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## Effects of Neurofeedback Training on Inhibitory Capacities in ADHD Children: A Single-Blind, Randomized, Placebo-Controlled Study

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# Effects of Neurofeedback Training on Inhibitory Capacities in ADHD Children: A Single-Blind, Randomized, Placebo-Controlled Study

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**ABSTRACT.** *Introduction.* Studies performed during the last decades suggest that neurofeedback (NF) training can effectively reduce symptomatology in children with Attention deficit hyperactivity disorder (ADHD). Yet questions remain concerning specific effects of NF training in ADHD children, because these studies did not use a randomized, placebo-controlled approach. To address this issue, such an approach was used in the present study to measure the impact of NF training on inhibitory capacities.

*Method.* Nine ADHD children (with no comorbidity), aged 8 to 13 years, were randomly assigned to either an experimental group ( $n = 5$ ) or a placebo group ( $n = 4$ ). For both groups, training protocols comprised 40 one-hr sessions (20 meetings of 2 sessions each). Sensorimotor rhythm/Theta training was used in the experimental group. Prerecorded sessions of the first author's EEG activity were used in the placebo group. Pre- and posttraining assessments consisted of the Conner's Parent Rating Scales (CPRS-R) and neuropsychological tests. A multiple case study strategy was applied for data analysis using a Reliable Change Index when applicable.

*Results.* One experimental participant was a dropout, and one placebo participant had to be discontinued due to adverse effects. The latter participant accepted to undergo posttraining evaluations; hence an Intention-to-Treat analysis was performed on this participant's data. Remaining participants showed significant improvements on the CPRS-R. Improvements were measured on the Variability measure of the CPT-II consistently across the placebo group and

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on the Inhibition Condition of the Stroop Task for all but one placebo participant. The same trend was found for the Inhibition/Switching Condition (Stroop Task) across the experimental group ( $n = 4$ ).

*Conclusion.* The small sample size precludes from evaluating specific neurofeedback effects. Still, the presence of placebo responses suggests that other factors, such as motivation or expectations, might contribute to the outcome of NF training in children with ADHD.

**KEYWORDS.** ADHD, inhibition, motivation, neurofeedback, placebo-controlled study, randomized, single-blind

### INTRODUCTION

Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder behaviorally diagnosed from a psychiatric perspective. In its latest clinical view, ADHD is defined along two distinct but correlated dimensions of symptoms, *inattention* and *hyperactivity-impulsivity* (*Diagnostic and Statistical Manual of Mental Disorders* [4th ed., text rev.; *DSM-IV-TR*]; American Psychiatric Association [APA], 2000). The exact causes to the disorder remain unclear, but researchers agree upon a multifactorial etiology comprising neurobiological and neuropsychological facets and resulting in fronto-striatal dysfunction associated with deficits in executive functioning (Clarke, Barry, McCarthy, & Selikowitz, 2001; Durston, 2003; Himelstein, Newcorn, & Halperin, 2000; Willis & Weiler, 2005).

The prevalence of ADHD is estimated around 3 to 7% (APA, 2000) and a recent meta-analysis showed this disorder to be worldwide (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). Often accompanied in children and adolescents by a string of impairments in social adjustment and academic achievement, ADHD persists in adulthood (Barkley, 1997). It is noteworthy that ADHD adults are at high risk of developing antisocial, addictive, mood, and/or anxiety disorders (Biederman et al., 2006).

Psychostimulant medications currently constitute the mainstream treatment of ADHD (Abikoff et al., 2004; Swanson et al., 2008a, 2008b). Nevertheless, a number of moderators (i.e., age, sex, and/or comorbid disorders) and mediators (i.e., acceptance

and/or attendance to treatment) have been shown to attenuate medication effects over time (MTA Cooperative Group, 1999). Furthermore, up to 30% of individuals with ADHD do not respond to psychostimulant drugs (Conners, 2002b; Wagner, 2002).

Neurofeedback (NF) is an operant conditioning procedure whereby an individual learns to self-regulate the electrical activity of his or her own brain. Initially developed as a treatment intervention for pathologies with underlying EEG dysfunctions such as epilepsy (Serman & Enger, 2006), NF is also used as a training tool to enhance specific cognitive states required in high-performance situations (Egner & Gruzelier, 2003; Landers et al., 1991).

EEG activity of ADHD children differs significantly from that of the normal population, with profiles showing deviations in slow wave or fast wave activity, mainly over centro-frontal regions (Chabot & Serfontein, 1996; Snyder & Hall, 2006). Regarding this issue, Clarke and colleagues (2001) found three distinct EEG clusters of children with ADHD. These clusters were characterized by (a) increased slow wave activity and deficiencies of fast wave, (b) increased high amplitude theta with deficiencies of beta activity, and (c) excessive beta. These findings indicate that children with ADHD do not constitute a homogenous group in EEG profile terms.

NF training in ADHD seeks to normalize aberrant EEG activity. J. F. Lubar and Shouse (1976) were the first to report behavioral improvements following EEG biofeedback in a hyperkinetic child. The NF protocol used by J. F. Lubar and Shouse, which was based on training conditioned increases of

the sensorimotor rhythm (SMR; 12–14 Hz), led to a reduction of hyperactivity. Subsequently, other investigators developed new NF protocols aimed at reducing both hyperactivity-impulsivity and inattention in ADHD children (Drechsler et al., 2007; Heinrich, Gevensleben, Freisleder, Moll, & Rothenberger, 2004; Leins et al., 2007; Lévesque, Beauregard, & Mensour, 2006; Linden, Habib, & Radojevic, 1996; J. O. Lubar & Lubar, 1984; J. F. Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Rossiter, 2004; Thompson & Thompson, 1998).

