Review on Frequency Neurofeedback on Autism Spectrum Disorder: Overview, Efficacy and Research Direction

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Abstract

Frequency neurofeedback (FNF) is a biofeedback method that targets frequencies between 1 and 50 Hz. The efficacy of FNF with autism has been labeled 'probably efficacious' in literature reviews in the last decade, despite new research pointing towards a higher standard. The aim of this review was to analyze key features of these studies, with a goal of determining the efficacy standard of FNF on autism and establishing a research direction. Electronic databases and literature reviews were used to collect a total of ten randomized and/or matched controlled trials. FNF reaches a Level 4 efficacy standard, with an impact on a broad range of factors including core autistic traits, social communication, emotional regulation, cognitive flexibility, executive function, behaviors of concern, attention, metabolic or thermal activity, and EEG e.g. decreased absolute power, mu rhythm, coherence and hyperconnectivity. Current evidence generalizes to male children, up to 18 years, with a low-average or higher intellectual functioning, with autism as the only diagnosis. A meta-analysis suggests a large superior effect when compared to wait list controls. Current research does not meet the higher efficacy standards outlined by Arns et al. (2020). Small samples plague most studies, and the maintenance of improvements post-training are yet to be assessed adequately. Eight recommendations are made.

Literature review

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Introduction

Frequency neurofeedback (FNF) is a biofeedback method that typically targets frequencies between 1 and 50 Hz. The evidence base for FNF on autism has been accumulating gradually over the last two decades since the first published trial by Jarusiewicz (2002). Coben et al. (2010) conducted a brief literature review of four studies and concluded that FNF is 'probably efficacious' based on the standards developed by the Association for Applied Psychophysiology and Biofeedback (La Vaque et al., 2002). However, a subsequent review by Holtmann et al. (2011) concluded that "the existing evidence does not support neurofeedback as a treatment that can be recommended for ASD core symptoms. Reviewed studies suggest that neurofeedback protocols that inhibit theta and reward beta activity or sensorimotor rhythm may hold promise for the treatment of ADHD-like symptoms in children with autism." Several literature reviews have been

published (e.g. Kumari & Sharma, 2020; van Hoogdalem et al., 2020) on the efficacy of FNF on autistic children. Van Hoogdalem et al. (2020) was a relatively brief review and included studies that did not employ FNF, such as Liu et al. (2017), which used a blood flow biofeedback intervention (HEG). Kumari & Sharma (2020) concluded that current research does not provide sufficient conclusive results of the efficacy of FNF on autism and social cognitive deficits.

Moreover, Arns et al. (2020) proposed analysing FNF studies based on stricter guidelines than those proposed by La Vaque et al. (2002). These guidelines, based on suggestions from Tolin et al. (2015), focus on two systematic reviews conducted in the last two years that report effect sizes, remission rates, safety and side-effect profiles, and cost-benefit analyses.

Based on these conflicting conclusions about FNF on autism, and the higher standards outlined by Arns et al. (2020), this article sought to analyze critically studies with an aim of establishing a reliable clinical efficacy standard, identifying strengths and limitations of the research, and providing future research direction. I will provide a broader analytical interpretation in contrast to previous literature reviews.

Method

Search strategy

The current author searched for scientific articles in Google Scholar and specific databases such as PubMed, Ovid MEDLINE, EMBASE, ERIC, and CINAHL using the key words: neurofeedback, neurofeedback training, neurotherapy, NF, NFT, NFB, autism, ASD, autism spectrum disorder. Search queries were adopted using Boolean operators AND and OR to locate studies. Additional references were identified in key literature reviews e.g., Coben et al. (2010), Marzbani et al. (2016), Holtmann et al. (2011), van Hoogdalem et al. (2020). English was chosen as the search language. Experimental articles up to May 2025 were analyzed.

Inclusion/exclusion criteria

Studies were included in this review if they were: a) peer-reviewed; b) written in English; c) had a reasonable sample size; and d) were a randomized and/or matched controlled trial. There were some studies that were excluded, including: Zivoder et al. (2015), where the sample was small (n=10) and the authors failed to report data even though findings were discussed; Darling (2007), where the paper was not peer-reviewed and his paper had a small sample size (n=6); and Mohammadi et al. (2019), which was not in English.

Summary of studies

Tables 1 shows ten studies reported across ten published articles with one article reporting two studies (Pineda et al., 2008) and one research group reporting follow-up data in a separate article (Kouijzer et al., 2009a, 2009b). The first published trial was in 2002 by Jarusiewicz. There is less than one study every two years being published in this field, which begs the question, why is the publication amount so low? FNF is still a relatively obscure and esoteric clinical therapy and is only beginning to attract research focus. A major barrier is the cost of equipment and training; FNF is a relatively complex therapy and to conduct research requires an experienced clinical researcher to govern the protocols using equipment and licences that cost at least $5,000USD. It entails a considerable commitment from the subject e.g. 10 hours plus of training time. Moreover, there is a growing number of studies on alternative neurofeedback methodologies such as Slow Cortical Potentials (SCP), fMRI neurofeedback,

and infra-low neurofeedback (ILF), which take focus from FNF as an intervention worthy of study.

