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EEG neurofeedback for executive functions in children with neurodevelopmental challenges

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[Intervention Protocol]

EEG neurofeedback for executive functions in children with neurodevelopmental challenges

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ABSTRACT

This is a protocol for a Cochrane Review (Intervention). The objectives are as follows:

To assess the effectiveness of EEG neurofeedback as treatment for inhibition and updating problems in children facing neurodevelopmental challenges.

BACKGROUND

Neurodevelopmental disorders encompass a range of conditions, each with cognitive challenges that become apparent during childhood. Historically, these have been conceptualised as various distinct disorders on the basis of clinical phenotype and the classification of symptom clusters in the *Diagnostic and Statistical Manual of Mental Disorders (DSM) Fourth Edition Text Revision (DSM-IV-TR)* or *Fifth Edition (DSM-5)*, or in the *International Classification of Diseases, 10th Revision (ICD-10)* (APA 2000; APA 2013; WHO 1993). Neurodevelopmental disorders include conditions such as intellectual disability; autism; Asperger syndrome; fetal alcohol spectrum disorder (FAS); fragile X syndrome; Down syndrome; and attention deficit hyperactivity disorder (ADHD) (Bishop 2008; Bishop 2010). However, existing classifications for neurodevelopmental disorders are not mutually exclusive, nor are these taxonomic systems intended to speak to causation – this presents a significant limitation when treatment options are considered.

Recent development of the Research Domain Criteria (RDoC) has encouraged scientists to undertake transdiagnostic reanalysis of disorders in the interests of identifying shared causal pathways and consequently informing more effective prophylactic or curative responses, or both (Coghill 2015). At the same time, emerging literature on neural circuitry is illuminating development of circuits that mediate core complex cognitive processes and behaviours in ways that cut across these diagnostic groups (Glahn 2016). Rather than distinct disorders, it is hypothesised that different phenotypes emerge as a result of the complex interplay of environment and biology within these neural circuits (Cuthbert 2010; Rutter 2010). For example, common cognitive functions that are implicated in a range of mental illnesses as well as in intellectual and behavioural disorders include reward response, emotional regulation, extinction, working memory, and response inhibition (Insel 2010a; Leckman 2010). In cognitive terms, these processes are summarised under the umbrella term ‘executive functions’ or cognitive control (Davidson 2006; Garvey 2016). They form the point of transdiagnostic intersect for several neurodevelopmental disorders (Doyle 2015; Insel 2010b).

Inhibition and updating dominate the executive function literature and are the cognitive functions that have received the most attention as key functions of executive control (Miyake 2000; St. Clair-Thompson 2006). As such, they form a secure conceptual platform for this review. Inhibition refers to the capacity to inhibit task-irrelevant distractors and to resolve conflict in pursuit of a particular goal (Barkley 1997; Hughes 2002; Miyake 2000; Padmanabhan 2015; Sergeant 2000). Inhibition is implicated in tasks such as the Stroop or Flanker task. Updating refers to the functioning of working memory and is implicated in coding of new information and, accordingly, in revising the information that is currently held in working memory. This function is targeted by tasks such as letter or digit memory and the Rey Auditory Verbal Learning Task (Kane 2003; Miyake 2000). Electroencephalographic (EEG) neurofeedback training provided to target inhibition and updating is described in the literature.

In sum, EEG neurofeedback treatment for neurodevelopmental disorders targeting the circuitry for inhibition or updating provides hope for prevention and remediation and will serve as the focus of this review.

Description of the condition

A considerable proportion of the population is affected by neurodevelopmental disorders including the following.

1. **Autism.** It is estimated that 1 in 160 children worldwide has a diagnosis of autism, which equates to more than 7.6 million disability-adjusted life-years and 0.3% of the global burden of disease (WHO 2013; WHO 2016a).
2. **ADHD.** It is estimated that 39 million people are affected by ADHD globally (WHO 2013). American reports suggest that approximately 11% of children between 4 and 17 years of age (6.4 million) are affected (Visser 2014). Australian figures suggest that 7.4% (298,000) of 4- to 17-year-olds who had a mental disorder between 2013 and 2014 suffered from ADHD (Lawrence 2015). Between 5 and 14 years of age, an estimated 3.4% of total years (1800 years) is lived in ill health or with disability, making ADHD the eighth leading cause of non-fatal loss of health for children in this age group in Australia (AIHW 2011).
3. **Intellectual disabilities.** An international meta-analysis in 29 countries indicated that, on average, 10.37 individuals among every 1000 people are affected (Maulik 2011). This is the seventh leading cause of non-fatal loss of health for children between birth and five years of age in Australia, with an estimated 4.3% of total years (700 years) lived in ill health or with disability (AIHW 2011).
4. **Down syndrome, FAS disorder, and fragile X syndrome.** These conditions have received little attention in statistical accounts; therefore, epidemiological data on these specific intellectual disorders are limited to prevalence rates. The incidence rate for Down syndrome is estimated to be around 1 in 1000 to 1 in 1100 live births worldwide (WHO 2016b). Western Australia reported estimates of FAS disorder of 0.4 per 1000 live births for the total population between 2000 and 2004 (Bower 2007). Roozen indicated that between 1990 and 2005 the reported occurrence of FAS disorder in Canada, Italy, and the United States was in between 30.52 and 47.13 per 1000, and, in South Africa, the prevalence of FAS disorder is particularly high, at 113.22 per 1000 (Roozen 2016). In the absence of life expectancy data for fragile X syndrome, and given the strong genetic component involved in development of this disorder, prevalence rates are expected to be the same across all age groups (Brown 2010). Leykin reported that numbers of Australian persons with fragile X syndrome were expected to range between 1362 and 4309 for a full mutation with intellectual disability, and Brown anticipated numbers of 13,466 and 87,137 with a permutation (Brown 2010; Leykin 2009). Crawford estimated that 1 in 3717 individuals of European descent is affected by this condition (Crawford 2002). Youings projected that 1 in 5530 persons in the United Kingdom would receive a diagnosis of fragile X syndrome, and, most recently, Coffee anticipated that fragile X syndrome would occur in 1 in 5161 males in the United States (Coffee 2009; Youings 2000). In sum, neurodevelopmental disorders exact a significant toll on individuals, families, and communities. Gaining an understanding of causal pathways with a view toward prevention or remediation should be seen as a priority.