Despite the clinical success reported in these studies, NF has been disregarded as a potential treatment for ADHD for nearly three decades. Methodological flaws (e.g., small sample sizes, lack of control group or participant randomization, absence of blindness of participants, parents and NF trainers, heterogeneity of the participants) are mostly responsible for this state of affairs (Leins et al., 2007; Loo & Barkley, 2005). However, NF is gaining interest with accumulating controlled and/or randomized studies showing positive outcomes on ADHD symptomatology. In keeping with this, a recent meta-analysis concluded upon NF efficacy and specificity as a treatment for ADHD, with a large effect size for inattention and impulsivity and a medium effect size for hyperactivity (Arns, De Ridder, Strehl, Breteler, & Coenen, 2009).

Of particular interest is the recent work by Gevensleben et al. (2009). This research team carried out a randomized controlled clinical trial involving sufficient participants to gain adequate statistical power. Participants were assigned to either a combined Theta/Beta and slow cortical potential NF training or a computerized attention skills training. Groups were as equivalent as possible in regards to the meta-cognitive regulation strategies applied, parental expectations and attitudes towards training. Treatment efficacy was assumed based on a greater improvement on behavioral rating scales (parent and teachers). Yet, as underlined by Gevensleben et al., unspecific training effects could have been at play due to the absence of a double-blind design and given that the rate of responders in the NF group (52%) was

equivocal. Although this study undeniably suggests clinical superiority of NF over computerized attention skills training, questions remain concerning specific effects of NF training due to the lack of a randomized, placebo-controlled, design (this methodology is considered the “gold” standard to isolate objectively the effects of treatments).

In this context, we decided to use a randomized, placebo-controlled, approach to isolate specific effects of NF training in ADHD children. We hypothesized that the experimental group would show significant improvement of inhibitory capacities compared to the placebo group. Because inhibition deficits in ADHD seem to affect attention and executive functions (Barkley, 1997), we also hypothesized that inhibition capacity improvement would be accompanied by amelioration of attention and executive functioning.

## MATERIAL AND METHODS

### Participants

To participate in the study, children had to fulfill *DSM-IV* criteria for ADHD combined-type with no comorbid disorder (APA, 2000), have an IQ of 90 or higher, and be 8 to 12 years of age. The diagnosis—which was based on a semistructured interview (Kiddie-SADS; Kaufman et al., 1997), Conners' Parent Rating Scales (CPRS-R; L; Conners, 2001), and neuropsychological tests—was confirmed either by a previous psychiatric or neuropsychological evaluation or by a child neuropsychologist specialized in pedopsychiatry (N.L.). During NF training, participants had to be medication free. Exclusion criteria were *DSM-IV* type I disorders, neurological conditions (i.e., epilepsy, dyslexia or learning disorders), and previous NF training.

Initially, inclusion criteria included an abnormal EEG pattern with an increased anterior theta and decreased posterior beta activity as measured by a quantitative electroencephalogram (qEEG), based on Clarke's EEG-defined subtypes of children with ADHD (Clarke et al., 2001). Twenty-one

children were recruited. All met the *DSM-IV* criteria for ADHD based on the Kiddie-SADS, but 7 children had to be excluded on the basis of their qEEG not being significantly deviant compared to the normal population. Due to time constraints and scarcity of parents interested in the project, inclusion criteria were revisited: The qEEG was less restrictive and a 13-year-old boy was enrolled in the study. Thereafter, children with all EEG-defined subtypes were included as long as the NF protocol used was not contraindicated, as confirmed by a BCIA-certified NF practitioner and clinical neuropsychologist (J.L.). All participating children showed increased anterior theta and decreased posterior beta activity, as measured by frequency analysis compared to a normative database (NeuroGuide 2.4; Applied Neuroscience Inc., St. Petersburg, FL).

In all, 9 participants fulfilled the inclusion criteria. Participants were randomly assigned to an experimental NF training group ( $n=5$ ) or a placebo group ( $n=4$ ). Table 1 summarizes the demographic and clinical characteristics of the participating children. The study, which was approved by the ethics research committees of the Faculté des Arts et Sciences—Université de Montréal, Hôpital Sainte-Justine and Laval's School Board, was conducted according to the declaration of Helsinki. Parents gave a written informed consent and were thoroughly explained the study design. Children were also asked for assent.

### Study Design

NF training sessions were supervised by trainers blind to the participant's condition. Pre- and postneuropsychological testing

was performed by the first author (É. P.-L.), who was not blind regarding the participant's condition (a randomized, single-blind, placebo-controlled design was thus used in this study). Protocols for the experimental and placebo groups were designed to be as similar as possible. These training protocols comprised forty 1-hr sessions (20 meetings of 2 sessions each, with a short break in between 2 consecutive sessions). Participants were seen three times per week for 7 to 9 weeks at the Department of Psychology, Université de Montréal. Most training took place during summer vacation (to minimize potential negative effects of stopping psychostimulant medications on academic achievement). A maximum of 1-week delay between 2 meetings was allowed during the summer when children were to leave for family vacations. For each participant, training sessions were held at the same times during the day. Parents were not allowed access to the training room and did not get feedback on their child's progress. Members of the placebo group were offered to undertake NF training once the study was completed.

### Neuropsychology Tests

For both groups of participants, neuropsychological tests were administered at most two weeks prior and after NF training. Full-scale IQs were estimated using the Wechsler Abbreviated Scale of Intelligence (1999, American norms). A range of tests were selected to measure specific cognitive abilities known to be affected in ADHD-combined type, namely, attention, motor inhibition, working memory, and planning (Nigg, 2005). Tests evaluating capacities

TABLE 1. Demographic and clinical characteristics of the participating subjects.

	Experimental Group					Placebo Group			
	ES1	ES2	ES3	ES4	ES5	PS1	PS2	PS3	PS4
Age	09:01	11:07	13:02	09:03	11:03	12:08	09:03	08:04	11:09
Gender	M	M	M	F	M	M	M	M	M
FSIQ	115	102	125	108	113	128	116	109	114
Treatment	MPH	MPH	MPH	MPH	MPH	AMP	N/A	MPH	N/A

FSIQ = Full Scale Intelligence Quotient; ES = Experimental Subject; PS = Placebo Subject; M = Male; F = Female; MPH = Methylphenidate; AMP = Amphetamine; N/A = not applicable.

beyond motor inhibition were deemed relevant since inhibitory deficits in ADHD are thought to affect attention and executive functions (Barkley, 1997).