Results

Sample characteristics

A glaring shortcoming of these studies is consistently small sample sizes, that equates with low-powered studies. Button et al. (2013) argue that low powered studies impact in three ways, including a) the low probability of finding true effects; b) the low positive predictive value when an effect is claimed; and c) an exaggerated estimate of the magnitude of effect. Points a) and b) reduce the probability of a true positive finding, and c) suggests that the effect is misleading because the finding is an outlier or the 'winner's curse'. The 'winner's curse' suggests that the first promising finding (e.g. Jarusiewicz, 2002) is an exaggerated positive result, and any attempts to replicate are difficult, presumably as they are not outliers. However, Jarusiewicz's study has been replicated several times, and Coben and Padolsky (2007) demonstrated a stronger effect with half the amount of FNF training using QEEG-derived protocols. This does not support the notion of a 'winner's curse' in the FNF research.

Larson and Carbine (2017) recommend calculating sample sizes before engaging in sampling, or at least calculating correlations between pre- and post-test measures to accurately calculate future sample sizes. This ensures that studies are sufficiently powered, reduce the probability of a false negative finding ('Type II' error), and ensure that effect sizes are not inflated. This has not happened consistently in EEG or ERP studies, and this recommendation should be followed when studying FNF on autistic populations.

There is also a possibility of publication bias, since only a handful of studies have been published since 2002. The publication rate has been low, and this may be due to many null findings not being published as they are unremarkable to scientific journals. Begemann et al. (2016) conducted a meta-analysis of five studies relating to FNF on autism and "showed a large superior effect of 0.85 (p=.003, 95% CI=0.29 to 1.40). Heterogeneity was moderate …, publication bias was not indicated." They added: "Four studies combined showed a large superior effect for neurofeedback compared to waiting list or skin conductance therapy (ES 0.80, p=.029, 95% CI=0.08 to 1.52) … Pineda et al (2008) found a large superior effect of neurofeedback over placebo treatment (ES 0.96, p=.039)" (p. 25). Moreover, Begemann et al. (2016) report a fail-safe N_R for ASD general symptomatology of 18 and a fail-safe N_R for passive treatment of 11. This suggests that many null finding studies (11 to 18 times greater than positive finding studies) are required to negate the positive effects reported in any given published study.

Based on the Begemann et al. (2016) review, assuming an effect size of .80, significance of .05 and power of .8, the estimated sample size per group is 26 (N=52). Researchers should account for dropout rates across groups, and aim for a final, eligible sample size of 26 and not an initial larger sample size at the beginning of a study. A participant of a FNF study is required to engage in several sessions each week, over a few months, which is a large commitment, and a moderate dropout is expected. The dropout group should also be compared with completing subjects to assess varying characteristics of these groups, that may influence the dependent variable. Carrick et al. (2018) failed to analyze completing participants compared with non-completing participants, which is a significant analytical flaw of this study (see limitations in Table 1).

A trend in the research is that autistic groups reported in Table 1 are usually children (4-17 years) who

Table 1. Randomized and/or matched controlled trials

Author(s)	Design & Measures	Sample	Protocol	Results	Limitations
Jarusiewicz (2002)	Matched controls, randomized to FNF or waitlist, pre-post ATEC, parent rated ATEC scores for both groups = moderate traits (score = mid 60s)	N=24 (initial sample = 40) 12 children with 12 matched controls (age, gender, severity) – one female in FNF and control groups 4-13 years age	C4-A1 Reward: 10-13 Hz (or lower) Inhibits: 2-7, 22-30 Hz 57% of sessions used this protocol 20-69 sessions 30-minute training	Experimental group showed reduction in ATEC by 26%. Sociability reduced by 33%, Speech/Language/Communication 29%, Health 26% and Sensory/cognitive awareness 17%. Control group reduced by 3% only. Full scale ATEC fell in experimental group from 65 to 48 and control group from 63 to 61. Parental reports from experimental group indicated improvements on a scale of 1-10 on socialisation (M=5), vocalisation (M=5), school work (M=5), anxiety (M=3), tantrums (M=4) and sleep (M=9).	Multiple protocols No sham or alternative treatment control group No blinding No follow up
Coben & Padolsky (2007)	Matched controls, pre-post test Wait list controls ATEC, GADS, GARS, BRIEF PIC -2, QEEG, IR Imaging Parent judgement ATEC baseline score = 45 for both groups (mild- moderate traits)	Experimental: n=37 (one drop out) 31 male, 6 females Mean age: 8.92 yrs Controls: n=12 Mean age: 8.19 yrs Matched: age, gender, race, handedness, ASD severity	QEEG assessment-based protocols (bipolar montages targeting temporal or frontal regions) Reward: somewhere between 5-16 Hz Inhibits: 1-6 Hz (92%), 7-14 Hz (68%), 18-30 Hz (100%) 20 sessions twice a week	Parent ratings: 89% parents rated improvements in ASD symptoms in experimental group. ATEC: Total score reduced by 40% in experimental group. Correlated with significant reductions in ASD behaviors, executive deficits, and ASD symptoms as reported on: GADS, BRIEF and the PIC-2. Significant improvements for the experimental group on composite measures of attention, visual perception and executive function. Improvement in language skills was significant. 76% experimental group decreased in cerebral hyperconnectivity. Increased self-regulation of metabolic or thermal activity (IR Imaging). Control group showed no significant change on parent rating of symptom severity, attention, parental judgements of treatment outcome.	No randomization Multiple protocols No placebo or alternative treatment control group No blinding Training time not specified No follow up

