see Gevensleben et al., 2009)" [emphases added; Lansbergen et al., 2011].

We reviewed Gevensleben et al. to ascertain what "active learning strategies" van Dongen-Boomsma/Vollebregt et al. are referencing that make NFB learning "***explicit***" rather than "***implicit.***" Gevensleben et al. state, "***Transfer trials***, i.e., trials without contingent feedback, were also conducted (about 40% at the beginning of a training block and about 60% at the end of a training block). ***The children of the NF group were required to practice their focused state (which was practiced in the sessions) at home, in different situations*** (one situation per day, e.g., 'try to be very focused while reading', 'try stay focused on the ball while playing football this afternoon')." Gevensleben et al.'s use of transfer trials and home practice is the same as that used in Gani et al.'s (2008) two-year follow-up study concluding that, "***Neurofeedback appears to be an alternative or complement to traditional treatments. The stability of changes might be explained by normalizing of brain functions that are responsible for inhibitory control, impulsivity and hyperactivity.***" Gevensleben et al. (2010) found a similar maintenance of NFB treatment gains in their 6-month follow-up assessment.

So again we ask, "What 'active learning' methodology did van Dongen-Boomsma/Vollebregt use?" They present no evidence of implementing Gevensleben et al.'s transfer trial strategies that do, in fact, make NFB learning "***explicit***" rather than "***implicit***." This second mid-stream adaptation by van Dongen-Boomsma/Vollebregt et al. is not only a confounding variable in their study, ***but by leaving it undefined, the authors tarnish the value of these researchers' thoughtful approach as well as the continued research efforts on how best to generalize NFB learning in the laboratory to everyday life situations***.

News Alert: ADHD Children are as Smart as Hungry Pigeons

While the Lansbergen/van Dongen-Boomsma/Vollebregt trilogy acknowledges that NFB "is based on the rationale that voluntary modulation of specific brain activity patterns can be learned by operant learning strategies" (Vollebregt et al., 2013), their methodology fails basic principles of operant conditioning. As behaviorists know, learning will not occur if the reinforcement criteria are made more or less stringent in order to maintain a high frequency of reward (e.g., Thorndike, 1932). The issue is not auto-thresholding versus "therapists" performing the same function, rather it is the failure to shape the EEG. For example, if hungry pigeons are fed a pellet approximately 8 out of 10 times they peck with the accuracy criteria made more or less stringent to maintain this high frequency of reward for 30, 40, or 100+ sessions, they will be no more accurate in pecking the target when they finish "treatment" than when they started, since pecking accuracy was not shaped.

Similarly, NFB subjects' EEGs were not reliably shaped, since regardless of their success at producing the desired EEG, they watched movies/played SonyPlayStation® videogames without disruption approximately 70 to 80% of the time. Furthermore, watching movies/playing videogames was likely far more reinforcing than striving to control the intermittent disruptions, particularly since most NFB subjects would soon learn that they could not control these disruptions on a sustained basis anyway, as their EEG thresholds were always being reset according to the authors' "about 80%" reward protocol regardless of how hard they tried . . . so why bother? ***The smart strategy for all subjects, therefore, was to simply ignore these minor disruptions, and instead, enjoy the entertainment similar to hungry pigeons happy to be fed for aimless pecking***.

This "***kick back and enjoy the show mindset"*** is what occurred, since in a subsequent article, the authors state, "Most participants of EEG-NF placebo-controlled RCTs conducted until now seem to experience the treatment as a placebo condition" and then cite all three articles from their trilogy along with one other (Vollebregt et al., 2014).

Given both the Arnold et al. and Lansbergen/van Dongen-Boomsma/Vollebregt et al. studies' flawed NFB training methodology, it is not surprising that their blinds were not broken and no evidence of specific effects found since both studies essentially compared sham treatments. Arnold et al.'s failure to shape NFB subjects' EEGs is made clear when they reported that "the sham group (***AS WELL AS ACTIVE GROUP***) showed no obvious EEG changes in a simple pre–post measure of theta/beta ratio" [emphasis added; Arnold et al., 2013a].

Furthermore, Vollebregt et al. found far more evidence of the ***NEGATIVE SHAPING*** of NFB subjects' EEGs away from their training targets than shaping in the desired direction. Their results section states, "EEG-data during the sessions were available for 10 children . . . Seven children showed a change in power towards ***ONE*** of the training targets. However, the variability between sessions was great and no children showed such a desired change in more than ***ONE*** frequency-band. ***Moreover***, ***ALL CHILDREN*** additionally showed a change in power ***AWAY FROM A TRAINING TARGET***" [emphases added; Vollebregt et al., 2013].

In the table of the EEG training targets for all 22 NFB subjects, 14 had two EEG training targets and 8 had three targets for an average of 2.4 EEG training targets per NFB subject (supplemental Table 2; van Dongen-Boomsma et al., 2013). While the authors report that there was EEG data available for only 10 NFB subjects, assuming that these subjects were representative of the NFB group as whole, there were approximately 24 EEG training targets on which data was available to evaluate the ability of their training methodology to shape subjects' EEG. Out of approximately 24 EEG training targets, Vollebregt et al. report a change in power in the desired direction in only seven (***29%***), with a minimum of ten changes in power in the negative direction and possibly 17 or more instances of the ***NEGATIVE SHAPING of NFB subjects' EEG*** (***range: 42 to 71+%***).

Therefore, despite the researchers instructing the NFB subjects "to attempt to self-regulate their brain activity by receiving positive feedback" and given that the "reward thresholds [were] manually adjusted so that the child was rewarded about 80% of the time***"*** (van Dongen-Boomsma et al., 2013), ***the ADHD children proved to be as smart as pigeons.*** The NFB subjects quickly learned that any of their attempts to "self-regulate their brain activity by receiving positive feedback" were futile because the reward criteria was intermittently being changed to maintain the researchers' "about 80%" positive feedback protocol. ***So like hungry pigeons happy to be fed for aimless pecking, the NFB subjects soon learned to just kick back and enjoy the show.***

This study's findings are as expected, since how can learning take place when the accuracy criteria necessary to receive "positive feedback" is continually changed to maintain a reward frequency of "about 80%" even when the participants are getting worse in performing the behavior to be learned? The Lansbergen/van Dongen-Boomsma/Vollebregt trilogy proves that it cannot, as did Thorndike, Skinner and many other behaviorists long before. ***Bad science confirms the good; how fitting.***

Subterfuge versus Acknowledging the Evidence

In their discussion section, Vollebregt et al. fail to acknowledge the evidence indicating that their training methodology negatively shaped NFB subjects' EEGs away from their training targets and then offered this fact as the most likely reason that their NFB protocol failed to separate from sham feedback. If, as Vollebregt et al. state, NFB "is based on the rationale that voluntary modulation of specific brain activity patterns can be learned by operant learning strategies," certainly evidence demonstrating the negative shaping of NFB subjects' EEGs warrants discussion by the authors. Instead of acknowledging this evidence, Vollebregt et al. only state in their discussion section that ***"NOT ALL desired training directions were met***" when they should have been forced by competent peer and editorial review to acknowledge the fact that there was far more evidence of the negative shaping of NFB subjects' EEGs away from their training targets than shaping in the desired direction. Such an acknowledgement is the minimum standard of researcher objectivity when discussing study findings.

Methodology Matters

Concerned about the proliferation of unsound practices, many leading researchers published a consensus statement on NFB and basic learning theory (Sherlin et al., 2011). Key points included:

- How readjusting the EEG threshold up and down during NFB sessions to maintain a high frequency of reward violates the principal of shaping and may in fact shape it, "***in the opposite direction than the desired training parameter***" as occurred in Lansbergen/van Dongen-Boomsma/Vollebregt et al.;
- Monetary or other secondary reinforcers should be based on success shaping the EEG, ***NOT*** mere participation in NFB;
- Encouraging strategies to promote generalization such as brief transfer trials where trainees attempt to activate the targeted cortical activity via self-regulation alone without real-time feedback and only learn if they were successful at the end of the trial; and
- Sessions should "stress exercise rather than entertainment" with the reinforcement leading to "knowledge of results" and informing "the learner whether the response was right or wrong and to what extent the brain signal changed."

While exemplifying unsound methodologies (including giving reinforcers for participation, not performance), these failed trials significantly advance scientifically-based NFB treatment by demonstrating that flawed practices fail to shape trainees' EEGs and consequently render outcomes indistinguishable from sham feedback. Using similarly flawed methodologies (e.g., re-thresholding to ensure subjects watched movies disruption-free 70 to 85% of the time and giving reinforcers for participation, not performance), Ogrim and Hestad (2013) also found such practices failed to shape trainees' EEGs.

The negative findings from these studies are in marked contrast to those from Leins et al. (2007) whose methods closely followed those in the consensus statement including the use of transfer trials and providing secondary reinforcers based on subjects' performance learning to self-regulate their EEG. This study compared theta/beta ratio and slow cortical potential training and found that both groups (a) learned to self-regulate cortical activity consistent with their NFB training protocol; (b) had significant improvements in the core symptoms of ADHD as well as IQ; (c) EEG self-regulation was maintained when reassessed six months and two years later; (d) a two-year follow-up found further significant improvements in behavior and attention; and (e) in each group, half of the children no longer met the criteria for ADHD (Gani, Birbaumer, & Strehl, 2008). Other evidence of NFB's specificity are studies finding significant neurophysiological effects including protocol-specific effects on event-related potential (ERP) components in attention tasks and neural substrates of selective attention imaged with fMRI (see Arns et al., 2014). In addition to Leins and Steiner finding strong evidence of sustained improvement in follow-up assessments of methodologically sound NFB procedures, every other NFB study that has included follow-up assessments has found similar evidence of sustained improvements in protocol-specified EEG and ADHD core symptoms. ***No other ADHD treatment has demonstrated credible evidence of sustained benefit following treatment termination***; ***NONE*** (Pigott & Cannon, 2014).

Conclusion

It is our assessment that the critics' claims that neurofeedback lacks sufficient evidence of efficacy and, therefore, cannot be considered a legitimate treatment for ADHD do not stand up to careful scrutiny. The critics making these claims display significant evidentiary bias in how they evaluate neurofeedback compared to treatments in which they have commercial interests (e.g., Barkley's website; ENHD's website), and the scientific rigor of methods supporting their conclusions are questionable.

The Sonuga-Barke et al. and Vollebregt et al. articles bring into question the scientific rigor adhered to during these studies' peer review and publication process. Sandra Loo, Russell Barkley, and the authors of the other two studies have all been invited to give a formal response to the methodological issues we have raised. Our view is that ***ALL*** authors should either defend the scientific integrity of the claims to which their names are assigned or formally request that their articles be retracted. Anything less is an abdication of scientific duty on their part and a de facto acknowledgement that they have no credible response.

In contrast to these critics' claims, the actual evidence is that neurofeedback, when administered in a way that is consistent with the principles of operant conditioning and using standardized treatment protocols (Arns et al., 2014), is the best currently available first-line treatment for ADHD (Pigott & Cannon, 2014). With full acknowledgement of our own commercial interests as neurofeedback professionals, this is a claim we will vigorously defend and invite critics of neurofeedback to challenge.

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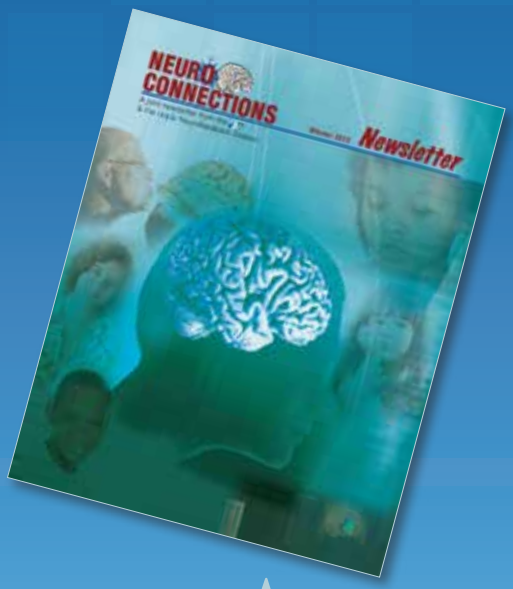

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NeuroRegulation

When Discussing Neurofeedback, Does Modality Matter?

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Abstract

Over the years, several new models and variations of neurofeedback (NF) have been developed. As such, NF has grown from traditional amplitude based modalities to now include slow cortical potential NF, as well as various approaches grounded in QEEG technology, including *z*-score NF models. These differing modalities have important implications in terms of outcomes, the number of sessions required, and treatment specificity. This, in turn, impacts clinical practice, research, and marketing considerations. In an effort to gain some perspective for where the field is today, a comparative review is presented to illustrate the importance of noting what particular modality is being referenced when discussing NF.

Keywords: neurofeedback, traditional NF, slow cortical potential NF, QEEG-guided NF, *z*-score NF

Introduction

If evaluating neurofeedback (NF) from an evidence-based perspective is important, and we propose that it is, then distinguishing between modalities is an essential component of that process. Over the years, several new models and variations of NF have been developed, which directly impacts practice implications in terms of outcomes, the number of sessions required, and treatment specificity. Thus, noting which modality of NF is being implemented is a necessary step in discussing any NF intervention.

In the 1970s, when NF was first being developed by the likes of Joe Kamiya, Barry Sterman, and Joel Lubar, the approach was generally the same, that of targeting amplitudes of specific frequency bands. However, since then, many other models, and variations of models, have been developed. While the standard for much of the history of NF was the use of only one or two electrode sites to train various EEG frequencies, today there are new models which use as many as 19 electrodes while incorporating real-time database metrics and targeting cortical regions of interest. Therefore, the current range of NF modalities include traditional amplitude-based NF (Theta-Beta, Sensory-Motor Rhythm [SMR], Alpha-Theta), slow cortical potential (SCP) NF, quantitative EEG (QEEG) guided NF (QNF), and *z*-score NF (ZNF) models including 4-channel, 19-channel surface, and l*ow resolution* brain electromagnetic tomography (LORETA) ZNF (also see Figure 1, which presents a graphic of this collection of models, in timeline format, to provide a better context for the development of these models over time).

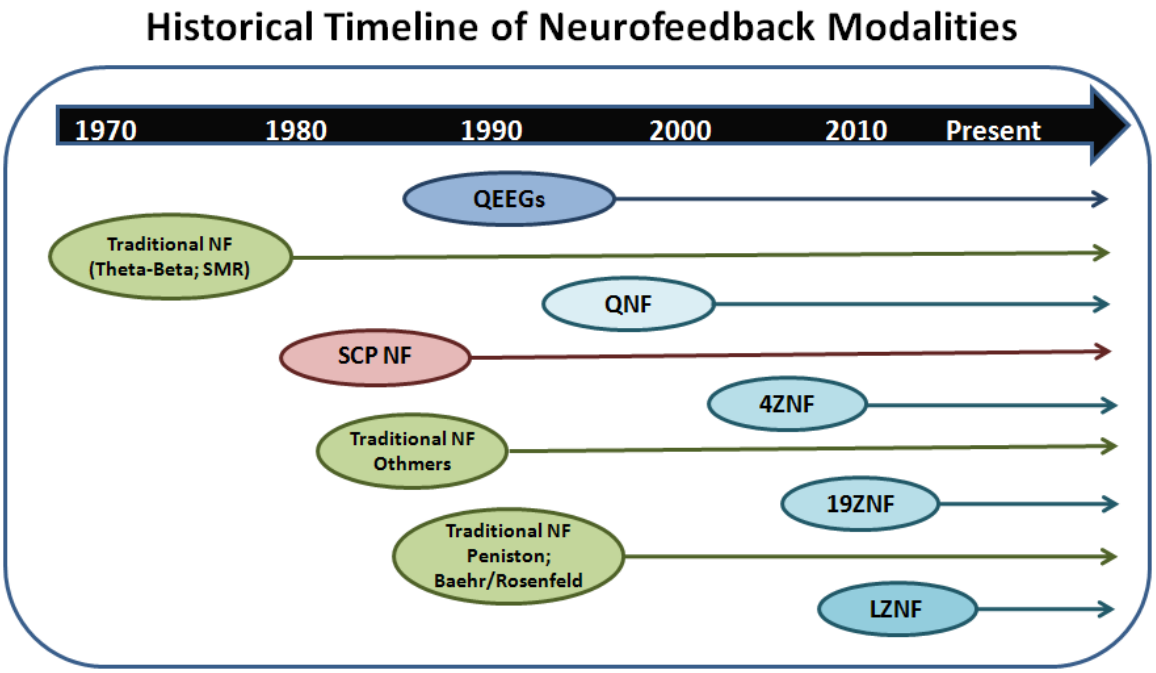

Figure 1. Timeline of when various neurofeedback modalities emerged.