The following tests were administered: Conners CPT-II (Conners, 2002a); Digit and Spatial Span (Kaplan, Fein, Kramer, Delis, & Morris, 1999); Verbal Fluency and Color Word Interference test (Delis, Kaplan, & Kramer, 2001); Key Search, Zoo Map (version 1 and 2) and Six Part test (BADS-C; Emslie, Wilson, Burden, Nimmo-Smith, & Wilson, 2003); Bells and Mesulam's Cancellation task; Child CAT (Brown-Peterson); the complete battery of the TEA-ch (except Code Transmission; Manly, Robertson, Anderson, & Nimmo-Smith, 1999); and D2 (Brickenkamp & Zillmer, 1998).

### **Training**

*Experimental group.* NF training was based on a protocol initially developed by J. O. Lubar and Lubar (1984), adapted by Thompson and Thompson (1998), and applied by Lévesque et al. (2006). SMR/Theta training over the right motor cortex (C4 electrode placement in the 10/20 system) has been shown to produce a decrease in impulsivity symptoms (Egner & Gruzeliér, 2004; J. F. Lubar & Shouse, 1976). Therefore, SMR/Theta training was used throughout treatment in order to control for a potential confounding variable associated to combined frequency protocols (e.g., SMR/Theta followed by Beta/Theta).

NF training was provided using the Biograph Infiniti software with ProComp2 Legacy Suite (Thought Technology Ltd., Montreal, Canada). Training sessions took place on an individual basis. Each session comprised about 30 min of NF. The remaining time concerned installing the sensors and coaching (i.e., requesting immobility and attention, providing positive reinforcing). The duration of the NF blocks increased from 3 min at the beginning of training to 5 min by mid-training. The same duration was kept for the rest of training.

EEG was recorded from C4, with reference placed on the left earlobe and ground

on the right earlobe. A sampling rate of 128 Hz with 2 s epochs was used. Skin impedance was less than 5 k $\Omega$ . Theta band was set at 4–8 Hz and SMR at 12–15 Hz.

At the beginning of each training session, a 1 min recording was used to adjust thresholds to facilitate the occurrence of rewarded behavior. Mean SMR level was decreased by 0.5 microvolt and mean theta level increased by 1 microvolt to augment the probability of generating activity beyond the requested threshold (i.e., above the SMR and below the Theta thresholds). The goal of the NF training was to decrease Theta activity (represented by a histogram to the left of the screen) to generate an auditory reward while increasing SMR activity (represented by an animation in the middle of the screen) to produce a visual reward.

No explicit instructions were given to the participants on how to self-regulate brain activity to achieve desired rewards. Nevertheless, they were told to concentrate, inhibit motor actions (i.e., fidgeting), and explore appropriate ways of controlling their brain activity. A positive approach was systematically used toward the participants and a pointlike reward schedule was applied: 1 point for adequate behavior and 1 point for an improved mean frequency as compared to the threshold (determined at the beginning of each NF session) was given at the end of every NF block (e.g., mean Theta amplitude of 12 when the threshold was at 13). A total of 100 points were required to win a prize (a toy, worth \$1, chosen according to the child's interests).

Trainers were volunteer undergraduate students. Each participant was trained by a maximum of two trainers. The trainers could only train participants belonging to the same group (experimental or placebo).

*Placebo group.* The NF training protocol used for the placebo group was identical to that utilized in the experimental group except for the origin of the brain activity displayed on the computer screen. Placebo group trainers presented 40 prerecorded sessions to the participants. These prerecorded sessions consisted of the first author's EEG activity during various cognitive tasks (e.g., speaking, reading, or writing).

### **Data Analysis**

Stringent inclusion criteria and the presence of a placebo group rendered the recruitment of participants extremely difficult. Due to a lack of statistical power (the target sample size was 12 for both groups), a Reliable Change Index (RCI) was used to analyze post- versus pretraining changes. Participants were compared to themselves before and after training with respect to normalized neuropsychological tests. No practice effects could be calculated (because the sample was too small) but were considered minimal due to the minimum 4-month delay between the two neuropsychological evaluation sessions.

Formulas (1) and (2) from Maassen, Bossema, and Brand (2009) were applied with  $P_i$  set at zero and variances, reliability coefficients, and/or standard error of measurements taken from the test's technical manuals when available. Tests with no adequate measures available were considered from a qualitative point of view. A critical value of  $\pm 1.96$  ( $p < .5$ ) is usually chosen to designate significant reliable change. This critical value can be assimilated to a  $z$  score and, hence, represents 2 standard deviations to the mean, classically established in psychology as the cutoff for pathological conditions. Still, certain conditions can be considered significantly abnormal, even if the 2 standard deviation criterion is not reached (Hannay & Lezak, 2004). For instance, in the case of the Conners' rating scales, clinical relevance is suggested at 1.5 standard deviations (Conners, 2001). Given this, neuropsychological changes were considered significant using a 1.5 standard deviation criterion.

### **RESULTS**

One participant of the experimental group was a dropout due to loss in motivation (ES5), and one member of the placebo group had to be discontinued by the first author (session 32/40) due to the outbreak over a week of adverse effects from coming to NF training (PS3; e.g., stomach pain and refusal to cooperate accompanied by a lot

of crying). The latter accepted to undergo posttraining evaluations; hence an Intention-to-Treat analysis was performed on this participant's data. Figure 1 represents the participant flow diagram recommended by the CONSORT Statement (Moher, Schulz, & Altman, 2001).