Difficulties with cognitive control are evident in the neurocognitive profiles of many individuals with different neurodevelopmental disorders and are implicated in the behavioural and emotional presentation of affected children (Happé 2006; Pennington 1996). For example, problems related to inhibition and updating are

present to varying degrees. The neurocognitive profiles of children with fragile X syndrome and ADHD most often indicate problems with inhibitory control, which overlap with clinical features of impulsivity and hyperactivity (Bari 2013; Happé 2006; Hooper 2008; Knox 2012; Oosterlaan 2005). Difficulties with updating are implicated in Down syndrome, fragile X syndrome, and intellectual disability, all of which have been associated with limited ability to hold, manipulate, and process incoming information (Dauhauer 2014; Hartman 2010; Hooper 2008).

Executive functioning, the central mechanism required for cognitive control, refers to the ability of the cognitive system to co-ordinate internal processes (e.g. perceptual selection and maintenance of contextual information) in pursuit of performance of specific tasks (e.g. reading a book) (Botvinick 2001). Although phenotypic presentations of difficulties in this area can be diverse, their impact on functioning generally aggregates and worsens as an individual gets older (Goldstein 2010; Masten 2005). Thus, when treatments for executive functioning challenges are considered, patient age is critical.

Executive functions develop rapidly during childhood (from about the age of six years) and adolescence (e.g. Anderson 2002). Not only does executive control typically improve during this time, but the nature of these functions is more fully differentiated (Brydges 2012; Brydges 2014a; Shing 2010). The growing neuroplasticity literature informs us that integral to the maturation of a child's nervous system are sensitive (but not necessarily critical) periods for development (Davis 2009; Happé 2014; Heim 2012; Knudsen 2004; Newport 2001; Perani 2003; Wachs 2014; Weber-Fox 1996). During these sensitive periods, the brain is particularly susceptible to change through experience, with potential for diminished remediation in adulthood. Therefore, considering treatment possibilities, such as EEG neurofeedback, during emerging stages of executive functions provides hope for remediating long-term dysfunction (Sonuga-Barke 2010). This developmental period accounts for the choice of age groups included in the present review. We will focus on children and adolescents between 6 and 18 years of age. As executive dysfunction plays a role in various disorders, review authors will not discriminate between disorders. Instead, we will focus on

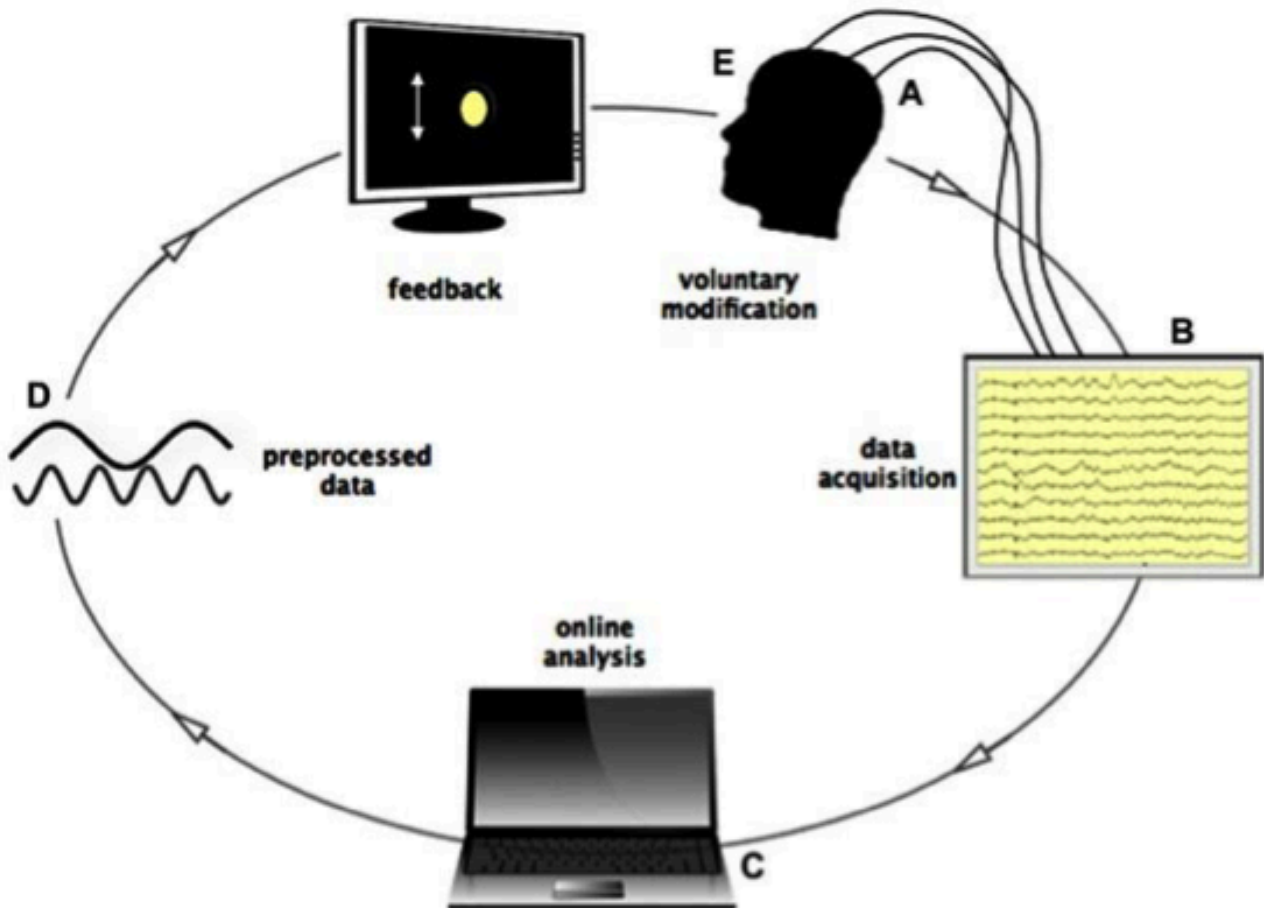
core executive functions targeted through EEG neurofeedback, specifically, inhibition and updating.

Description of the intervention

Cognitive-behavioural therapy, behavioural intervention, medical treatment, or a combination of at least two of these is currently employed to manage symptoms of neurodevelopmental disorders (Ageranioti-Bélanger 2012; Fabiano 2009; Hsia 2014; Moskowitz 2011; Murawski 2015; Narzisi 2014; Reichow 2011; Scheifes 2013; Weston 2016). Conclusions regarding the effectiveness of non-invasive symptomatic treatment approaches are often limited by methodological weaknesses, such as lack of methodological rigour or lack of randomisation during group allocation, and future research is needed to further evaluate treatment options (Ozonoff 1998; Reid 2015; Walters 2016). The current alternative to non-invasive approaches is pharmacological intervention. The pharmacological treatment pathway serves as a popular means of symptom control, particularly for children with ADHD (Banaschewski 2006; Faraone 2010; Scheifes 2013). Although pharmacological interventions may be deemed a moderately effective treatment option, side effects (e.g. headaches, dizziness, reduced appetite, growth restriction), lack of certainty around potential long-term risks, reappearance of symptoms after discontinuation of treatment, and non-response to medication have sparked the search for non-invasive long-term treatments that can be provided without negative consequences (Graham 2011; Heinrich 2007; Jensen 2007; Murray 2008).

