Review of Studies on Amplitude Neurofeedback with Autism: Critical Overview, Efficacy Level and Research Direction

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Abstract

The efficacy of amplitude neurofeedback (aNF) with autism has been labeled 'probably efficacious' in reviews in the last decade, despite new research pointing towards a higher standard. The aim of this review was to critically analyse key features of these studies, with an aim of determining aNF efficacy with autism and establishing research direction in this field. Electronic databases and literature reviews were used to collect a total of 13 controlled trials or comparative studies. An analysis showed that aNF reaches a Level 4 efficacy standard with emerging Level 5 efficacy, with an impact on a broad range of factors including core autistic traits, social communication, emotion regulation, executive function, behaviours of concern, attention, and EEG. A replication of one of two key studies by an independent research team is required to claim Level 5 efficacy. Current efficacy standards generalise to male children, up to 18 years, with a low-average or greater intellectual functioning, with no co-morbid conditions. Improvements are maintained or increase after 12 months with approximately 50% of subjects responding to aNF when using standardised protocols. Sample sizes were typically small, although a meta-analysis suggests a large superior effect when compared to a wait list. Five recommendations are made.

Keywords: Neurofeedback, neurofeedback training, autism, ASD, literature review, efficacy.

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1. Introduction

The evidence base for 'amplitude neurofeedback' (aNF) with autism has been accumulating gradually over the last 18 years since the first research trial by Jarusiewicz (2002). Coben et al. (2010) conducted a short literature review of four studies and concluded that aNF 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. The 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." At the time of writing this article, two literature reviews were published (Kumari & Sharma, 2020; van Hoogdalem et al., 2020) on the efficacy of aNF amongst children with autism. Van Hoogdalem et al. was a relatively short review and included studies that were not aNF, such as Liu et al. (2017), which used a blood flow biofeedback intervention (i.e. HEG). Kumari & Sharma concluded that current research does not provide sufficient conclusive results about the efficacy of aNF with autism and social cognitive deficits.

Based on these somewhat conservative (and sometimes conflicting) conclusions about aNF, this article sought to analyse research studies using a rigorous approach with an aim of establishing level of clinical efficacy, identifying strengths and limitations of the research, and providing research direction. I will provide a broader analytical interpretation in contrast to Kumar and Sharma, and van Hoogdalem et al.

2. Method

2.1. Search Strategy

The current author searched for scientific articles in Google Scholar using the key words: neurofeedback, neurofeedback training, neurotherapy, NF, NFT, NFB, autism, ASD, autism spectrum disorder. I used a combination of these key words to locate studies and then found additional references in key literature reviews e.g., Coben et al. (2010), Marzbani et al. (2016), Holtmann et al. (2011), van Hoogdalem et al. (2020). Further studies were searched in specific databases such as PubMed, Ovid MEDLINE, EMBASE, ERIC, and CINAHL.

2.2. Inclusion/exclusion criteria

I included studies in this review that were: a) peer-reviewed; b) written in English; c) had a reasonable sample size; and d) were a controlled trial (randomised, nonrandomised, matched) or a comparative study of interventions. 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 they discussed findings; 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 written in English.

2.3. Summary of studies

Tables 1 and 2 show 13 studies reported across 13 articles with one article reporting two studies (Pineda et al., 2008) and one research group completing a follow-up study in a separate article (Kouijzer et al., 2009a, 2009b). Nine studies were randomised or matched controlled trials. Four studies used a comparative or controlled methodology without randomisation. The first published trial was in 2002 by Jarusiewicz. Therefore, two studies every three years are being published in this field. Why more studies are not being published on aNF is an interesting question. Is aNF still an obscure therapy that does not attract research attention? Maybe the barrier is the cost of equipment and training; aNF is a relatively complex therapy and to conduct research requires a senior therapist to govern the protocols using equipment and licences that cost from $5,000USD. It also involves a considerable commitment from the subject e.g. 10 hours or more of training time. An equally plausible explanation is that, due to low powered studies (i.e. small sample sizes), there are many studies being filed by researchers or rejected by journals, as they are supporting the null hypothesis. I discuss this in greater detail in the next section.

3. Results

3.1. Sample characteristics

An obvious characteristic of this research body is small sample sizes, which leads to low-powered studies. Button et al. (2013) describe that low powered studies have three impacts, 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. The first two impacts reduce the chances of reaching a true non-null finding, and the last 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 of aNF (e.g. Jarusiewicz, 2002) is an exaggerated positive result, and any attempts to replicate these findings are difficult, presumably as they are not outliers. It is important to note that Jarusiewicz study has been replicated several times, and Coben and Padolsky (2007) demonstrated a stronger effect by using qEEG-derived protocols. This does not support the idea of a 'winner's curse' in the aNF research, although it precludes the opportunity to make strong conclusions about the magnitude of the effect when sample sizes are consistently small.

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 yet in EEG or ERP studies, and this recommendation should be considered in research focused on autism using aNF.

There is also a possibility of publication bias, since only a handful of studies have been published since 2002. The publication rate is low and could be the result of many false negatives not being published as they are unremarkable to a scientific

journal and its community. Begemann et al. (2016) conducted a meta-analysis of five studies relating to aNF for 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. found a large superior effect of neurofeedback over placebo treatment (ES 0.96, p=.039)" (p. 25).

Based on the Begemann et al. 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 consider dropout rates across groups, and aim for a final, eligible sample size of 26 and not an initial sample size of 26 per group. This suggests that the initial sample per group might be 35 to 50 subjects, depending on estimated dropout rates. A participant of an aNF study is required to engage in (at least) weekly sessions, over 10-20 weeks, which is a large commitment and a moderately high dropout should be expected. The dropout group should also be compared with completing subjects to assess for different characteristics of these groups.

