

Neurofeedback for Attention-Deficit/Hyperactivity Disorder: Meta-Analysis of Clinical and Neuropsychological Outcomes From Randomized Controlled Trials

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Objective: We performed meta-analyses of randomized controlled trials to examine the effects of neurofeedback on attention-deficit/hyperactivity disorder (ADHD) symptoms and neuropsychological deficits in children and adolescents with ADHD.

Method: We searched PubMed, Ovid, Web of Science, ERIC, and CINAHAL through August 30, 2015. Random-effects models were employed. Studies were evaluated with the Cochrane Risk of Bias tool.

Results: We included 13 trials (520 participants with ADHD). Significant effects were found on ADHD symptoms rated by assessors most proximal to the treatment setting, that is, the least blinded outcome measure (standardized mean difference [SMD]: ADHD total symptoms = 0.35, 95% CI = 0.11–0.59; inattention = 0.36, 95% CI = 0.09–0.63; hyperactivity/impulsivity = 0.26, 95% CI = 0.08–0.43). Effects were not significant when probably blinded ratings were the outcome or in trials with active/sham controls. Results were similar when

only frequency band training trials, the most common neurofeedback approach, were analyzed separately. Effects on laboratory measures of inhibition (SMD = 0.30, 95% CI = –0.10 to 0.70) and attention (SMD = 0.13, 95% CI = –0.09 to 0.36) were not significant. Only 4 studies directly assessed whether learning occurred after neurofeedback training. The risk of bias was unclear for many Cochrane Risk of Bias domains in most studies.

Conclusion: Evidence from well-controlled trials with probably blinded outcomes currently fails to support neurofeedback as an effective treatment for ADHD. Future efforts should focus on implementing standard neurofeedback protocols, ensuring learning, and optimizing clinically relevant transfer.

Key words: ADHD, neurofeedback, nonpharmacological treatment, meta-analysis, risk of bias

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Attention-deficit/hyperactivity disorder (ADHD) is a common neurodevelopmental disorder characterized by age-inappropriate and impairing inattention and/or hyperactivity/impulsivity.^{1,2} Among currently available treatment options, psychostimulant and nonstimulant medications are efficacious, at least in the short term, and widely used.³ Nonpharmacological interventions—both dietary and psychological—have also been extensively investigated.^{4–7} Among nonpharmacological approaches, neurofeedback has been considered a promising ADHD treatment strategy since the early 1970s.^{8–10} When applied to ADHD, neurofeedback is intended to reduce ADHD symptoms by targeting aberrant patterns of brain activity thought to underpin the condition. Neurofeedback is implemented through the training of self-regulation using operant reinforcement procedures; learning of self-regulation is thus a key mechanism. To achieve this aim,

electroencephalogram (EEG) indices of interest are converted into visual or acoustic signals and fed back automatically in real time to the patient. For instance, cortical activity may be represented by the height or speed of a ball, plane, or cartoon character presented using animation on a computer screen. In this case, learning occurs when the object rises, falls, or advances more quickly in response to patients' regulated changes in brain activity. Two general neurofeedback approaches have been used to treat ADHD: frequency band training (FBT) and slow cortical potential training (SCP). When applied to ADHD, the former is intended to target alterations in cortical electrical oscillations thought to be associated with ADHD, namely elevations of slow, relative to fast, brainwave activity, especially in the frontal lobes (e.g., theta versus beta frequency¹¹). The latter aims to regulate cortical excitation thresholds by focusing on activity generated by external cues (similar to event-related potentials), focusing primarily on EEG components registered in the late latency range, that is, several seconds after the cue. For instance, this form of training has been used to target the contingent negative variation (CNV) that occurs during this time window and is involved in effective preparation, decision



Supplemental material cited in this article is available online.

making, and time estimation, which have all been found to be deficient in individuals with ADHD, or at least in subgroups of them.^{12,13}

The efficacy of nonpharmacological treatments for ADHD, including neurofeedback, has been subject to a number of earlier meta-analytic reviews.¹⁴⁻¹⁶ However, these have sometimes been difficult to interpret because of the inclusion of studies with weak experimental designs (e.g., no control arm, nonrandom allocation, or the use of nonblinded measures), as discussed by Sonuga-Barke *et al.*¹⁷ On behalf of the European ADHD Guidelines Group (EAGG), Sonuga-Barke *et al.*¹⁷ attempted to address these limitations through a meta-analysis of nonpharmacological interventions that included only randomized controlled trials (RCTs). It also addressed the issue of blinding by comparing outcomes rated by individuals judged to be most proximal to the therapeutic setting (often parents poorly blinded and invested in the therapeutic outcome) and those provided by reporters judged to be probably blinded. They found that the effects of neurofeedback on ADHD total symptoms based on most proximal ratings were highly significant (standardized mean difference [SMD] = 0.59, 95% CI = 0.31, 0.87). However, when only probably blinded measures were used, the effects became nonsignificant (SMD = 0.29; 95% CI = -0.02 to 0.61). More recently, Micolaud-Franchi *et al.*¹⁸ followed a similar approach, focusing their analyses on ADHD core symptoms, but with a smaller set of studies (n = 5) limited to trials with particular control conditions. As in Sonuga-Barke *et al.*,¹⁷ they found a significant, positive effect of neurofeedback on ADHD core symptoms when considering most proximal raters. Probably blinded scores were attenuated and were significant only for symptoms of inattention.