One participant had to stay on medication during the experiment because his NF training took place during the academic year. His medication regimen was methylphenidate extended-release taken at breakfast. He came to NF training between 8 a.m. and 10 a.m. before taking his medication of the day; therefore he was considered off medication, because the amount of medication in system is negligible and far from clinical efficacy at 24-hr postmedication intake (Biedermann, 2002; Quinn, Bode, Reiz, Donnelly, & Darke, 2007). During the final debriefing, all parents, but one who refused to come, denied knowing to which group their child belonged.

### **CPRS-R: L**

RCI for each scale of the questionnaire was calculated using the CPRS-R: L technical manual's standard error of measurement according to sex and age. Table 2 shows RCIs obtained per scale and per participant, with values representing standard deviation scores of the difference pre-post training (i.e., value  $\geq |1.5|$  suggests a significant difference). Differential individual patterns of change emerged across the two groups, ranging from change occurring on all scales (Subject PS1) to change occurring on one scale (Subject ES3). All but one participant in the placebo group showed an improvement on the hyperactivity dimension, suggesting that participating in the study, regardless of group, decreased hyperactive behavior according to parental ratings. The latter participant, who is the individual who had to be discontinued from the study, demonstrated a significant deterioration on various scales posttraining. This suggests that his mother's ratings reflected the behavior resultant of adverse effects of his coming to NF training. Overall, more improvement was noted across scales in the placebo group.

FIGURE 1. Participant flow diagram.

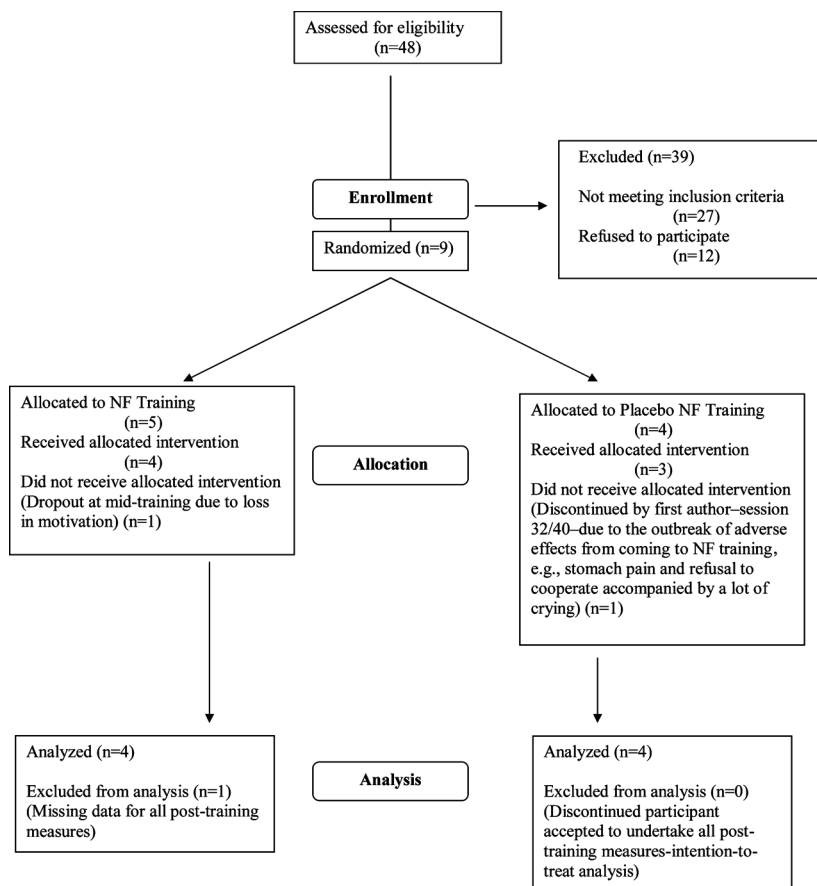


TABLE 2. Reliable Change Index values of relevant improved scales on the Conners’ Parent Rating Scale Revised Long version (CPRS-R:L).

	Experimental Group				Placebo Group			
	ES1	ES2	ES3	ES4	PS1	PS2	PS3	PS4
Oppositional Cognitive Problems/ Inattention	<b>-3.40</b>	-0.42	0.45	<b>-3.30</b>	<b>-6.32</b>	<b>-5.52</b>	2.22	<b>-4.25</b>
Hyperactivity Restless-Impulsive CGI	-1.35	<b>-2.03</b>	0.33	<b>-6.37</b>	<b>-2.67</b>	<b>-3.38</b>	0.00	<b>-4.06</b>
Total CGI	<b>-2.34</b>	<b>-2.34</b>	<b>-1.62</b>	<b>-5.22</b>	<b>-6.46</b>	<b>-2.34</b>	1.82	<b>-2.81</b>
Inattention DSM-IV	<b>-1.96</b>	-0.49	-0.50	<b>-3.90</b>	<b>-4.99</b>	-1.47	1.46	<b>-4.41</b>
Hyperactive-Impulsive DSM-IV	<b>-1.91</b>	-0.38	-0.40	<b>-4.69</b>	<b>-4.38</b>	<b>-1.52</b>	2.28	<b>-4.19</b>
Total ADHD DSM-IV	-0.44	<b>-2.20</b>	0.92	<b>-6.92</b>	<b>-3.20</b>	<b>-3.52</b>	1.33	<b>-3.52</b>
	<b>-1.78</b>	<b>-1.78</b>	-0.95	<b>-5.19</b>	<b>-6.63</b>	<b>-1.78</b>	0.88	<b>-3.57</b>
	-1.44	<b>-2.59</b>	0.00	<b>-8.00</b>	<b>-6.21</b>	<b>-3.45</b>	1.77	<b>-4.60</b>

Bold = significant reliable change of at least 1.5 standard-deviation as an improvement of behavior; CGI = Conners’ Global Index; DSM = Diagnostic Statistical Manual.