An interesting pattern in the research is that autistic groups that are reported in Tables 1 and 2 are usually young males (5-18 years) who are verbal with an IQ score >70. Autism is more often diagnosed in males, which would explain the higher ratio of males studied versus females. The incidence rates of autism in males and females are often attributed to genetics, however there is some argument for under diagnosis with females (who camouflage deficits) and diagnostic bias towards males. Either way, future research could focus on factors such as gender, IQ level (including intellectual deficit, ID, versus non-ID) and verbal abilities (i.e. non-verbal, minimally verbal, verbal). To summarise, the findings suggest that we can generalise the evidence to young males under 18 years, who are verbal with an IQ above 70. This would capture a large proportion of the population with autism, but would not generalise to most in the autistic community.

3.2. Design

A major consideration when evaluating the efficacy of aNF is the type of methodological designs used in the research outlined in Tables 1 and 2. The factors to consider include selection process and allocation (i.e. self-selected, randomisation), blinding (i.e. participant only, double blinded) and type of control (i.e. waitlist, active, matched). What is important is to control for nonspecific effects such as contextual (e.g. subject is engaging in an experiment and expects to improve; positive regard for therapist; EEG displays are perceived by the subject as scientific and therefore efficacious), extraneous (e.g. subject watches a screen for 30 minutes, 30-50 times and improves due to focussing and paying attention for long periods of time), repeated reinforcement (e.g. researcher/therapist give verbal reinforcement, audio-visual feedback unrelated to EEG data), and, specifically with children with autism, the routine of attending regular sessions, which can prove stabilising by satisfying the 'need for sameness'. The gold standard in biofeedback is a double-blinded, randomised, and sham or active control (van Doren et al., 2019). The active control group receives a cognitive training or EMG biofeedback to control for nonspecific effects e.g. placebo.

The first feature to note is that four studies used randomised controlled designs with only Pineda et al. (2008) using a randomised, double-blind [parent and technicians were blinded] procedure in Study 2, and showed a mu suppression in the experimental group, as well as decreased reaction and response times, and reductions on the ATEC subscale 'Social Cognitive Awareness'. The control group did not show these changes. Carrick et al. (2018) utilised a randomised, sham control, subject blinded methodology, reporting improvements in the active group on executive functioning scales, reduction in problem behaviours, improvements in communication and a normalisation of qEEG data. It is worth noting that the ATEC produced mixed results with improvements for the active group on the 'Sociability' subscale, with a worsening on 'Speech and Sensory/Cognitive Awareness'. One explanation of the worsening on the 'Sensory/Cognitive Awareness' subscale is related to the sites trained, namely, prefrontal and occipital lobes, which could have caused an over activation of those sites related to thinking and visual sensory input. The importance of double blinding in this study is less important than laboratory-based studies, since all the training was completed at home and contact with the researchers was minimised, thereby reducing the impact of researcher-based factors such as higher verbal reinforcement towards the active group. It is also worth mentioning that Carrick et al. used the home-based Mente device that delivers feedback via an audio component only, which implies that density-based training (daily sessions) can improve functioning with audio only feedback.

Goodman et al. (2018) introduced an active control group (heart rate variability, HRV, training), which also involved feedback albeit heart rate variation. This is a useful study that compares aNF with HRV, however a design shortcoming is that they combined HRV with aNF without employing an aNF-only group. The assumption is that the effects of aNF are additive. There were mixed results to interpret with this study: the HRV group improved in emotion regulation and social behaviour, whilst the HRV+aNF group showed decreases in mu suppression, emotional lability and autistic traits. This group also improved in heart rate variability by the end of training, whilst the HRV group did not. The reduction in mu suppression contradicts research showing an increase in this biomarker. Moreover, the combination of HRV and aNF had a multiplicative effect or aNF training improved brain (piriform cortex) to heart (baroreceptor reflex) synchronisation, which improved heart rate variability in the aNF-HRV group. Alternatively, the low total sample of 15 subjects may explain these mixed and unintuitive results.

3.3. Measurement

Researchers have mostly used the Autism Treatment Evaluation Checklist (ATEC), which is available free of charge and has been validated as an outcome measure for therapies with autistic populations. The ATEC has added advantages 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 associated with maturation only.

Nearly all studies have used a pre- and post-therapy methodology, spanning over a few months to six months, and only one study has engaged in long-term follow-up (Kouijzer et al., 2009a, 2009b). Kouijzer (2009b) reported that any immediate

behavioural and cognitive improvements from aNF were maintained (and some improved significantly more) at a 12-month follow-up. The authors did not use the ATEC so it is unclear if this follow-up improvement was due to aNF or maturation. Ryan et al. (2019) cite van Doren et al.'s meta-analysis which reported that ADHD symptoms reduce even further from post-neurofeedback treatment to a 12-month follow-up, due to sleep improving over time (a mediating factor). Intuitively, one could predict that the positive effects of therapy would diminish over an extended period after therapy, however Ryan et al. (2019) and Kouijzer's research suggest that the opposite can occur.

A further strength of this research has been the use of specific, standardised 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 standardised diagnostics and measures, there are some areas that are yet to be measured, that are important for people with a 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 neurodiverse populations (e.g. ASQoL, ASC-ASD).

Another strength of this field is the use of multiple sources of data to correlate the effects of aNF 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 increase social awareness of non-verbal cues, and measure brain functioning (e.g. fMRI) or a biomarker (e.g. event-related potential, ERP) that reflect those changes.

