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 occurence 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 (Daunhauer 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.

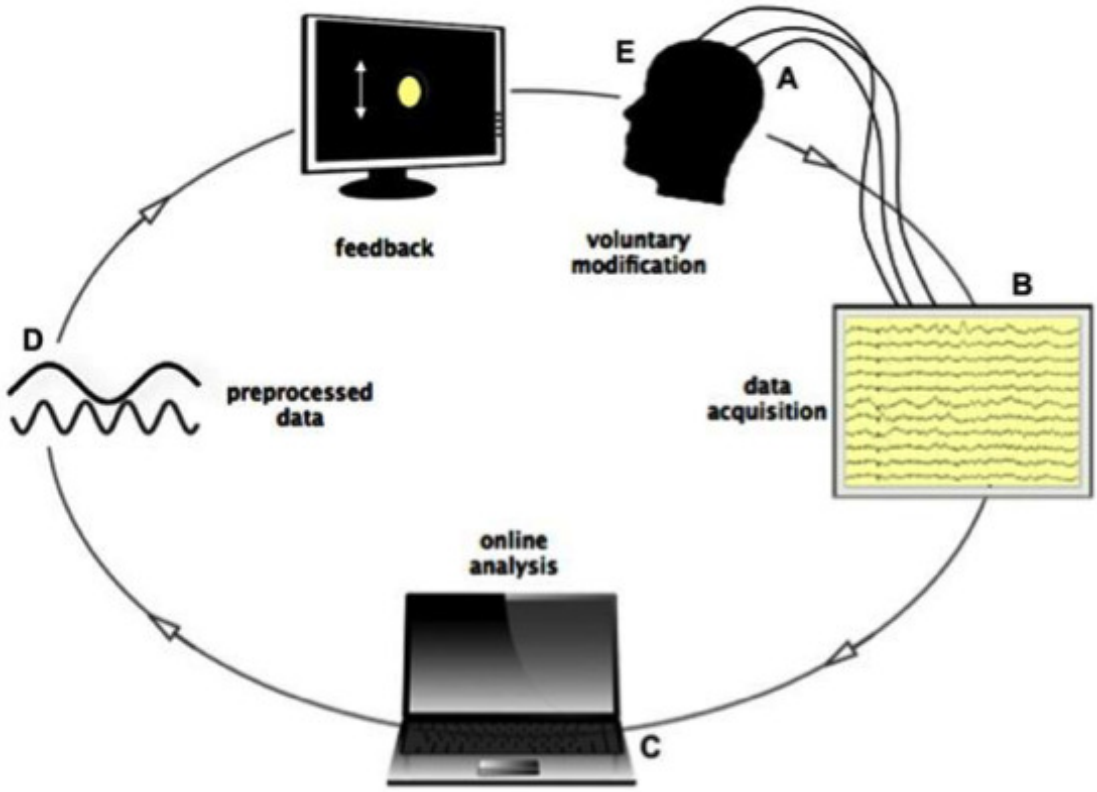
Executive functions develop rapidly during childhood (from about the age of six years) and adolescence (e.g. Anderson 2002). Not only does executive control typically improve during this time, but the nature of these functions is more fully differentiated (Brydges 2012; Brydges 2014a; Shing 2010). The 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).