TABLE 3. Reliable Change Index values of improved scales of the Conners' Continuous Performance Tests (CPT-II).

	Experimental Group				Placebo Group			
	ES1	ES2	ES3	ES4	PS1	PS2	PS3	PS4
Omissions	<b>-4.77</b>	-0.73	<b>-6.65</b>	<b>-12.06</b>	<b>-5.25</b>	-0.79	5.16	1.47
Hit Reaction Time	0.57	-1.30	0.43	1.57	-0.88	<b>-2.97</b>	<b>-2.09</b>	-0.10
Standard Error								
Variability	1.43	<b>-43.27</b>	<b>-13.30</b>	14.70	<b>-29.65</b>	<b>-102.05</b>	<b>-62.71</b>	<b>-2.86</b>
Detectability (d')	0.47	<b>3.84</b>	<b>12.02</b>	0.00	-0.82	<b>6.72</b>	<b>4.24</b>	0.51

Bold = significant reliable change of at least 1.5 standard-deviation as an improvement of behavior.

### Neuropsychological Test

RCIs were calculated using technical manual's standard error of measurement according to sex and age for Conners CPT-II (Conners, 2002a), age for Digit and Spatial Span and Verbal Fluency and Color Word Interference test (Delis et al., 2001), and variances and reliability coefficients for Key Search, Zoo Map (version 1 and 2) and Six Part test (BADs-C; Emslie et al., 2003). Tables 3 and 4 show RCIs obtained per scale or test and per participant, with values representing standard-deviation scores of the difference pre-post training (i.e., value  $\geq |1.5|$  suggests a significant difference). The great variability in RCI values can be explained either by the magnitude of change in the participant's raw scores pre- versus posttraining (e.g., CPT-II Variability Scale-PS2: pre = 80.46 vs. post = 8.3; ES2: pre = 39.13 vs. post = 7.31), or by the normative standard error of measurement (SEM) that varies according to specific scales (e.g.,

CPT-II Variability Scale: SEM = 0.5 vs. CPT-II Hit Reaction Time: SEM = 49.53). For the remaining neuropsychological tests, RCIs could not be computed due to a lack of sufficient technical information concerning the tests.

All participants showed change on at least one measure, with equivalent change occurring in both groups. Improvement on the Variability measure (CPT-II) was measured across the placebo group and on the Inhibition Condition of the Stroop Task for 3 of the 4 placebo participants. The same trend was found for the Inhibition/Switching Condition (Stroop Task) across the experimental group. No change was measured on Digit and Spatial Span or the BADs-c's Zoo Maps and Six Part tests (see Table 4).

Qualitative appreciation of the direction of change on neuropsychological tests for which RCIs could not be calculated suggested that all participants improved on a cancellation task (i.e., fewer omissions;

TABLE 4. Reliable Change Index values of improved neuropsychological tests.

	Experimental Group				Placebo Group			
	ES1	ES2	ES3	ES4	PS1	PS2	PS3	PS4
<i>Verbal Fluency</i>								
Condition 1: Letter	-0.44	<b>2.87</b>	<b>5.94</b>	-1.75	0.99	<b>1.75</b>	<b>3.35</b>	-0.48
Condition 2: Category	0.00	1.09	-1.61	-4.24	<b>3.56</b>	<b>4.24</b>	-0.75	1.09
<i>Stroop</i>								
Condition 3: Inhibition	<b>-10.35</b>	1.72	-0.43	<b>-13.37</b>	1.72	<b>-7.33</b>	<b>-9.05</b>	<b>-2.16</b>
Condition 4: Inhibition/Switching	<b>-3.87</b>	<b>-2.39</b>	<b>-1.84</b>	<b>-4.79</b>	0.18	<b>-3.68</b>	0.18	0.00
<i>BADS-c</i>								
Key Search	0.00	0.23	-0.94	0.23	0.94	<b>2.11</b>	<b>2.11</b>	0.00

Bold = significant reliable change of at least 1.5 standard-deviation as an improvement of behavior. BADs-c = Behavioral Assessment of Dysexecutive Syndrome-Children.

experimental group [EG]: range = 1–5; placebo group [PG]: range = 1–9), whereas all but one participant in the placebo group had better scores on the Brown-Peterson (i.e., increased number of total correct answers: EG: range = 2–9; PG: range = 4–11) and Sky Search attention component (i.e., increased scale score; EG: range = 1–5; PG: range = 1–4). All but one participant in the experimental group showed better scores for the total number of errors (commission and omissions) on the D2 test (i.e., fewer errors; EG: range = 5–15; PG: range = 3–10).

### ***NF Training Point Schedule***

On average, participants in the experimental group received 11.14/12 ( $\pm 0.87$ ) points per session across the 40 sessions of training, whereas participants in the placebo group received 9.9/12 ( $\pm 1.15$ ) points per session.

### ***Qualitative Observations***

Participants and/or parents were asked to evaluate motivation levels to train, to participate in the study, and their belief in NF training on a Likert-type scale ranging from 0 (*not at all*) to 10 (*a lot*) (see Table 5). Participants' motivation to NF training was equivalent in both groups ( $M \pm SD$ : EG =  $6.87 \pm 2.93$ , PG =  $6.25 \pm 2.98$ ), whereas parental rating of motivation for training and belief in NF were higher in the placebo group (motivation: EG =  $7 \pm 1.73$ ; PG =  $9 \pm 1.41$ , belief in NF effect: EG =  $7 \pm 1$ ; PG =  $8 \pm 0$ , belief in child's brain change: EG =  $4 \pm 1.7$ ; PG =  $8 \pm 0$ ). All the parents of the participants accepted to enter the study because they were seeking an alternative approach to medication. Parental support was quite different across participants, ranging from what appeared as limited (i.e., parents dropping child at training or child coming alone; no involvement expressed) to as strong and constant (i.e., across the entire NF training parents valued the presence and the efforts of the child; family organization revolved around the NF session).