Technical advances have seen the development of EEG neurofeedback as a promising, non-pharmacological mode of intervention that can be used to help train, prevent or remediate participants' cognitive impairment at the source. EEG neurofeedback is commonly conceptualised as computer game-based training of awareness or control of cognitive state that can be achieved by providing participants with real-time feedback on their own brain states (Figure 1). It is thought that participants can learn to modify or control targeted brain-state activity, inducing neural plasticity, which leads to improved self-regulation in daily activities.

Figure 1. Neurofeedback intervention loop. This figure is adapted from Bagdasaryan and Le Van Quyen. It depicts a simplified overview of neurofeedback that is delivered via electroencephalography (EEG) (Bagdasaryan 2013). During the neurofeedback session, the individual’s brain signal is acquired through the EEG equipment (A, B). The software processes the incoming brain signal and provides information about the degree of alignment between the participant’s real-time brain activity and predetermined training goal parameters (C, D). This information is presented to the participant as visual or auditory feedback in real time, to continuously update the participant about modulation of his or her own brain activity (E; Bagdasaryan 2013; Huster 2014).

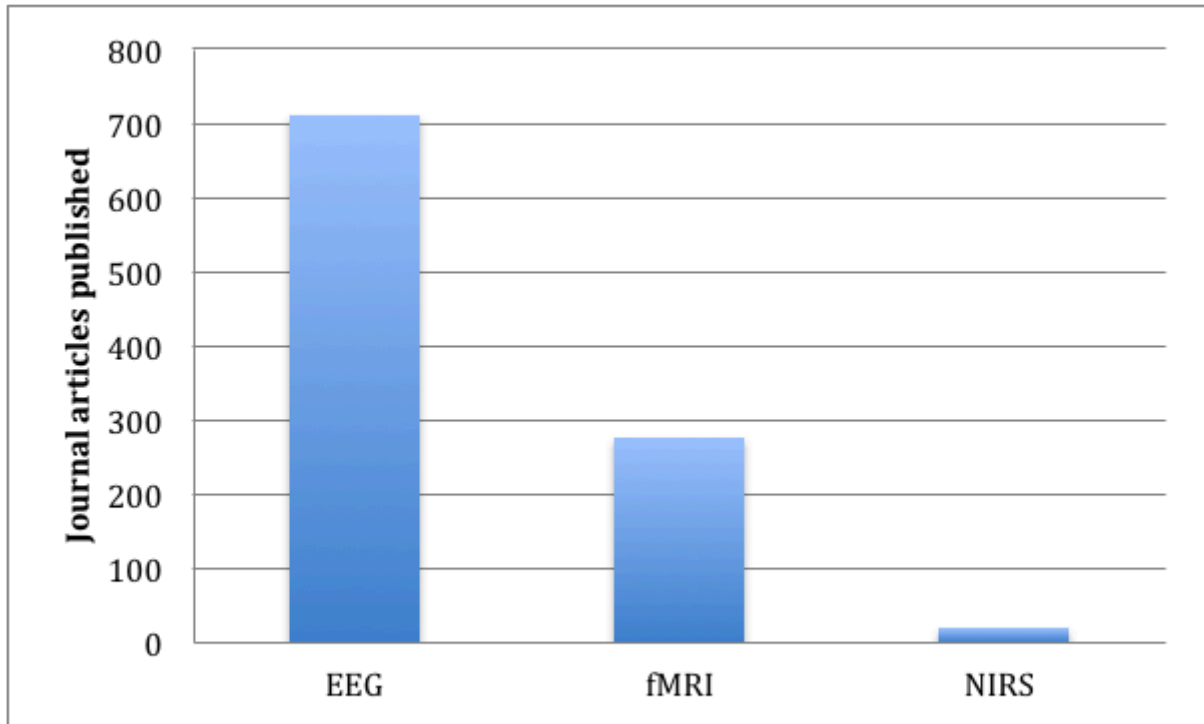


EEG neurofeedback is not dependent on complex verbal instructions; therefore, this brain-training intervention can be effectively implemented cross-culturally and in groups with language and communication impairment. It is designed to be embedded in a game format, which offers face validity as a treatment for children. Currently, neither implementation of this approach in the community nor training of healthcare providers is monitored by an accredited organising body. Instead, implementation of EEG neurofeedback is based on the personal preferences of providers and consumers, which makes a systematic review of the evidence base for this treatment a critical task.

EEG neurofeedback training comprises a range of elements. A fundamental technical component of this intervention is the technology that is used to monitor the degree of alignment between the participant’s brain activity and the pre-set goal parameter. Various brain-imaging techniques have been utilised for the intervention, such as near-infrared

spectroscopy (NIRS), functional magnetic resonance imaging (fMRI), and electroencephalography (EEG) (Egner 2001; Marx 2015; Scharnowski 2015). Experimental research to appraise various brain-imaging techniques is ongoing; however, to date, no one technique has been identified as the superior method for obtaining neurofeedback, nor is compelling evidence available to support the use of one technique over another. Therefore, we argue that to get the most accurate picture of changes in brain activity, such as those required in a microanalysis of learning, a measure that can capture the most fine-grained changes in milliseconds rather than seconds is preferable (Sauseng 2008). Because it offers the highest temporal resolution of all known techniques, EEG is the only technology that meets this criterion. Additionally, EEG is less costly and is more widely used than alternatives (Figure 2). Its use is also more feasible than, for example, fMRI when healthcare providers are working with children, as the experience of being inside an MRI machine can be unsettling and may disrupt optimal task completion.

Figure 2. Publication rates between 2006 and 2016 of journal articles examining EEG-, fMRI-, and NIRS-neurofeedback, as indexed by Scopus. Footnotes EEG: electroencephalogram; fMRI: functional magnetic resonance imaging; NIRS: near-infrared spectroscopy.