Table 1. (continued): . Randomized and/or matched controlled trials

Author(s)	Design & Measures	Sample	Protocol	Results	Limitations
Pineda et al. (2008) Study 1 Study 2	*Study 1:* Randomized, sham controls, pre-post QEEG, Mu Suppression Index (MSI), TOVA, ATEC, Apraxia Imitation Scale Participants & parents blinded *Study 2:* Study 1 design with participants, parents and technicians blinded	*Study 1:* 7 Males, 7-17 years (one drop out) Mean age: 9.3 yrs Experimental=4, Sham control=3 IQ>80 *Study 2:* 19 ASD participants, 7-17 years Mean age=9.8 Sham control, n=10 (all males) Experimental n=9 (6 males) IQ>80 ASD diagnosis verified	*Study 1:* C4-A2 + EMG (trapezius shoulder muscle) Reward: 8–13 Hz Inhibit EMG: 30-60 Hz Placebo received EMG and artificial mu-like signal 8–13 Hz feedback 30 sessions, three times per week 30-minute training *Study 2:* Same as Study 1 except placebo received 10–13 Hz signal	*Study 1:* Experimental group showed decreased coherence at C3/C4 for most frequency bands; placebo group showed increased coherence. All frequencies significantly decreased for experimental group; increased for placebo group at C4. TOVA: reaction time decreased by 70% in experimental group; no change in placebo group. ATEC: decrease in Sensory/Cognitive Awareness subscale in experimental group (reduced autistic traits); increase in the control group. Mu: experimental group reduced Mu power over training; no change for control group. Apraxia Imitation Scale: increased imitation but no significant difference between groups. *Study 2:* MSI: 75% of experimental group showed significant suppression following training in the Hand, Crayon, Social, and Happy face videos. There was an average of 32% and 26% increased suppression in the Hand and Crayon conditions, respectively, and a 33% increased suppression between initial and post training in the Social condition. No subject in the placebo group showed suppression in these conditions following training. TOVA: reaction time z-scores decreased in experimental group; the control group increased. Similar trend with total commission errors. ATEC: experimental group showed score reductions on Sensory/Cognitive Awareness subscale; control group showed no changes. No correlation between MSI and ATEC scores. Apraxia Imitation Scale: Improved imitation at post. No effect of training on imitation.	*Study 1:* Very small sample Technicians not blinded Auto-thresholding at constant 75% reward ASD diagnosis not verified No follow up *Study 2:* No follow up

Table 1. (continued): . Randomized and/or matched controlled trials

Author(s)	Design & Measures	Sample	Protocol	Results	Limitations
Kouijzer et al. (2009a) with follow-up study (2009b)	Matched controls, pre-post CPT, TOSSA, TOL, neurocognitive battery, parent report (15 items)	N=14 children with ASD (12 males) 8-12 years Mean age: 10.1 yrs Inclusion criteria: IQ>70 Seven children per group Matched diagnosis, age, sex, IQ	C4-A1 7 X 3 min training periods, 1 min rest between periods Reward: 12-15 Hz Inhibit: 4-7 Hz	TOSSA auditory: FNF: 30% increase in correct responses; Control group: no change. Interference effects (written names): FNF group: 55% reduction; Control group: 24%. Set shifting, concept generation and goal setting showed similar trends. Parents reported improved communication, social interaction and typical behavior in FNF group, but not controls. All parents would recommend FNF to others. Behavioral improvements were maintained after 12 months.	No randomization Small sample
Kouijzer et al. (2013)	Randomized, waitlist, pre-post 6-month follow up Blinded subjects and parents ADI, SCQ, Clinical Global Impression, Tower of London, Stroop test, TOSSA, Digit Span, WISC, Trail Making, QEEG	FNF group: n=13 (10 males) Mean age: 15.3 yrs Skin conductance (SC) group: n=12 (9 males) Mean age: 14.5 yrs Wait list: n=13 (11 males). Mean age: 15.9 yrs Inclusion Criterion: IQ=80+ Exclusion Criterion: Comorbidities	CZ-mastoid or FCZ- mastoid Reward: 50-80% - increased reward time if subject not motivated Inhibit: anywhere from 2-9 Hz 23-40 sessions (twice a week) 7 X 3 min training periods, 1 min rest between periods	Subjects with a negative correlation between FNF or SC amplitude and the number of sessions were classed as EEG- or SC-regulators; subjects who did not show a correlation were classed as EEG- or SC-non regulators. Blinding was analyzed to be true for FNF and SC groups. SCQ: EEG-regulators showed improvements on absolute score for full scale and all subscales, however they were not significantly higher than EEG non-regulators, who showed varied results of improvement or worsening across subscales. No change on Clinical Global Impression scores (as rated by technicians). EEG-regulators improved cognitive flexibility on Trail Making Test, but SC-regulators did not. Improvements increased at 6-month follow-up. No differences on social communication, inhibition, planning, attention, working memory or executive function. QEEG data: No significant differences across groups. Treatment expectancy did not influence outcomes since FNF and SC groups were prepared for training identically and participants were unaware of what the feedback related to i.e. EEG. Parents were blinded. Nonspecific effects were accounted for.	Small sample Technicians not blinded Higher reward rate if subject not motivated No reward bandwidth, inhibits only No sham controls Successful subjects (EEG regulators) separated from non-responders