## ***DISCUSSION***

In this study, a randomized, single-blind, placebo-controlled approach was used to isolate specific effects of NF training in ADHD children. Our primary hypothesis stated that the experimental group would show significant improvements of inhibitory capacities compared to the placebo group. According to our secondary hypothesis, inhibition capacity improvements would be accompanied by amelioration of attention and executive functioning in the experimental group relative to the placebo group.

In regards to inhibitory capacities, on the CPRS-R: L all but one participant in the placebo group showed improved hyperactivity behavior, whereas no specific improvement was noted across group on inhibition measure of the CPT-II. As for the Stroop Task, improvement was found on the Inhibition/Switching Condition for the experimental group and on the Inhibition Condition for the placebo group. Yet the small sample size of the study could not enable a complete adequate testing of aforementioned hypothesis. Nevertheless, these results preclude the rejection of the null hypothesis, as significant improvements have been measured in both groups.

### ***Changes in the Experimental Group***

The improvement in inhibition capacities as a consequence of NF training is consistent with the results of a number of previous studies (Drechsler et al., 2007; Heinrich et al., 2004; Leins et al., 2007; Lévesque et al., 2006; Linden et al., 1996; J. F. Lubar et al., 1995; J. O. Lubar & Lubar, 1984; Rossiter, 2002; Thompson & Thompson, 1998). Moreover, in line with our secondary hypothesis, amelioration was measured with respect to attention measures. For instance, a reduction in the number of omissions in the CPT-II was noted along with improvement on tests measuring executive functions (e.g., Letter Condition and/or Category Condition for the Verbal Fluency test, working memory for Digit Span and Spatial Span). These results suggest that

TABLE 5. Subjective evaluation of child and parent motivation for training and parent belief in NF training on a Likert-type scale.

	Experimental Group				Placebo Group			
	ES1	ES2	ES3	ES4	PS1	PS2	PS3	PS4
<i>Subject's motivation ratings</i>								
Beginning	5	7.5	9	4	10	n/a	n/a	3
End	10	7.5	2	10	7	n/a	n/a	7
<i>Parent's ratings</i>								
Motivation for training	8	n/a	8	5	10	n/a	n/a	8
Belief in the effect of NF training	6	n/a	7	8	8	n/a	n/a	8
Belief in child's brain change following NF	5	n/a	2	5	8	n/a	n/a	8
Parental support	Constant	Adequate	Limited	Adequate	Strong constant	Limited	Strong constant	Fluctuant

Scale range: 0 (not at all) – 10 (a lot); n/a = not available.

NF training of specific EEG frequency bands can lead to cognitive enhancement.

### ***Changes in the Placebo Group***

In the placebo group, significant improvement was noted on the Hyperactivity scale of the CPRS-R: L as well as the Variability measure (CPT-II) and the Inhibition Condition of the Stroop Task. The amelioration in inhibition capacity in both groups of participants suggests that improvement in ADHD symptomatology may be related to factors other than self-regulation of selective EEG frequency bands. For instance, a participant's characteristics, such as high intellectual functioning (e.g., PS1), or behavioral constraint (e.g., having to be attentive 2 hr in a row for 20 sessions) might be sufficient to increase attentional capacities.

Alternatively, the results of the neuroimaging studies of placebo effect demonstrate that beliefs and expectations can markedly modulate neurophysiological activity in brain regions involved in various mental functions, including cognition (Beauregard, 2007). It thus appears conceivable that the placebo treatment led to changes in the participant's brain activity that accompanied cognitive enhancement measured on the CPT-II and Stroop Task. Furthermore, parent's belief in the effect of NF training and perception of child's brain changed activity was higher in the placebo group. This possibly decreased between group differences on the Conners questionnaire.

### ***Motivation as a Key Factor in NF Training?***

Motivation (intrinsic and extrinsic) may be a key factor in NF training. Various lines of evidence indicate that dopamine is crucially involved in both ADHD and motivation (Nieouillon & Coquerel, 2003). Of interest, motivation to perform on a task has been shown to positively correlate with inhibitory capacities (Carlson & Tamm, 2000). These findings suggest that motivation probably influences to a considerable extent the outcome of NF training. In this

regard, the case of PS1 is particularly interesting. His motivation to train was the highest of all participants. Likewise, his father's motivation to participate in the project was considerable. PS1 was exceptionally applied at finding adequate self-regulation strategies. During poststudy debriefing, his father mentioned how much PS1 had benefited from the training and emphasized the fact that his son was able to go to school off medication. This case also supports the view held by Drechsler et al. (2007) that parental support may represent one of the crucial variables at play during NF training.

### ***Limitations of the Study***

Applying stringent methodological criteria, we sought to recruit "pure" cases of ADHD children displaying increased anterior theta and decreased posterior beta activity. Initially, 21 children were recruited. All these children met the *DSM-IV* criteria for ADHD based on the Kiddie-SADS, but 7 had to be excluded because their QEEG did not appear abnormal as compared to a normative database. In addition, parents' initial enthusiasm dampened considerably when they realized that their child had a 50% chance of being assigned to a placebo group (even if a conventional NF training was offered once the study was completed). These problems largely explain the small sample size in this study. The use of a multicentric approach with an alternative design, such as using time series, medicated participants or even medication nonresponders, could circumvent such a limitation.

In other respects, the first author (E. P.-L.), who administered pre- and posttraining the CPRS-R: L and the neuropsychological tests, was not blind regarding the participant's condition. This represents a potential confounding variable, because she could have overestimated either group's performance posttraining. Nevertheless, the use of objective standardized neuropsychological tests administered according to their respective manuals should have minimized such potential confound.

Another limitation concerns the use of volunteer undergraduate students as NF trainers. These trainers had minimal training in operant condition and NF. Because we cannot assume that the trainers interacted the same way as licensed professionals with years of NF training, it is conceivable that this lack of experience might be partially responsible for the lack of difference between groups.