NF models based on Theta-Beta bands and SCP have robust evidence-based research support; however, treatment sessions number from thirty to over forty and the specificity is limited to surface neuroregulation. In contrast, the evidence-based research is limited in newer models, such as the 19-channel and LORETA *z*-score models, which have been around since 2006 when the NF *landscape* changed to include ZNF. Yet, treatment outcome reports of the 19-channel and LORETA *z*-score models indicate that treatment goals can be achieved in fewer sessions, as more networks and deeper structures can be targeted (Thatcher, 2013; Wigton, 2013). Hence, depending on the modality, the amount of treatment sessions and specificity will make a difference in how NF is understood and/or is identified as a choice of treatment. For this reason, when addressing NF, the length of treatment and specificity are important in order to identify what modality of NF is being referenced. This is also important in terms of being an informed consumer of NF research in the literature (scientific).

In 2012, the American Academy of Pediatrics (AAP) elevated biofeedback (to include NF) to a *Level 1/Best Support* recommended intervention. However, the three NF studies evaluated to make this determination (PracticeWise, 2012) only included Theta-Beta and SCP NF (Beauregard & Levesque, 2006; Gevensleben et al., 2009; Levesque, Beauregard, & Mensour, 2006), and did not include newer models (i.e., QNF or ZNF). As a result, it is important to be aware of the scope of this recommendation. Moreover, the non-inclusion of newer models (such as QNF or ZNF) may highlight the need for further empirical support, with solid methodologies, so as to elevate the evidence-based support matching that of Theta-Beta and SCP models; because the modality of NF used guides the treatment outcome, specificity and the number of sessions, which may vary between modalities.

Although all NF is informed by a basic framework of operant conditioning and learning theory (Sherlin et al., 2011), the variation in the application of that framework presents the NF clinician and researcher with many choices. Consequently, this comparative review is presented to gain some perspective of where the field is today with respect to the traditional NF, SCP NF, QNF, and ZNF models.

It is important to note there are other neuroregulation applications, which are also frequently referred to as NF, and are beyond the scope of this article. These include hemoencephalography (HEG), *low-energy neurofeedback system* (*LENS)*, low intensity pulsed electromagnetic field (pEMF) stimulation, audio-visual entrainment (AVE), and real-time functional magnetic resonance imaging (fMRI) feedback, some of which may be considered adjuncts to NF treatment. So, due to the growing variety of neuroregulation techniques included under the overarching umbrella of NF, with differences in treatment outcome, number of sessions, and specificity, it is critical to be clear about which modality is being referred to when discussing NF.

Synopsis of NF Modalities, Treatment Outcomes, Number of Sessions, and Specificity.

Traditional NF. In the context of this review, the term *traditional* is used to describe the models that are primarily amplitude based. These are the longest standing iterations of NF and can be traced back to the original founders of the field (Kamiya, 1968; Sterman & Friar, 1972; Lubar & Shouse, 1976), including models where the targeted frequencies are Theta-Beta, SMR, or Alpha-Theta. While most NF practitioners who have been in the field for more than a decade are likely to be familiar with these models, newer entrants who may start with learning more recent NF models may not. Therefore, those not familiar with the historical aspects of NF are encouraged to acquaint themselves with the beginning works in the NF field (see Budzynski, 1999; Evans & Abarbanel, 1999; Robbins, 2000; Thatcher & Lubar, 2009). Other models, which have developed from this perspective, are symptom-based approaches such as the earlier Othmer models (Othmer, Othmer, & Kaiser, 1999) where, generally, one frequency band is rewarded (either 12–15 Hz or 15–18Hz), while two other frequencies (Theta and High Beta) are inhibited. Still other models, which can be traced back to Kamiya's work, target parietal Alpha and/or Theta frequencies to enhance relaxation and creativity states (Budzynski, 1999), which Peniston and Kullkosky (1990, 1991) developed further, leading to treatment models for post-traumatic stress disorders and alcoholism. Finally, Baehr, Rosenfeld, and Baehr (1997) created protocols with the goal of balancing frontal alpha as a treatment for depression.

Slow Cortical Potential (SCP) NF. As described by Mayer, Wyckoff, and Strehl (2013), SCPs are very slow brain activation electrical shifts, generated subcortically and cortically, which alternate between being electrically negative or positive. Further, central to this model is an event related potential termed the contingent negative variation (CNV), such that

reduced CNVs have been found to be associated with attention deficit hyperactivity disorder (ADHD) symptoms; and SCP feedback has been shown to lead to an increased CNV. Therefore, in SCP NF, with an active electrode at Cz, the training goal is to generate shifts between surface-positive and surface-negative SCPs. The sessions typically consist of two to four runs made up of approximately 40 trials in each run. SCP NF is different from many traditional NF models in that there is only a single protocol used, with only slight variants, and has been designated by Mayer et al. as a *one size fits all* approach. Reports investigating the self-regulation of SCPs date back to the mid to late 1980s (Roberts, Rockstroh, Lutzenberger, Elbert, & Birbaumer, 1989; Rockstroh, Birbaumer, Elbert, & Lutzenberger, 1984; Rockstroh et al., 1993).

Traditional NF and SCP NF treatment outcomes, number of sessions, and specificity. Over time, the Theta-Beta and SMR variations of traditional NF, together with SCP NF, have come to be some of the most researched, specifically for the condition of ADHD (Arns, Heinrich, & Strehl, 2014). This is further demonstrated by many reviews and meta-analyses available (Arns, de Ridder**,** Strehl, Breteler, & Coenen, 2009; Arns et al., 2014; Brandeis, 2011; Gevensleben, Rothenberger, Moll, & Heinrich, 2012; Lofthouse, Arnold, Hersch, Hurt, & DeBeus, 2012; Mayer et al., 2013; Niv, 2013; Pigott, De Biase, Bodenhamer-Davis, & Davis, 2013). Overall, this collection of literature provides strong support for NF being considered efficacious for ADHD.

While the traditional NF models may vary with respect to which EEG frequencies are trained, elements common to them are targeting amplitudes and a need for 40+ sessions, on average, for successful clinical outcomes. Regarding SCP, in their review of SCP NF studies for ADHD, Mayer et al. (2013) report the number of sessions ranged from 25 to 35. Thus, while SCP NF is frequently investigated and reported alongside traditional NF, it is possible that SCP NF may require somewhat fewer sessions that traditional NF.

However, with a reliance on only one or two electrode placements, specificity in traditional and SCP NF is limited. At any individual electrode location, the EEG recording includes diffuse sources (both close and distant) that are picked up from other brain areas (Thatcher, 2013). Thus, with traditional NF, EEG recordings from an electrode at Cz will actually include a blend of the EEG signal from all other areas of the scalp, to varying degrees. Yet, traditional NF still employs choices of different frequencies at varying sites, depending on the particular symptoms or case presentation. However, with SCP NF, generally there is a single electrode on the central strip, typically at Cz, with a singular protocol applied to all clients (Mayer et al., 2013).

QEEG-guided NF (QNF). In their recent review of NF for ADHD, Arns et al. (2014) suggest that research efforts should focus on NF protocols that are tailored to the individual, as many studies have suggested clinical outcomes can be improved with a personalized approach to NF. QNF allows for a tailored approach to individual treatment. QNF is grounded in QEEG technology dating back to the late 1980s; it has been improved through the years, so that the treatment can be tailored given the particular QEEG baseline, clinical status, and history of the client (Arns, Drinkenburg, & Kenemans, 2012). This then allows for individualized treatments of differing electrocortical presentations, even with the same or similar overarching diagnoses (Hammond, 2010). Multiple reports in the literature support this clinical approach indicating how training the deviant *z*-scores towards the mean (i.e., normalize the QEEG) yields clinical benefit (Arns et al., 2012; Breteler, Arns, Peters, Giepmans, & Verhoeven, 2010; Collura, 2008a; Orgim & Kestad, 2013; Surmeli, Ertem, Eralp, & Kos, 2012; Surmeli & Ertem, 2009, 2010; Walker, 2009, 2010a, 2011, 2012a). Therefore, QNF still targets amplitudes of frequency bands, but does so in an effort to

normalize the excessive *z*-score deviations shown in the QEEG that correspond to the overall clinical picture.

QNF treatment outcomes, number of sessions, and specificity. QNF studies are well represented in peer-reviewed literature; however, no meta-analysis of QNF has been identified to-date. Yet, the current QNF studies in the literature do report positive treatment outcomes. Moreover, while traditional and SCP NF mostly focus on ADHD symptoms, QNF studies report covering a wider range of symptoms, syndromes and disorders that include, behavior aspects, mood, cognitive dysfunction, epilepsy, head injuries, autism spectrum, migraines, learning disorders, schizophrenia, post-traumatic stress disorder, Down syndrome, and intellectual disability (Arns et al., 2012; Breteler et al., 2010; Coben & Myers, 2010; Huang-Storms, Bodenhamer-Davis, Davis, & Dunn, 2006; Koberda, Hillier, Jones, Moses, & Koberda 2012; Surmeli et al., 2012; Surmeli & Ertem, 2007, 2009, 2010, 2011; Walker, 2009, 2010b, 2011, 2012b, 2013). This literature collection largely represents retrospective investigations from clinical settings.

While some QNF studies provide results from research where the number of sessions was in the range of around 20 sessions (Breteler et al., 2010; Walker, 2009) others have reported as many as 50 or 100 sessions (Surmeli & Ertem, 2009, 2011). However, as with traditional NF, the number of sessions for QNF typically remains on an average of 40 (Thatcher, 2013; Wigton, 2013).

Specificity with QNF, however, improves by being able to individually tailor the protocol to the needs of the client as directed by the QEEG findings and clinical assessment. This is accomplished with protocols that target specified sites with a goal of normalizing identified deviant *z*-scores. Thus, this NF modality takes into account the heterogeneity of QEEG patterns, more so leveraging the sensitivity, reliability, and specificity of the QEEG, as discussed by Hammond (2010).

***Z*-Score NF (ZNF).** Rather than targeting frequency amplitudes, as with traditional NF or QNF, in ZNF, the training targets are the calculated real-time QEEG *z*-scores integrated into the NF software. As a result, real-time QEEG assessment metrics can be paired with operant conditioning and incorporated into the NF session (Collura, 2014; Thatcher, 2013). Thus, ZNF capitalizes on the statistical foundation for a normal distribution, where a value converted to a *z*-score is a measure of the distance from the mean of a population, such that the mean represents a range considered to be typical (Collura, 2014). This brings a new dynamic to the NF process. The focus is no longer on making more or less of a particular frequency, but rather moving excessive live (i.e., real-time) *z*-score metrics towards the mean (i.e., $z = 0$), thereby placing more emphases on normalizing the QEEG values. ZNF allows for more metrics to be targeted in a NF protocol as active training components — up to ten frequency bands, both absolute and relative power, frequency power ratios, and the connectivity metrics of asymmetry, coherence, and phase lag. Yet, consistent with being an outgrowth of the QNF model, normalization goals are still governed by the presenting clinical picture. ZNF is one of the newest models, but even within this category, three distinctions can be made to include 4-channel ZNF, surface 19-channel ZNF, and LORETA ZNF; each are discussed as follows:

4-Channel ZNF (4ZNF). The 4ZNF model was the first iteration and was introduced in 2006, with the total number of metrics available to train being 248 (Collura, 2014). However, while the reference to *4-channel* implies only four channels can be trained, there are also options for one or two channel ZNF training as well; thus this can be considered an *up to* 4 channel

model. Here the *z*-scores are calculated from the surface recordings; yet, there is still access to all metrics/components as described above (i.e., power, connectivity, etc.).

19-Channel ZNF (19ZNF). 19ZNF was made available as an outgrowth from 4ZNF in 2009, and the total number of metrics for protocol selection is 5700 (Collura, Thatcher, Smith, Lambos, & Stark, 2009). Again, in 19ZNF, the surface EEG recording is the source for the real-time *z*-score calculations. However, unique to 19ZNF, in addition to the linked-ears montage, is the availability to directly train the Laplacian montage. Yet, as with 4ZNF, so long as the linked-ears montage is applied, any number of channels up to 19 can be included in a protocol, thus allowing the use of any number of electrodes from one to 19.

LORETA ZNF (LZNF). While there have been investigations of non-*z*-score LORETA NF (examples include Cannon, Lubar, Sokhaze, & Baldwin, 2008; Cannon, Congedo, Lubar, & Hutchens, 2009; Cannon, Baldwin, Diloreto, Phillips, Shaw, & Levy, 2014), LZNF was first introduced in 2010. Even though similar in many respects, there is a fundamental difference between surface ZNF and LZNF. Surface ZNF calculates the *z*-score of identified EEG metrics at various 10-20 electrode sites, whereas with LZNF, the *z*-score is calculated for a particular collection of current source density (CSD) voxels. This makes it possible to conduct NF with the *z*-scores of the calculated location of deeper cortical dipole generating regions or structures (i.e., Brodmann areas, cingulate gyrus, precuneus, etc.). Consequently, isolated cortical regions of interest can be identified for normalization with this modality.

ZNF treatment outcomes, number of sessions, and specificity. Currently, case reviews, technical reports, and discussion about ZNF can be found in books (Collura, 2014; Thatcher, 2012), and publications such as that of Collura (2008b), Genardi (2012), Koberda (2012), Thatcher (2008, 2013), and Wigton (2013). Additionally, there have been conference presentations such as Koberda, Moses, Koberda, and Koberda (2012a), Rutter (2011), and Wigton (2010). Nevertheless, while it is hoped there are more studies in preparation, currently, there are few examples of *empirical investigations* of ZNF in *peer-reviewed* literature evaluating the outcomes of ZNF. There are two such publications that investigate 4ZNF, the first of which is a study by Collura, Guan, Tarrant, Bailey, and Starr (2010) that presented a collection of clinical reports from six clinicians covering 24 successful cases, with an average of 21 sessions per case, and all clients reported clinical improvement. The second publication, by Hammer, Colbert, Brown, and Ilioi (2011), is a randomized, controlled, single-blind study with small sample size (n = 3 and n = 5) that suggests 4ZNF is beneficial for insomnia. For 19ZNF, examples include two single case study evaluations: Hallman (2012) described a case of a child with fetal alcohol syndrome where 80 sessions of 19ZNF brought about remarkable improvements and QEEG normalization; then, Koberda, Moses, Koberda, and Koberda (2012b) described a case where both 19ZNF and LZNF were used with a 23-year-old male, where improvements in cognitive and QEEG assessments were achieved in 15 sessions. For LZNF, one study presented a review of four cases: Koberda, Koberda, Bienkiewicz, Moses, and Koberda (2013) applied LZNF to treat chronic pain and all cases reported improvement.