Applying the same meta-analyses protocol used in recent EAGG reviews of behavioral interventions⁵ and cognitive training,⁴ we here extend the focus of meta-analytic evidence relating to neurofeedback for ADHD in a number of ways. First, we included, among the outcomes, not only specific ADHD behavioral dimensions (i.e., inattention and impulsivity/hyperactivity) but also ADHD-related neuropsychological deficits such as inhibitory dysfunction. The latter may be important, as they may take us closer to neural mediators of the behavioral effects of neurofeedback.⁹ Second, we addressed the relative efficacy of different types of neurofeedback by restricting subanalyses to specific types of treatment protocols, namely, FBT. Third, we examined the impact of different aspects of trial design (e.g., use of a sham/placebo design) or pragmatic “dosage” characteristics of neurofeedback implementation (i.e., number of sessions). Fourth, we addressed the crucial question of whether neurofeedback-related learning at the neural level was investigated and/or demonstrated in available trials.⁹ Fifth, we examined whether the neurofeedback protocols used in these studies could be considered “standard” in terms of the criteria discussed by Arns *et al.*,¹⁹ which include elements related to EEG bands/measures, electrode placement and type, and feedback following learning. Finally, we applied, for the first time in a meta-analysis of neurofeedback for ADHD, a rigorous assessment of study bias, namely, the Cochrane Risk of Bias tool (RoB).²⁰

METHOD

The EAGG protocol was originally registered on the International Prospective Register of Systematic Reviews (PROSPERO; <http://www.crd.york.ac.uk/PROSPERO>, protocol number: CRD42011001393). As in previous work,^{4,5} the original protocol was adapted to take account of the broader scope of this systematic review/meta-analysis. Most crucially, given that the scope of this analysis included neuropsychological measures, the mandatory requirement for studies to have ADHD symptoms-related outcomes no longer applied (i.e., we included also studies presenting only neuropsychological outcomes).

Inclusion and Exclusion Criteria

To ensure high levels of methodological adequacy as recommended by the Cochrane group and to avoid the inevitable bias caused by dependence on investigators agreeing to provide data from unpublished studies,²⁰ only published studies were included. Only RCTs using neurofeedback training were retained. Participants in the trials were required to be between 3 and 18 years of age and to have a diagnosis of ADHD (any subtype) or hyperkinetic disorder (HKD) or to meet accepted cut-offs on validated ADHD symptom rating scales. Trials that selected children with ADHD who had rare comorbid disorders (e.g., Fragile X syndrome) were excluded. Control conditions allowed were “treatment as usual,” “wait list,” “active,” or “placebo/sham” (i.e., involving other forms of alternative training regimen). As per the EAGG protocol, trials in which neurofeedback was compared only with optimized medication or in which neurofeedback was added to optimized medication were excluded. Trials in which medication was part of background normal clinical provision in either the control or the active arm were included.

Search Strategy

Details about the search strategy/syntax for each database are reported in Supplement 1, available online. The final search was updated on August 30, 2015. Independent searches were conducted by 2 authors (S.C. and M.F.), leading to the same number of references.

Outcome Measures

To provide analytical robustness and in line with previous EAGG meta-analyses,^{4,5,17,20} analyses of outcome domains were considered reliable only if at least 5 RCTs were available. The planned outcomes included the following: ADHD symptoms (total ADHD and inattention and hyperactivity/impulsivity symptoms separately), neuropsychological laboratory-based measures, measures of academic functioning, and rating of severity of symptoms of comorbid conditions (e.g., oppositional defiant disorder or anxiety disorders).

Study Selection

Retrieved references were independently screened and blindly double coded for eligibility by 2 authors (S.C. and M.F.). Any disagreement was resolved by a senior author (E.S.-B.).

Study Bias Assessment

Study quality was assessed independently by pairs of raters from the authorship group using the Cochrane RoB tool.²⁰ The RoB domains included selection bias, performance bias, detection bias, attrition bias, and other bias. Any disagreement was resolved through consensus.

Data Extraction and Statistical Analysis

Trial information was entered into RevMan 5.3.²¹ Data extraction was independently performed and cross-checked by the first 2 authors. SMD was calculated as mean pre- to posttreatment change in the intervention group minus the mean pre- to posttreatment change in the control group, divided by the pooled pretest standard deviation with a bias adjustment.²² SMDs for each trial were combined using the inverse variance method. Given the inherent heterogeneity of studies, random effects models were used. The I^2 statistic was calculated to estimate between-trial SMD heterogeneity. When multiple measurements were available for an outcome, the most frequently reported outcome across trials or the outcome that was judged to better tap the core of the construct was selected. To be consistent with the EAGG protocol, we considered as probably blinded those outcomes rated by an individual who was likely unaware of treatment allocation. Most proximal ratings were based on assessors close to the therapeutic setting and often not blinded. These ratings typically constituted a trial's own primary outcome measure and were therefore the assessment most available for analysis.

Selection of most proximal and probably blinded ratings was based on independent judgments and consensus of 3 authors (S.C., M.F., and E. S.-B.). Where 2 or more probably blinded ratings were available (which was sometimes the case on sham-controlled trials), what was deemed the best probably blinded outcome was selected for analysis, as in Cortese *et al.*⁴ When there were 2 or more neuropsychological outcome measures, the one most frequently reported across the relevant trials was selected. Four sensitivity analyses were conducted. The first included only trials using an active or sham control. The second was restricted to FBT trials. The third examined the effect of co-treatment with medication and was restricted to studies with no/low levels of medication (<30% of participants on medication, as per EAGG protocol). The final sensitivity analysis included only studies meeting the criteria defining a standard neurofeedback methodology as described by Arns *et al.*¹⁹ (see Supplement 2, available online). Meta-regression was conducted to assess the effects of number of training sessions. Publication bias was assessed with funnel plots and Egger tests. Analyses were conducted using RevMan 5.3²¹ and STATA 13.1.²³

RESULTS

A total of 13 trials²⁴⁻³⁶ met entry criteria. Figure 1 presents the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) selection flowchart. Table S1, available online, provides a list of excluded papers with reasons for exclusion. Retained studies included a total of 520 participants with ADHD. Table 1 gives information about the characteristics of the retained trials. Table 2 summarizes results of all analyses. There were fewer than 5 studies available to examine some of the planned outcomes such as working memory, parent ratings of executive functioning (e.g., Behaviour Rating Inventory of Executive Function³⁷), academic functioning (e.g., reading or arithmetic ability), or comorbid conditions (e.g., ODD, anxiety). Therefore, we did not perform analyses for such outcomes.