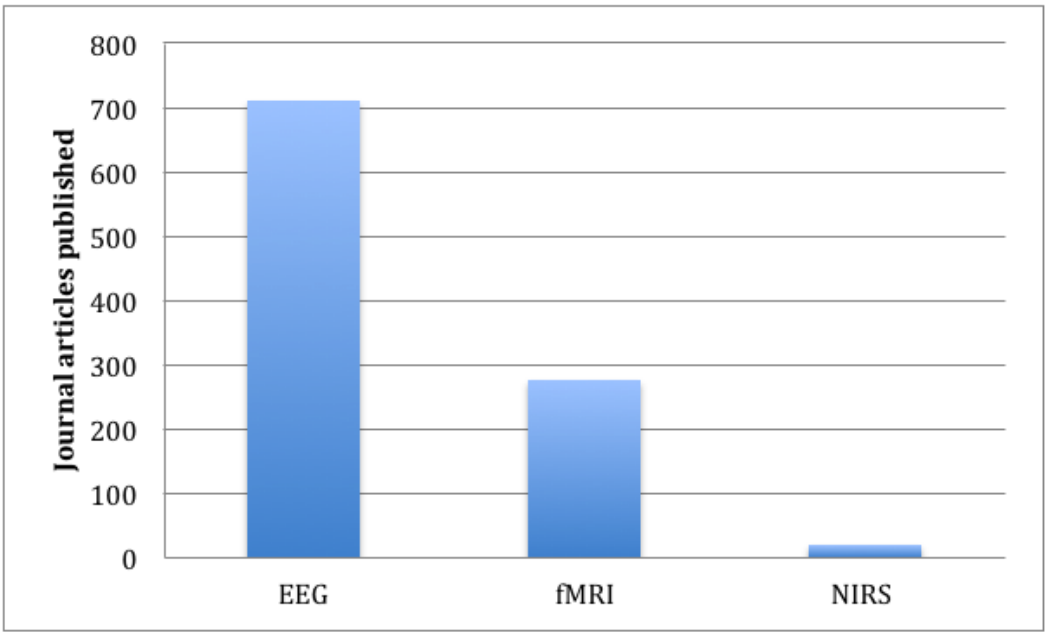


EEG neurofeedback is not dependent on complex verbal instructions; therefore, this brain-training intervention can be effectively implemented cross-culturally and in groups with language and communication impairment. It is designed to be embedded in a game format, which offers face validity as a treatment 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 micro-analysis 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.FootnotesEEG: 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 neu-

ral 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 rigourous 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. Pittburgh 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, pairwise 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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Additional references

Ageranioti-Bélanger 2012

Ageranioti-Bélanger S, Brunet S, D'Anjou G, Tellier G, Boivin J, Gauthier M. Behaviour disorders in children with an intellectual disability. *Paediatrics & Child Health* 2012; **17**(2):84–8. [PUBMED: PMC3299352]

AIHW 2011

Australian Institute of Health and Welfare. Australian Burden of Disease Study: impact and cause of illness and death in Australia 2011. www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=60129555476 (accessed 27 July 2016).

Anderson 2002

Anderson P. Assessment and development of executive function (EF) during childhood. *Child Neuropsychology* 2002;**8**(2):71–82. [DOI: 10.1076/chin.8.2.71.8724; PUBMED: 12638061

APA 2000

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)*. 4th Edition. Washington (DC): American Psychiatric Association, 2000.

APA 2013

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)*. 5th Edition. Washington (DC): American Psychiatric Association, 2013.

Arnold 2013

Arnold LE, Lofthouse N, Hersch S, Pan X, Hurt E, Bates B, et al. EEG neurofeedback for ADHD: double-blind sham-controlled randomized pilot feasibility trial. *Journal of Attention Disorders* 2013;**17**(5):410–9. [DOI: 10.1177/1087054712446173; PMC3939717; PUBMED: 22617866

Bagdasaryan 2013

Bagdasaryan J, Le Van Quyen M. Experiencing your brain: neurofeedback as a new bridge between neuroscience and phenomenology. *Frontiers in Human Neuroscience* 2013;**7**(680):1–10. [DOI: 10.3389/fnhum.2013.00680; PMC3807564

Bai 2014

Bai O, Huang D, Fei DY, Kunz R. Effect of real-time cortical feedback in motor imagery-based mental practice training. *NeuroRehabilitation* 2014;**34**(2):355–63. [DOI: 10.3233/NRE-131039; PUBMED: 24401829

Banaschewski 2006

Banaschewski T, Coghill D, Santosh P, Zuddas A, Asherson P, Buitelaar J, et al. Long-acting medications for the hyperkinetic disorders. A systematic review and European treatment guideline. *European Child & Adolescent Psychiatry* 2006;**15**(8):476–95. [DOI: 10.1007/s00787-006-0549-0; PUBMED: 16680409

Bari 2013

Bari A, Robbins TW. Inhibition and impulsivity: behavioral and neural basis of response control. *Progress in Neurobiology* 2013;**108**:44–79. [DOI: 10.1016/j.pneurobio.2013.06.005; PUBMED: 23856628