In terms of number of sessions, ZNF employs simultaneous training of multiple *z*-score metrics (i.e., power, connectivity) at many more electrode sites (up to 19), within either surface or LORETA frameworks. This allows for more neuroregulation and enhanced QEEG normalization. Consequently, in clinical reports of ZNF it is suggested that positive clinical outcomes can be achieved in an average of 10 to 20 sessions (Collura et al., 2010; Koberda et al., 2012b; Wigton, 2013).

The available *z*-score metrics and multiple channels of the ZNF modalities greatly enhance NF specificity. With surface 4ZNF and 19ZNF, the dysregulated areas and rhythms can be isolated for normalization, and even more so with the Laplacian montage (Thatcher, 2013). With LZNF, brain networks and hubs can be identified and targeted as regions of interest for training. By directly targeting these regions of interest, in a *z*-score framework, it is reported that LZNF allows for specificity and localization similar to that of fMRI methods (Thatcher, 2013). This allows for the linkage of functional systems with the presenting clinical symptoms, such as when identified brain regions (with deviant *z*-scores) are being reinforced towards the mean (i.e., $z = 0$) to promote increased stability and homeostasis in brain function (Koberda et al., 2013).

Conclusion

In sum, while all NF is grounded in operant conditioning, it is clear there is a wide range of approaches today. As a result, misunderstandings and misinterpretations can happen if the modality being addressed is not noted. This can have implications for clinical practice, research, and marketing, as has been addressed through this review. For instance, although the AAP recommended that biofeedback/NF be elevated to a *Best Support* intervention, would all NF modalities be paid if NF is accounted for as a managed care paid intervention (even though the decision seems to be based on traditional NF)? In research, each NF modality may have different levels of efficacy, number of sessions, and treatment specificity, which directly impacts methodology and implications. And, in marketing, a frequent question of NF consumers is how long does the treatment take or how many sessions are needed? In answering these questions, it is clear that the treatment and amount of sessions is guided by the NF modality utilized. Thus, when discussing NF, does modality matter? Yes, it does matter.

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NeuroRegulation

Toward Development of Sham Protocols for High-Definition Transcranial Direct Current Stimulation (HD-tDCS)

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Abstract

High-definition transcranial direct current stimulation (HD-tDCS) is a noninvasive cortical stimulation (NICS) technique that, due to the utilization of multi-electrode stimulation, may enable development of sham conditions characterized by indistinguishable scalp sensations compared to active conditions, with little or no cortical influence. We sought to contribute to the development of an optimal sham electrode configuration for HD-tDCS protocols by gathering ratings of overall sensation reported by participants during different electrode configurations and current intensities. Twenty healthy participants completed a magnitude estimation task during which they rated their “overall sensation” in 1-minute intervals during five 5-minute stimulation conditions. A 5 x 5 (Time x Stimulation condition) analysis of variance (ANOVA) was conducted to determine if sensation measurements differed over time, and how this varied by condition. Null hypothesis significance tests and equivalence tests were conducted to determine which sham conditions were statistically indistinguishable from the experimental condition. The ANOVA revealed main effects for Time and Stimulation condition. Planned comparisons, comparing each sham condition to the experimental condition (4x1 ring configuration, 2 mA), revealed differences in sensation ratings for all but one condition (Sham 1x1A); no sham conditions were found to be statistically equivalent to the experimental condition. Our HD-tDCS findings build upon previous NICS reports of differences in sensation ratings between sham versus experimental conditions when traditional “ramping down” approaches were used. Alternative multi-electrode configurations that manipulate electrode placement to shunt current across the scalp warrant further investigation as valid blinding methods.

Keywords: HD-tDCS; sham; sensation; tDCS

Introduction

Noninvasive cortical stimulation (NICS) techniques, such as transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS), are useful for investigating brain-behavior relationships in healthy populations and have also begun to be used therapeutically in clinical populations (Brunoni et al., 2012; Fregni & Pascual-Leone, 2007; Williams, Imamura, & Fregni, 2009). TMS directly alters cortical excitability via application of a magnetic stimulus to the scalp that travels through overlying matter to influence discrete cortical areas. Depending largely upon temporal patterning and stimulus intensity, cortical effects can be inhibitory or excitatory (Chrysikou & Hamilton, 2011; Vallence & Ridding, 2013). tDCS modulates cortical excitability via application of a weak electrical stimulus to the scalp (1–2 mA) through two surface electrodes. The weak stimulus travels through overlying matter to diffusely and indirectly influence cortical excitability. Much remains to be learned about dose-response relationships for tDCS, but in general the area under the cathodal surface electrode is more inhibitory and the area under the anodal surface electrode is more excitatory (Chrysikou & Hamilton, 2011; Vallence & Ridding, 2013).

Just as placebo trials are fundamental for proving drug effectiveness in pharmacological research, the ability to blind both experimenters and subjects to stimulation condition is important for unbiased interpretation of NICS results and is accomplished via “sham” forms of NICS. Pharmacological investigations often employ an active placebo (to induce side effects in absence of target effect) to avoid unintended unblinding of participants by absence of side effects (e.g., Moncrieff, Wessely, & Hardy, 2004). Similarly, TMS investigations often utilize active sham conditions (e.g., change in angle of coil orientation with unchanged or reduced stimulus intensity) that produce comparable scalp sensations with reduced cortical effect (e.g., Deng & Peterchev, 2011; Loo et al., 2000). tDCS protocols generally utilize the “fade in – short stimulation – fade out” approach, where the current is ramped down following a brief period of delivery designed to induce initial sensations that are thought to fade (Gandiga, Hummel, & Cohen, 2006). In fewer instances, active control conditions (e.g., current delivery to cortical area thought to be unimportant to experimental task; Boggio et al., 2008) or low-current conditions (e.g., 0.1 mA; Coffman, Trumbo, & Clark, 2012) are utilized for comparison.

It is uncertain that subjects are truly blinded during sham conditions using current NICS techniques. Recent investigations of sham TMS revealed that a greater proportion of subjects in active experimental groups guess correctly which condition they received (Broadbent et al., 2011), and special care must be taken when designing tDCS trials (e.g., selection and preparation of electrodes that determine sensation in the active phase; Minhas, Datta & Bikson, 2011; Dundas, Thickbroom, & Mastaglia, 2007) to avoid significant differences in sensory side effects and severity between experimental and sham tDCS conditions (e.g., Kessler, Turkeltaub, Benson, & Hamilton, 2012). High-definition tDCS (HD-tDCS) is a new NICS technique that improves current focality and intensity using multiple gel-based electrodes, similar to those used in electroencephalography (EEG), to deliver electrical stimulation (Datta et al., 2009; Dmochowski, Datta, Bikson, Su, & Parra, 2011). The parameters for an acceptable sham HD-tDCS are being explored (Borckardt et al., 2012). With HD-tDCS, it is possible to manipulate electrode configuration to purposefully shunt current across the scalp. This could facilitate development of active sham conditions whereby current is continually applied and resultant scalp sensations are indistinguishable from active experimental conditions, with little or no cortical modulation. The purpose of this study is to contribute to the development of an optimal sham condition for HD-tDCS

protocols by gathering detailed ratings of sensations experienced by participants during different electrode configurations and current intensities.

Materials and Methods

Subjects

Twenty healthy participants between the ages of 18 and 75 years (M = 30.3 years; 9 females) participated in this experiment. The University of South Carolina Institutional Review Board approved this study.

HD-tDCS

Stimulation was delivered using High-Definition electrode insets (model HD2, Soterix Medical, Inc.) that are safe and well tolerated for currents up to 2.0 mA (Borckardt et al., 2012; Minhas et al., 2010; Villamar et al., 2013). Prior to electrode placement, a mild anesthetic (1–2 mL Lanacane, active ingredient 6% benzocaine) was applied to the scalp under HD-insets to reduce scalp irritation and sensations. Sintered Ag/AgCl electrodes were then immersed in conductive jelly (Signa gel®, Parker Laboratories) inside the insets. 1.0 to 2.0 mA HD-tDCS was administered via a battery-powered constant current stimulator that was connected to the electrodes through a Multi-Channel Stimulation Adapter (Soterix Medical, Inc.).

There were five conditions with two electrode montages for this study. Each condition was 5 minutes in duration. To guard against order effects, partial counterbalancing was employed, and conditions were administered in random order to each participant with at least a 1-minute break between each condition. Three conditions involved the 4x1 ring montage (Figure 1a), with the cathode electrode centered over the left inferior parietal lobe and 4 anode electrode returns circling the target region: (a) Exp4x1 - experimental condition, subjects received 5 minutes of 2.0 mA — this montage was selected because it has been modeled and used in clinical research to provide focal cortical stimulation (e.g., Datta et al., 2009; Borckardt et al., 2012; Villamar et al., 2013); (b) Sham4x1A - 45 seconds of 2.0 mA ramped down to 1.0 mA for the remaining time (active fade sham); and (c) Sham4x1B - 45 seconds of 2.0 mA ramped down to zero current for the remaining time (inert fade sham). The two remaining conditions involved an active sham, using the 1x1 montage (Figure 1b) where the anode and cathode electrodes were placed immediately adjacent to the other in order to shunt at least part of the current across the scalp: (a) Sham1x1A - 5 minutes of 2.0 mA, and (b) Sham1x1B - 5 minutes of 1.5 mA. Pilot testing revealed near floor ratings for sensation for 1x1 at 1.0 mA, so this was not pursued as a viable sham option for our experimental montage (see Discussion). Participants did not perform tasks during HD-tDCS administration.

The experimental and sham montages (electrode positions and current applied) were modeled in a single individual using methods described previously (Datta et al., 2009). Current density at the scalp (reflecting sensation) and electric field at the cortex (reflecting neuromodulation) were predicted for both 4x1 and 1x1 configurations, and are illustrated in Figure 1 (a and b).

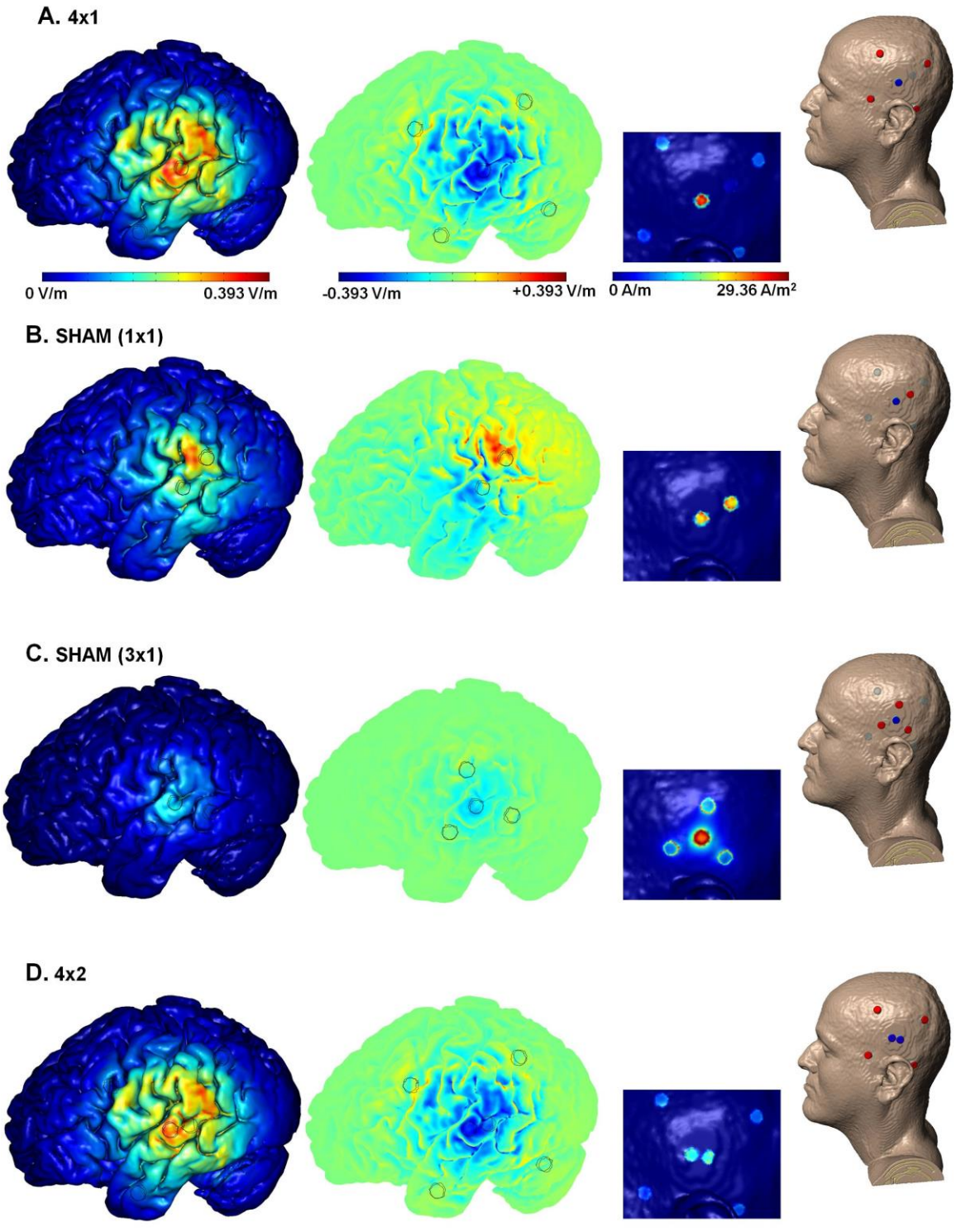

Figure 1. High-resolution 1 mm3 MRI-derived FEM simulations of current flow using 4 electrode montages. Cortical electric field magnitude (1st panel), radial cortical electric field (which considers inward/outward flow; 2nd panel), and current density at the skin (indicative

of sensation; 3rd panel) for each montage (4th panel) are considered. The colored circles on the rendered image (4th panel) indicate the status of the electrode in the loaded cap: blue = cathode, red = anode, gray = inactive. A) The control experimental montage, 4x1 HD-tDCS with 2 mA applied through the center cathode, was used experimentally and in the simulation as a standard for comparison. B) The proximal Sham1x1 montage, with 2 mA, resulted in reduced, but not negligible, cortical current flow and moderately reduced skin current density compared to the control case, which is consistent with experimental findings. C) The proximal Sham3x1 montage, with 2 mA, resulted in maximal skin shunting as indicated by low brain electric field, but skin current density comparable to the control case, suggesting this montage should be further evaluated as an active sham. D) The 4x2 HD-tDCS montage with 1 mA current applied through each of the center cathodes (2 mA total) results in comparable electric field as the control case, but significantly reduced current density. This result is consistent with preliminary findings that stimulation with up to 1 mA per electrode approaches sensation floor for most subjects, such that the 4x2 HD-tDCS montage may be explored as a new test condition with no active (current flow) sham required.