When most proximal assessments were the outcome, there was a small-to-moderate (SMD < 0.5) but significant effect on inattention, impulsivity/hyperactivity, and total ADHD symptoms (Figure 2). In sensitivity analyses

considering only trials with an active/sham control, the effects dropped to non-statistically significant levels for total ADHD and inattention symptoms. The effect was significant for hyperactivity/impulsivity but with a small effect size (SMD = 0.25), and the 95% CI was wide (CI = 0.03–0.47). When probably blinded outcomes were analyzed, effect sizes for ADHD outcomes dropped further, and none were significant (Figure 3). Results were also not significant when considering only probably blinded measures from trials with active/sham control (Table 2). When considering only trials evaluating FBT, results were significant for ADHD total (SMD = 0.37, 95% CI = 0.09–0.64) and hyperactive/impulsive symptoms (SMD = 0.26, 95% CI = 0.06–0.46) from most proximal raters, but not for ADHD inattentive symptoms or any ADHD symptoms rated by probably blinded assessors. There were insufficient trials ($n = 2^{30,32}$) for an analysis focused on SCP training (2 studies also used both SCP and FBT^{31,33}). When pooling only trials with no/low medication, results were significant only for ADHD inattentive symptoms, most proximal (SMD = 0.59, 95% CI = 0.31–0.88) and hyperactive/impulsive symptoms, most proximal (SMD = 0.32, 95% CI = 0.06–0.58).

When the analysis was restricted to only the 7 trials that met Arns *et al.*¹⁹ criteria for use of standard protocol, the SMDs increased for most proximal outcomes, although the 95% CIs were wide (Table 2). There were only 3 trials^{27,30,31} that both used a standard protocol and had probably blinded measures. In an exploratory analysis with these 3 trials, the effect on total ADHD symptoms was significant (SMD = 0.36), although the 95% CI was large (0.04–0.69).

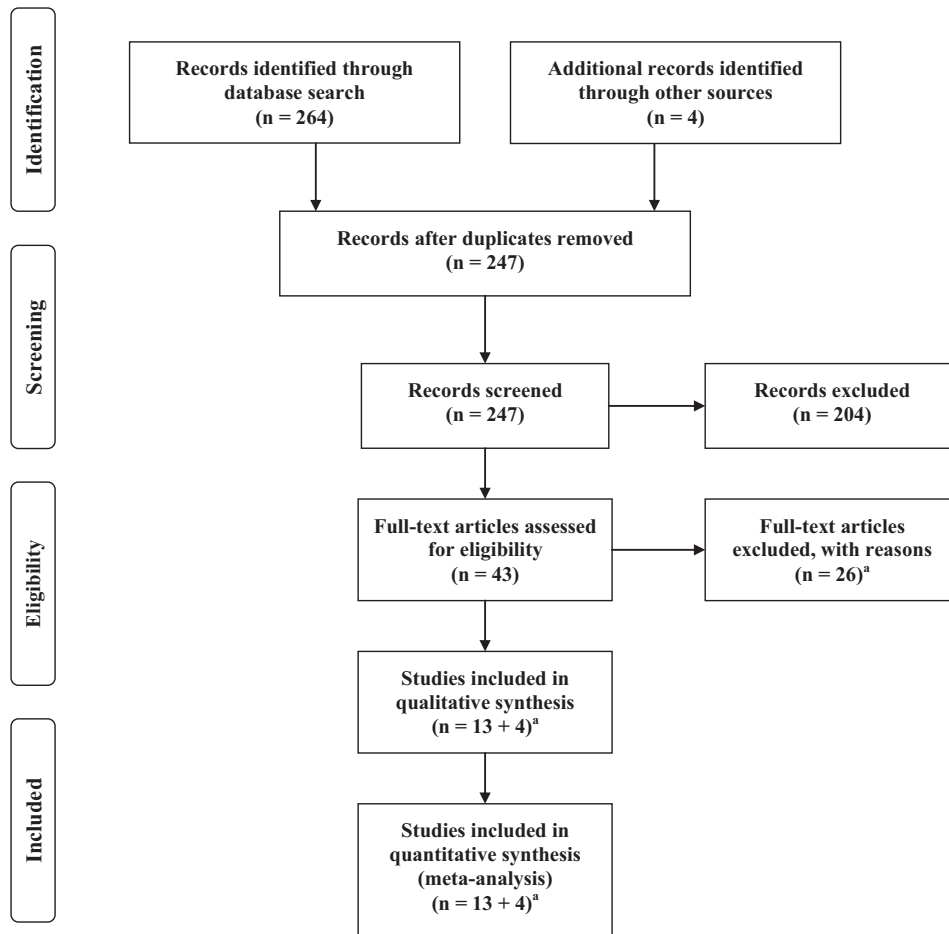
The analysis of neuropsychological outcomes required the pooling of diverse neuropsychological measures within general domains (Table 1). Effects on laboratory measures of inhibition (SMD = 0.30, 95% CI = –0.10 to 0.70) and attention (SMD = 0.13, 95% CI = –0.09 to 0.36) were not significant.