A potential weakness of these studies is the use of parent-only based ratings which can be defined as 'most proximal' and 'least blinded' ratings. Cortese et al. (2016) showed that the experimental effects of neurofeedback for ADHD diminished to non-significance when teacher ratings were analysed across multiple studies, since teacher ratings are presumably 'less proximal' and 'probably blinded'. This phenomenon was also replicated with sham controls where there were blinded raters. Van Doren et al. (2019) argue against the 'proximal-blinded' concept suggesting that parents rate different cognitive and behavioural aspects of ADHD than teachers, or they are rating the ADHD behaviours in a different context e.g. home. 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 aNF and skin conductance groups (both groups were identically prepared and did not know the feedback they were receiving). An effect was demonstrated with blinded parents with an added sophistication of differentiating subjects who can EEG regulate compared to those who cannot. Seven out of 13 subjects in the aNF group were identified as regulators, suggesting that approximately 50% of cases will respond to aNF when using standardised protocols. Outcomes were maintained at a six-month follow up.

Either way, future studies should explore proximal raters (e.g. teachers versus parents) and assess whether they produce different results, or studies should blind raters to reduce the self-fulfilling prophecy effect.

Lastly, Ryan and Files (2019) adopted an easy to apply measure of change that is tailored for a therapy setting that could be used in research called the Goal Attainment Scale (GAS) method. This could be used alone or with other measures to assess level of change, targeting a specific behaviour of concern, functional behaviour or emotional disturbance. The GAS method ranks change from mild to large improvements and specifies what change would look like for each individual person. It moves away from standardising, however it has the benefit of being specific to each individual (i.e. person centred) and therefore has strong face validity.

Table 1: Randomised (or matched) controlled trials

Author(s)	Design & Measures	Sample	Protocol	Results
Jarusiewicz (2002)	Matched controls, pre-post ATEC, parent rated	N=24 12 children with 12 matched controls (age, gender, severity) 4-13 years age	C4-A1 Rewarding: 10-13 Hz (or lower) Inhibiting: 2-7, 22-30 Hz 30 mins training, 20-69 sessions	Experimental group improved by 26% on ATEC. Sociability improved 33%, speech/language/communication 29%, health 26% and sensory/cognitive awareness 17%. Control group improved by 3%. 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).
Coben & Padolsky (2007)	Matched controls, pre-post test 20+ sessions twice a week ATEC, GADS, GARS, BRIEF PIC-2, qEEG Parent rated	37 children with ASD Experimental: n =25 84% males Matched: age, gender, race, handedness, ASD severity	qEEG assessment-based protocols (bipolar and unipolar) Reward band range: 5-16 Hz Inhibits bands: 1-6 Hz (92%), 7-14 Hz (68%), 18-30 Hz (100%)	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 behaviours, 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.

Table 1 (Continued): Randomised (or matched) controlled trials

Author(s)	Design & Measures	Sample	Protocol	Results
Pineda et al. (2008) Study 1 Study 2	Study 1: Sham controls, randomised, pre-post qEEG, MSI, TOVA, ATEC, Apraxia Imitation Scale Participants & parents blinded Study 2: Same as Study 1 Participants, parents and technicians blinded	Study 1: 7 Males, 7-17 years Mean age=9.3 Experimental=4, Control=3 IQ>80 Study 2: 19 ASD participants, 7-17 years Mean age=9.8 Placebo control, n=10 (all males) Experimental n=9 (6 males) IQ>80	Study 1: C4-? + EMG (trapezius) Rewarding: 8–13 Hz Inhibiting EMG: 30-60 Hz Placebo received EMG and artificial mu-like signal 8–13 Hz feedback 15 hours (30 sessions over 10 wks) Study 2: Same as Study 1 except placebo received 10–13 Hz signal	Study 1: Amplitude (uV) significantly decreased across all bandwidths (delta, theta, alpha, etc) for experimental group, and increased for placebo group at C4. TOVA: reaction time/response time decreased by 70% in experimental group, no change in placebo group. Error rates decreased in experimental group only. ATEC: increase in Social Cognitive Awareness subscale in experimental group, with a decrease in control group. Mu: experimental group showed reduction in mu power compared with control group due to training. 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/response time z-scores decreased in experimental group; the control group increased. ATEC: experimental group showed score reductions on Social Cognitive Awareness subscale, whilst control group showed no changes.
Kouijzer et al. (2009a) with follow-up study (2009b)	Matched controls, pre-post CPT, TOSSA, TOL, neurocognitive battery, parent reports (15 items)	N=14 children with ASD (12 males) 8-12 years Mean age: 10.1 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 Rewarding: 10-13 Hz Inhibiting: 4-7 Hz	TOSSA auditory: aNF **group:** 30% increase in correct responses; Control group: no change. Interference effects (written names): aNF group: 55% reduction; Control group: 24%. Set shifting, concept generation and goal setting showed similar trends. Parents reported improved communication, social interaction and typical behaviour in aNF group, but not controls. All parents would recommend aNF to others. Behavioural improvements were maintained after 12 months.
Kouijzer et al. (2013)	Waitlist, pre-post 6-month follow up, randomised, blinded subjects and parents ADI, Tower of London, Stroop test, TOSSA, Digit Span, WISC, Trail Making	aNF group: n=13 (10 males) Mean age: 15.3; Skin conductance (SC) group: n=12 (9 males). Mean age: 14.5 Wait list: n=13 (11 males). Mean age: 15.9 Inclusion Criteria: IQ>79	CZ-mastoid or FCZ-mastoid Rewarded 50-80% of time and increased reward time if participant was not motivated (*design flaw*) 29-40 sessions (twice a week)	EEG-regulators improved in cognitive flexibility on Trail Making Test, but SC-regulators did not. Maintenance of the effect at 6-month follow-up. No differences on social communication, inhibition, planning, attention, working memory or executive function. Treatment expectancy did not influence outcomes since aNF and SC groups were prepared for training identically and participants were unaware of what they were receiving feedback for. Parents were also blinded. Nonspecific effects accounted for in design and analysis.
Goodman et al. (2018)	Active controls, randomised, pre-post HRV (Group 1, N=7), HRV + aNF (Group 2, N=8) SRS-2, Mu Suppression Index (MSI), ATEC, ERC, Spence Anxiety (SPS) Parent rated	15 ASD children (13 males) 9-18 years M=12.4 yrs Extremely low to high IQ IQ equal across groups Randomised on age, gender, IQ	C4-? Rewarding: 8-13 Hz 4 preliminary sessions of HRV; 12 hours of training for HRV and HRV+aNF, DVD training 70%-80% reward frequency (*flaw*) 10-20 mins / day breathing at home	Group 1: increase in ER subscale equal to improvements in emotion regulation. Significant increase in SRS, indicating improvements in social behaviour. No change in Lability/Negativity ERC subscale, Spence or ATEC. Group 2: significant decrease in L/N subscale, suggesting improvements 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 over training, Group 1 did not.
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, M=12.5 7 typically developed (5 males) 8-17 years, M=10.6	C4-? Rewarding: 8-13 Hz Inhibiting: 4-8 Hz, 13-30 Hz 20-30 hours of training DVD training	ASD group had higher activation in the right IPL (BA 40) after aNF. Controls had widespread lower activation after aNF, 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 between the ASD and controls before aNF were absent at post-assessment. Increased activation correlated with decreased SRS scores (lower symptom severity). ATEC: decreased symptom severity with increased post-activation state. ADOS: increased task activation changes were correlated with lower initial ASD severity. No correlation on ADI.