Sensation Ratings

Participants completed a magnitude estimation task (no modulus) during which they rated "overall sensation" in 1-minute intervals during each condition. A left-to-right visual analog scale was used, ranging from "no sensation" to "maximum sensation". Participants were instructed to make subsequent ratings relative to the first rating. To ensure accurate rating at designated time points, only overall sensation was assessed rather than requiring subjects to track multiple sensations (which can be subjective, difficult to disentangle, and not experienced by every participant). All ratings were scaled as within-subject in reference to the individual maximum across time and condition (rating at each time/maximum rating for that participant at any time, in any condition) to allow for cross-subject comparisons, given differences in individual sensitivity.

Results

A 5 x 5 (Time x Stimulation condition) analysis of variance (ANOVA) was conducted to determine if sensation measurements differed over time and/or by condition. There was a main effect for Time, $F(4, 76) = 33.738$, $p < .001$, and Stimulation condition, $F(4, 76) = 5.576$, $p = .001$. The interaction effect was not significant ($p = .063$). See Figures 2a and 2b for sensation mean and standard deviation for each condition. A 5 x 5 (Time x Order) ANOVA did not reveal main effects for order ($p = .511$) nor an interaction ($p = .128$).

Planned paired samples t-tests were performed for each time point to determine which sham conditions were significantly different from Exp4x1. Significant differences (Holm-Bonferroni corrected at each time point) in the following sham conditions compared to the Exp4x1 were observed: Sham1x1B at time points 1 and 3 ($p = .012, .005$), Sham4x1A at time points 3 through 5 ($p = .006, .004, .008$) and Sham4x1B at time points 2 through 5 ($p < .001$). Sham 1x1A showed no significant differences at any time point. Effect sizes (Cohen's adjusted d) were calculated for each comparison and are displayed in Figure 2c; most effect sizes (13 of 20 comparisons) are medium to large.

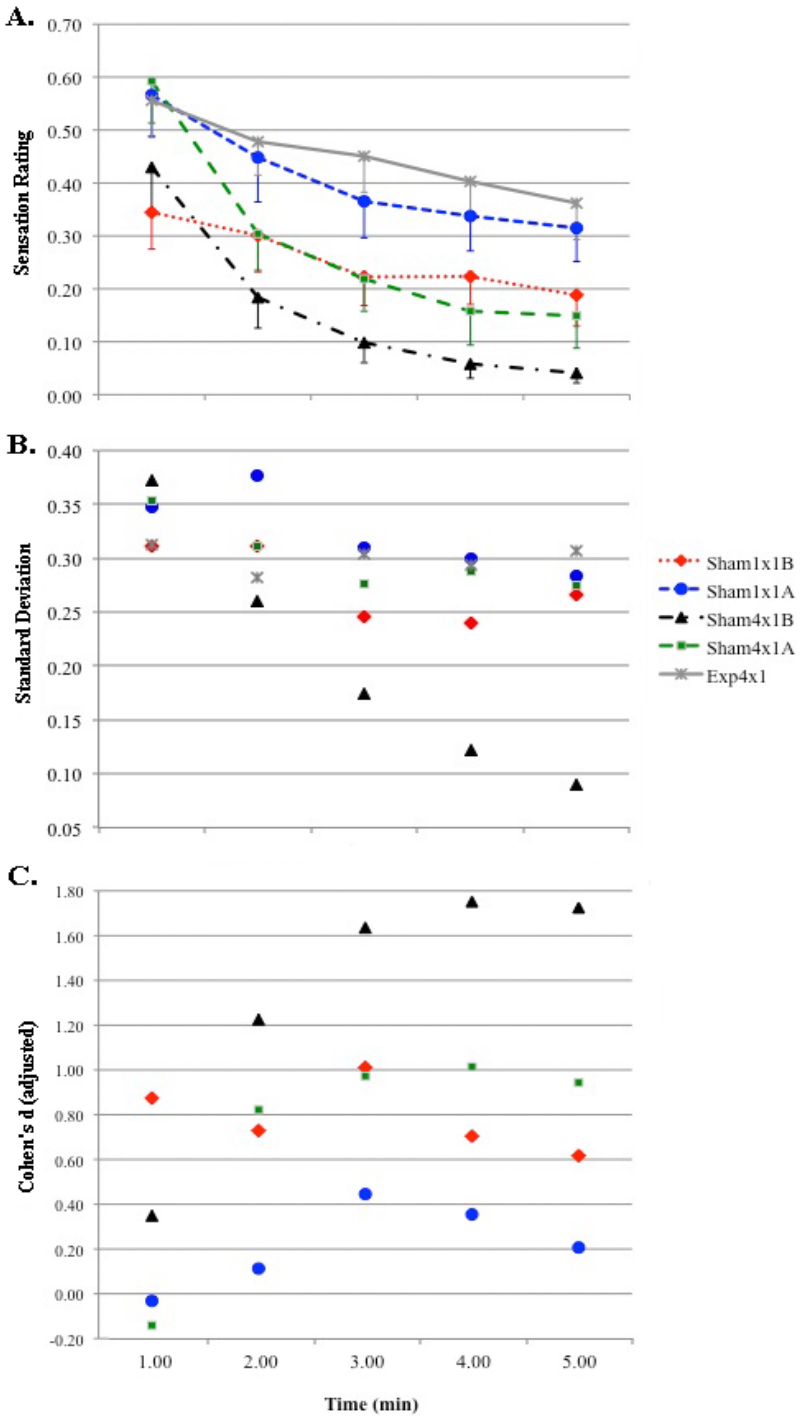

Figure 2. A) Sensation curves (created with average ratio values) per condition. The x-axis represents time; the y-axis represents sensation ratio measurement. B) Standard deviation of mean ratio values per condition. The x-axis represents time; the y-axis represents standard deviation. C) Effect sizes for each sham-to-experimental condition comparison. The x-axis represents time; the y-axis represents Cohen's adjusted *d*.

Equivalence testing utilizing the confidence interval approach (Tryon, 2001; Rusticus & Lovato, 2011) was performed to determine which sham conditions could be considered equivalent to the experimental condition. The *a priori* equivalence interval (*I*; mean +/- 0.5 *SD*) was established to identify the boundaries of a range of values that might indicate a practically significant difference. The 90% confidence intervals (CIs) from the ANOVA were used to define the maximum probable difference (MPD) between the two means. Thus, mean differences were classified as: within *I* (statistically equivalent), partially overlapping *I* (statistically indeterminate), or outside *I* (statistically non-equivalent). No MPDs at any time point were classified as equivalent (see Table 1). MPDs for Sham4x1B at time points 2 through 5 were statistically non-equivalent, and all other comparisons were statistically indeterminate.

Table 1

Equivalence Test Results for Paired Differences at Each Time Point

	Time1 (T1)		**T2**		**T3**		**T4**		**T5**	
	Equivalence Interval (*I*)									
Exp4x1	+/- .1566		+/- .1410		+/- .1522		+/- .1466		+/- .1536	
	90% Confidence Intervals of Paired Differences (paired with Exp4x1)									
	Lower	Upper	L	U	L	U	L	U	L	U
Sham4x1A	-.179	.106	.058	.290	.101	.362	.116	.375	.089	.337
Sham4x1B	-.071	.324	.163*	.426*	.232*	.473*	.235*	.455*	.208*	.433*
Sham1x1A	-.185	.164	-.117	.176	-.020	.190	-.034	.164	-.078	.172
Sham1x1B	.079	.344	.044	.310	.104	.352	.043	.316	.020	.328

Note. The equivalence interval (*I*) is +/- half the SD of the mean for Exp4x1 at each time point. The 90% confidence intervals (lower to upper bounds) of the paired differences are presented as the maximum probable difference (MPD).

Discussion

The essential challenge with shamming all NICS is that energy delivered to the brain must pass, typically at higher intensity, through the scalp. Traditional sham approaches reduce the applied energy (i.e., intensity and/or duration), often ramping down soon after the start of stimulation. In the current study, we found that when using electrode configurations identical to the experimental condition, sensation differed significantly at one or more time points, both for current ramped down to 1.0 (active fade sham) and to zero (inert fade sham). Using a novel, active sham approach, which shunts a portion of the current through the scalp, we

found no significant differences in scalp sensation ratings between the sham and experimental conditions when the current remained constant at 2 mA; absence of differences has historically been the criteria for acceptance of a sham condition in previous research (e.g., Gandiga, Hummel, & Cohen, 2006). However, it should be noted that this montage did not meet the more stringent criteria for statistical equivalence (though only exceeding MPD bounds by < +/- .04 at all time points) and resulted in greatly reduced, but perhaps not negligible, current induced in the cortex (see Figure 1b), the behavioral effects of which are unknown. Even with the variability of sensation ratings reported by our participants (a major limitation of investigations of such a subjective experience), it is clear that the inert fade sham condition is probably not the best candidate for blinding (i.e., significantly different, non-equivalent, consistently low sensation ratings across participants during zero current), particularly for crossover investigations where participants receive both sham and experimental conditions.

Given these results, other sham configurations that capitalize on scalp shunting of current and/or that match the scalp sensations of experimental conditions should be investigated further. For example, a proximal Sham3x1 montage (see Figure 1c) should result in increased current shunting and comparable scalp sensations, and could be further explored as a viable sham. Alternatively, given that 1 mA resulted in floor or near floor sensation ratings for subjects in our pilot study, we feel it is plausible to reduce sensation in active/experimental conditions to negligible levels. This could be accomplished by applying current in parallel using HD electrode “functional sets” (e.g., splitting 2 mA across two adjacent center HD electrodes; see Figure 1d), which results in a comparable electric field to the experimental condition according to our model (compare with Figure 1a). If the target current were delivered in this manner, then sham development would be trivial and equivalence likely readily attained, as sensation ratings should be at or near floor for the active condition and require no active current flow for the sham condition.

In future investigations, attention should not only be paid to scalp sensations, but also to behavioral effects that could occur due to the small amount of current induced in the cortex. While modeling provides a best guess of how much current may have reached the cortex during these different conditions, an examination of the relationship between estimated electric field and physiological and/or behavioral relevance has not been clearly characterized and was not addressed in this study. In addition, future research should employ designs that carefully consider the pharmacokinetic properties of the local anesthetic used in scalp preparation. Because precise information about the half-life and duration of effect of benzocaine is unknown, we are unable to make post hoc inferences about the relationships between the amount of anesthetic applied, time post application, sensation ratings, and current delivery in this study.

This is the first NICS investigation to employ equivalence testing, and as such, we used rationale and criteria supported by related literature to determine the bounds of the MPD. Whether or not our strategy was too conservative to detect equivalence is unknown (but suspected) and future work should include development of standards for equivalence testing specific to NICS research in order to increase trial rigor in NICS research to ensure adequate sham development. Equivalence testing is a more rigorous approach that, if utilized, could prevent the continued development and use of NICS sham conditions that, though they do not result in significant differences, are still able to be detected by participants as different (and thus are not true shams). Our behavioral and modeling findings suggest that the flexibility of multi-electrode HD-tDCS should permit improvements in both active (reduced

sensation) and sham conditions (equivalent sensation with negligible current) that will lead to enhanced quality and interpretability of NICS research.

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NeuroRegulation

Neurofeedback Practitioner Factors Related to Client Adherence

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Abstract

Introduction. This study systematically identified, extracted, and organized neurofeedback (NFB) practitioner factors connected to client adherence. It is important to understand this connection because increased adherence leads to improved NFB outcomes. A previous NFB conceptual framework and previous NFB client adherence findings were used to guide the current study.

Method. One hundred and ninety-eight NFB practitioners completed online surveys gathering demographic information and ratings of practice behaviors and characteristics. For data set analyses, this study utilized SPSS version 20 for descriptive statistics, frequencies, means, standard deviations, ranges, Pearson product-moment correlation analyses, and independent samples *t*-tests.

Results. Findings indicated that the following significantly correlated with client adherence: (a) practitioner technical and interpersonal techniques; (b) practitioner commitment to improving technical and interpersonal skills; and (c) practitioner confidence displayed during sessions. Results also indicated commitment correlated separately with techniques and confidence. These results suggested that practitioners engaging in self-NFB sessions reported significantly higher adherence rates compared to practitioners not engaging in self-NFB sessions. Findings demonstrated that practitioners conducting ≧ 40 monthly NFB sessions reported significantly higher adherence rates compared to practitioners conducting < 40 monthly NFB sessions.

Conclusion. This study concluded that practitioners with commitment to improving their technical and interpersonal expertise leads to increased confidence during NFB sessions, ultimately improving adherence and outcome rates. When averaging 40 or more NFB sessions with clients per month, practitioners provide themselves with continued opportunities to practice current and new technical and interpersonal skills. By conducting self-NFB, practitioners develop their own descriptions of physiological regulation and share their own results with clients, which in turn builds rapport and increases therapeutic bonds leading to higher adherence.

Keywords: Practitioners, neurofeedback, EEG biofeedback, brain-computer interface, adherence

Introduction

Neurofeedback, electroencephalographic (EEG) biofeedback, or brain-computer interface merges advanced technology and operant conditioning to teach individuals to influence their EEG patterns leading to improved physiological regulation and psychological functioning. For the purposes of this paper, the term neurofeedback (NFB) was utilized; however, this term also refers to electroencephalographic biofeedback and brain-computer interface. These terms were also utilized in the literature reviews for this paper.

Research has demonstrated the positive effects of NFB on various physiological and psychological disorders. Arns, de Ridder, Strehl, Breteler, and Coenen's (2009) neurofeedback meta-analysis reported large effect sizes for impulsivity and inattention and a medium effect size for hyperactivity. A randomized controlled trial with a six-month follow-up of children with ADHD indicated significant academic improvements for the NFB intervention group compared to the pharmacological intervention group (Meisel, Servera, Garcia-Banda, Cardo, & Moreno, 2013). Niv (2013) reviewed NFB effectiveness research for various disorders and concluded that NFB demonstrated superior or equivalent outcomes when compared to alternative or no treatment. To organize continued advancement of NFB research, Yucha and Montgomery (2008) published an evidence-based framework, and Hammond (2011) provided an extensive review of NFB research findings.

In addition to NFB efficacy and effectiveness research, current literature highlights the importance of exploring practitioner and client relationships, establishing NFB practice guidelines, identifying properly trained practitioners, highlighting NFB learning principles, and understanding potential directions for future practice and research growth (Aguilar-Prinsloo & Lyle, 2010; Hammond & Kirk, 2008; Hammond et al., 2011; Sherlin et al., 2011; Lyle, 2012). This study pursued these recommendations through investigating practitioner factors related to client adherence within NFB settings. A crucial aspect of NFB feasibility and effectiveness research included exploring client adherence since discontinuation of recommended NFB treatment plans negatively affects physiological and psychological outcomes. The World Health Organization (2003) defined adherence as client behaviors that correspond with a collaborative plan of action developed with health care practitioners. Current adherence literature demonstrates that 20-30% of clients do not fill their first medication prescription or attend their first therapy appointment, 50% of clients drop out of behavioral and medication treatments, and 25-50% drop out of services during the first year of treatment (World Health Organization, 2003; Fischer et al., 2011). Specifically, previous research indicated client adherence problems exist within NFB settings with adherence connected to practitioner quality of work life, frequency of NFB sessions, practitioner NFB knowledge levels, commitment to practice improvement, and mentorship (Larson, Ryan, & Baerentzen, 2010; Larson, Cothran, Drandorff, Morgan, & Ryan, 2012).

Based on previous mental health practitioner literature (Grencavage & Norcross, 1990; Larson, Ryan, & Baerentzen, 2010; Larson, Cothran, Drandorff, Morgan, & Ryan, 2012; Tracey, Lichtenberg, Goodyear, Claiborn, & Wampold, 2003; Wampold, Mondin, Moody, Benson, & Ahn, 1997), this study explored connections between client adherence and practitioner variables including: NFB techniques; commitment; empathy, confidence, friendliness, optimism, monthly NFB sessions, and self-NFB sessions. First, variables utilized in this study are provided, and then specific measurement details of each variable are described in the methods section of this paper. Second, a current literature review and a rationale for including these variables in this study are provided. Third, the study hypotheses are offered.