EEG is a non-invasive method that is used to measure the brain's electrical activity along the scalp via sensors or electrodes. During setup of EEG neurofeedback, these electrodes are placed on the head of the participant, and training goal parameters define features of the EEG signal that are to be trained (i.e. EEG frequency); the number of electrodes; the placement location of electrodes on the scalp; and the number, duration, and frequency of sessions (e.g. to decrease theta/alpha activity and increase beta activity, one electrode at Cz for 12 sessions overall, split into three 45-minute sessions per week; to increase sensorimotor rhythm and suppress theta activity, individualised training at, for example, C3 and C4, for 30 sessions overall, split into two 20-minute sessions per week; and to increase sensorimotor rhythm and suppress theta activity, electrode placement at C4 for 40 sessions overall, split into 20 meetings of two 60-minute sessions each with a short break in between (Arnold 2013; Lansbergen 2011; Perreau-Linck 2010)). Once EEG neurofeedback equipment is set up, the participant's neural variability is recorded and analysed in real time, and the degree of alignment between set parameters and the person's neural activity is communicated in the form of visual or auditory feedback. Visual feedback may consist of a blurry picture of an animated character. Only when the participant's ongoing neural variability matches goal parameters would the picture become

clear. Alternative visual feedback may be received in the form of virtual reality tasks, animated games, waveforms, or graphs (e.g. Linden 1996). Auditory feedback may comprise tones that change in volume, pitch, or duration as recorded activity fluctuates (Egner 2002).

The elaborate setup of the intervention poses interesting challenges for comparison or control groups in EEG neurofeedback research. As the technology is integral to EEG neurofeedback, the most rigorous and the only form of placebo for which blinding is possible is sham EEG neurofeedback. During sham EEG neurofeedback, the participant receives feedback unrelated to his or her own performance but based on pre-recorded or artificial EEG activity. Apart from the technical elements that make it challenging to find a placebo or carry out blinding, participants unsuccessfully attempt to learn to modulate their (fictitious) neural activity, often resulting in poor compliance or frustration. Therefore, rather than subjecting participants to a blinding trial, EEG neurofeedback research frequently implements alternative comparators such as conventional treatments (i.e. active, non-invasive control trials, for example, behavioural management interventions) and wait-list controls, by which participants wait for their active treatment intervention (Sonuga-Barke 2013).

Uncertainty in the literature pertains to the measurable effect of EEG neurofeedback, as well as to effects of technology, target frequency, electrode location, feedback type, number of sessions, and session duration on the efficacy of the intervention. Each of these intervention components forms a critical part of the intervention. In theory, any changes in the composition of these parts can influence the efficacy of EEG neurofeedback and may constitute distinct therapeutic approaches. In the absence of an evidence base for EEG neurofeedback, clinical practice currently operates on the basis of literature that has produced favourable EEG neurofeedback outcomes in the past (e.g. [Arnold 2013](#); [Lubar 1995a](#)). Each component needs further research to explore its influence on the intervention process. With a growing number of EEG neurofeedback studies and approaches, it is not clear which frequency, electrode location, number of sessions, technology, or feedback type provides the best EEG neurofeedback therapy for children with executive functioning problems. As a starting point, we will look at the current literature and will investigate the fundamental effectiveness of EEG neurofeedback.

How the intervention might work

Traditionally, researchers have conceptualised the neurofeedback loop as a learning process, which follows behavioural learning mechanisms of operant conditioning. Operant conditioning studies (e.g. Schedules of Reinforcement) have shown that targeted participant behaviour can be regulated by providing positive reinforcement immediately after the targeted behaviour occurs ([Ferster 1957](#)). It is of great importance that the relationship between the behaviour and the reinforcement is clear to the learner. As such, the timing of presentation of the reinforcer is crucial, as even small delays (as little as a second) can decrease the strength of the association between the reinforcer and the target behaviour that is to be reinforced ([Skinner 1958](#)). Use of EEG in the neurofeedback paradigm has enabled researchers to seek more immediate and more secure associations via measurable aspects of behaviours and reinforcers, such as by targeting the specific oscillations that underlie clinical symptoms like impulsivity or hyperactivity as the independent variable ([Gevensleben 2012](#)). It is therefore conceivable that the capacity for EEG neurofeedback to provide subsecond feedback may make it especially efficient as an approach to behaviour modification and brain plasticity, as compared with mental regulation unassisted by feedback ([Bai 2014](#); [Beatty 1974](#)). Nevertheless, it should be noted that this potential advantage of high temporal resolution of EEG over other neurofeedback techniques has not been demonstrated.

Progressing technology has enabled researchers to seek clues in an attempt to describe EEG neurofeedback mechanisms from a biological viewpoint. As mentioned earlier, during EEG neurofeedback, the participant is provided with feedback about differences between target parameters and their actual neural activity. In theory, through this feedback, the participant can learn to modulate brain activity towards the target parameter. Fundamental to this step is the idea that, during training, the participant learns to memorise the neural or behavioural state at the time of the reward, which facilitates reproduction of this same pattern in the future. Currently, the mechanisms that underpin this learning process have not been fully illuminated. However, principles of neuroplasticity may provide further clues to the causal pathway.

Neuroplasticity refers to the unique ability of the brain to grow neurons and to alter neural connections in response to experiences ([Siegel 2010](#)). Imaging studies have indicated, for example, how training in activities such as music, exercise, or meditation can have a lasting impact on brain structure or function, or both ([Vance 2010](#); [Zatorre 2013](#)). This finding highlights the fact that repeated, activity-dependent experiences can have a lasting impact on the brain ([Ganguly 2013](#)). Converging evidence suggests that reinforcing a particular oscillatory pattern through EEG neurofeedback training increases the likelihood that the same pattern will be reproduced more easily in the future ([Lubar 1995b](#); [Ros 2010](#)). For the beta rhythm, for example, this effect is robust enough to be detected up to three years after EEG neurofeedback training ([Engelbregt 2016](#)). This supports the fundamental premise of EEG neurofeedback that the brain can be conditioned to exhibit certain oscillatory patterns.

Theoretically, this phenomenon might be explained by a combination of previously established plasticity mechanisms, such as associative and homeostatic forms ([Ros 2014](#)). The principle of associative (i.e. Hebbian) plasticity suggests that “synapses that fire together, wire together and synapses that fire apart, wire apart” ([Knoblauch 2012](#)). Neurophysiological evidence indicates that the amplitude of EEG oscillations is augmented by the number of neurons (or synaptic potentials) ([Musall 2014](#)). Therefore, with repeated training, the connections between neuron populations that are amplified or synchronised to create a particular oscillatory pattern would strengthen, facilitating generation of this pattern in the future. Common to all these theories is the hypothesis that modification of neural circuitry is possible and is likely to result in observable behavioural changes.