Table 1 (Continued): Randomised (or matched) controlled trials

Author(s)	Design & Measures	Sample	Protocol	Results
Carrick et al. (2018)	Sham control, randomised, subject blinded qEEG, posturography, Parent rated: ATEC, SRS-2, BRIEF, ABC, QABF	N = 34 Active: n=17 Control: n=17 28=Male, 6=Female 4-17 years Diagnosed ASD, exclusion criteria: comorbidities	aNF using Mente device (binaural auditory feedback, sound volume) FP1-O1, FP1-O2, FP2-O1, FP2-O2 Delta (1-3 Hz), theta (4-7 Hz), alpha (8-13 Hz), beta (14-19 Hz), high beta (20-35 Hz) Rewarding alpha, inhibiting all other bandwidths 45 minutes daily over 12 weeks	ATEC: Behaviour worsened for Active group on the Speech and the Sensory/Cognitive Awareness scale, and improved on Sociability. Health/Physical Behaviour 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, in particular in the Shift, Initiate, Organizational of Materials, Behavioural Regulation Index, Metacognition Index, and in the Global Executive Composite. ABC: Active group showed significant reduction at post-test in autistic behaviours. QABF: Active group showed significant reduction in problem behaviours 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 improved normalisation in Active group only across all bandwidths.
Sokhadze et al. (2014)	Nonactive controls, pre-post Experimental group: rTMS+aNF Control group: Waitlist ERP, EEG, RBS-R, ABC	High functioning autism Aged 10-21 years (34 males) Inclusion criteria: IQ>80. Experimental=20, Mean age = 14.7 Waitlist=22, Mean age = 14.2	rTMS=10 mins at DLPFC, left, right and bilateral application (1 Hz, 180 pulse), followed by aNF (FPz-A1), 18 sessions Rewarding: 30-45 Hz (gamma) Inhibiting: 18-30 Hz. DVD mode	Linear increase in relative gamma power over 18 sessions; theta/low beta ratio showed significant linear decrease over in experimental group. RBS-R: Significant decrease in repetitive/restricted, compulsive, and ritualistic/sameness behaviours at post-test for experimental group. Focused attention index increased over 18 sessions and correlated negatively with theta-low beta ratios. Commission error and total errors decreased in experimental group only. Post-error reaction time increased in experimental condition but not for waitlist. ERP: lower amplitude to nontargets for N100, N200, P300, higher amplitude to nontargets for P2d ABC: Experimental group showed significant reductions in the Lethargy/Social Withdrawal subscale. Hyperactivity subscale showed greater reductions.
Pineda et al. (2014)	Pre-post design Control group: typically developed Parental surveys, SRS, ATEC, Vineland-II, EEG, MSI	13 Children with ASD (10 males) 7-17 years (M=11.4) 11 TD (7 males) 8-17 YEARS (M=10.2)	C4-A2 Rewarding: 8-12 Hz Inhibiting: 4-8 Hz, 13-30 Hz ASD and TD groups received 30 hours of aNF (1-2 sessions/wk). Sessions 45-60 mins.	EEG: Improvements, particularly in 8–12 Hz band, in the ASD group was greater than TD group post-training. SRS: Improvements in ASD group post-training ATEC: decreased scores at post-test in ASD group; TD group increased in absolute value. Vineland: no change. The occipital cluster showed a main effect of video, indicating that suppression effects occurred only in the social play condition. ASD group showed enhanced mu suppression and a decrease mu suppression in fronto-central sites. TD participants increased mu power from pre- to post-training, whereas ASDs decreased mu power.
Friedrich et al. (2015)	Pseudo random assignment to Group 1 or Group 2. MSI, RMET, emotion imitation task VABS, SRS, ATEC (parent rated)	13 ASD 6-17 years old (M=11 years) Group 1 mu suppression only Group 2 mu suppression or enhancement	C3-? and C4-? 16 sessions, 60 minutes, 2-3 times a week. Group 1: positive feedback for enhancing mu power in the social interaction and nonsocial gaming. Group 2 feedback for decreasing mu power during the social interactions and for enhancing mu power during the nonsocial gaming. Both groups: Inhibiting theta and beta	Biomotion condition: more mu suppression in the post-test at C3 for both groups. Social condition: Group 2 showed more mu suppression than Group 1 at C4. RMET: Lower MSI was correlated with higher % correct responses and shorter reaction times. Subjects had significantly more correct responses in the post- than in the pre-test in the emotion task suggesting improvement in emotion recognition. Group 2 showed shorter reaction time in the post-test on the emotion task. EIT: Group 2 had more mu suppression in the post-test and more than Group 1. Group 1 exhibited greater mu suppression in the pre- than in the post-test and more than Group 2 in the pre-test. In the post-test, in the positive condition, activation was higher at the zygomaticus major between 700 and 1000 ms than at the corrugator supercilii indicating smiling. In the negative condition, activation was higher at the corrugator supercilii than at the zygomaticus major between 400 and 600 ms, indicating frowning. Parent ratings: Improvements in coping with daily life, social skills / relationships and communication. VABS: Parents rated more adaptive in the daily living, socialisation and communication domains at post-test. SRS: Parents rated more socially responsive in the post-test than in the pre-test. Social motivation subscale improved significantly at post-test. ATEC: Parents reported significant reductions in ASD symptoms at post-test. Sociability and Behaviour subscales improved significantly.