Definitions of Variables

Throughout this section of the paper, the primary variables are typed in bold to provide easy reference for the reader. The World Health Organization (2003) defined client adherence as client behaviors corresponding with a collaborative plan of action developed with health care practitioners. This study defined techniques as practitioner abilities utilizing both NFB technology and interpersonal skills. This paper identified commitment as the level of importance practitioners place on learning new NFB technology and interpersonal skills. Empathy included the ability to display active understanding of a client's situation, and confidence described self assurance in providing effective therapeutic treatment during NFB sessions. This paper defined friendliness as providing comfortable and engaging conversations during sessions and optimism as maintaining a positive outlook throughout the therapeutic process. Monthly sessions included the total number of NFB sessions that practitioners provided each month. Self-NFB sessions included the total number of NFB sessions that practitioners apply to themselves each month.

Study Rationale

Client adherence literature demonstrates alarming rates of failure to attend first appointments and high dropout rates for both behavioral and medication treatments; however, increasing adherence rates improves health and psychological outcomes (World Health Organization, 2003; Fischer, 2011). Previous research indicated that client adherence problems exist within NFB settings (Larson, Ryan, & Baerentzen, 2010); with this evidence of client adherence problems, the current study proposes that is important to continue adherence research within NFB in order to improve health outcomes. Previous research connected client adherence to frequency of monthly NFB sessions, NFB techniques, and commitment to practice improvement (Larson, Cothran, Drandorff, Morgan, & Ryan, 2012). Substantiating previous research and building understanding of NFB adherence, this paper proposes to investigate these variables. This paper postulates that self-NFB is related to adherence because practitioners who use NFB themselves are able to develop their own descriptions of physiological and psychological regulation. Doing self-NFB also allows practitioners to engage in self-disclosure about similar NFB experiences and outcomes. By sharing their own results with clients, practitioners build rapport and improve the therapeutic bond leading to improved adherence rates.

This study also investigates empathic, confident, friendly, and optimistic qualities because practitioners reported the importance of these traits within NFB settings in previous studies (Larson, Ryan, & Baerentzen, 2010; Larson, Cothran, Drandorff, Morgan, & Ryan, 2012). Other research also identified these items as important therapist qualities (Grencavage & Norcross, 1990; Wogan, & Norcross, 1985). Imel and Wampold's (2008) psychotherapy common factors framework organized the four NFB practitioner characteristics of empathic, confident, friendly, and optimistic. Imel and Wampold defined common factors as practitioner characteristics, role, client bond, context, and relationship qualities, which are separate from the specific therapy method being applied. A meta-analysis reported that up to 70% of client outcomes can be explained by common factors rather than method of therapy (Wampold, Mondin, Moody, Stich, Benson, & Ahn, 1997). Since their framework includes a broad range of factors and this study was only focused on practitioner factors, this study modified the common factors model into a common NFB practitioner factors model that included four practitioner factors. This study offers the following hypotheses based on previous literature and rationales.

Research Hypotheses

1. Empathic, confident, friendly, and optimistic scores will be separately correlated with adherence scores.
2. Adherence will be separately correlated with techniques, commitment, and confidence.
3. The group with high rates of monthly sessions will report higher adherence rates compared to the group with low rates of monthly sessions.
4. The group that completes self-NFB sessions will report higher adherence rates compared to the group that does not complete self-NFB sessions.

Method

Participants and Procedure

With Illinois Institute of Technology institutional review board approval, the study team recruited NFB practitioners through discussion boards and email distribution. The announcement directed participants to an online survey that included a consent process. This study collected 198 usable practitioner surveys and utilized SPSS Version 20.0 to complete study analyses. Two research assistants entered the surveys into two separate SPSS files; discrepancies were resolved by comparing the two files and original surveys. A five-step data set cleaning process was utilized to identify errors, missing data, and outliers, and to ensure data met assumptions for the analyses (Mickey, Dunn, & Clark, 2004). Descriptive statistics, frequencies, means, standard deviations, ranges, Pearson product-moment correlation analyses, and independent samples *t*-tests were calculated for SPSS data set analyses.

Instrumentation

This study collected responses to the 65-item NFB Practitioner Survey, which can be found in Appendix A. This survey was developed by utilizing findings from previous NFB practitioner investigations (Larson, Ryan, & Baerentzen, 2010; Larson, Cothran, Drandorff, Morgan, & Ryan, 2012; Larson, In Press). This survey included demographic information and ratings on practitioner characteristics. The variables from the 65-item survey that were used for the remaining analyses are described below. The following variables utilized one survey question: gender (item #1), age (item #2), education (item #3), mental health license (item #4), health care license (item #5), experience (item #6), continuing education (item #7), monthly NFB sessions (item #8), and self-NFB (item #17). The following variables utilized two or more survey questions. Client adherence was calculated by subtracting monthly dropouts (item #10) from successful monthly closures (item #9). This study measured techniques by adding the scores of two survey questions: “How would you rate your current knowledge about neurofeedback technology?” (item #11) and “How would you rate your interpersonal skills with clients?” (item #12). Both were measured on a 7-point Likert scale with the anchors of “1 = poor” to “7 = excellent”. These questions gathered practitioner perspectives of their own knowledge levels rather than testing their knowledge or obtaining someone else’s rating of their knowledge. Commitment was measured by adding the scores of two survey questions: “How would you rate your commitment to learning about neurofeedback technology?” (item #13) and “How would you rate your commitment to improving interpersonal skills with clients?” (item #14). Both were measured on a 7-point Likert scale with the anchors of “1 = poor” to “7 = excellent”.

Using 7-point Likert scales, ability, priority, ease, and frequency were measured for: empathic, confident, friendly, and optimistic. For example, “During a neurofeedback session, what is your satisfaction level with your ability to be confident?” (1 = very dissatisfied to 7 = very satisfied); “During a neurofeedback session, what is your priority level for being confident?” (1 = not a priority to 7 = essential priority); “During a neurofeedback session, what is your level of difficulty or ease with being confident?” (1 = very difficult to 7 = very easy); “During a neurofeedback session, how often are you confident?” (1 = not at all to 7 = frequently). The same method of measurement was used for the remaining three factors of empathic, friendly, and optimistic. This study added the four scores from each question to obtain a composite factor score. For example, the composite confident score = confident ability score + confident priority score + confident ease score + confident frequency score. Composite scores for empathic, confident, friendly, and optimistic factors were used for the remaining analyses of this study.

Results

Table 1 presents demographic information for research subjects utilized in this study. For 198 subjects, percentages for gender, education, mental health licensure, and healthcare licensure were provided; in addition, means and standard deviations were provided for age, years practicing NFB, number of NFB sessions monthly, and continuing education.

Table 2 provides means, standard deviations, and ranges for variables utilized in the remaining analyses. The variables included: adherence, techniques, commitment, and confidence. These results were used for the Pearson product-moment correlation analyses.

Table 3 provides Pearson product-moment correlations for adherence, techniques, commitment, and confidence. Results indicated significant correlations between variables of interest in this study, and implications are discussed within the conclusion section.

An independent samples *t*-test was conducted to compare adherence in the no self-NFB condition and the self-NFB condition. There was a significant difference in the scores for no self-NFB (M = 4.01, SD = 9.21) and self-NFB (M = 7.89, SD = 16.28) conditions, $t(196) = -2.09$, $p = 0.038$. These results suggest that self-NFB affects client adherence; specifically, results suggest that when practitioners engage in self-NFB, their clients’ adherence increases. An independent samples *t*-test was conducted to compare adherence in the fewer than 40 monthly NFB sessions condition and the ≧ 40 monthly sessions condition. There was a significant difference in the scores for the ＜40 monthly sessions (M = 3.08, SD = 10.60) and the ≧ 40 monthly sessions (M = 6.62, SD = 12.12) conditions, $t(196) = -2.16$, $p = 0.032$. These results suggest that frequency of monthly sessions affects client adherence; specifically, results suggest that when practitioners conduct ≧ 40 monthly sessions, their client adherence increases.

Table 1

Demographic Information for Neurofeedback Practitioners (N =198)

Item	*M*	*SD*	%
Gender			
Female	--	--	48.00
Male	--	--	52.00
Total	--	--	100.00
Education			
Associates	--	--	1.00
Bachelors	--	--	7.60
Masters --		--	39.90
Doctorate	--	--	51.50
Total	--	--	100.00
Mental Health Licensure			
License --		--	76.30
Non-License	--	--	23.70
Total	--	--	100.00
Healthcare Licensure			
License --		--	69.20
Non-License	--	--	30.80
Total	--	--	100.00
Age	55.70	11.19	--
Years Practicing NFB	9.96	7.61	--
Monthly Sessions	62.45	69.82	--
Monthly Continuing Education	6.00	7.38	--

Table 2

Means, Standard Deviations, and Range of Adherence, Techniques, Commitment, Confidence, Monthly Sessions, and Self-NFB Scores (N =198)

Measure	*M*	*SD*	Range
Adherence	5.03	11.57	-5.00 – 95.67
Techniques	11.31	1.57	7.00 – 14.00
Commitment	12.04	1.81	7.00 – 14.00
Confidence	23.79	3.11	14.00 – 28.00
Monthly Sessions	62.45	69.82	0.00 – 400.00
Self-NFB	2.6	1.41	1.00 – 6.00

Table 3

Findings from Correlations of NFB Practitioners' Adherence, Techniques, Commitment, and Confidence Scores (N=198)

Scale	A	T	Com	Con
A	--	.15*	.16*	.18*
T	--	--	.55**	.43**
Com	--	--	--	.46**

Note. A = Adherence, T = Techniques, Com = Commitment, Con = Confidence, *$p < .05$, **$p < .01$.

Discussion

The first hypothesis was partially supported by the Pearson product-moment correlation analysis findings; a significant correlation between confidence and adherence was found. Clients may be less likely to drop out and be more likely to complete training recommendations when practitioners engage with confidence about applying NFB training sessions, planning a course of treatment, and describing outcomes. Practitioners that display high levels of confidence during NFB sessions provide clients with reassurance, which in turn increases the likelihood of attending future sessions. Practitioners who model confidence also promote therapeutic relationships that augment recommended NFB treatment goals. Activities that may contribute to increasing practitioner confidence include: attending NFB workshops, utilizing mentorship opportunities, completing NFB certifications, increasing NFB technology knowledge, and increasing interpersonal skills. This study did not find significant relationships between client adherence and empathic, friendly, and optimistic traits. Potentially, these three factors do not influence client decisions about continuing NFB sessions. It is also possible that the current study design, measurement methods, and analyses may be limited in measuring and identifying empathic, friendly, and optimistic traits as factors related to adherence. Further research on adherence may include surveying clients on practitioner factors that promote treatment adherence.

The second hypothesis was supported by the correlation analysis findings; significant and separate correlations between adherence and techniques, commitment, and confidence were found. In addition, significant and separate correlations between commitment, techniques, and confidence were indicated. Commitment to improving NFB techniques and interpersonal skills increases adherence rates leading to higher rates of positive outcomes. Commitment also increases confidence in practice skills that lead to improved adherence rates. Practitioners displaying confidence and commitment during sessions may influence clients to increase their own commitment to and confidence about the NFB process. When clients expect and experience positive outcomes, treatment adherence and willingness to complete NFB therapy goals increases.

The third hypothesis was supported by an independent samples *t*-test. Group one with ≧ 40 monthly sessions reported significantly higher adherence rates compared to group two with＜40 monthly sessions. Results suggested that the frequency of monthly sessions affects client adherence. It is possible that increasing the number of monthly sessions provides more opportunities to improve NFB application skills, which in turns produces successful outcomes leading to higher client adherence. To improve client adherence, practitioners may focus on strategies that increase the time available to complete NFB sessions. One strategy may include hiring/contracting personnel to coordinate scheduling, billing, marketing, and other administrative tasks that take time away from running sessions.

The fourth hypothesis was supported by an independent samples *t*-test; there was a significant difference in the scores for the no self-NFB sessions group versus the self-NFB sessions group. Results suggested that practitioners completing self-NFB reported higher adherence rates. When practitioners practice self-NFB they are able to develop their own descriptions of physiological and psychological regulation, which in turn, allows them to use these descriptions to discuss the NFB process with clients. While self disclosing NFB experiences and outcomes, practitioners build rapport and improve the therapeutic bond leading to higher adherence rates.

A wealth of robust research has indicated positive results of NFB therapy on client outcomes; however, this paper also emphasized the importance of exploring the influence of practitioner factors on adherence. Client adherence can be influenced by practitioners that display confidence during sessions, average 10 or more sessions per week, practice self-NFB, and maintain a commitment to improving techniques and interpersonal skills. These study findings offer guidance for future adherence research and for understanding adherence from a practitioner's viewpoint. Incorporating these findings within mentorship contacts, NFB workshops, and/or university courses may improve awareness of factors influencing adherence. Providing brief reviews about NFB adherence within educational settings may initiate discussions about problems and potential adherence strategies among new or experienced practitioners. Mentors, trainers, and teachers providing opportunities to discuss adherence problems prepare practitioners to incorporate adherence solutions within their practice. Future NFB client adherence research may include testing the feasibility and the impact of incorporating adherence components within NFB sessions. Comparing NFB education sessions with and without adherence training components may produce fruitful insights connected to improving NFB adherence outcomes. Future research may focus on exploring client perceptions of practitioner levels of commitment, techniques, and confidence within NFB sessions, since exploring client viewpoints of adherence may also improve an understanding of process and outcome factors. With these findings, the development of a NFB practitioner common-factors framework to organize practitioner factors may improve efficiency for future adherence and outcomes research.

These findings are not a comprehensive list of variables that influence adherence. This study collected practitioner self-perceptions and did not collect client data; this leads to limitations in generalization and ability to connect practitioner self-perceptions with client adherence. Additional factors may have been missed due to the study design, sample size, and method of data collection. Overall, this study attempted to identify practitioner self-perceptions connected to adherence for future NFB research.

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Appendix A: NFB Practitioner Survey

1. What is your gender?
   Male
   Female
2. What is your age?
3. What is your highest level of education?
   High School
   Associate
   Bachelor
   Master
   Doctorate
4. Are you a licensed mental health practitioner in your state?
   Yes
   No
5. Are you a licensed healthcare practitioner in your state?
   Yes
   No
6. How many years of neurofeedback experience do you have?
7. For an average month, how many hours of continuing education do you complete?
8. For an average month, how many neurofeedback sessions do you provide?
9. For an average month, how many clients do you have successfully completing their neurofeedback treatment?
10. For an average month, how many clients quit neurofeedback training before completing their neurofeedback treatment?

How would you rate your current knowledge about neurofeedback technology?

Poor	Fair	Good	Very Good	Excellent
1	2	3	4	5	6	7

How would you rate your current interpersonal skills with clients?

Poor	Fair	Good	Very Good	Excellent
1	2	3	4	5	6	7

How would you rate your current commitment to learning about neurofeedback technology?