Barkley 1997

Barkley RA. Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin* 1997;**121**(1):65–94. [PUBMED: 9000892]

Beatty 1974

Beatty J, Greenberg A, Deibler WP, O'Hanlon JF. Operant control of occipital theta rhythm affects performance in a radar monitoring task. *Science* 1974;**183**(4127):871–3. [DOI: 10.1126/science.183.4127.871; 4810845

Bishop 2007

Bishop DVM. Curing dyslexia and attention-deficit hyperactivity disorder by training motor co-ordination: miracle or myth?. *Journal of Paediatrics and Child Health* 2007;**43**(10):653–5. [DOI: 10.1111/j.1440-1754.2007.01225.x; PMC2835859; UKMS28929

Bishop 2008

Bishop D, Rutter M. Chapter 3. Neurodevelopmental disorders: conceptual issues. In: Rutter D, Bishop DVM, Pine DS, Scott S, Stevenson J, Taylor E, et al. editor(s). *Rutter's Child and Adolescent Psychiatry*. 5th Edition. Oxford (UK): Blackwell Publishing Ltd, 2008:32–41. [DOI: 10.1002/9781444300895.ch3

Bishop 2010

Bishop DVM. Which neurodevelopmental disorders get researched and why?. *PLoS One* 2010;**5**(11):e15112. [DOI: 10.1371/journal.pone.0015112; PMC2994844; PUBMED: 21152085

Botvinick 2001

Botvinick MM, Braver TS, Barch DM, Carter CS, Cohen JD. Conflict monitoring and cognitive control. *Psychological Review* 2001;**108**(3):624–52. [PUBMED: 11488380]

Bower 2007

Bower C, Rudy E, Callaghan A, Cosgrove P, Quick J, Nassar N. *Report of the Birth Defects Registry of Western Australia 1980-2006*. Perth (Western AU): Birth Defects Registry, 2007.

Brown 2010

Brown L. *The Prevalence of Fragile X-Associated Disorders in Australia*. Canberra (AU): NATSEM, University of Canberra, 2010.

Brydges 2012

Brydges CR, Reid CL, Fox AM, Anderson M. A unitary executive function predicts intelligence in children. *Intelligence* 2012;**40**(5):458–69. [DOI: dx.doi.org/10.1016/j.intell.2012.05.006

Brydges 2014a

Brydges CR, Fox AM, Reid CL, Anderson M. The differentiation of executive functions in middle and late childhood: a longitudinal latent-variable analysis. *Intelligence* 2014;**47**:34–43. [DOI: dx.doi.org/10.1016/j.intell.2014.08.010

Brydges 2014b

Brydges CR, Fox AM, Reid CL, Anderson M. Predictive validity of the N2 and P3 ERP components to executive functioning in children: a latent-variable analysis. *Frontiers in Human Neuroscience* 2014;**8**:1–10. [DOI: 10.3389/fnhum.2014.00080; PMC3929846

CNG 2013

Kerson C, Collaborative Neurofeedback Group. A proposed multisite double-blind randomized clinical trial of neurofeedback for ADHD: need, rationale, and strategy. *Journal of Attention Disorders* 2013;**17**(5):420–36. [DOI: 10.1177/1087054713482580; PUBMED: 23590978

Coffee 2009

Coffee B, Keith K, Albizua I, Malone T, Mowrey J, Sherman SL, et al. Incidence of fragile X syndrome by newborn screen for methylated FMR1 DNA. *American Journal of Human Genetics* 2009;**85**(4):503–14. [DOI: 10.1016/j.ajhg.2009.09.007; PMC2756550; PUBMED: 19804849

Coghill 2015

Coghill D. Commentary: we've only just begun: unravelling the underlying genetics of neurodevelopmental disorders - a commentary on Kiser et al (2015). *Journal of Child Psychology and Psychiatry* 2015;**56**(3):296–8. [DOI: 10.1111/jcpp.12399; PUBMED: 25714739