Finally, the considerable amount of reward points received by the members of the placebo group might have diminished the difference of effect size between groups with respect to the improvement of inhibitory capacities associated with NF training. Yet rewarding substantially participants in the placebo group was necessary to minimize the development of a state of learned helplessness in these individuals who were faced with the impossibility to adequately control mock brain activity.

### CONCLUSION

The results of this preliminary study suggest that factors such as motivation, expectations, and parental support might contribute to the outcome of NF training in children with ADHD. This conclusion, however, should be considered cautiously given the small sample size for this study.

### REFERENCES

- Abikoff, H., Hechtman, L., Klein, R. G., Weiss, G., Fleiss, K., Etcovitch, J., et al. (2004). Symptomatic improvement in children with ADHD treated with long-term methylphenidate and multimodal psychosocial treatment. *Journal of American Academy of Child & Adolescent Psychiatry, 43*, 802–811.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: American Psychiatric Press.
- Arns, M., de Ridder, S., Strehl, U., Breteler, M., & Coenen, A. (2009). Efficacy of neurofeedback treatment in ADHD: The effects on inattention, impulsivity and hyperactivity: A meta-analysis. *Clinical EEG and Neuroscience, 40*, 180–189.
- Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121*(1), 65–94.
- Beauregard, M. (2007). Mind does really matter: Evidence from neuroimaging studies of emotional self-regulation, psychotherapy, and placebo effect. *Progress in Neurobiology, 81*, 218–236.
- Beauregard, M. (2009). Effect of mind on brain activity: Evidence from neuroimaging studies of psychotherapy and placebo effect. *Nord Journal of Psychiatry, 63*(1), 5–16.
- Biederman, J. (2002). Practical considerations in stimulant drug selection for the attention-deficit/hyperactivity disorder patient—efficacy, potency and titration. *Today's Therapeutic Trends, 20*, 311–328.
- Biederman, J., Monuteaux, M. C., Mick, E., Spencer, T., Wilens, T. E., Silva, J. M., et al. (2006). Young adult outcome of attention deficit hyperactivity disorder: A controlled 10-year follow-up study. *Psychological Medicine, 36*, 167–179.
- Brickenkamp, R., & Zillmer, E. (1998). *The D2 Test of Attention manual*. Berlin: Hogrefe & Huber.
- Carlson, C. L., & Tamm, L. (2000). Responsiveness of children with attention deficit-hyperactivity disorder to reward and response cost: Differential impact on performance and motivation. *Journal of Consulting and Clinical Psychology, 68*, 73–83.
- Chabot, R. J., & Serfontein, G. (1996). Quantitative electroencephalographic profiles of children with attention deficit disorder. *Biological Psychiatry, 40*, 951–963.
- Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2001). EEG-defined subtypes of children with attention-deficit/hyperactivity disorder. *Clinical Neurophysiology, 112*, 2098–2105.
- Conners, C. K. (2001). *Conners' Rating Scales—Revised*. Toronto, Canada: Multi-Health Systems Inc.
- Conners, C. K. (2002a). *CPT-II, Continuous Performance Test II*. Toronto, Canada: Multi-Health Systems Inc.
- Conners, C. K. (2002b). Forty years of methylphenidate treatment in Attention-Deficit/Hyperactivity Disorder. *Journal of Attention Disorder, 6*, S17–S30.
- Delis, D., Kaplan, E., & Kramer, J. (2001). *Delis-Kalpan Executive Function System examiner's manual*. San Antonio, TX: Harcourt Assessment, Inc.
- Drechsler, R., Straub, M., Doehnert, M., Heinrich, H., Steinhausen, H. C., & Brandeis, D. (2007). Controlled evaluation of a neurofeedback training of slow cortical potentials in children with Attention Deficit/Hyperactivity Disorder (ADHD). *Behavioral and Brain Functions, 26*, 3–35.
- Durston, S. (2003). A review of the biological bases of ADHD: What have we learned from imaging