Direct evidence that neurofeedback training led to learning was gathered in only 4 trials^{27,29,33,36} and was positive for only 1 trial.²⁷ In 1 study,²⁹ there was a partial learning effect. In Heinrich *et al.*,³² there was indirect evidence of learning: the increase in the contingent negative variation (a slow cortical potential) suggests that children learned what was trained.

Studies varied considerably in terms of risk of bias (Figures S1 and S2, available online). For approximately half of the RoB domains across studies, the level of risk was unclear. For those categories that could be determined, 8 trials were rated as having a high risk of bias in at least 1 domain. In general, the major concern (i.e., high risk of bias) related to blinding of participants, personnel, and assessors. Among “other bias,” funding was not clearly stated in a sizable portion of studies.

Funnel plots and Egger test results (Figures S3 and S4, available online) suggested little evidence of publication bias, although the number of trials was insufficient to establish a reliable estimate. Finally, meta-regression analyses did not support a relationship between the number of training sessions and most proximal or probably blinded outcomes (Figures S5 and S6, available online).

FIGURE 1 Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram of selection of studies (last search updated on August 30, 2015). Note: Reasons for exclusion of each article are reported in Table S1. ^aFour articles included in the search results (^{29,35,36,46}) refer to the same sample as referred to by another four articles (^{44,43,45,31}, respectively), so the PRISMA flowchart does not count them twice.



DISCUSSION

The rationale for the use of neurofeedback for ADHD rests on the idea that promoting normalization or self-regulation of brain activity will translate into improved cognitive and behavioral control that is deficient in individuals with ADHD. A previous meta-analysis¹⁷ based on a limited number of trials (n = 8) was inconclusive with regard to the efficacy of neurofeedback for ADHD symptoms. Although there were moderate but highly significant effects (SMD = 0.59; 95% CI = 0.31–0.87) for ADHD outcomes rated by most proximal assessors, these were not significant when only probably blinded outcomes were considered (SMD = 0.29; 95% CI = –0.02 to 0.61). The current meta-analysis, including an additional 5 RCTs, provides what is likely to be a more reliable estimate of the effects of neurofeedback for ADHD. Importantly, the additional statistical power also allowed us to explore the impact of neurofeedback type, the effect of type of control, the impact of number of neurofeedback sessions, and the value of using standard

neurofeedback procedures. Although it does not provide a definitive statement as to the value of neurofeedback, the current analysis has clarified a number of issues.

In general, the effect size estimates in the current analysis are substantially smaller compared to the previous one by Sonuga-Barke *et al.*¹⁷ This is due to the smaller effect sizes reported in the most recent trials not included in Sonuga-Barke *et al.*¹⁷ For instance, SMDs dropped for most proximal and probably blinded total ADHD outcomes by 41% and 49%, respectively, although the analysis based on most proximal ratings of ADHD core symptoms remained significant. Crucially, as in the previous meta-analysis,¹⁷ when the risk of biased effect size estimates was reduced either by selecting probably blinded outcomes or by limiting analysis to trials with a high-quality control arm (sham or active), effects were no longer significant. Importantly, and in contrast to previous meta-analyses,^{15,18} there was no evidence for a particular benefit with regard to inattention symptoms, which have previously been hypothesized to be

TABLE 1 Characteristics of Studies Included in Meta-Analysis

Trial ^a	Design			Training				Sample			Outcomes		
	Control	Type NF	Standard Protocol ^b	Sessions, n	Follow-Up (mo)	N ^c	Meds (%)		ADHD Symptoms		Neuropsychological Outcomes		
							f	c	M-PROX	P-BLIND	Inhibitory Control	Attention	
Arnold ²⁶	Sham placebo ^d	FTB ^e	no	40	2	25	0	0	Parent	Teacher		BRC	
Bakhshayesh ²⁷	EMG biofeedback	FTB	yes	30	6 ^f	11	0	22	Parent	Teacher	CPT (commission)	CPT (omission)	
Beauregard ²⁸	No treatment	FTB ^g	yes	40	N/S	17	18	0	Parent		Counting Stroop (interference)	CPT (integrated visual and auditory)	
Bink ²⁹	TAU	FTB ^h	no	37	12	45	44.4	193.2 ± 39.6 (I)	Parent		Color Stroop (Interference)	D2 attention (total correct)	
Christiansen ³⁰	Self-manag.	SCP	yes	30	12	58 (tot.)	61.5	194.4 ± 40.8 (C)	Parent				
Gevensleben ³¹	AT	SCP + FTB ^k	yes	36	6	26	23 ^{i,j}		Parent	Teacher	ANT, Orienting (data from ⁴⁶ , that refers to the same sample of ³¹)	ANT, Conflict (data from ⁴⁶ , that refers to the same sample of ³¹)	
Heinrich ³²	WL	SCP ^l	yes	25	N/S	13	46.1	90-165	Parent		CPT (commission)	CPT (omission)	
Holtmann ²⁵	AT ^m	FTB ^l	yes	20	N/S	9	44.4		Parent		Stop Signal Task		
Linden ²⁴	WL	FTB	yes	40	N/S	20	79.4 ⁿ	123.6 ± 14.4 (mean)	Parent				
Maurizio ³³	EMG biofeedback	SCP + FTB ^o	no	36	N/S	14	0	110 (mean)	Parent				
Steiner ^{34p}	AT ^m , WL ^q	FTB ^h	no	23	N/S	8	0		Parent	Teacher		D2 attention (total score)	
Steiner ³⁵	CT, WL ^q	FTB ^h	no	40	6 ^r	11	7.6 ⁱ	102-154.8	Parent	Teacher			
						34	8.3 ⁱ	148.8 ± 10.8	Parent	Teacher			
						36	60	100.8 ± 13.2 (I)	Parent	BOSS			
							55.5	100.8 ± 13.2 (C)					