Table 1 (Continued): Randomised (or matched) controlled trials

Author(s)	Design & Measures	Sample	Protocol	Results
Ryan & Files (2019)	Ex-post facto design Group 1: aNF only Group 2: aNF + traditional therapies Group 3: Traditional therapies only Goal Attainment Scale (GAS)	132 clients (75% male) 5-64 years (M=13) ASD = 55% ASD plus comorbidities = 26% Non-ASD = 19%	EEG mini-map based protocols. Three protocol groups: Group 1: T4-F4 Group 2: SMS (Sensory motor strip) Group 3: Other 30 mins training once per week.	Mean improvement across 132 clients on GAS was 1.8 and the median improvement was 2. GAS scores ranged from 0 to 3. No clients scored -1 (regression in symptoms). The aNF group scored higher on the GAS compared with traditional therapies and with aNF combined with traditional therapies; differences were not significant. There were no significant differences with gender, diagnosis, aNF protocol and main problem. There was a significant positive correlation between total number of sessions and outcome scores (weak correlation).

3.4. Protocols

A criticism of this body of evidence is the lack of detail provided by authors in relation to montage and training protocol. For example, Table 1 shows that in four of the nine studies, the signal site is reported but not the reference site. The reference site is compared with the signal site, and, in theory, could play a role in the outcome of training. The fact that there is no data at present means that research could be invested in this area:- that is, does the reference site play a role in outcome?

A criticism in the field is the lack of standardisation for training. The problem with this criticism is that preliminary evidence supports EEG-based protocols, that is, the therapist determines the protocol based on individual EEG data e.g. qEEG (see Coben & Padolsky, 2007). An opinion paper is required on this topic to guide researchers with regards to standardisation. If the opinion paper suggests standardisation of protocols for all subjects in a study, then the researcher must accept that the effect size will be lower and sample size estimates will increase. There is the added complexity that autism is associated with a spectrum of behavioural concerns, which may explain the wide variety of protocols across studies, and the advantages of individualised protocols based on EEG data. This makes it difficult to standardise and expect an effect size that is truly reflective of the efficacy of aNF. In the ADHD field there is a relatively standardised approach to train for frontal-central slowing (Fz-A1, Cz-A1) or hyperkinetic behaviour (SMR training) and even in that field there is a movement towards 'precision medicine' (Arns et al., 2014).

Ryan and Files (2019) supply descriptive statistics of problems and problem behaviours in a clinic setting. Of a sample size of 132 cases, 42% targeted meltdowns/emotion regulation, 17% anger / aggression and 22% anxiety. If these categories are typical of the prevalence of problems with autistic children, a study could be designed around these three categories and exploring protocols that work versus those that do not. Ryan and Files recommend the use of T4-F4 to tackle meltdowns, anger, and aggression. This protocol matches with known temporal and frontal lobe dysfunction in autism.

Researchers have also been fixated on equipment and their characteristics. In my opinion, the factors that matter in aNF are not the equipment and their idiosyncrasies. What probably matters is the protocol, how much reward is provided, how immediate the feedback is, type of feedback, and the bandwidths rewarded and inhibited. This is a topic of process, which is the essence of aNF. The technician will regulate 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. This requires the technician to have a minimum level of experience and competency. There are competency-based standards when it comes to aNF training, however it is unclear if these standards have been met in the research.

Finally, the most common protocol is to reward alpha frequencies (i.e. 8-13 Hz) with an aim of targeting the mirror neuron system (MNS) and reducing disruptions that are often seen in the autistic brain. 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 neurofeedback sessions, with increased mu suppression in key networks in the brain approaching a typically-developed child i.e. default mode network (DMN), theory of mind network (ToMN). I will discuss the importance of Pineda et al.'s 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 wave, testing hypotheses other than ones derived from a Theory of Mind (ToM).