Poor	Fair	Good	Very Good	Excellent
1	2	3	4	5	6	7

14. How would you rate your current commitment to improving your interpersonal skills with clients?

Poor	Fair	Good	Very Good	Excellent
1	2	3	4	5	6	7

15. My satisfaction level with my work life related to neurofeedback is?
   0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%
16. My burnout level related to my neurofeedback practice is?
   0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%
17. What is your frequency of doing neurofeedback training on yourself?
   Not at all, Once a month, Once every other week, Once a week, Two times a week, Three times a week, Four times a week, Five times a week, Six times a week, Everyday

NOTE: Survey participants rated 12 traits for questions 18, 19, 20, & 21. Each question had 12 separate responses for a total of 48 items.

During a neurofeedback session, what is your satisfaction level with your ability to be...
(a) ethical, (b) attentive, (c) empathic, (d) calm, (e) observant, (f) humorous, (g) analytical, (h) confident, (i) friendly, (j) realistic, (k) optimistic, (l) careful

Very Dissatisfied	Dissatisfied	Neutral	Satisfied	Very Satisfied
1	2	3	4	5	6	7

19. During a neurofeedback session, what is your priority level for being...

(a) ethical, (b) attentive, (c) empathic, (d) calm, (e) observant, (f) humorous, (g) analytical, (h) confident, (i) friendly, (j) realistic, (k) optimistic, (l) careful

Not a priority	Low Priority	Somewhat Priority	Neutral Priority	Moderate Priority	High Priority	Essential Priority
1	2	3	4	5	6	7

20. During a neurofeedback session, what is your level of difficulty or ease with being...
(a) ethical, (b) attentive, (c) empathic, (d) calm, (e) observant, (f) humorous, (g) analytical, (h) confident, (i) friendly, (j) realistic, (k) optimistic, (l) careful

Very Difficult	Difficult	Somewhat Difficult	Neutral	Somewhat Easy	Easy	Very Easy
1	2	3	4	5	6	7

21. During a neurofeedback session, what is your satisfaction level with your ability to be...
(a) ethical, (b) attentive, (c) empathic, (d) calm, (e) observant, (f) humorous, (g) analytical, (h) confident, (i) friendly, (j) realistic, (k) optimistic, (l) careful

Not at all		Occasionally				Frequently
1	2	3	4	5	6	7

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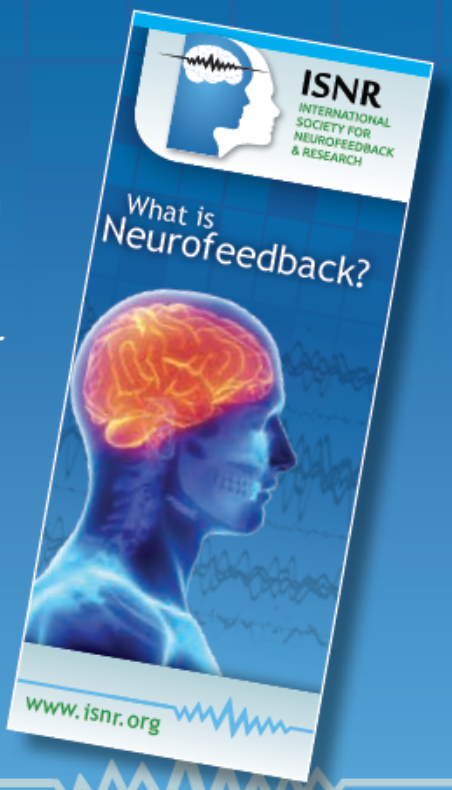

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NeuroRegulation

The Coordinated Allocation of Resource (CAR) Electrophysiological Patterns of Recalling Names of Faces in Children, Adolescents and Adults and the Central Processing Unit (CPU) of the Brain

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Abstract

The quantitative EEG (QEEG) has proven to be an important methodology in the understanding of brain functioning. The Coordinated Allocation of Resource (CAR) model maintains that cognitive effectiveness depends on the employment of a specific set of resources for specific cognitive tasks, which overlap in some situations. The model employs the flashlight metaphor in understanding the coherence and phase relations between locations. The metaphor asserts that each location can function as a flashlight that sends out a “beam” to the other locations within a frequency. The “beam” can involve all the other locations or be a mini-flashlight that involves only selected locations. The task of recalling names of faces was examined in the context of the CAR model.

The developmental changes that occur during the encoding of names of faces include increases in diffusely located communication connections involving theta (4–8 Hz) and alpha (8–13 Hz), increases in the relative power values of the beta variables (13–64 Hz), peak frequency of beta1 (13–32 Hz) and alpha, decreases in communication patterns involving the beta2 (32–64 Hz) and delta (0–4 Hz) frequencies as well as decreasing values of variables involving the lower frequencies (delta, theta), relative power values of alpha and magnitudes of alpha, beta2 and peak amplitudes of beta2.

The face-name task is both a verbal and visual task as the participant is hearing the name while he looks at the photograph. Variables that relate to success during the encoding task involve diffuse increases in flashlight activity from F7 and T3 across all frequencies to and between central locations. The QEEG variables that relate to immediate and delayed recall success involve flashlights from T3 across 4 frequencies, F7 involving 3 frequencies and the appearance of a heuristic “central processing unit” involving frontal (F3, Fz, F4), central (C3, Cz, C4) and posterior (P3, Pz, P4) locations.

Key Words: quantitative EEG, memory, memory for names of faces, name memory, coordinated allocation of resource model (CAR), central processing unit (CPU), encoding

Introduction

Face-name recall represents a specific cognitive task for memory functioning. General memory functioning concepts such as working memory (WM) and episodic, semantic and declarative memory are relevant to the task. Previous functional Magnetic Resonance Imaging (fMRI), Positron Emission Tomography (PET) and electroencephalographic (EEG) research in these conceptual areas have focused on locations and frequencies.

Neuroanatomical issues have dominated a substantial portion of the research in this area. Location differences were reported for word recognition (posterior portion of the left middle and inferior temporal gyri) and face recognition (right lingual and fusiform gyri) in a PET study (Kim et al., 1999). The amygdala and insula have been studied for the role of emotion in the recognition of faces (Gobbini & Haxby, 2007). The fMRI response pattern for faces and objects involving “the ventral temporal cortex are widely distributed and overlapping” (Haxby et al., 2001). It has also been asserted that there is a hierarchical system that involves occipito-temporal regions in the extrastriate visual cortex that mediates the visual analysis of faces (Haxby, Hoffman, & Gobbini, 2000, 2002).

Other locations that have been implicated in fMRI studies of working memory include the left prefrontal cortex, left posterior parietal cortex and hippocampus (Oztekin, McElree, Staresina, & Davachi, 2009). Support for the role of the parietal lobe in episodic memory has been reported in fMRI studies (Wagner, Shannon, Kahn, & Buckner, 2005). Verbal and visual working memories have also been shown in clinical studies of brain lesions to involve the dorsolateral prefrontal cortex (Barbey, Koenigs, & Grafman, 2013). Based on neurophysiological and neuroimaging studies, the prefrontal cortex has been hypothesized to be involved with the recovery of information (Miller & Cohen, 2001; Petrides, 2005). Several researchers have emphasized the role of the medial temporal lobe in episodic memory retrieval based on anatomical (Squire, 1992) and neuroimaging studies (Andrews-Hanna, Saxe, & Yarkoni, 2014; Diana, Yonelinas, & Ranganath, 2007).

The hemispheric encoding / retrieval asymmetry (HERA) model (Tulving, Kapur, Craik, Moscovitch, & Houle, 1994) asserts that the right frontal cortex is involved in the retrieval of episodic (versus semantic) information more than the left PFC (Habib, Nyberg, & Tulving, 2003). The right prefrontal cortex role in episodic memory retrieval has received support from others (Henson, Shallice, & Dolan, 1999). Studies of episodic retrieval using lists of items consistently find activations in the prefrontal cortex (Gilboa, 2004). Other research has focused on the same age bias in face recognition (Anastasi & Rhodes, 2005; Rhodes & Anastasi, 2012; Rhodes, Castel, & Jacoby, 2008).

Electrophysiological studies offer a different functional viewpoint on the subject and have focused on arousal measures (frequency amplitudes), communication variables (phase, coherence) in addition to location information. For example, EEG differences in word and face recognition have been reported for the alpha and beta frequencies in temporoparietal locations (Burgess & Gruzelier, 1997). The theta and alpha frequencies have been shown to be critically involved in memory processes (Jacobs, Hwang, Curran, & Kahana, 2006; Klimesch, 1999). Invasive electrophysiological monitoring has indicated phase locking in the 3–4 Hz “theta” range between the retrosplenial cortex and the medial temporal lobe during autobiographical retrieval (Foster, Kaveh, Dastjerdi, Miller, & Parvizi, 2013). The phase

locking was limited only to the theta frequency of the 0–20 Hz range studied, and there was no significant relation between theta amplitudes and phase locking. However, the 3–4 Hz frequency range is typically considered in the delta frequency.

Theta amplitudes and theta phase relations have been shown to be significantly involved in memory functioning (Klimesch, 1999; Mizuhara, Wang, Kobayashi, & Yamaguchi, 2004, 2005; Mizuhara & Yamaguchi, 2007; Sarnthein, Petsche, Rappelsberger, Shaw, & von Stein, 1998; Sauseng et al., 2002). Theta phase coding has also been shown to be relevant for long-term memory formation and working memory (Lee, Simpson, Logothetis, & Rainer, 2005; Siapas, Lubenov, & Wilson, 2005).

Phase alpha has been shown to be relevant to memory functioning (Klimesch, 1999) while phase beta has been demonstrated to be relevant to cognitive processing, memory processing and working memory (Gross et al., 2004; Tallon-Baudry, Bertrand, & Fischer, 2001; von Stein & Sarnthein, 2000). The traumatic brain injured participant has deficit coherence and phase beta2 (32–64 Hz) activity, which is related to impaired memory functioning (Thornton, 2003).

Theta-gamma (30–100 Hz) synchronization (phase and coherence) during declarative memory consolidation in the hippocampal and parahippocampal regions has been reported (Axmacher, Mormann, Fernandez, Elger, & Fell, 2006). The researchers assert that "synchronization in the gamma frequency range has to be accompanied by a stimulus-locked phase reset of ongoing theta oscillations." A literature review of the area reported that the "gamma frequency hypothesis" implies that synchronized activity in the gamma range induces memory processes more successfully than both slower (e.g., beta) and faster activity (e.g., ripple [~ 200 Hz]; Buzsaki, Leung, & Vanderwolf, 1983; Engel & Singer, 2001). Increases in the power of both gamma and theta activity in diffuse locations have been reported during successful memory encoding (Sederberg, Kahana, Howard, Donner, & Madsen, 2003) as well as during successful encoding and retrieval (Gruber, Tsivilis, Montaldi, & Muller, 2004). The role of the gamma frequency has also been studied in the recognition of familiar stimuli such as faces and buildings (Zion-Golumbic, Golan, Anaki, & Bentin, 2008). Other electrophysiological studies have used event related potentials to activation differences when discriminating faces (Zheng, Mondloch, Nishimura, Vida, & Segalowitz, 2011) and face versus non-face stimuli (Zheng, Mondloch, & Segalowitz, 2012).

The brief literature review demonstrates a diverse set of findings varying by task and implicating several frequency ranges and locations. The tasks employed are generally restricted in terms of locations studied, frequency ranges, time periods analyzed and specific tasks employed. An alternate method to the problem of recalling someone's name would be a task which requires spontaneous free recall of the person's name after a short exposure (face-name recognition and recall). This research was designed to address the issue of how does someone recall an individual's name after a short exposure to their face and name, a common situation in many social and business situations. The investigation is one of discovery and confirmation of the previous research.

Methods

The methodology in this research does not employ the typical baseline versus task analysis methodology (fMRI, PET studies, etc.), but rather a correlational analysis between performance and absolute values of the QEEG variables. The senior author considers this approach to be the preferred approach to understanding brain electrophysiology due to several problems with the baseline versus task approach.

The first problem of the methodology is the assumption of what the activation means. Implicity, it has been considered to relate to performance in some positive manner. However, it is possible that the activation has: (a) no relation; (b) a negative relation; or (c) a necessary component but unrelated to performance in addition to the possibility that (d) activation does relate to performance. Some researchers have addressed this issue and have successfully related the activation levels to performance. Thornton and Carmody (2009) demonstrated that the normal brain does not necessarily activate the appropriate QEEG resources (those related to success during the task) to be successful at the task, thus an “inefficient” brain. In one case of a brain injury, the participant activated frontal beta2 relative power more than the control group, and that increase was negatively related to memory performance (Thornton, 2014). Thus, it cannot be assumed that an activation pattern is inherently relevant to success at the task, despite its compelling appearance. In a group of participants with mild cognitive impairment (MCI), the QEEG absolute power measures were negatively related to Mini-Mental Status Exam (MMSE) scores and were significantly higher in the MCI group compared to the control group. The coherence values were higher in the MCI group during a working memory task (and not at rest), but these values were not related to the MMSE scores (Jiang, 2005).

The second problem is the implicit assumption that if the brain activates a connection, a higher activation of that connection between the two locations will relate to higher cognitive performance on the cognitive task. This would be true if the absolute value of the variable relates to success (examined in this research). The alternative interpretation would be that somehow the brain records the amount of change in a variable and that record and degree of change relates to performance. For example, would a change from 40 to 60 be more predictive than a change of 70 to 75 or a raw score of 80 during the actual task? The assertion that the change is more important for functioning would be arguing that a 60 value is preferred to the value of 75 or 80 for the coherence number, contrary to common sense. It is more logical and simpler (Occam’s razor) to assume that it is the raw value during the task that is critical rather than the change from a previous state. The statement assumes present cognitive functioning levels are determined not by present neurophysiological variables but by past levels, a very contra-intuitive statement.

Participants

Participants were recruited (*N* = 167) at a general mental health clinic in response to advertising or word-of-mouth recruiting efforts, and they received a financial incentive ($25) or intention to enter a treatment program. The mean age was 31.2 years and ranged from 7.75–72.4 years. There were 80 females and 87 males. The average education level was 12.6 years. There were 79 non-clinical individuals, 65 head-injured individuals and 23 other clinical individuals with no diagnosis in the sample.

Table 1

Participant Characteristics

Sample Size	Age Mean (Mos.) (*SD*) and Range	Male	Female
167	374.9 (211) / 31.2 Yrs. Range: 93–869 Mos. 7.75–72.4 years	87	80

Procedure

Cognitive evaluation / measures. Each participant received an activation QEEG evaluation, which was conducted by the senior author (Thornton, 2014b), during which they participated in 26 cognitive tasks. The name / face task was presented approximately 15 minutes into the evaluation. The participants were presented with a laminated sheet containing 10 pictures of the faces of individuals. The first and last names for each photograph were verbalized to the participants. The participants were allowed 15 seconds to study the picture and internally record the association. After the 15 seconds, the second face picture was named. The procedure continued until all 10 faces had been named. The participants closed their eyes for 60 seconds to memorize the association. They then opened their eyes and recalled the names of the 10 faces, which had been re-arranged on a different laminated sheet. After a delay period (during which other tasks were presented) the participants were asked to recall (to themselves) the names of the faces. They then opened their eyes and named the faces shown in random order. Participants were given a score of 1 for each first name and last name they recalled. The total maximum score was 40. Thus, the QEEG was recorded during the studying phase (150 seconds), and the immediate (60 seconds) and delayed (60 seconds) recall tasks.