TABLE 1 Continued

Trials ^a	Design		Training		Sample			Outcomes		
	Control	Type NF	Standard Protocol ^b	Sessions, n	Follow-Up (mo)	N ^c	Meds (%)	ADHD Symptoms	Neuropsychological Outcomes	Attention
VanDongen ⁴⁵ Vallebregt ^{3,6s}	Placebo NF	FTB ^t	no	30	6 ^v	22 19	54.5 73.7	126.0 ± 26.4 (T) Investigator Teacher	M-PROX P-BLIND Inhibitory Control	Attention

Note: Studies are listed in alphabetical order. ADHD = attention-deficit/hyperactivity disorder; ANT = Attention Network Test; AT = attention training; BOSS = Behavioral Observation of Students in Schools; BRC = brain resource center computer-based named neuropsychological test; C = control group; CPT = continuous performance test; CT = cognitive training; EMG = electromyography; FTB = frequency theta/beta; NF = neurofeedback; N/S = not specified; SCP = slow cortical potential; SMR = sensory motor rhythm; SST = Stop Signal Test; T = treatment condition; TAU = treatment as usual; WL = waiting list.

^aIn alphabetical order, followed by study reference number.

^bStandard methodology described elsewhere.¹⁹

^cN is the number of individuals in the treatment (T) and control (C) conditions.

^dEqual intensity and duration.

^eEEG recorded from an electrode placed at position Cz vs. the ears as reference

^fResults of the follow-up were not published in this article.

^gSMR+beta.

^hSMR.

ⁱChildren were off medication 48 hours prior to all the assessments.

^jNumber in each group not specified.

^kSMR+beta Cz-ears.

^lCalculated from Cz vs mastoids.

^mStandard computer format.

ⁿA total of n=27 were medicated (no indication of how many in each group).

^oB=beta, [ORETA Tomography.

^pWe used parent #1 because parent #2 measures were available for a smaller sample (n = 9 for parent #1 and n = 5 for parent #2 in NF and n = 11 for parent #1 and n = 9 for parent #2 in the waiting list group); a discussion of the outcomes selected from this study is available elsewhere.^{47,48}

^qThe WL arm was used as comparator in the analyses.

^rThe results of the follow-up phase are published elsewhere.⁴³

^sThese 2 articles refer to the same study and present analyses on different outcomes.

^tIndividualized, mainly T/B, B = SMR.

^uData reported only at endpoint (1.5 wk).

TABLE 2 Summary of Results

Outcome	Trials Included	Measure	Trials, n	Effect of Intervention			Heterogeneity		
				SMD	95% CI	p	I ²	p	
ADHD									
symptoms									
Total	All	MPROX	13	0.35	0.11, 0.59	.004	41	.06	
		PBLIND	8	0.15	−0.08, 0.38	.20	0	.74	
	Active/sham	MPROX	7	0.22	−0.08, 0.52	.14	35	.16	
		PBLIND	6	0.20	−0.05, 0.45	.12	0	.66	
	FBT	MPROX	9	0.37	0.09, 0.64	.01	36	.13	
		PBLIND	5	0.03	−0.29, 0.35	.84	0	.76	
	Low medication	MPROX	7	0.39	−0.01, 0.79	.05	48	.08	
		PBLIND	5	0.26	−0.01, 0.54	.06	0	.76	
	Standard protocol	MPROX	7	0.45	0.02, 0.88	.04	53	.05	
		PBLIND	7	0.06	−0.24, 0.36	.70	41	.12	
	Inattention	All	MPROX	11	0.36	0.09, 0.63	.009	43	.07
			PBLIND	7	0.06	−0.24, 0.36	.70	41	.12
Active/sham		MPROX	6	0.26	−0.10, 0.63	.16	49	.08	
		PBLIND	5	0.21	−0.09, 0.50	.17	16	.31	
FBT		MPROX	9	0.33	0.00, 0.67	.05	51	.04	
		PBLIND	5	−0.04	−0.37, 0.28	.79	19	.29	
Low medication		MPROX	6	0.59	0.31, 0.88	.0001	0	.63	
		PBLIND	5	0.55	0.01, 1.09	.05	58	.05	
Standard protocol		MPROX	5	0.55	0.01, 1.09	.05	58	.05	
		PBLIND	5	0.15	−0.15, 0.44	.33	6	.37	
Hyper/imp		All	MPROX	10	0.26	0.08, 0.43	.004	0	.80
			PBLIND	7	0.17	−0.05, 0.39	.13	0	.59
	Active/sham	MPROX	6	0.25	0.03, 0.47	.03	0	.92	
		PBLIND	5	0.15	−0.11, 0.41	.26	0	.80	
	FBT	MPROX	8	0.26	0.06, 0.46	.01	0	.62	
		PBLIND	5	0.15	−0.15, 0.44	.33	6	.37	
	Low medication	MPROX	5	0.32	0.06, 0.58	.02	0	.44	
		PBLIND	5	0.15	−0.15, 0.44	.33	6	.37	
	Neuropsychological								
	test performance								
	Attention	All	Objective	8	0.13	−0.09, 0.36	.26	0	.72
		FBT	Objective	5	0.11	−0.16, 0.38	.43	0	.76
Inhibition	All	Objective	6	0.30	−0.10, 0.70	.15	56	.05	