At a more technical level, during EEG neurofeedback, electrodes that are placed on the scalp measure the synchronised, rhythmical fluctuations of local field potentials of groups of neurons, also known as neural oscillations. These oscillations arise from the excitatory postsynaptic potentials (EPSPs) of large groups of neurons, resulting in a measurable EEG signal at scalp level ([Nunez 2000](#)). Synchronised oscillatory activities are associated with cognitive abilities such as inhibition (for a review, see Klimesch), updating of working memory, and temporary maintenance of information in working memory, which suggests that neural oscillations are a fundamental functional mechanism in cortical computation ([Deiber 2007](#); [Klimesch 2006](#); [Sauseng 2010](#)).

Why it is important to do this review

Executive functions play a critical role in everyday life. Performance of complex tasks, academic achievement, and later success in life are mediated by the development of executive functioning ([Garavan 1999](#); [Miyake 2000](#); [St. Clair-Thompson 2006](#)). EEG neurofeedback treatment provides hope for prevention and remediation of difficulties in the area of executive functioning for children with neurodevelopmental disorders. New EEG neurofeedback protocols are continuously being tested to determine what most effectively reduces or remediates executive functioning difficulties in neurodevelopmental disorders such as inhibition and updating (e.g. [Kouijzer 2009](#)). However, commercial use of EEG neurofeedback is currently outpacing the evidence base. The economic cost of this intervention is high for parents, but patient desperation is also high. A history of poorly researched interventions for children (e.g. studies by Bishop and Stephenson) has encouraged the profession to take greater accountability in

establishing the effectiveness of new treatments as a matter of priority and professional ethics (Bishop 2007; Stephenson 2008). Use of EEG neurofeedback as an intervention for children with neurodevelopmental problems has reached just such a critical juncture. It is imperative that the evidence base for these interventions is now put to the test.

This systematic review is the first of its kind and therefore will make a unique contribution to the EEG neurofeedback literature. It is preliminary to any further investigations of the cost-effectiveness or feasibility of this intervention. If the effectiveness of EEG neurofeedback is supported by this review, it is conceivable that additional research will be conducted to identify its applicability to other mental health conditions or age groups.

OBJECTIVES

To assess the effectiveness of EEG neurofeedback as treatment for inhibition and updating problems in children facing neurodevelopmental challenges.

METHODS

Criteria for considering studies for this review

Types of studies

All randomised controlled trials (RCTs) (i.e. random allocation of participants to treatment, control, or follow-up groups) and quasi-RCTs (i.e. allocation of participants to intervention or control groups via date of birth, etc.).

Types of participants

Children or adolescents, or both, aged 6 to 18 years with executive functioning difficulties in the domains of inhibition and updating. We will identify these neurodevelopmental challenges in the literature via the clinical diagnosis of a neurodevelopmental disorder such as intellectual disability, autism spectrum disorder (ASD), FAS disorder, fragile X syndrome, Down syndrome, and ADHD, as specified by the *Diagnostic and Statistical Manual of Mental Disorders (DSM) Fourth Edition Text Revision (DSM-IV-TR)* or *Fifth Edition (DSM-5)*, or by the *International Classification of Diseases, 10th Revision (ICD-10)* (APA 2000; APA 2013; WHO 1993).

Excluded participants

We will exclude participants with severe brain damage, epilepsy, Tourette's syndrome, or any condition in which the focus of neurofeedback intervention is not specific to executive functions (e.g. to remediate damage, seizures, or tics), as well as participants with non-neurodevelopmental comorbidities (e.g. depression).

We will include participants with neurodevelopmental challenges as well as those with other mental health problems, as long as data provided for participants with neurodevelopmental challenges can be considered separately.

Types of interventions

EEG neurofeedback (also referred to as EEG biofeedback), regardless of protocol (target frequency), feedback type (visual vs aural), and session number and duration, used as treatment for improving levels of inhibition and updating (or both) in children with neurodevelopmental challenges. We will include studies administering EEG neurofeedback in combination with another

intervention only when the cointervention is administered to both groups.

For reasons outlined earlier in this protocol, control groups will include sham feedback (i.e. feedback that is unrelated to the participant's neural activity at the time of intervention administration), treatment as usual (e.g. behavioural management intervention), and wait-list control (see [Description of the intervention](#)).

Types of outcome measures

Primary outcomes

1. Changes in participant EEG profiles (e.g. event-related potential (ERP), specifically, N2 for inhibition and P3 for updating (Brydges 2014b; Donchin 1988; Luck 2014; Polich 2007))
2. Changes in inhibition (e.g. Stroop Color and Word Test: Children's Version) and changes in updating (e.g. Rey Auditory Verbal Learning Test) (Golden 2002; Schmidt 1996)
3. Adverse effects (e.g. Pittsburgh Side Effects Rating Scale) (Pelham 1999) (It is important to note that we will consider only outcome assessments for which the outcome assessor was blinded.)

Secondary outcomes

1. Changes in behavioural performance (e.g. hyperactivity and impulsivity, as measured by self-report measures such as Conners-3 (Conners 2011)) (Again, we will consider only outcome assessments for which the outcome assessor was blinded.)

Search methods for identification of studies

Electronic searches

We will search the electronic databases and trials registers listed below, and will not limit our searches by language, date, or publication type.

1. Cochrane Central Register of Controlled Trials (CENTRAL; current issue) in the Cochrane Library, which includes the Cochrane Developmental, Psychosocial and Learning Problems Specialised Register.
2. MEDLINE Ovid (1946 onwards).
3. MEDLINE In-Process and Other Non-Indexed Citations Ovid (current issue).
4. MEDLINE Epub Ahead of Print Ovid (current issue).
5. Embase Ovid (1974 onwards).
6. CINAHL Plus EBSCOhost (Cumulative Index to Nursing and Allied Health Literature; 1937 onwards).
7. PsycINFO Ovid (1806 onwards).
8. Science Citation Index - Expanded Web of Science (SCI-EXPANDED; 1970 onwards).
9. Social Sciences Citation Index Web of Science (SSCI; 1970 onwards).
10. Conference Proceedings Citation Index - Science Web of Science (CPCI-S; 1990 onwards).
11. Conference Proceedings Citation Index - Social Science & Humanities Web of Science (CPCI-SS&H; 1990 onwards).
12. *Cochrane Database of Systematic Reviews* (CDSR; current issue), part of the Cochrane Library.

13. Database of Abstracts of Reviews of Effects (DARE; current issue), part of the Cochrane Library.
14. Epistemonikos (www.epistemonikos.org/nl/advanced_search).
15. WorldCat (OCLC) (www.worldcat.org/default.jsp).
16. ClinicalTrials.gov (www.clinicaltrials.gov).
17. World Health Organization (WHO) International Clinical Trials Registry Platform (ICTRP; apps.who.int/trialsearch).