Table 1. (continued): . Randomized and/or matched controlled trials

Author(s)	Design & Measures	Sample	Protocol	Results	Limitations
Goodman et al. (2018)	Active controls, randomized, pre-post HRV (Group 1, N=7), HRV + FNF (Group 2, N=8) SRS-2, Mu Suppression Index (MSI), QEEG, ATEC, ERC, Spence Anxiety (SPS) Parent rated	15 ASD children (13 males) 9-18 years Mean age: 12.4 yrs (Ave. age across the groups: 12.1 to 12.5 yrs) Average IQ across groups: 87 to 94 full- scale score Randomized on age, gender, IQ	C4-? Reward: 8-13 Hz 4 preliminary sessions of HRV 12 hours of training for HRV and HRV+FNF groups over six weeks DVD training 70%-80% reward rate 10-20 mins / day breathing at home at participant RF	Group 1 and Group 2 post training reduced on L/N & ER subscales, and SRS and ATEC full scale scores. Group 1: increase in ER subscale, indicating improvements in emotion regulation. Significant decrease in SRS, indicating a reduction in social impairments. No change in L/N subscale, or Spence or ATEC full scale scores. Group 2: significant decrease in L/N subscale, suggesting reduction in lability. Significant decrease in ATEC full scale, suggesting reduction in autistic traits. No change in ERC full scale, Spence or SRS. Group 1 showed a small increase in mu suppression post-training, and Group 2 showed a large decrease in mu suppression post-training. Reduced suppression was greatest over the central, parietal and occipital lobes. Group 2 showed improvements in HRV factors over training, Group 1 did not. Group 2 showed higher alpha power at post training compared with Group 1. Group 2 showed less mu suppression at post training; Group 1 showed no change. Less mu suppression at post training in Group 2 observed in central and occipital cortices.	No treatment control group Omission of a FNF-only group High reward rate – 70-80% Autothreshold: threshold adjusted to deliver reward of 70-80% No follow up Study could have inhibited 8-13 Hz also
Datko et al. (2018)	Matched controls (age, gender, IQ), pre-post Parent surveys pre and post training, parent-completed ATEC, SRS, ADOS, ADI, fMRI	10 ASD children (7 males) IQ>80 8-17 years Mean age: 12.5 yrs 7 typically developed (5 males) 8-17 years Mean age: 10.6 yrs	C4-? Reward: 8-13 Hz Inhibit:4-8 Hz, 13-30 Hz Two sessions per week 45-minute sessions Mean training hours: 26.4 hours for ASD group; 17.2 for TD group DVD training	ASD group showed a significant reduction on ATEC full scale score (38.7 down to 27.2). ASD group showed a significant reduction in SRS full scale score (79.5 down to 70.6). There was no absolute change in the TD group SRS score. ASD group showed higher activation in the right IPL (BA 40) after FNF. Controls had widespread lower activation after FNF, including in bilateral precentral gyrus (BA 4 & 6), right IFG (BA 44), left IPL and supramarginal gyrus (BA 40), and bilateral occipital areas (BA 17). Differences in ASD and TD groups before FNF were absent post assessment. Increased activation for ASD group was correlated with decreased SRS scores (improved social functioning) and decreased ATEC scores (reduced symptoms of autism). ADOS: greater task activation changes were correlated with lower initial ASD severity. A stronger effect was observed with higher severity of autism. No correlation on ADI. ***Results show that FNF targeting mu rhythm in autistic adolescents has a neurophysiological basis in the mirror neuron network.***	Small sample No follow up No sham controls TD group mitigates non-specific effects No blinding Neurological changes overrides no blinding