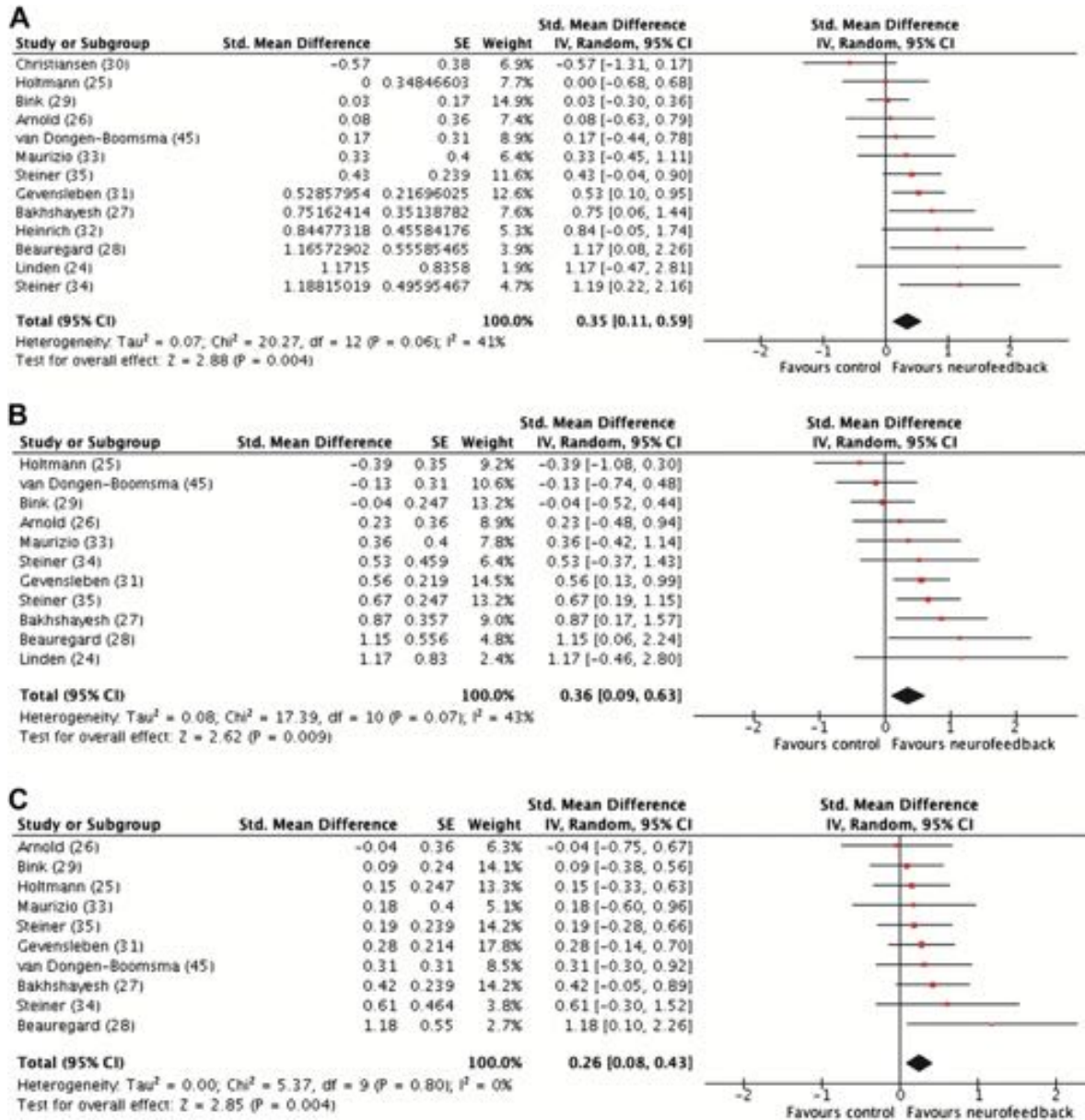
Note: Pooled standardized mean differences (SMD) are shown for each outcome. Positive SMDs indicate that neurofeedback is more efficacious than control condition. Significant effects are indicated in boldface type. There were insufficient (n < 5) trials for the following: standard neurofeedback (NF), probably blinded rater (PBLIND) total (tot; n = 3), PBLIND inattention (In; n = 3); standard protocol, most proximal rater (MPROX), hyperactivity/impulsivity (hyp/imp; n = 4), and PBLIND (tot, In, hyper/imp) (n = 3); trials using a version of frequency band training including theta–beta ratio training (FBT), inhibition (n = 4 trials); low medication, inattention, PBLIND (n = 4) and hyp/imp, PBLIND (n = 4). Active/sham = only trials with an active/sham control arm; ADHD = attention-deficit/hyperactivity disorder; All = all trials meeting inclusion criteria with available measures; SMT = self-management therapy; TAU = treatment as usual.

more amenable to neurofeedback. This was also reflected in our failure to find effects on laboratory measures of attention such as the continuous performance test.