We will use the strategy provided in [Appendix 1](#) to search MEDLINE, and we will adapt it appropriately for searches of the other databases. When papers are published in a language other than English, we will contact the study author to request reports translated into English.

Searching other resources

Grey literature

We will search the websites listed below for unpublished studies in this field.

1. The Association for Applied Psychophysiology and Biofeedback, Inc. (aapb.org).
2. The Biofeedback Federation of Europe (bfe.org).
3. EEG Spectrum International (eegspectrum.com/ADHD-ADD).
4. International Society for Neurofeedback and Research (isnr.org).
5. Applied Neuroscience Society of Australasia (appliedneuroscience.org.au).

Reference lists

We will search the reference lists of all eligible studies included in this review for additional relevant studies that meet our inclusion criteria (see [Criteria for considering studies for this review](#)).

Data collection and analysis

Selection of studies

Two review authors (JL and SB) will individually examine the titles and abstracts of records yielded by the search and will assess them against the inclusion criteria of this review ([Criteria for considering studies for this review](#)). For all studies that meet the inclusion criteria, or for which more information is needed to assess eligibility, we will obtain the full-text reports, and both review authors (JL and SB) will separately reassess these studies against the inclusion criteria. For full-text reports that are not written in the English language, or for data that are not available in the article, we will contact study authors for further information, to help us ascertain the eligibility of these studies for inclusion in the present review. We will record reasons for inclusion or exclusion of all studies separately, and JL and SB will discuss discrepancies between views. When conflicting views cannot be reconciled, these review authors will consult the entire research team until a consensus is reached. Before any studies are selected, we will pilot-test application of the eligibility criteria. Finally, for maximum transparency during this selection process, we will complete a PRISMA flow diagram ([Liberati 2009](#)).

Data extraction and management

Two review authors (JL and SB) will independently extract and enter the following data from each study onto an electronic spreadsheet specifically designed for this Cochrane Review:

participant details (age, gender, executive function problems); intervention details (number of sessions, session duration, follow-up, electrode location(s), frequency parameter, aural or visual feedback mode); study location; type of study (RCT, quasi-RCT); intervention procedures (treatment allocation, blinding); type of control group (sham feedback, treatment as usual, wait-list control); and outcome measure data. Before any data are extracted, we will pilot test the application of spreadsheet categories, to ensure that relevant and comprehensive data are collected. We will resolve disagreements amongst ourselves that might occur during the data extraction process. When conflicting views cannot be reconciled, we will consult the entire research team until a consensus decision is reached.

Assessment of risk of bias in included studies

Two review authors (JL and SB) will independently assess the risk of bias of each included study, using the Cochrane 'Risk of bias' tool ([Higgins 2017](#)). We will assess each study as having low, high, or unclear risk of bias in relation to each of the 'Risk of bias' domains described below. JL and SB will record each rating separately and will discuss discrepancies between views. When conflicting views cannot be reconciled, we will consult the entire research team until a consensus agreement is reached.

Cochrane 'Risk of bias' tool

The domains described below form the 'Risk of bias' assessments for RCTs and quasi-RCTs.

Sequence generation

We will describe the method used in each study to generate the participant allocation sequence and will assess whether this sequence should have produced comparable participant groups.

Review authors' judgement: Is the participant allocation sequence truly random, and what is the resulting risk of allocation bias to experimental or control groups?

1. Low risk of bias: Study authors described the random component in the allocation sequence of participants (e.g. computer random number generator, random number table).
2. High risk of bias: Study authors described a non-random component in the allocation sequence of participants (e.g. allocation by date of birth or by judgement of the investigator).
3. Unclear risk of bias: The process of randomisation was not described in sufficient detail to permit a judgement of low or high risk of bias.

Allocation concealment

We will describe the measures that were used to conceal the allocation process from participants and from investigators and will determine whether this allocation to a particular group or training schedule could have been foreseen before, or during, participation by participants or investigators.

Review authors' judgement: Is the participant allocation sequence truly concealed, and what is the resulting risk of allocation bias due to inadequate concealment?

1. Low risk of bias: The allocation procedure was adequately concealed from participants and investigators (e.g. telephone allocation).

2. High risk of bias: Participants or investigators could have foreseen their allocation (e.g. when allocation was based on the judgement of the clinician or on the date of birth of participants).
3. Unclear risk of bias: The allocation process was not described in sufficient detail to permit a judgement of low or high risk of bias.

Blinding of participants and personnel

We will describe all modes of blinding participants and staff from any knowledge of the intervention that a participant received.

Review authors' judgement: Are participants and personnel adequately blinded from any knowledge of the type of intervention that participants received, and what is the resulting risk of performance bias?

1. Low risk of bias: Lack of blinding (no blinding or incomplete blinding) is present, but it is clear that this lack of blinding is unlikely to influence the outcome; or participants and personnel have been blinded, and it is unlikely that this blinding has been interrupted.
2. High risk of bias: Lack of blinding (no blinding or incomplete blinding) is present, and outcomes are likely to have been influenced by lack of blinding; or participants and personnel have been blinded, but it is likely that this blinding has been interrupted, which has influenced the outcome.
3. Unclear risk of bias: The blinding process was not described in sufficient detail to permit the judgement of low or high risk of bias, or this outcome was not addressed in the study. Owing to the learning component in EEG neurofeedback (see [Description of the intervention](#)), we expect that most studies will fall into this category.

Blinding of outcome assessment

We will describe all modes of blinding outcome assessors from any knowledge of the intervention that a participant received.

Review authors' judgement: Are outcome assessors adequately blinded from any knowledge of the type of intervention that participants received, and what is the resulting risk of detection bias?

1. Low risk of bias: The outcome assessment was not blinded, but it is clear that this lack of blinding is unlikely to influence the outcome measurement; or the outcome assessment has been blinded, and it is unlikely that this blinding has been interrupted, which has influenced outcome measurements.
2. High risk of bias: The outcome assessment was not blinded, and outcomes are likely to have been influenced by the lack of blinding, or the outcome assessment has been blinded, and it is likely that this blinding has been interrupted, which has influenced outcome measurements.
3. Unclear risk of bias: The blinding process was not described in sufficient detail to permit the judgement of low or high risk of bias, or this outcome was not addressed in the study. Owing to the learning component in EEG neurofeedback (see [Description of the intervention](#)), we expect that most studies will fall into this category.

Incomplete outcome data

We will describe the completeness of outcome data, including information on participant attrition, exclusions, re-inclusions for

analyses, and participant numbers for each intervention group, as well as any withdrawals from study groups.