Table 1. (continued): . Randomized and/or matched controlled trials

Author(s)	Design & Measures	Sample	Protocol	Results	Limitations
Carrick et al. (2018)	Randomized, sham controls, subject blinded (no technician blinding required) QEEG, posturography, Parent rated: ATEC, SRS-2, BRIEF, ABC, QABF	N = 34 28=Male, 6=Female Active: n=17 Control: n=17 4-17 years Diagnosed ASD Exclusion criteria: comorbidities	FNF using Mente device (binaural auditory feedback - volume) FP1-O1, FP1-O2, FP2- O1, FP2-O2 Delta (1-3 Hz), theta (4-7 Hz), alpha (8-13 Hz), beta1 (14-19 Hz), beta2 (20-35 Hz) Reward Beta 1, inhibit all other frequencies 45 minutes daily over 12 weeks	ATEC: Behavior worsened for Active group on the Speech and the Sensory/Cognitive Awareness subscales, and improved on Sociability. Health/Physical Behavior improved for Control group. Total score did not change. No change to SRS-2 scores across the groups. BRIEF: Active group showed significant improvements at post-test on Shift, Initiate, Organization of Materials, Behavioral Regulation Index, Metacognition Index, and in the Global Executive Composite. ABC: Active group showed significant reduction at post-test in autistic behaviors. QAFB: Active group showed significant reduction in problem behaviors i.e. Escape, Nonsocial Reinforcement and total score. Active group parents rated significant improvements in communication and social skills. No change in Control group. QEEG showed normalisation in Active group across all bandwidths. Controls no change.	No follow up >50% drop out in Active and Control groups and no analysis of drop out characteristics
Wang et al. (2024)	Randomized, sham controls, parents and experimenters were blinded of treatment type (i.e. pitched as "attention" training) SRS, PEP-3, ATEC, CARS -2 FNF group had a higher baseline SRS score	FNF group: n=17 (14 males) Mean age: 55 months Sham group: n=24 (17 males) Mean age: 52 months Inclusion Criteria: IQ>79	FPZ-left mastoid Mu suppression training in FNF group 60 sessions (twice a week) 30-minute sessions Feedback delivered by a mobile headset, driven by AI-powered EEG algorithms. FNF was conducted at home and at the clinic.	FNF group experienced significantly greater improvements than sham controls on expressive language subscale of the PEP-3. They also experienced significantly greater improvements on the Sensory/Cognitive awareness subscale of the ATEC. The full-scale ATEC score approached significance i.e. the FNF group showed significantly greater reductions on the total ATEC score. The SRS showed no change between treatment groups, possibly due to baseline differences i.e. FNF group had a significantly higher baseline SRS score.	Mu training was conducted at FPZ (not at C3 or C4 where Mu manifests) No follow up

are verbal with an IQ score >70. Males have been selected in greater proportion in these studies, usually on a ratio of 3:1 or 4:1. Autism is diagnosed more often in males, which explains the higher ratio of males to females studied. The incidence rates of autism in males and females are often attributed to genetics, however there is some argument of the under diagnosis of females, due to a varying presentation, and diagnostic bias towards males (Tsirgiotis et al., 2023). In spite of these arguments, future research could focus on factors such as biological sex, IQ level (including intellectual deficit, ID, versus non-ID) and verbal abilities (i.e. non-verbal, minimally verbal, verbal).

To summarise, the findings suggest a generalization to young males under 18 years, who are verbal with an IQ above 70. This would capture a reasonable proportion of the population with autism but does not generalize to adults with autism, or autistic females of any age.

Design

A major area if evaluating the efficacy of FNF is the type of methodological designs employed in the research. The factors to consider include selection process and allocation (i.e. self-selected, randomization), blinding (i.e. participant and parent only, or double blinding, including FNF technician) and type of control (i.e. waitlist, active, matched). Controlling nonspecific factors is very important, such as: contextual (e.g. subject is engaging in an experiment and expects to improve, positive regard for technician, EEG display is perceived as scientific and therefore efficacious); attention effort (e.g. participant watches a screen for 30 minutes, 30-50 times and improves due to focussing for long periods of time); repeated reinforcement (e.g. technician gives verbal reinforcement, audio-visual feedback unrelated to EEG data); and, specifically with autism, the routine of attending regular sessions, which can satisfy the 'need for sameness' or a routine. The gold standard in biofeedback is a double-blind randomized sham or active controlled design (van Doren et al., 2019). The active control group receives cognitive training or EMG biofeedback to control for nonspecific effects e.g. placebo.

Four studies used the gold standard of a blinded randomized sham control (Pineda et al., 2008, studies 1 and 2; Carrick et al., 2018; Wang et al., 2024) with another study controlling for nonspecific effects by adopting a typically developed control group (Datko et al., 2018). The Carrick et al. (2018) study is problematic because the dropout rate was greater than 50% in the experimental and control groups with no reported data analysis of dropouts versus completing participants across the groups. If the experimental group dropouts failed to respond to FNF, then the data from this subgroup may have negated the effect reported in the study. Datko et al. (2018) is also problematic because the researchers could have incorporated sham controls for both ASD and typically developed groups. The two Pineda et al. (2008) and the Wang et al. (2024) studies are consistent in findings, that is, a reduction in the ATEC Sensory/Cognitive Awareness subscale.

It is worth mentioning that Wang et al. (2024) used a technician delivered and home-based AI-driven device that delivered audio-only feedback, with a reduction in core autistic traits as measured by the ATEC.