Previously, Sonuga-Barke *et al.*¹⁷ have argued that the substantial drop in SMDs between most proximal and probably blinded analyses is likely to be the result of biases in perception in favor of the active treatment when one relies on observations by raters aware of treatment allocation. However, there are some other explanations. For instance, it is possible that probably blinded ratings are, for some reason, less sensitive to change than most proximal measures, perhaps either because of the instruments used or the person rating. Teachers, for instance, may be less sensitive to change than parents. However, essentially the same questionnaires were completed by both of these types of raters. It is also possible that proximal ratings accurately reflected real

improvements in the setting in which the treatment was delivered, but these effects failed to generalize to more distal settings in which blinded measures were recorded. This explanation seems unlikely, as probably blinded measures were also collected in the treatment setting for some trials, and these followed the same pattern. The type of neurofeedback protocol implemented in the trials did not seem to be an important factor in determining the results. When we restricted the analysis to trials using some form of FBT such as that focusing on alteration of the ratio between slow theta and faster beta oscillations, there was no increase in the effects of treatment. There was an insufficient number of trials (n = 2)^{30,32} using SCP training exclusively to state any firm assertion about this approach. The results raise the question of whether current training protocols have the appropriate treatment target. For instance, the

FIGURE 2 Forest plots for meta-analysis of effects on attention-deficit/hyperactivity disorder (ADHD) core symptoms assessed by most proximal (MPROX) raters. Note: A) ADHD total symptoms; B) inattentive symptoms; C) hyperactive/impulsive symptoms. SE = standard error; Std = standardized.

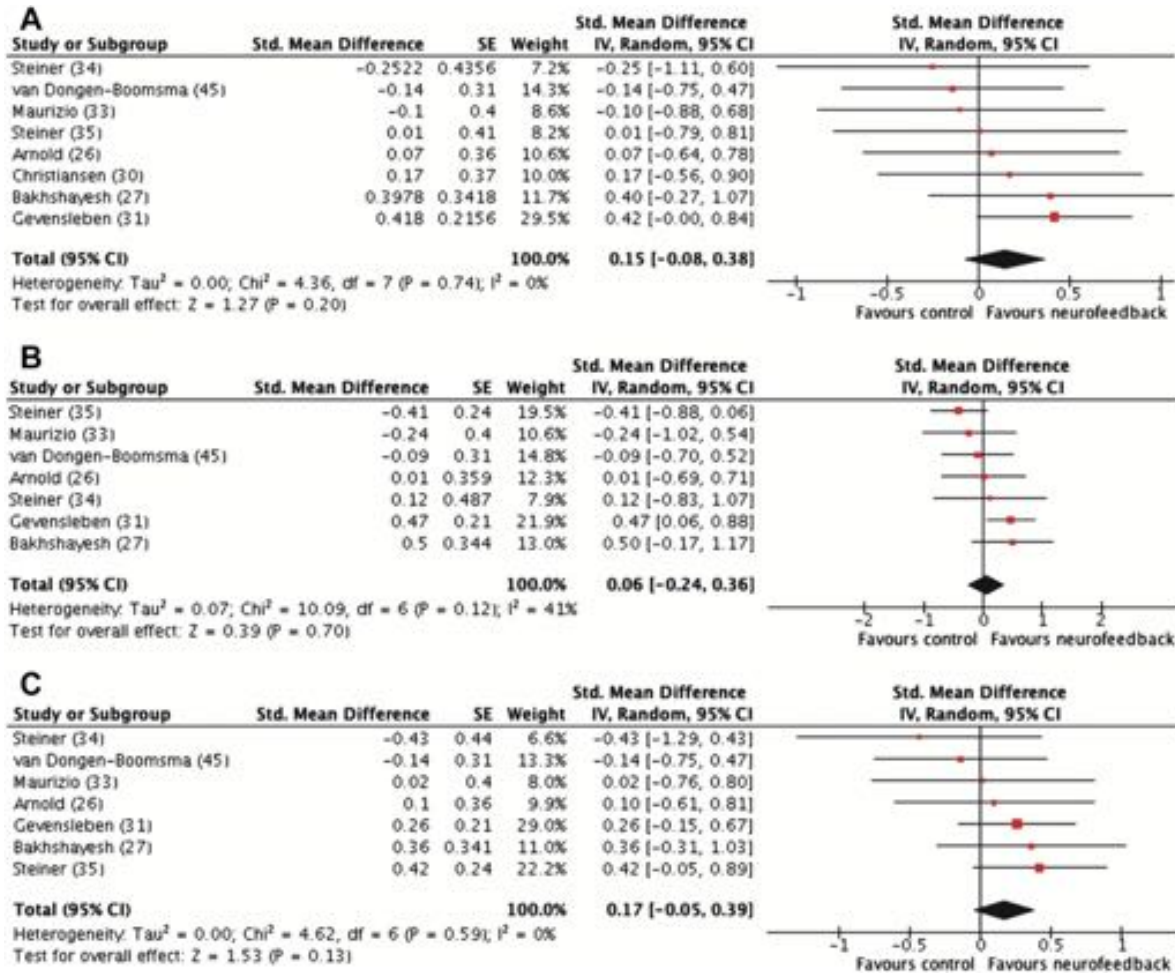


developmental stability of EEG frequency band alterations in ADHD from childhood into adulthood has been questioned,^{11,38,39} although the most commonly observed ADHD effects with regard to evoked brain responses relate to early rather than later components not targeted in current neurofeedback protocols. Therefore, the rationale for theta-beta feedback has been very critically discussed.^{9,40}

The value of a treatment meta-analysis is, of course, constrained by the methodology of the trials that it includes. It is therefore possible that the results of our meta-analysis reflect the methodological weaknesses of the included

studies rather than the weakness of neurofeedback as such. Indeed, the current set of 13 trials, taken as a whole, had a number of methodological short-comings. First, only 4 studies tested,^{27,29,33,36} and then only 1 study,²⁷ reported directly and positively whether neurofeedback training had actually led to learning as indexed by changes/improvement at the electrophysiological level. In the 1 positive trial,²⁷ the mediating role of changes in the electrophysiological signature leading to changes at the symptom level was not investigated. This is a crucial point, because if neurofeedback cannot bring about the expected changes at