Quantitative EEG (QEEG) measures. This research employs the following frequency definitions: Delta (0–4 Hz), Theta (4–8 Hz), Alpha (8–13 Hz), Beta1 (13–32 Hz), and Beta2 (32–64 Hz). The QEEG variables involve two sets of data. The first set concerns “activation / arousal” variables, which involve specific cortical locations and frequencies with reference to magnitude (M), relative power (RP), peak frequency (PF), and peak amplitude (PKA). The second set examines the amplitude correlation coefficients between locations with concepts of phase (P) and Spectral Correlation Coefficient (SCC; Lexicor Medical Technology). The QEEG data were examined for artifact (eye movements, muscle activity, etc.), and epochs that contained the artifacts were marked for deletion.

Activation / Arousal Measures.

RP: Relative Magnitude/Microvolt or Relative Power: The relative magnitude of a band defined as the absolute microvolt of the particular band divided by the total microvolt generated at a particular location across all bands.

M: Absolute Magnitude*:* The average absolute magnitude (as defined in microvolts) of a band over the entire epoch (one second).

PA: Peak Amplitude: The peak amplitude of a band during an epoch (defined in microvolts).

PF: Peak Frequency: The peak frequency of a band during an epoch (defined in frequency).

Connectivity Measures.

C: Coherence or Spectral Correlation Coefficients (SCC): The average similarity between the waveforms of a particular band in two locations over the epoch (one second). The SCC variable is conceptualized as the strength or number of connections between two locations and is a correlation of the magnitudes.

P: Phase: The time lag between two locations of a particular band as defined by how soon after the beginning of an epoch a particular waveform at location #1 is matched in location #2. References in the figures employ a combination of letters. For example, **CA** refers to coherence (SCC) alpha and **RPA** refers to relative power of alpha.

Results

Memory Scores

The memory scores ranged from 0 to 40 (M = 11.6, SD = 8.04). Given that the maximum potential score was 40 points (20 for immediate recall and 20 points for delayed recall) the task was very difficult for the participants. The correlations between age, education or sex and total memory score were non-significant.

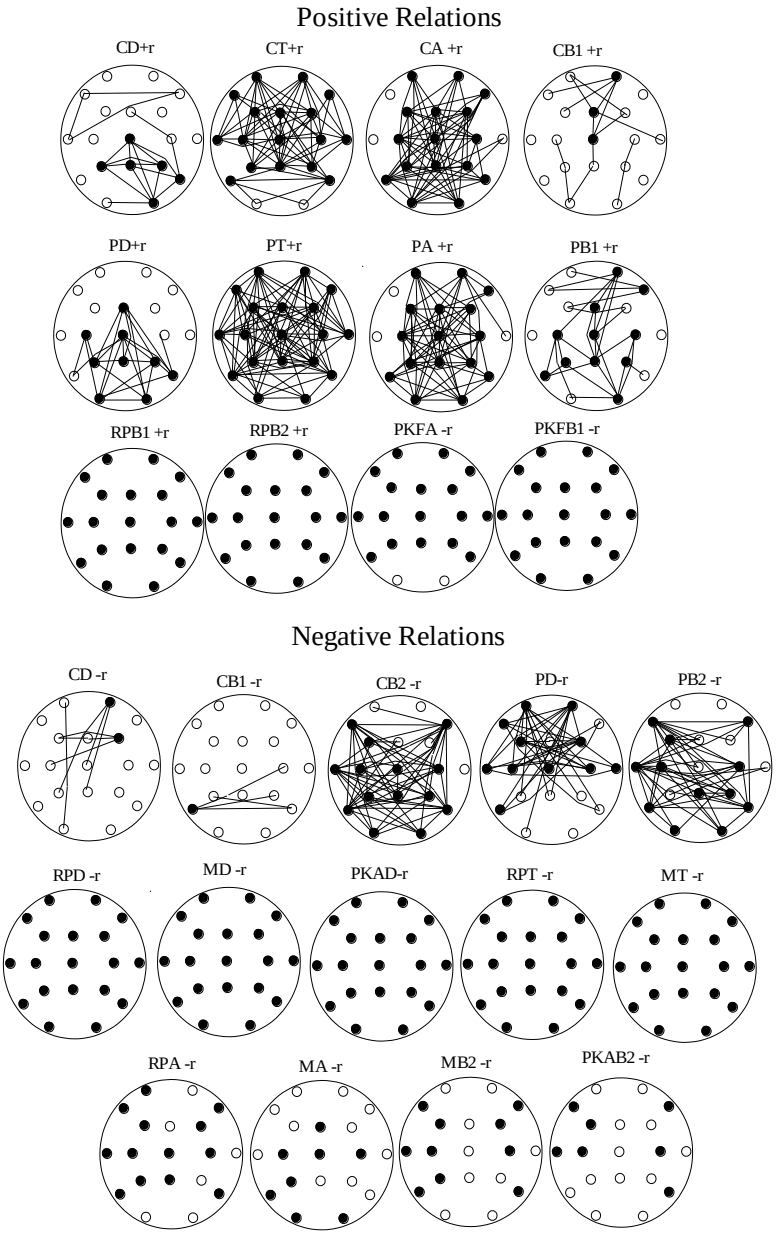
Developmental Changes in QEEG Measures

Figure 1 presents the developmental patterns that were evident during the encoding task. All the lines were significant. The locations that involved 3 or more significant SCC or phase relations were indicated by a blackened circle to indicate a possible “source” of the signal and to provide greater clarity of the response patterns. Only frequencies that had at least one “source” were included in the figures. The “+” sign indicates a positive relation between the QEEG variable and the variable under investigation. The “–” sign indicates a negative relation. The individual significant groupings were arranged according to frequencies to provide a clearer presentation of the results. A specific blackened circle could be considered the source of the signal. However, there are overlapping connections, which renders it difficult, on occasion, to determine the source. A location with a greater number of significant connections might be considered a “source”. For the purposes of the following discussion, a “source” will be assumed if it has a preponderance of significant connections.

As Figure 1 indicates, the most significant connection pattern increases are in the alpha and theta frequency (SCC and phase), posterior CD and PD, and frontal CB1 and PB1. Notably absent are increases in F7 and T3 SCC and phase alpha, which are critical variables related to task performance. There are also broad increases in the beta variables (RPB1, RPB2, PKFB1) and alpha (PKFA). The negative developmental trends are decreases of SCC and phase (beta2) and decreases in frontal / central PD. Concomitant with these communication pattern decreases are diffusely located decreases in variables involving the lower frequencies (delta, theta), multiple locations for alpha variables (RP, MA) and diffuse locations for MB1, MB2, PKAB2 with a dominant focus on the left hemisphere (LH) locations. One possible interpretation of the patterns, albeit with some data inconsistencies, is that development results in a pruning of the brain into the more central frequencies—a “centralization” trend. This is evident in the decreases in the phase delta and phase and SCC beta2 values and corresponding decreases in the lower frequencies and decreases in

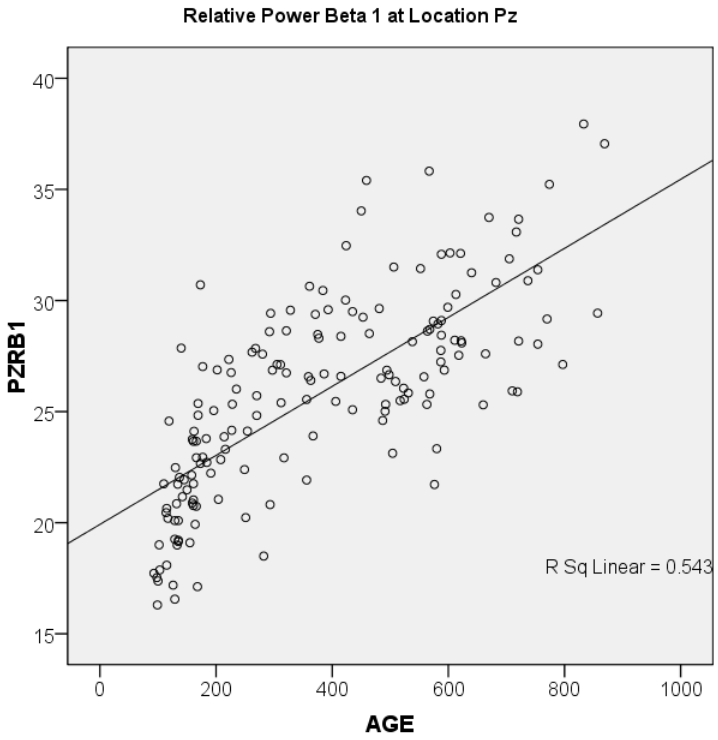
magnitude values of the beta2 frequency. Alternatively, the decreases in the SCC and phase beta2 values may represent the electrophysiological underpinnings of the cognitive decline in the elderly.

Figure 1. Relations Between QEEG Variables and Development.



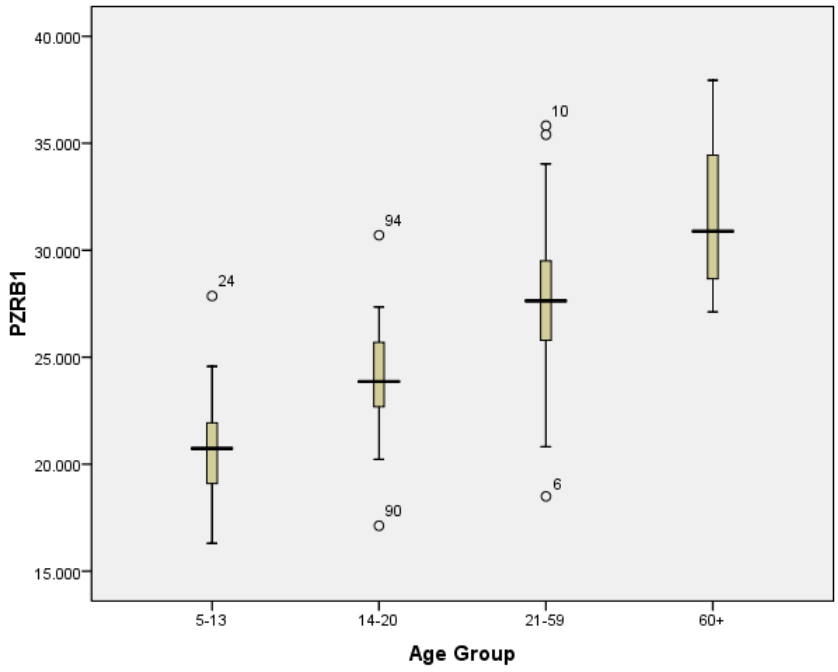
Note. CD = Coherence Delta; CT = Coherence Theta; CA = Coherence Alpha; CB1 = Coherence Beta1; CB2 = Coherence Beta2; PD = Phase Delta; PT = Phase Theta; PA = Phase Alpha; PB1 = Phase Beta1; PB2 = Phase Beta2; RPD = Relative Power Delta; RPT = Relative Power Theta; RPA = Relative Power Alpha; RPB1 = Relative Power Beta1; RPB2 = Relative Power Beta2; MD = Magnivolts Delta; MB1 = Magnivolts Beta1; MB2 = Magnivolts Beta2; PKFA = Peak Frequency Alpha; PKFB1 = Peak Frequency Beta1; PKAD = Peak Amplitude Delta; PKAB2 = Peak Amplitude Beta2

Figure 2. Scatterplot of Distribution of Pz Relative Power of Beta1 (13–32 Hz) and Age (Months)



There was a steady increase with age in the relative power of B1 at location PZ, with no evidence of a leveling with age and no decline in older years. While the linear fit was a significant model, the best regression model for the association of relative power and age was a logarithmic fit, $F(1, 165) = 242.20$, $p < .001$, adjusted $R^2 = .60$. Changes in RPB1 were examined by age groups: 7–13 years, 8–20, 21–59, and 60+ years. A one-way analysis of variance revealed a significant effect for age group, $F(3, 163) = 61.15$, $p < .001$. Post-hoc examinations using the Scheffe method showed significant (all $p < .005$) differences between all pairwise comparisons. Figure 3 shows the boxplots of RPB1 at Pz for the age groups.

Figure 3. Boxplot of Relative Power Beta1 at Location Pz by Age Group.

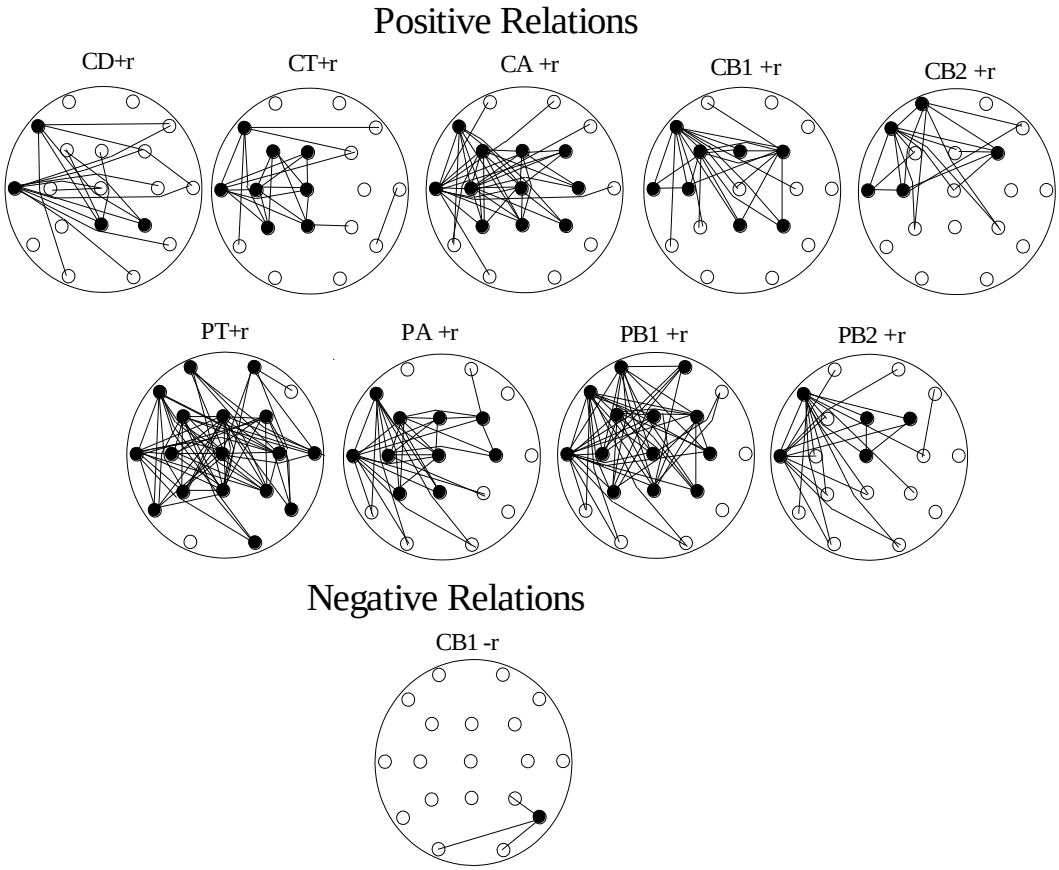


QEEG Measures Related to Memory Scores

Figure 4 shows the variables that are correlated with performance during the encoding names-faces task. The figure reflects significant involvement of the F7 and T3 SCC and phase flashlight activity from the delta to beta2 frequency, with the phase values dominant. There are also significant relations involving central and frontal locations (CA, CB1, CB2, PT, PA, PB1). The variables overlapping with development involve PT (temporal and central locations), PA (central locations), CA (central locations), and CD (posterior locations). Interestingly, PB2 (F7 and T3) decrease with age and yet are positively associated with performance.