Measurement

One of the strongest features of this body of research is the use of standardized psychometric measures. Researchers mostly employed the Autism Treatment Evaluation Checklist (ATEC), which is available free of charge and has been validated as a treatment outcome measure for autistic populations (Rimland & Edelson, 1999). The ATEC has an advantage of providing normative scores based on age, and

accounts for change based on maturation. If a study uses the ATEC and has a one-year follow-up methodology, then actual change scores must be compared with change scores due to maturation alone.

All studies have used a pre- and post-therapy methodology, spanning over a few months to six months, except one study that reported post-training follow-up data (Kouijzer et al., 2009a, 2009b). Kouijzer (2009b) reported that any immediate behavioral and cognitive improvements from FNF were maintained (and some participants improved even more) at a 12-month follow-up. These findings are consistent with van Doren et al.'s (2019) meta-analysis which reported that ADHD symptoms reduce even further from post-FNF treatment to 12-month follow-up, due to the mediating factor of sleep improving.

Another strength of this research is the use of specific, standardized psychometric and diagnostic measures of social functioning (e.g. SDS), IQ (e.g. WISC), attention (e.g. TOVA), autism diagnostics and traits (e.g. ADI, CARS), emotion regulation (e.g. ERC), anxiety (e.g. Spence Anxiety), medical diagnostics (e.g. fMRI, QEEG) and cognitive functioning (e.g. BRIEF). Whilst this is a relatively comprehensive use of standardized psychometrics, there are some areas that are yet to be measured, that are important for people with disability, such as: quality of life, education and learning, employment, activities of daily living (ADLs) and mood (i.e. depression). Researchers should aim to use measures that are sensitive to change and applicable to neurodivergent populations (e.g. ASQoL, ASC-ASD).

A further strength of this field is the use of multiple sources of data to correlate the effects of FNF and pinpoint a mechanism. For example, Datko et al. (2018) correlated psychometric measures (e.g. autistic traits, ATEC, social functioning, SDS) with diagnostic data (e.g. ADOS, ADI) and fMRI. Future studies should aim to target an area of dysfunction (e.g. social deficits), hypothesise an area of training that would reduce a dysfunction (e.g. mu rhythm using C4-A1), and measure brain functioning (e.g. fMRI) or a biomarker (e.g. event-related potential, ERP) that reflect those changes. The ultimate test of FNF efficacy is: a) EEG changing in the direction predicted by the protocol (i.e. increased mu suppression, increased SMR); b) emotional, cognitive and/or behavior change that is correlated with EEG change; and c) maintenance of these changes following the withdrawal of the treatment i.e. self-regulation in the absence of brain-derived feedback.

A potential weakness of these studies is the use of parent-only ratings which are defined as 'most proximal' and 'least blinded' ratings. Cortese et al. (2016) showed that the experimental effects of FNF for ADHD diminished to non-significance when teacher ratings were analyzed across multiple studies, since teacher ratings are presumably 'less proximal' and 'probably blinded'. This phenomenon was also replicated with sham controls with blinded raters. Van Doren et al. (2019) argue against the 'proximal-blinded' concept suggesting that parents rate different cognitive and behavioral elements of ADHD than a teacher, or ADHD behaviors are rated in a different context e.g. home, community. Furthermore, Table 1 shows that Pineda et al. (2008) and Carrick et al. (2018) used sham controls and still found an effect, whilst Kouijzer et al. (2013) used a double-blinded procedure for FNF and skin conductance groups (both groups were identically prepared and were unaware of the true feedback they were receiving). An effect was demonstrated with blinded parents, however the separation of experimental participants into regulators versus non-regulators is slightly dubious. Seven out of 13 subjects in the FNF group were identified as regulators, suggesting that approximately 50% of cases will respond to FNF when using standardized protocols. Outcomes were maintained at a six-month follow up.

Standardization

There is a call for standardization of training in FNF research both in terms of protocols and delivery of training (Arns et al., 2020). In regards to delivery of training, Table 1 shows a wide variety of approaches to training, including auto-thresholding (Pineda et al., 2008) and/or high reward delivery (Goodman et al., 2018). The problem with auto-thresholding or high reward delivery is that a constant reward is delivered regardless of EEG output, which violates the 'successive approximation' approach required in FNF to train EEG frequencies to normative or desirable power limits.

There is also a significant issue associated with standardized protocols. Coben and Padolsky (2007) showed that using QEEG-derived protocols increased the magnitude of the effect with half the number of sessions when compared with Jarusewicz's original study. That is, if standardized protocols are utilized, then the effect will be smaller than if QEEG-derived protocols are adopted. There is the additional dilemma that autism is associated with a spectrum of behaviors, which may explain the wide variety of protocols across studies, and the advantages of individualized protocols based on QEEG data. It is disadvantageous to standardize protocols and expect an effect size that is truly reflective of the efficacy of FNF. In the ADHD field there is a relatively standardized approach to target fronto-central slowing (Fz-A1, Cz-A1) or hyperkinetic behavior (SMR training), and even in that field there is a movement towards 'precision medicine' (Arns et al., 2014).