FIGURE 3 Forest plots for meta-analysis of effects on attention-deficit/hyperactivity disorder (ADHD) core symptoms assessed by probably blinded (PBLIND) raters. Note: A) ADHD total symptoms; B) inattentive symptoms; C) hyperactive/impulsive symptoms. SE = standard error; Std = standardized.



the neural level, then treatment effects are more likely to be artifacts of some other, nonspecific aspect of the training. Such a situation may be similar to cognitive training targeting but not improving working memory, or to a drug with an established neurotransmitter profile not reaching the corresponding neural target system in the patient's brain. On the other hand, tests for learning of neural self-regulation, and addressing relations between learning and clinical improvement, would also need to consider more complex models and alternative mechanisms, for example allowing for initial, delayed, and nonlinear types of learning and translation,⁴¹ before concluding that effects are nonspecific. Further research should address whether possible neuronal modifications underpin putative behavioral changes in ADHD symptoms following neurofeedback.

A number of groups have defined what constitutes a standard neurofeedback protocol in terms of the number of training sessions, the reinforcement parameters operating, the EEG montage, etc. (see Vernon *et al.*⁴² for a justification).

Interestingly, only 54% of the studies in the current analysis met such a threshold. When we restricted the analysis to that subset of trials, our results were somewhat mixed; the effect size for total ADHD and inattention increased by about 20%. However, for all most proximal outcomes, 95% CIs were wide and close to nonsignificance. Unfortunately, there were only 3 studies with a standard protocol including probably blinded ratings,^{27,30,31} so that firm conclusions on the value of standardized protocols as defined by Arns *et al.*¹⁹ cannot be drawn. In addition, the level of methodological rigor specifically related to RCT conduct, as explored by the RoB tool, was in general unclear. The level of blinding was unclear or insufficient in many studies. In addition, 1 particularly striking omission in the majority of trials was the report of possible potential conflicts of interest. This would seem to be a major oversight in the current literature, given the growing presence of neurofeedback training companies in the commercial treatment marketplace. Given these limitations, it seems that without evidence for the learning of self-

regulation, and given the widespread use of nonstandard neurofeedback protocols, it is difficult to draw definitive conclusions about the ultimate value of neurofeedback approaches.

The EAGG has also recently completed meta-analyses for behavioral interventions and cognitive training using the same core protocol as used here.^{4,5} There are striking similarities but also some differences between the results of the present meta-analysis and those from these recent EAGG meta-analyses. In terms of effects on ADHD symptoms, neurofeedback, cognitive training, and behavioral interventions show almost identically sized positive and statically significant effects on total ADHD symptoms scores rated by most proximal assessors (SMD: behavioral interventions, 0.35; cognitive training, 0.37; neurofeedback, 0.35). Furthermore, in all 3 cases, the effects drop substantially to nonsignificant levels when probably blinded outcomes are used. This is more evident for behavioral interventions where the effects drop to 0 but is also substantial for neurofeedback and cognitive training (SMD = 0.15 and 0.20, respectively). However, 1 quite striking difference among the 3 meta-analyses is that both behavioral interventions and cognitive training had predictable positive effects on outcomes other than ADHD. For instance, for working memory training, there were highly significant effects on neuropsychological measures of working memory, whereas behavioural interventions improved parenting rated by independent observers and had positive effects on probably blinded measures of conduct problems. By contrast, we did not find evidence for effects of neurofeedback on neuropsychological outcomes.

Caution is required when interpreting these findings, given a number of limitations in addition to the issues raised with regard to the nature of the trials above. First, effect size estimates may be inflated because of the failure to report intention to treatment analyses in most trials. Second, there were insufficient trials measuring important outcomes such as working memory task performance, academic skills, general functional impairment, IQ, and other mental health problems such as conduct problems. Third, few trials included long-term outcomes (Table 1) to allow an evaluation of the extent to which effects on clinical symptoms grew over time or effects on neuropsychological processes persisted. Fourth, no trial recruited participants based on the presence of ADHD-related deficits in EEG signature (i.e., altered theta-beta ratio). This may limit the chance for neurofeedback-related improvements in symptoms. Fifth, it was necessary to pool data from diverse measures from different tasks to have sufficient trials to analyze neuropsychological functions. Although in principle the pooled measures tapped the same neuropsychological domain, this inevitably was a somewhat arbitrary process that likely increased SMD heterogeneity. In addition, ADHD is a pathophysiologically heterogeneous disorder, and distinct EEG subtypes have been described (e.g., cortical hyperarousal versus hypoarousal subtypes). Patients might require more specific and tailored training targeting different deficits associated with ADHD. Combining

different neuroimaging approaches with neurofeedback training might be a useful approach in the future. Finally, the range of number of sessions across studies may have been too restricted to allow the detection of possible effect of session number.

In summary, the current meta-analysis shows that evidence from well-controlled trials with probably blinded outcomes does not support neurofeedback as an effective treatment for ADHD, in terms of either ADHD symptoms or other cognitive correlates. Future research should focus on the following: identifying the most appropriate electrophysiological treatment target; increasing the use of standard EEG and learning protocols; developing new methods to optimize the chances that neurofeedback leads to learning at the brain level; and identifying predictors of treatment response for individual patients or at least in distinctive subgroups of children. ϵ

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