There is an overall appearance of the left lateral locations (F7, T3, T5) sending signals to each other and into central locations (F3, Fz, F4; C3, Cz, C4; P3, Pz, P4). These central locations do not receive any direct sensory input during the cognitive task and yet are significantly involved in successful performance. These locations could be heuristically conceptualized as a Central Processing Unit (CPU).

Figure 4. QEEG Variables During Encoding Task Relations to Total Memory Score

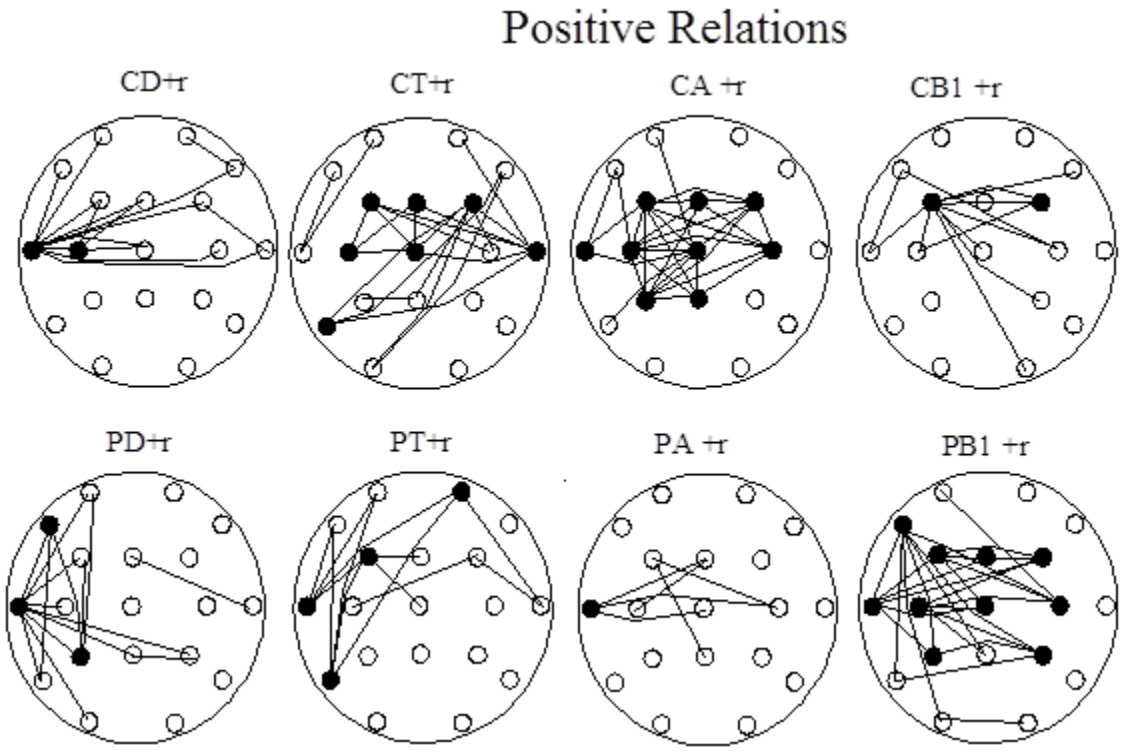


Note. CD = Coherence Delta; CT = Coherence Theta; CA = Coherence Alpha; CB1 = Coherence Beta1; CB2 = Coherence Beta2; PT = Phase Theta; PA = Phase Alpha; PB1 = Phase Beta1; PB2 = Phase Beta2

Figure 5 shows the variables associated with performance during the quiet recall task. The figure reflects the importance of the T3 flashlight (CD, CA, PD, PT, PA, PB1), which is evident during the encoding task. Many of the PB1 variables are involved in success during both the encoding and recall tasks. The dominant locations involve (F3, Fz, F4, C3, Cz, and C4) for CT, CA, and PB1. The posterior (O1, O2), frontal (Fp1, Fp2) and right hemisphere lateral locations (T6, P4, T4, F8) appear to be minimally involved. As in the encoding task, the lower frequencies (delta, theta) do not appear to negatively affect performance. The dominant frequencies involved in the CPU involve CT, CA, and PB1.

The difference between the input and immediate recall variables is a narrowing of the variables involved in successful performance, with the focus in CPU locations and CA and PB1. The right temporal (T4) projection activity involving CD and PT are implicated in both the input and immediate recall task.

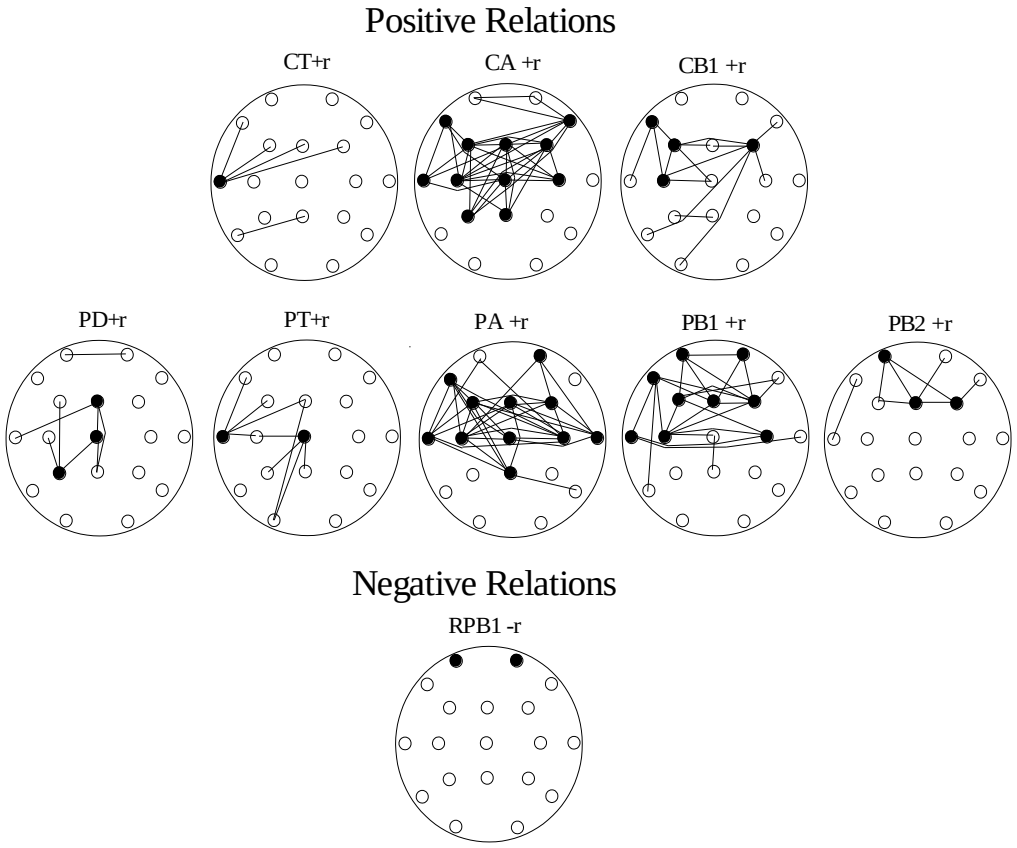
Figure 5. QEEG Variables During Silent Immediate Recall Task - Relations to Total Memory Score



Note. CD = Coherence Delta; CT = Coherence Theta; CA = Coherence Alpha; CB1 = Coherence Beta1; CB2 = Coherence Beta2; PD = Phase Delta; PT = Phase Theta; PA = Phase Alpha; PB1 = Phase Beta1; PB2 = Phase Beta2

Figure 6 shows the variables that are related to performance during the delayed recall task, which employs the delayed recall score (not the total memory score). The results point to the T3 location (CT, CA, PA, PB1). The variables that overlap across the input, immediate and delayed recall task and are involved in successful performance are T3 (CT, PT, CA, PA, PB1), and the CPU, which involves CA, PA, CB1 and PB1. The only negative effect involves Fp1 and Fp2 RPB1 values. The data suggests a further focusing of the variables in the CPU locations with CA and PA being the critical variables.

Figure 6. QEEG Variables During Silent Delayed Recall Task Relations to Delayed Recall Memory Score



Note. CT = Coherence Theta; CA = Coherence Alpha; CB1 = Coherence Beta1; CB2 = Coherence Beta2; PD = Phase Delta; PT = Phase Theta; PA = Phase Alpha; PB1 = Phase Beta1; PB2 = Phase Beta2; RPB1 = Relative Power Beta1

Discussion

This study investigates the associations of brain activation during encoding and recall of the names of novel faces. Developmental changes are addressed by examining QEEG measures as a function of age in the participants ranging from 8 to 72 years. Associations of QEEG measures and performance are also examined to identify the activation pattern associated with better memory for face names. Given that the photographs were of individuals in the age range of 20–40 years, we examined an age effect in brain activation. An example of this examination was the measure of relative power in the beta1 frequency band at location Pz during the encoding phase. The systematic changes in relative power with age were evident. The scatterplot of QEEG relative power and age did not suggest a potential effect age bias. The explanation for the increase in relative power may be found in the reduction of power in the delta and theta bands in childhood. However, the differences between adolescents, ages 13–20 years, and both younger and older adults, would not be

explained by a reduction in theta and delta power in childhood. Therefore, the increase in RPB1 in adulthood may be an indication of the allocation of resources to the encoding task. However, the increase in relative power in encoding was not associated with better performance in recall.

The large developmental increases are most evident in the coherence and phase theta and alpha relations, relative power of beta values (13–64 Hz), and peak frequency values (alpha, beta1). There are decreases in the beta2 coherence and phase values and frontal / central phase delta values as well as decreases in variables related to delta, theta, alpha and beta2 values. The decreases in SCC and phase values associated with the highest and lowest frequencies might be conceptualized as a “centralization” trend of the brain. However, the decreases in coherence and phase beta2 values may also represent an important variable to investigate in terms of the cognitive decline in the elderly. The lack of increases in the alpha coherence and phase values from the T3 and F7 locations (critical variables in this task) presents a problematic finding that is difficult to understand.

The dominant pattern of successful performance on the names of faces tasks is flashlight activity from the F7 and T3 locations across all frequencies during the initial encoding task as well connection activity in the heuristic CPU. Neither the arousal levels involving the lower frequencies (delta, theta) nor the beta frequencies appear to be related to performance, contrary to most of the results for the other cognitive tasks (Thornton, unpublished). The negative relation of T6CB1 during the encoding task presents an interesting pattern, as previous research has focused on implicit positive activations. The presence of negative activation level patterns can’t be discerned from a methodology that does not examine the activation level patterns to performance (i.e., the general activation versus baseline methodology).

As noted previously, there is a pattern of left locations (F7, T3, T5) communicating with each other and sending signals into the central locations (F3-Fz-F4; C3-Cz-C4; P3-Pz-P4). The involvement of the central locations could be heuristically conceptualized as the central processing unit (CPU) of the brain. The specific function of the individual connections is beyond the scope of this research or the available data. In addition, the goal of identifying specific functions is reminiscent of localization theories in psychology, with all the limitations inherent in that approach. From the EEG biofeedback point of view, the specific function of a connection is not as relevant as the standard deviation difference from a normative reference group and the relation of that variable to performance. The CPU heuristic concept, however, could present a new model for effective intervention. The CPU appears to involve more left hemisphere (LH) locations than right hemisphere (RH) in the face-name task.

What is additionally important in this data is that the coherence and phase values for all the frequencies are involved in memory processing, contrary to previous research that has focused predominantly on theta phase relations (Mizuhara, Wang, Kobayashi, & Yamaguchia, 2005; Mizuhara, Wang, Kobayashi, & Yamaguchi, 2004; Sarnthein, Petsche, Rappelsberger, Shaw, & VonStein, 1998). The involvement of all the frequencies redefines how we think about the relation between cognition and the quantitative EEG. The role of left temporal (T3), left and right PFC and parietal locations in memory functioning was supported in the results in terms of connectivity. The occipital locations appear minimally related to successful performance.

The involvement of the CPU was also evident in the reading memory results (Thornton & Carmody, in press) and intermittently in results for other cognitive abilities (Thornton,

unpublished data). The dominant recall flashlight location was T3 (across almost all frequencies). It is clear from the figures that successful completion of the task involves multiple connections across frontal and central locations.

This research presents an interesting understanding of how the brain functions in response to a very cognitively demanding task. It is clear from the results that the brain is a complex system that involves multiple locations and interrelationships between these locations. The CAR model and CPU concept appear to be useful concepts in describing how the brain functions in the name – face learning task. The results presented in this research may be of some benefit to the field of EEG biofeedback when addressing patients with a problem in face-name learning.

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Book Review

Neurotherapy and Neurofeedback: Brain-Based Treatment for Psychological and Behavioral Problems. By Theodore J. Chapin and Lori A. Russell-Chapin. (Routledge, 2014.)

Neurofeedback and Neurotherapy is a very good introductory text to these two elements of modern day biofeedback treatment and its integration into the world of psychotherapy. This fact that the authors explore and explicate both aspects of the book's title makes this book unique in the field. It is not too difficult to find books that delineate the workings of the technique of neurofeedback, nor do you have to look too hard to find texts that highlight the growing body of knowledge of how neurology informs developmental and mental disorders. However, the integration of the technique and practice of Neurotherapy in one small volume is a wonderful addition to the literature. As Allen Ivy states in his foreword to the book:

> Neurofeedback *normalizes* disregulated brains. However, Neurotherapy must be conducted by an ethical and well-trained professional. Sadly, there are many "certification" programs that run through the many complexities of neurofeedback much too quickly. Quality technical equipment that is fully up to date is required. Ethical practice demands that neurotherapists be licensed in their helping profession, seek BCIA certification, and receive supervision from a certified neurotherapist. The Chapins emphasize these points quite well. (p. x)

Chapters 1 through 5 provide the reader with an excellent introduction to the history and development of neurofeedback and Neurotherapy. While some may quibble over their particular delineation of the distinctions in these two concepts, it is both refreshing and important that the distinction be made and discussed. Their discussion is succinct but still relatively complete. In addition, these chapters provide an excellent review or introduction to the basic neuroscience underlying the growing area that we are calling Neurotherapy. They do a commendable job of connecting basic neuroscience to the possible sources of mental and developmental dysfunction and dysregulation. They connect the dots both in regard to neuroanatomy and to the connection of neurological function to human relational development. They include the ideas of important thinkers such as Bowlby and Schore and their conceptualizations related to attachment.

Chapters 6 through 8 are more specifically focused on the technique and workings of neurofeedback. They offer good, in-depth explanations about adequate equipment, appropriate knowledge and skill in application and ability to operate systems, as well as understanding how to make the connection between the technique and the problem. They are well aware that training a brain is more than just sticking some wires on a head and letting the client play games on the screen. They spend a reasonable amount of time discussing assessment, treatment planning and determining if anything good is happening. They also connect the dots by linking treatment protocols and their application to specific cases.

Finally, the authors provide a handy review of neurofeedback efficacy research and answer the question that neurotherapists are often asked, “Does it work?” The list is not comprehensive and it could be improved by also offering some responses to some of the literature and public statements that are made diminishing the efficacy of neurofeedback. For many beginners in neurofeedback, the critics seem to have science behind them and how it could be that there are two such wildly differing opinions is confusing. Recent rebuttals written by some of the leaders in the field (such as that contained in this inaugural issue of NeuroRegulation) could significantly help the newcomers to the field spread the word on the effectiveness of neurofeedback. The final chapter of the book looks to the future of Neurotherapy and offers some good reflections and potential directions for where this field might grow and improve in its ability to help the ill to become healthy and others to simply enhance their abilities.

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