3.5. Outcome and efficacy level

All studies show a positive effect when aNF is compared with controls, under random or non-random assignment, with pre- and post-testing. A first impression suggests that even with 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). aNF straddles psychological and neuroscientific theories (through biological psychology), and the research has pursued simple and pragmatic outcomes such as reducing social deficits and behaviours of concern, instead of proving 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 aNF with 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). Given there have been a number of studies published since Coben and Padolsky's literature review, it begs the question of where the efficacy level is for aNF based on the current evidence.

La Vaque et al. (2002) outline 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). With an aim of establishing the highest possible efficacy of aNF, I will only consider the studies in Table 1, RCTs and matched controlled studies. Based on this body of studies, aNF with autism has an emerging Level 5 efficacy because there have been two studies with a sham/placebo control, although the studies were slightly different (Carrick et al., 2018; Pineda et al., 2008). aNF reaches Level 4 efficacy which is labeled "efficacious":- using a no-treatment control group or sham control group (superior to), or alternative therapy (equivalent to); treating a specific problem; valid and clearly specified outcome measures; appropriate data analysis; and the superiority or equivalence has been shown in two independent research settings. Table 1 reveals independent RCTs or matched control studies that, taken as a whole, meet the criteria of Level 4 efficacy of aNF. The efficacy of aNF generalises to average functioning males with IQs in the low-average range and upwards, with no comorbid conditions.

Further on this point, there have been some conflicting findings in the research. For example, Carrick et al. (2018) found that the 'Sensory/Cognitive Awareness' subscale worsened on the ATEC in the experimental group, whilst in most other studies it improved. However, the reduction in total ATEC and the 'Sociability' subscale has been replicated in independent studies. The discrepancies on the 'Sensory/Cognitive Awareness' subscale in the Carrick et al. study may be due to a small sample size, and/or the treatment differed slightly from other studies (e.g. audio feedback only, home-based training), and/or the sites being trained were quite different to other research i.e. prefrontal/occipital lobes.

3.6. 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 changes in EEG and other factors e.g. behaviours of concern. The concept of understanding the mechanisms of neurofeedback has been recommended in the ADHD research (Arns., et al, 2014). The clear leader in training specific protocols that link theory and neurophysiological mechanisms is Pineda and his colleagues. They have shown that training mu suppression (in the 8-13 Hz bandwidth) during emotion-focused neurofeedback training at C4, causes connectivity changes in the brain in key networks like the DMN and ToMN. This links elegantly 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), which is known for its role in regulating cognitive and emotional processes associated with cognitive control and executive function (Bush et al., 2000). Kouijzer et al. (2009a) also write about the relationship between the activation of the default mode network (DMN)/ACC during task demand, which improves performance, and how ASD is associated with an 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.

4. Conclusion and Recommendations

The current paper broadly reviewed 13 studies of aNF with autism, and there are nine randomised or matched controlled trials that report consistent positive findings on core autistic traits. The areas of improvement using aNF include diagnostic autistic traits (e.g. ADOS), biomarkers (i.e. EEG, mu suppression), social functioning and cognitive awareness, emotional regulation, executive function and cognitive flexibility, attention, communication, and behaviours of concern. There are several non-randomised controlled or comparative studies that also demonstrate the positive effect of aNF. Despite small samples, trials show that aNF reaches a Level 4 efficacy standard with emerging Level 5 efficacy. These efficacy standards apply to male children, up to 18 years, with a low-average or greater intellectual functioning and with no co-morbid conditions. Improvements are maintained long-term with approximately 50% of subjects responding to aNF using standardised protocols. Further studies are required to generalise these positive findings to females, adults, and intellectually and verbally impaired autistic cases (Recommendation 1). Ryan and Files (2019) have reported data from an observational study that suggest these findings generalise, however further studies using randomised procedures are desirable. Researchers could also explore broader 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 standardise outcome measurement.

To reach a Level 5 efficacy, the studies conducted by Pineda et al. (2008) and/or Carrick et al. (2018) need replication using a larger sample size accounting for dropouts (Recommendation 2). These studies have not been replicated by an independent research group and the leading researcher in our field is Pineda and his colleagues. There should also be a focus on using least proximal raters, like teachers or carers, instead of most proximal raters like parents.

With regards to protocols, there is evidence that qEEG-based protocols produce greater improvements than standardised protocols. This is pertinent for a clinical practice. Researchers and practitioners who do not have qEEG at their disposal should use 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 3). Researchers have mainly rewarded alpha wave (8-13 Hz), whilst inhibiting slow wave (2-7 Hz) and fast wave (15-30 Hz). Protocols that have shown promise in other studies (like Ryan & Files, 2019) include T4-F4 and T4-P4. Researchers could also investigate if rewarding frequencies other than alpha wave might produce positive outcomes. Hey (2020) reported a series of case studies with neurotypical adults diagnosed with mental

disorders who were rewarded in the delta frequencies that reduced core symptoms (Recommendation 4). However, this approach if taken would be highly exploratory but could broaden the limited amount of evidence-based protocols for autism.

The research field of aNF has been relatively broad in investigation in the last decade. 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. This is the idea that some autistic brains respond to aNF, others do not. This concept is extremely promising in that EEG regulators could be reliably identified at intake and selected for aNF, whilst non-regulators could save time, money and energy by not engaging in aNF that is unlikely to work. 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 cognitive and behavioural factors (Recommendation 5). Practitioners would be interested in assessment markers that could reliably differentiate those who probably respond to aNF, and those who would not. This important shift in research focus will elevate aNF from a status of 'probably efficacious' to 'highly efficacious', with guidelines on what predictable changes in functioning can be demonstrated, on whom, and in what way.