Researchers have also focused on equipment and their characteristics. In my opinion, the factors that matter in FNF are not the equipment and their idiosyncrasies. What matters is the montage, how much and in what way reward is delivered, duration of training, number of training sessions to achieve a treatment response, training density (e.g. frequency of sessions per week), the immediacy of the feedback, type of feedback (i.e. audio, tactile, visual), and the bandwidths rewarded and inhibited. These factors are considered in a recent paper by Bazanova et al. (2025). This is a matter of process, which is the essence of FNF. The technician regulates settings in a reflexive manner to guide the brain to function efficiently and optimally during training. In operant conditioning terms, 'successively approximate' or 'shape' to efficient electrical activity in specific frequencies. This requires the technician to have a minimum level of experience and competency. There are competency practice standards for FNF training (Biofeedback Certification International Alliance, BCIA, 'Blueprint of Knowledge Statements', 2018), however it is unlikely these standards were met in the extant research. A more detailed analysis and discussion of FNF research on healthy subjects is provided elsewhere (Rogala et al., 2016).

Finally, the most common protocol was to reward alpha frequencies (i.e. 8-13 Hz) with an aim of targeting the mirror neuron system (MNS) and training mu suppression to impact brain functioning when observing social interactions or movement. This connection was elegantly demonstrated by Pineda and his colleagues (see Courellis et al., 2019), in which they down trained mu rhythm during emotion-focused FNF sessions, with increased mu suppression in key networks with the brain approaching a typically developed child. I will discuss the importance of Pineda et al.'s (2008) research in the Theory section. The point here is that researchers could expand on our understanding of causal mechanisms by training frequencies other than alpha and testing hypotheses other than Theory of Mind (ToM).

Outcome and efficacy level

All studies show a positive effect when FNF is compared with controls, under random or matched

assignment, for pre- and post-testing. A first impression suggests that even with some publication bias this represents a positive conclusion of the evidence base. Moreover, publication bias against null-hypothesis findings may suggest more about a tendency to support accepted psychological theories (and reject findings that weaken our belief in these) than a mere aversion to null findings (Ferguson & Heene, 2012). FNF straddles psychological and neuroscientific theories (in other words, biological psychology), and the research has pursued simple and pragmatic outcomes such as reducing social deficits and behaviors of concern, instead of testing psychological theories like a ToM.

If we assume that publication bias is a minor problem in this field, then we can consider the efficacy level of FNF on autism. Coben and Padolsky (2007) write: "Our study may be the first step in establishing a Level 3 criteria rating of neurofeedback as probably efficacious in the treatment of ASD. We replicated another controlled study (Jarusiewicz, 2002). A broader range of outcome measures confirmed the reduction of ASD symptomatology following neurofeedback" (p.18). There have been further studies published since Coben and Padolsky (2007), which means the overall efficacy level of FNF is re-considered here.

To begin with, FNF autism research cannot be assessed using the stricter guidelines outlined in Arns et al. (2020) because there are no systematic reviews completed in the last two years. The last systematic review was completed by Begemann et al. (2016). Therefore, we can only assess this body of research using the lower standards outlined by La Vaque et al., which outlines an efficacy framework for psychophysiological interventions with five levels of efficacy. The first level is anecdotal or case studies through to the fifth level of "Efficacious and Specific. The investigational treatment has been shown to be statistically superior to credible sham therapy, pill, or alternative bona fide treatment in at least two independent research settings" (p.280).

All studies in Table 1 show positive findings with some inconsistent results. There are two studies that meet the gold standard methodology, including Pineda et al. (2008) and Wang et al. (2024). Both studies employed a double blinded randomized sham-controlled methodology. Both studies targeted mu rhythm using a suppression approach, with results consistent across the studies i.e. reduced scores on the ATEC Sensory/Cognitive Awareness subscale. The Wang et al. showed a reduction in the full scale ATEC score that approached significance (p=.082). The Wang et al. (2024) study failed to employ the video conditions (e.g. Hand, Crayon, Social), utilized by Pineda et al. (2008), who showed that mu suppression was higher in the treatment group when watching these videos following FNF compared with controls. Level 5 'efficacious and specific' cannot be claimed, rather Level 4, 'efficacious' only, because this component was not replicated by Wang et al. (2024).

Theory and causal mechanisms

A weak aspect of the research has been the practice of describing autism according to the DSM-5 nomenclature and then demonstrating a reduction in autistic traits without any connection to a theory that may explain the disorder, or demonstrating neuro-mechanisms that underlie change in EEG and other factors e.g. behaviors of concern. The concept of understanding the mechanisms of FNF has been recommended in the ADHD research (Arns., et al, 2014). The clear leader in training specific protocols that link theory and neurophysiological mechanism is Pineda and his colleagues. They have shown that training mu suppression (8-13 Hz) during emotion-focused FNF training at C4, causes connectivity changes in the brain in key networks like the DMN and ToMN. This elegantly links to the psychological theory of ToM developed by Baron-Cohen et al. (1985). There have also been papers targeting executive dysfunction (first proposed by Minshew & Goldstein, 1998; Pennington & Ozonoff,

1996). Kouijzer et al. (2009a) showed that inhibiting theta and rewarding beta targeted under-connectivity in the anterior cingulate gyrus (ACC), recognised as a brain region that regulates cognitive and emotional processes associated with cognitive control and executive function (Bush et al., 2000). Kouijzer et al. (2009a) also write of the relationship between the activation of the default mode network (DMN)/ACC during task demand, which improves performance, and how ASD is associated with a hypoactivation of the DMN/ACC under attentional demand.