- studies? *Mental Retardation and Developmental Disability Research Reviews*, 9, 184–195.
- Egner, T., & Gruzelier, J. H. (2003). Ecological validity of neurofeedback: Modulation of slow wave EEG enhances musical performance. *NeuroReport*, 14, 1221–1224.
- Egner, T., & Gruzelier, J. H. (2004). EEG biofeedback of low beta band components: Frequency-specific effects on variables of attention and event-related brain potentials. *Clinical Neurophysiology*, 115(1), 131–139.
- Emslie, H., Wilson, F. C., Burden, V., Nimmo-Smith, I., & Wilson, B. A. (2003). *Behavioral Assessment of the Dysexecutive Syndrome for Children manual*. Bury St Edmunds, England: Thames Valley Test Company.
- Gevensleben, H., Holl, B., Albrecht, B., Vogel, C., Schlamp, D., Kratz, O., et al. (2009). Is neurofeedback an efficacious treatment for ADHD? A randomized controlled clinical trial. *Journal of Child Psychology & Psychiatry*, 50, 780–789.
- Hannay, J., & Lezak, M. (2004). The Neuropsychological examination: Interpretation. In M. Lezak, D. Howieson & D. Loring (Eds.), *Neuropsychological assessment*, 4th edition, (pp. 133–156). New York: Oxford University Press.
- Heinrich, H., Gevensleben, H., Freisleder, F. J., Moll, G. H., & Rothenberger, A. (2004). Training of slow cortical potentials in attention-deficit/hyperactivity disorder: Evidence for positive behavioral and neurophysiological effects. *Biological Psychiatry*, 55, 772–775.
- Himelstein, J., Newcorn, J. H., & Halperin, J. M. (2000). The neurobiology of attention-deficit hyperactivity disorder. *Frontiers in Bioscience*, 5, D461–D478.
- Kaplan, E., Fein, D., Kramer, J., Delis, D. & Morris, R. (1999). *WISC-III-PI manual*. San Antonio, TX: Harcourt Assessment, Inc.
- Kaufman, J., Birmaher, B., Brent, D., Rao, U., Flynn, C., Moreci, P., et al. (1997). Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL): Initial reliability and validity data. *Journal of American Academy of Child & Adolescent Psychiatry*, 36, 980–988.
- Landers, D. M., Petruzzello, S. J., Salazar, W., Crews, D. J., Kubitz, K. A., Gannon, T. L., et al. (1991). The influence of electrocortical biofeedback on performance in pre-elite archers. *Medicine & Science in Sports & Exercise*, 23(1), 123–129.
- Leins, U., Goth, G., Hinterberger, T., Klinger, C., Rumpf, N., & Strehl, U. (2007). Neurofeedback for children with ADHD: A comparison of SCP and Theta/Beta protocols. *Applied Psychophysiology & Biofeedback*, 32, 73–88.
- Lévesque, J., Beauguegard, M., & Mensour, B. (2006). Effect of neurofeedback training on the neural substrates of selective attention in children with attention-deficit/hyperactivity disorder: A functional magnetic resonance imaging study. *Neuroscience Letters*, 394, 216–221.
- Linden, M., Habib, T., & Radojevic, V. (1996). A controlled study of the effects of EEG biofeedback on cognition and behavior of children with attention deficit disorder and learning disabilities. *Biofeedback & Self Regulation*, 21(1), 35–49. [Erratum, 1996, 21(3), 297]
- Loo, S. K., & Barkley, R. A. (2005). Clinical utility of EEG in attention deficit hyperactivity disorder. *Applied Neuropsychology*, 12(2), 64–76.
- Lubar, J. F., & Shouse, M. N. (1976). EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): A preliminary report. *Biofeedback & Self Regulation*, 1, 293–306.
- Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & O'Donnell, P. H. (1995). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in T.O.V.A. scores, behavioral ratings, and WISC-R performance. *Biofeedback & Self Regulation*, 20(1), 83–99.
- Lubar, J. O., & Lubar, J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. *Biofeedback & Self Regulation*, 9(1), 1–23.
- Maassen, G. H., Bossema, E., & Brand, N. (2009). Reliable change and practice effects: Outcomes of various indices compared. *Journal of Clinical and Experimental Neuropsychology*, 31, 339–352.
- Manly, T., Robertson, I. H., Anderson, V., & Nimmo-Smith, I. (1999). *TEA-CH: The Test of Everyday Attention for Children manual*. Bury St Edmunds, UK: Thames Valley Test Company Limited.
- Moher, D., Schulz, K. F., & Altman, D. G. (2001). The CONSORT statement: Revised recommendations for improving the quality of reports of parallel-group randomised trials. *Lancet*, 357, 1191–1194.
- MTA Cooperative Group. (1999). Moderators and mediators of treatment response for children With attention-deficit/hyperactivity disorder: The Multimodal Treatment Study of children with Attention-deficit/hyperactivity disorder. (1999). *Archives of General Psychiatry*, 56, 1088–1096.
- Nieoullon, A., & Coquerel, A. (2003). Dopamine: A key regulator to adapt action, emotion, motivation and cognition. *Current Opinion Neurology*, 16(Suppl. 2), S3–S9.
- Nigg, J. T. (2005). Neuropsychological theory and findings in Attention-Deficit/Hyperactivity Disorder: The state of the field and salient challenges

- for the coming decade. *Biological Psychiatry*, 57, 1424–1435.
- Polanczyk, G., de Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: A systematic review and meta-regression analysis. *American Journal of Psychiatry*, 164, 942–948.
- Quinn, D., Bode, T., Reiz, J., Donnelly, G. A. E., & Darke, A. (2007). Release methylphenidate and immediate-release methylphenidate in children with attention-deficit/hyperactivity disorder. *Journal of Clinical Pharmacology*, 47, 760–766.
- Rossiter, T. (2004). The effectiveness of neurofeedback and stimulant drugs in treating AD/HD: Part I. Review of methodological issues. *Applied Psychophysiology & Biofeedback*, 29, 95–112.
- Snyder, S. M., & Hall, J. R. (2006). A meta-analysis of quantitative EEG power associated with attention-deficit hyperactivity disorder. *Journal of Clinical Neurophysiology*, 23, 440–455.
- Serman, M. B., & Egner, T. (2006). Foundation and practice of neurofeedback for the treatment of epilepsy. *Applied Psychophysiology and Biofeedback*, 31(1), 21–35.
- Swanson, J., Arnold, L. E., Kraemer, H., Hechtman, L., Molina, B., Hinshaw, S., et al. (2008a). Evidence, interpretation, and qualification from multiple reports of long-term outcomes in the Multimodal Treatment study of Children With ADHD (MTA): part I: Executive summary. *Journal of Attention Disorders*, 12(1), 4–14.
- Swanson, J., Arnold, L. E., Kraemer, H., Hechtman, L., Molina, B., Hinshaw, S., et al. (2008b). Evidence, interpretation, and qualification from multiple reports of long-term outcomes in the Multimodal Treatment Study of children with ADHD (MTA): Part II: supporting details. *Journal of Attention Disorders*, 12(1), 15–43.
- Thompson, L., & Thompson, M. (1998). Neurofeedback combined with training in metacognitive strategies: Effectiveness in students with ADD. *Applied Psychophysiology & Biofeedback*, 23, 243–263.
- Wagner, K. D. (2002). Management of treatment refractory attention-deficit/hyperactivity disorder in children and adolescents. *Psychopharmacology Bulletin*, 36(1), 130–142.
- Wechsler Abbreviated Scale of Intelligence. (1999). *WASI Manual*. San Antonio, TX: Harcourt Assessment.
- Willis, W. G., & Weiler, M. D. (2005). Neural substrates of childhood attention-deficit/hyperactivity disorder: Electroencephalographic and magnetic resonance imaging evidence. *Developmental Neuropsychology*, 27(1), 135–182.