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References

Arns, M., Heinrich, H., & Strehl, U. (2014). Evaluation of neurofeedback in ADHD: The long and winding road. *Biological Psychology, 5*, 108-115. https://doi.org/10.1016/j.biopsycho.2013.11.013

Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a "theory of mind"? *Cognition, 21*(1), 37-46. https://doi.org/10.1016/0010-0277(85)90022-8

Begemann, M., J.R. Florisse, E., Lutterveld, R., Kooyman, M., & Sommer, I. (2016). Efficacy of EEG neurofeedback in psychiatry: A comprehensive overview and meta-analysis. *Translational Brain Rhythmicity, 1(1),* 19-29. https://doi.org/10.15761/TBR.1000105

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences, 4*(6), 215-222. https://doi.org/10.1016/S1364-6613(00)01483-2

Button, K. S., Ioannidis, J. P. A., Mokrysz, C., Nosek, B. A., Flint, J., Robinson, E. S. J., & Munafò, M. R. (2013). Power failure: why small sample size undermines the reliability of neuroscience. *Nature Reviews Neuroscience, 14*(5), 365-376. https://doi.org/10.1038/nrn3475

Carrick, F. R., Pagnacco, G., Hankir, A., Abdulrahman, M., Zaman, R., Kalambaheti, E. R., & Oggero, E. (2018). The treatment of autism spectrum disorder with auditory neurofeedback: A randomized placebo controlled trial using the Mente autism device. *Frontiers in Neurology, 9*, 537. https://doi.org/10.3389/fneur.2018.00537

Coben, R., Linden, M., & Myers, T. E. (2010). Neurofeedback for autistic spectrum disorder: a review of the literature. *Applied Psychophysiology & Biofeedback, 35*(1), 83-105. https://doi.org/10.1007/s10484-009-9117-y

Coben, R., & Padolsky, I. (2007). Assessment-Guided neurofeedback for autistic spectrum disorder. *Journal of Neurotherapy, 11*(1), 5-23. https://doi.org/10.1300/J184v11n01_02

Courellis, H. S., Courelli, A. S., Friedrich, E. V. C., & Pineda, J. A. (2019). Using a novel approach to assess dynamic cortical connectivity changes following neurofeedback training in children on the autism spectrum. In L. M. Oberman & P. G. Enticott (Ed.), *Neurotechnology & brain stimulation in pediatric psychiatric & neurodevelopmental disorders* (pp. 253-276). Academic Press. https://doi.org/10.1016/B978-0-12-812777-3.00011-8

Cortese, S., Ferrin, M., Brandeis, D., Holtmann, M., Aggensteiner, P., Daley, D., & Zuddas, A. (2016). Neurofeedback for attention-deficit/hyperactivity disorder: Meta-analysis of clinical and neuropsychological outcomes from randomized controlled trials. *Journal of the American Academy of Child & Adolescent Psychiatry, 55*(6), 444-455. https://doi.org/10.1016/j.jaac.2016.03.007

Darling, M. (2007). School-based neurofeedback for autistic spectrum disorder. *Neurofeedback for ASD*, 1-7. https://www.eeginfo.com/research/articles/NeurofeedbackforASD.pdf

Datko, M., Pineda, J. A., & Müller, R. A. (2018). Positive effects of neurofeedback on autism symptoms correlate with brain activation during imitation and observation. *European Journal of Neuroscience, 47*(6), 579-591. https://doi.org/10.1111/ejn.13551

Ferguson, C. J., & Heene, M. (2012). A vast graveyard of undead theories: Publication bias and psychological science's aversion to the null. *Perspectives on Psychological Science, 7*(6), 555-561. https://doi.org/10.1177/1745691612459059

Friedrich, E. V., Sivanathan, A., Lim, T., Suttie, N., Louchart, S., Pillen, S., & Pineda, J. A. (2015). An effective neurofeedback intervention to improve social interactions in children with autism spectrum disorder. *Journal of Autism & Developmental Disorders, 45*(12), 4084-4100. https://doi.org/10.1007/s10803-015-2523-5

Frith, U., & Happé, F. (1994). Autism: beyond "theory of mind". *Cognition, 50*(1), 115-132. https://doi.org/10.1016/0010-0277(94)90024-8

Goodman, M. S., Castro, N., Sloan, M., Sharma, R., Widdowson, M., Herrera, E., & Pineda, J. A. (2018). A neurovisceral approach to autism: Targeting self-regulation and core symptoms using neurofeedback and biofeedback. *NeuroRegulation, 5*(1), 9-9. https://doi.org/10.15540/nr.5.1.9

Hey, C. (2020). Neurofeedback and counseling as integrative treatment. *Asia Pacific Journal of Neurotherapy, 2*(1), 6-11. https://apjnt.org/list-of-papers

Holtmann, M., Steiner, S., Hohmann, S., Poustka, L., Banaschewski, T., & Bölte, S. (2011). Neurofeedback in autism spectrum disorders. *Developmental Medicine & Child Neurology, 53*(11), 986-993. https://doi.org/10.1111/j.1469-8749.2011.04043.x

Jarusiewicz, B. (2002). Efficacy of neurofeedback for children in the autistic spectrum: A pilot study. *Journal of Neurotherapy, 6*(4), 39-49. https://doi.org/10.1300/J184v06n04_05

Kouijzer, M. E., van Schie, H. T., Gerrits, B. J., Buitelaar, J. K., & de Moor, J. M. (2013). Is EEG-biofeedback an effective treatment in autism spectrum disorders? A randomized controlled trial. *Applied Psychophysiology & Biofeedback, 38*(1), 17-28. https://doi.org/10.1007/s10484-012-9204-3