There has been no research on the third key psychological theory of autism called a 'weak Central Coherence' (Frith & Happe, 1994). Part of the problem may lie in quantifying 'Central Coherence' and in positing which brain networks are associated with deficits in this capacity.

Conclusion & recommendations

The current paper reviewed ten studies of FNF on autism that were randomized and/or matched controlled trials, and report consistent positive findings on core autistic traits and other factors. The areas of improvement using FNF include diagnostic autistic traits (e.g. ADOS), biomarkers (i.e. EEG, mu suppression), social functioning, cognitive awareness, emotional regulation, executive function and cognitive flexibility, attention, communication, and behaviors of concern. Despite small sample sizes, studies show that FNF reaches Level 4 on La Vaque et al.'s (2002) categorisation, that is, 'efficacious'. This efficacy standard applies to males, up to 18 years, with a low-average or greater intellectual functioning and with a single diagnosis of autism. Improvements are maintained long-term with approximately 50% of subjects responding to FNF using standardized protocols. However, the maintenance of improvement post training has not been replicated, and follow-up data should be collected in all studies moving forwards (*Recommendation 1*). Further studies are required to generalize these positive findings to females, adults, and intellectually and verbally impaired autistic cases (*Recommendation 2*). Researchers could also explore alternative outcome measures such as quality of life, education, employment, ADLs and mood i.e. anxiety, depression. The use of EEG connectivity (as described by Courellis et al., 2019) could also be used pervasively to standardize outcome measurement.

To reach a stronger conclusion of Level 5 'efficacious and specific', the Pineda et al. (2008) study needs to be replicated with a specific focus on mu rhythm changes under the observation of movement and social conditions, with post-training follow-up outcome data (*Recommendation 3*). To achieve the stricter standards outlined by Arns et al. (2020), two independent systematic reviews are required within a two-year period, calculating and analysing variables such as effect size, remission rates, safety and side effects, and cost-benefit analyses (*Recommendation 4*).

With regards to protocols, there is evidence that QEEG-derived protocols produce stronger improvements than standardized protocols. Researchers need to use a manualised approach to FNF, which would include the following protocols shown to be efficacious:- C4-A1, C3-?, CZ-mastoid, FCZ -mastoid, FPz-A1, and the two-channel protocol used by Carrick et al., namely, FP1-O1, FP1-O2, FP2-O1, FP2-O2 (*Recommendation 5*). Researchers have mainly rewarded alpha wave (8-13 Hz), whilst inhibiting slow wave (2-7 Hz) and fast wave (15-30 Hz). Researchers could also investigate if positive treatment outcomes are achieved by rewarding frequencies other than alpha (*Recommendation 6)*. Hey (2020) reported a series of case studies with neurotypical adults diagnosed with mental disorders who were rewarded for delta frequencies that reduced core problem symptoms. However, this approach is highly exploratory and should be conducted with a degree of caution.

The research field of FNF has been relatively broad in investigation in the last two decades. The concept of an 'EEG regulator', first published by Kouijzer et al. (2013) is a promising area of enquiry, that has specific implications for a clinical practice. However, it can also be dubious because researchers can simply demarcate responders from non-responders to increase the probability of finding an effect between treatment and controls. The notion of a regulator is only legitimate in research and clinical practice if there is a variable that differentiates a responder from a non-responder, apart from just improving on a dependent variable. It is legitimate and meaningful if an autistic participant can be differentiated at the start of treatment and precluded if unlikely to respond based on a pre-defined independent variable. The next stage of this research is to identify factors that can discriminate regulators from non-regulators, such as demographics (e.g. gender), biomarkers (e.g. ERP), psychophysiology, and/or cognitive and behavioral factors (*Recommendation 7*). Bazanova et al. (2025) outlined research on potentially differentiating factors such as personality traits, cognitive functioning and locus of control.

Moreover, a mediating factor which may warrant future research is the role of oxidative stress (*Recommendation 8*), which leads to a higher degree of inflammation and excitotoxicity in autistic children (Liu et al., 2022). Improved neural regulation using FNF may stabilise the autonomic nervous system and reduce oxidative stress, causing a reduction in core autistic behaviours and traits. It could elucidate an underlying mechanism of FNF.

Research into differentiating and mediating factors will shift focus away from the fundamental question of whether FNF is efficacious with autism, towards a more profound understanding of the mechanisms and factors that make FNF work with specific autistic populations.

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Declaration of conflicting interests

The author declares that he has several EEGer licences and operates a clinic that profits from using FNF on autistic cases. He currently presides as the applications secretary on the BCIA-A.

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