Review authors' judgement: Are incomplete data handled adequately, and what is the resulting risk of attrition bias?

1. Low risk of bias: There is no indication of missing data; if data are missing, the same numbers of data points are missing across intervention groups; the data have been imputed suitably; or reasons for the missing data are unlikely to have influenced the outcome.
2. High risk of bias: Uneven numbers of data points are missing across intervention groups; data have been imputed through an unsuitable approach; or reasons for missing data are likely to have influenced the outcome.
3. Unclear risk of bias: Study authors did not provide sufficient information that permits a judgement of low or high risk of bias (e.g. no reasons for missing data provided), or this outcome was not addressed in the study.

Selective reporting

To assess any reporting bias, we will examine whether all prespecified outcomes have been reported. When this is not the case, we will contact researchers to ask for non-reported findings.

Review authors' judgement: Are there indications of selective outcome reporting, and what is the resulting risk of reporting bias?

1. Low risk of bias: Study protocol is available, and outcomes prespecified in the protocol have been reported; when the protocol is not available, it is clear that all expected outcomes have been reported.
2. High risk of bias: Not all of the outcomes prespecified in the protocol were reported; measurements were used that were not prespecified; outcomes were reported that were not prespecified; reporting of outcomes was incomplete; or study authors failed to include results for a particular outcome.
3. Unclear risk of bias: Study authors did not provide sufficient information to permit a judgement of low or high risk of bias. It is expected that most studies will fall into this category.

Allegiance bias

We will report any concerns of allegiance bias not otherwise covered by above-mentioned components of the 'Risk of bias' tool. Currently, no consensus has been reached on what constitutes an effective measurement of allegiance bias. The procedure most often used to document and evaluate this type of bias was developed by Gaffan and colleagues ([Gaffan 1995](#)). However, in line with the critique provided by Leykin and colleagues, we will focus on evaluation of the treatment protocol by its developers or, in this case, on sponsorship by neurofeedback equipment owners, to measure high, low, or unclear risk of allegiance bias ([Leykin 2009](#)).

1. Low risk of bias: no evidence that study authors developed the protocol; study was not sponsored by neurofeedback equipment owners.
2. High risk of bias: evidence that study authors developed the protocol; study was sponsored by neurofeedback equipment owners.
3. Unclear risk of bias: information provided points towards allegiance bias, but insufficient details prevent a judgement of

whether low or high risk of bias is present; evidence of this bias is insufficient.

Measures of treatment effect

Continuous data

For continuous data, we will calculate the mean difference (MD), when possible (i.e. when the same outcome variables were assessed via the same measurement scale). When investigators assessed the same outcome variables through different modes of data collection (i.e. different scales, various scoring methods), we will calculate the standardised mean difference (SMD). We will present both the MD and the SMD with 95% confidence intervals (CIs).

Dichotomous data

For dichotomous data, we will compute the risk ratio (RR) for each outcome and the 95% CI to describe the probability that a particular outcome is going to occur.

Unit of analysis issues

Cluster-randomised trials

To our knowledge, no EEG neurofeedback studies have randomised groups or clusters of participants, rather than individuals; therefore, we do not expect to find any cluster-randomised trials during our search. Should cluster-randomised trials become available in the future, we will assume that researchers have adjusted for clustering in their results. For trials that have not previously adjusted for clustering, we will attempt to calculate an estimate of the intracluster correlation coefficient (ICC) by using the formula provided in the *Cochrane Handbook for Systematic Reviews of Interventions* (Higgins 2011). If we are unable to calculate the ICC (owing to lack of information), we will request further information from study authors or will attempt to calculate the ICC using data from comparable studies or available resources. We will examine the impact of variation in ICCs via a [Sensitivity analysis](#) and will discuss outcomes in the Discussion section of the review.

Cross-over trials

We do not anticipate identifying any cross-over trials. However, if we do, we will use data from the first period only, given the possibility of a carry-over effect, and lack of available information concerning the time taken for any intervention effects to fade or 'wash out'.

Studies with multiple interventions

We will combine all EEG groups and will conduct a simple, pair-wise comparison with all control groups. For participants who continue to receive medication, we will consider data only if participants in both intervention and control groups continue to receive medication. We will conduct a sensitivity analysis to examine the potential effects of differences in participants' medication or dosage, or both, on trial results (see [Sensitivity analysis](#)).

Multiple reports

When multiple reports describe the same study, we will take extra care to ensure that only independent findings are reported. If it is unclear whether reports include independent findings, we will contact the report authors to ask for clarification.

Dealing with missing data

We will record attrition and missing outcome data for each study and will contact study authors to request missing outcome data. When study authors do not provide data for missing summary statistics (e.g. standard deviations), we will base our calculations on other reported outcomes, when possible. When study authors do not provide data for missing specified outcomes, we will attempt to conduct an intention-to-treat (ITT) analysis by including participants randomised into a trial, irrespective of the group. If an ITT analysis is not possible, we will conduct an available case analysis using only participants whose outcome data are known. We will examine the impact of missing data on the main analyses via a [Sensitivity analysis](#) and will discuss outcomes in the Discussion section of the review.

Assessment of heterogeneity

We will assess clinical and methodological heterogeneity by comparing the effects of distribution of key participant traits (e.g. distribution of sex, age, severity of executive functioning difficulties), protocol factors (e.g. target frequency, electrode location, feedback type, number of sessions, session duration), and trial factors (e.g. randomisation) on the efficacy of the intervention. We will employ forest plots to identify any statistical heterogeneity (overlap of CIs) and will quantify this by computing I^2 and Chi^2 statistics (Deeks 2011). Although I^2 of 50% is a reasonable indication of heterogeneity, substantial heterogeneity will be clearly exemplified by I^2 of 65% (Section 9.5.2; Deeks 2011). The P value for the Chi^2 test must be less than 0.10. We will employ the Tau^2 statistic as a measure of between-study variability.

In the event of very high heterogeneity, we will identify studies that are contributing to high heterogeneity and will exclude them. If exclusion does not successfully remove the heterogeneity, we will not present outcomes of meta-analyses for this variable. We will transparently record all actions and reasons for exclusion.

Assessment of reporting biases

Before we include any studies, we will assess risk of allocation, detection, performance, attrition, and reporting biases, as outlined in the [Assessment of risk of bias in included studies](#) section above. Additionally, when we include more than 10 studies, we will prepare funnel plots to assess publication bias. We will visually inspect these plots for skewness. When we find evidence of an asymmetrical funnel plot, we will apply Egger's test (Egger 1997).