Kouijzer, M. E. J., de Moor, J. M. H., Gerrits, B. J. L., Buitelaar, J. K., & van Schie, H. T. (2009b). Long-term effects of neurofeedback treatment in autism. *Research in Autism Spectrum Disorders, 3*(2), 496-501. https://doi.org/10.1016/j.rasd.2008.10.003

Kouijzer, M. E. J., de Moor, J. M. H., Gerrits, B. J. L., Congedo, M., & van Schie, H. T. (2009a). Neurofeedback improves executive functioning in children with autism spectrum disorders. *Research in Autism Spectrum Disorders, 3*(1), 145-162. https://doi.org/10.1016/j.rasd.2008.05.001

Kumari, M. & Sharma, A. (2020). Neurofeedback training for social cognitive deficits: A systematic review. *International Journal of Online & Biomedical Engineering, 16*(10), 151-171. https://doi.org/10.3991/ijoe.v16i10.15923

Larson, M. J., & Carbine, K. A. (2017). Sample size calculations in human electrophysiology (EEG and ERP) studies: A systematic review and recommendations for increased rigor. *International Journal of Psychophysiology, 111*, 33-41. https://doi.org/10.1016/j.ijpsycho.2016.06.015

La Vaque, T. J., Hammond, D. C., Trudeau, D., Monastra, V., Perry, J., Lehrer, P., Matheson, D., & Sherman, R. (2002). Template for developing guidelines for the evaluation of the clinical efficacy of psychophysiological interventions. *27*(4), 273-281. https://doi.org/10.1023/A:1021061318355

Liu, N., Cliffer, S., Pradhan, A. H., Lightbody, A., Hall, S. S., & Reiss, A. L. (2017). Optical-imaging-based neurofeedback to enhance therapeutic intervention in adolescents with autism: methodology and initial data. *Neurophotonics, 4*(1), 011003. https://doi.org/10.1117/1.NPh.4.1.011003

Marzbani, H., Marateb, H. R., & Mansourian, M. (2016). Neurofeedback: A comprehensive review on system design, methodology and clinical applications. *Basic & Clinical Neuroscience, 7*(2), 143-158. https://doi.org/10.15412/J.BCN.03070208

Minshew, N. J., & Goldstein, G. (1998). Autism as a disorder of complex information processing. *Mental Retardation and Developmental Disabilities Research Reviews, 4*(2), 129-136. https://doi.org/10.1002/(SICI)1098-2779(1998)4:2<129::AID-MRDD10>3.0.CO;2-X

Mohammadi, R., Narimani, M., Abolghasemi, A., & Taklavi, S. (2019). Comparison of the effectiveness of treatment and education for autistic and related communication handicapped children (TEACCH) and neurofeedback on the promotion of cognitive, social, and daily living activities in children with autistic spectrum disorders. *Middle Eastern Journal of Disability Studies, 9*, 126-126. http://jdisabilstud.org/article-1-1788-en.html

Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology & Psychiatry*, 37(1), 51-87. https://doi.org/10.1111/j.1469-7610.1996.tb01380.x

Pineda, J. A., Brang, D., Hecht, E., Edwards, L., Carey, S., Bacon, M., & Rork, A. (2008). Positive behavioral and electrophysiological changes following neurofeedback training in children with autism. *Research in Autism Spectrum Disorders, 2*(3), 557-581. https://doi.org/10.1016/j.rasd.2007.12.003

Pineda, J. A., Carrasco, K., Datko, M., Pillen, S., & Schalles, M. (2014). Neurofeedback training produces normalization in behavioral and electrophysiological measures of high-functioning autism. *Philosophical Transactions of the Royal Society B, 369*(1644), 20130183. https://doi.org/10.1098/rstb.2013.0183

Ryan, A., & Files, N. (2019). The efficacy of neurofeedback and traditional therapies with autism spectrum disorder in a clinic setting. *Asia Pacific Journal of Neurotherapy*, 1(2), 14-20. https://apjnt.org/list-of-papers

Ryan, B., Vollebregt, & Arns. (2019). What's sleep got to do with it? Circadian rhythm sleep disorder, ADHD and neurofeedback. *Asia Pacific Journal of Neurotherapy, 1*(1), 85-98. https://apjnt.org/list-of-papers

Sokhadze, E. M., El-Baz, A. S., Tasman, A., Sears, L. L., Wang, Y., Lamina, E. V., & Casanova, M. F. (2014). Neuromodulation integrating rTMS and neurofeedback for the treatment of autism spectrum disorder: an exploratory study. *Applied psychophysiology & biofeedback*, *39*(3-4), 237–257. https://doi.org/10.1007/s10484-014-9264-7

van Doren, J., Arns, M., Heinrich, H., Vollebregt, M. A., Strehl, U., & K. Loo, S. (2019). Sustained effects of neurofeedback in ADHD: a systematic review and meta-analysis. *European Child & Adolescent Psychiatry*, *28*(3), 293-305. https://doi.org/10.1007/s00787-018-1121-4

van Hoogdalem, L. E., Feijs, H. M. E., Bramer, W. M., Ismail, S. Y., & van Dongen, J. D. M. (2020). The effectiveness of neurofeedback therapy as an alternative treatment for autism spectrum disorders in children. *Journal of Psychophysiology*, 1-14. https://doi.org/10.1027/0269-8803/a000265

Zivoder, I., Martic-Biocina, S., Kosic, A. V., & Bosak, J. (2015). Neurofeedback application in the treatment of autistic spectrum disorders (ASD). *Psychiatria Danubina, 27 Suppl 1*, S391-394. PMID: 26417802.