Data synthesis

To conduct the meta-analysis, we will pool outcome data through Review Manager 5 (RevMan 5) (Review Manager 2014). Owing to the nature of our study design, we will consider the likelihood of heterogeneity in our data as high (e.g. data from varying EEG neurofeedback protocols, participants with different disorders and from different study designs).

Given the high probability of significant heterogeneity in our results, we will apply a random-effects model. We will conduct subgroup analyses to systematically investigate heterogeneity (see [Subgroup analysis and investigation of heterogeneity](#)).

'Summary of findings'

We will create a 'Summary of findings' table using a combination of RevMan 5 ([Review Manager 2014](#)) and GRADEprofiler (GRADEpro; [GRADEpro GDT 2015](#)). In this table, we will present effects of EEG neurofeedback (1) on underlying ERPs of executive function performance, (2) on executive function performance as measured by psychometric tests, (3) in relation to overall well-being of participants with adverse effects recorded, and (4) on behavioural performance. Additionally, we will include the number of participants and a rating of the quality of evidence based on GRADE criteria derived using GRADEpro ([GRADEpro GDT 2015](#); [Guyatt 2006](#); [Schünemann 2006](#)). Two review authors (JL and SB) will independently rate the quality of evidence according to one of four levels (high, moderate, low, or very low). For example, we will rate the quality of evidence from a RCT as high; however, presence of risk of bias (e.g. design limitations, limitations in the implementation of studies that are likely to introduce bias), indirectness of evidence (e.g. indirect effects on the population, intervention or control groups, or outcomes), imprecision (e.g. wide CIs due to small sample sizes), inconsistency of results (e.g. unexplained heterogeneity), and/or reporting bias (e.g. publication bias; failure to report outcomes) may lower the GRADE rating. Both review authors will make notes to guide their judgements to ensure a transparent rating procedure.

Subgroup analysis and investigation of heterogeneity

Subgroup analyses are observational in nature, and any conclusions drawn are intended only to highlight potential areas of future research ([Deeks 2011](#)). When sufficient outcome data are available, we will carry out subgroup analyses and investigations of heterogeneity for each outcome (see [Types of outcome measures](#)), with consideration of the following points.

1. Investigation of the effectiveness of EEG neurofeedback as a function of frequency, session number, session duration, electrode location, or feedback type.
2. Investigation of the effectiveness of EEG neurofeedback as a function of the control group against which it is compared.
3. Investigation of the effectiveness of EEG neurofeedback as a function of the childhood disorder, as defined by diagnostic criteria (DSM-IV-TR; DSM-5; ICD-10) ([APA 2000](#); [APA 2013](#); [WHO 1993](#)).
4. Investigation of the effectiveness of EEG neurofeedback as a function of age.
5. Investigation of the interaction between intervention factors (e.g. session numbers) and dropout rates.

Sensitivity analysis

Our goal is to draw robust conclusions regarding the questions that we ask in this review. When methodological choices of individual studies or trial analyses might compromise the robustness of our conclusions, we will conduct sensitivity analyses. Specifically, we anticipate that we will be able to conduct sensitivity analyses for the situations listed below.

1. Comparison of variable findings from RCTs and quasi-RCTs.
2. Studies with high or unclear risk of bias as indicated by the 'Risk of bias' assessment.
3. Studies with concurrent psychopharmacological treatment.
4. Variation in ICCs for analyses pertaining to cluster-randomised controlled trials.
5. Studies with missing data.

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APPENDICES
Appendix 1. Ovid MEDLINE search strategy

- 1 Neurofeedback/
- 2 Biofeedback, Psychology/
- 3 (neurofeedback or neuro-feedback).tw,kf.
- 4 (biofeedback or bio-feedback).tw,kf.
- 5 or/1-4
- 6 Electroencephalography/
- 7 (electroencephalograph\$ or electro-encephalograph\$ or EEG).tw,kf.
- 8 or/6-7
- 9 Feedback/
- 10 (feedback\$ or feed-back\$).tw,kf.
- 11 or/9-10
- 12 8 and 11
- 13 5 or 12
- 14 neurodevelopmental disorders/ (651)
- 15 ((neurodevelopment\$ or neuro-development\$) adj3 (disorder\$ or disab\$ or challeng\$ or condition\$)).tw,kf.
- 16 child development disorders/
- 17 developmental disabilities/
- 18 (developmental\$ adj3 (disab\$ or disorder\$ or impair\$)).tw,kf.
- 19 exp child development disorders, pervasive/
- 20 autis\$.tw,kf.
- 21 asperger\$.tw,kf.
- 22 exp "Attention Deficit and Disruptive Behavior Disorders"/
- 23 attention deficit\$.tw,kf.
- 24 (hyperactiv\$ or hyper-activ\$).tw,kf.
- 25 impulsiv\$.tw,kf.
- 26 (ADHD or ADDH or "AD/HD" or TDAH).tw,kf.
- 27 intellectual disability/
- 28 (intellectual\$ adj3 (disab\$ or disorder\$ or impair\$)).tw,kf.
- 29 (mental\$ adj3 (disab\$ or impair\$ or handicap\$ or retard\$)).tw,kf.
- 30 learning disab\$.tw,kf.
- 31 Down Syndrome/
- 32 Down\$ syndrome\$.tw,kf.
- 33 Fetal Alcohol Spectrum Disorders/
- 34 F?etal Alcohol.tw,kf.
- 35 Fragile X Syndrome/
- 36 (FRAXE or FRAXA).tw,kf.
- 37 "Fragile X".tw,kf.
- 38 or/14-37
- 39 13 and 38

CONTRIBUTIONS OF AUTHORS

JKL (guarantor) drafted the protocol with feedback from all other review authors.

DECLARATIONS OF INTEREST

Jasmin K Landes (JKL) reports that this review forms part of her PhD thesis. Co-authors Dr Corinne Reid and Professor Michael Anderson are JKLs PhD supervisors. This supervisor-student relationship is a pre-existing arrangement to this Cochrane Review.

Corinne L Reid - none known.

Martijn Arns (MAR) reports research grants and options from Brain Resource (Sydney, Australia); owns stock in and serves as Chief Scientific Officer of the NeuroCare Group (Munich, Germany) and Director and Researcher of Research Institute Brainclinics (Nijmegen, Netherlands); is a consultant on a National Institute of Mental Health, US-funded iCAN study (CNG 2013); and is a co-inventor on four patent applications (A61B5/0402; US2007/0299323, A1; WO2010/139361 A1; one pending) related to EEG, neuromodulation, and psychophysiology (not related to neurofeedback). MAR declares no ownership or financial gains for these patents - just authorship.

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