Conners 2011

Conners CK, Pitkanen J, Rzepa SR. Conners Comprehensive Behavior Rating Scale. In: Kreutzer JS, DeLuca J, Caplan B editor(s). *Encyclopedia of Clinical Neuropsychology*. 3rd Edition. New York (NY): Springer, 2011:678–80. [link.springer.com/referenceworkentry/10.1007/978–0–387–79948–3˙1536]

Crawford 2002

Crawford DC, Meadows KL, Newman JL, Taft LF, Scott E, Leslie M, et al. Prevalence of the fragile X syndrome in African-Americans. *American Journal of Medical Genetics* 2002;**110**(3):226–33. [DOI: 10.1002/ajmg.10427; PUBMED: 12116230

Cuthbert 2010

Cuthbert BN, Insel TR. Toward new approaches to psychotic disorders: the NIMH research domain criteria project. *Schizophrenia Bulletin* 2010;**36**(6):1061–2. [DOI: 10.1093/schbul/sbq108; PMC2963043

Daunhauer 2014

Daunhauer LA, Fidler DJ, Hahn L, Will E, Lee NR, Hepburn S. Profiles of everyday executive functioning in young children with Down syndrome. *American Journal on Intellectual and Developmental Disabilities* 2014;**119**(4):303–18. [DOI: 10.1352/1944-7558-119.4.303; PMC4512669; PUBMED: 25007296

Davidson 2006

Davidson MC, Amso D, Anderson LC, Diamond A. Development of cognitive control and executive functions from 4 to 13 years: evidence from manipulations of memory, inhibition, and task switching. *Neuropsychologia* 2006;**44**(11):2037–78. [DOI: 10.1016/j.neuropsychologia.2006.02.006

Davis 2009

Davis MC, Luecken L, Lemery-Chalfant K. Resilience in common life: introduction to the special issue. *Journal of Personality* 2009;**77**(6):1637–44. [DOI: 10.1111/j.1467-6494.2009.00595.x; PUBMED: 19796066

Deeks 2011

Deeks JJ, Higgins JP, Altman DG, editor(s). Chapter 9: Analysing data and undertaking meta-analyses. In: Higgins JP, Greens S, editor(s). Cochrane Handbook for Systematic Reviews of Interventions Version 5.1.0 (updated in March 2011). The Cochrane Collaboration, 2011. Available from handbook.cochrane.org.

Deiber 2007

Deiber MP, Missonnier P, Bertrand O, Gold G, Fazio-Costa L, Ibañez V, et al. Distinction between perceptual and attentional processing in working memory tasks: a study of phase-locked and induced oscillatory brain dynamics. *Journal of Cognitive Neuroscience* 2007;**19**(1):158–72. [DOI: 10.1162/jocn.2007.19.1.158; PUBMED: 17214572

Donchin 1988

Donchin E, Coles MGH. Is the P300 component a manifestation of context updating. *Behavioural and Brain Sciences* 1988;**11**(3):357–427. [DOI: 10.1017/S0140525X00058027

Doyle 2015

Doyle AE. Commentary: insights from across diagnostic boundaries: ADHD in RDoC era - a commentary on Scerif and Baker (2015). *Journal of Child Psychology and Psychiatry* 2015;**56**(3):274–7. [DOI: 10.1111/jcpp.12401; PUBMED: 25714738

Egger 1997

Egger M, Davey Smith G, Schneider M, Minder C. Bias in meta-analysis detected by a simple, graphical test. *BMJ* 1997;**315**(7109):629–34. [DOI: dx.doi.org/10.1136/bmj.315.7109.629; PMC2127453; PUBMED: 9310563

Egner 2001

Egner T, Gruzelier JH. Learned self-regulation of EEG frequency components affects attention and event-related brain potentials in humans. *Neuroreport* 2001;**12**(18):4155–9. [PUBMED: 11742256]

Egner 2002

Egner T, Strawson E, Gruzelier JH. EEG signature and phenomenology of alpha/theta neurofeedback training versus mock feedback. *Applied Psychophysiology and Biofeedback* 2002;**27**(4):261–70. [PUBMED: 12557453]

Engelbregt 2016

Engelbregt HJ, Keeser D, van Eijk L, Suiker EM, Eichhorn D, Karch S, et al. Short and long-term effects on sham-controlled prefrontal EEG-neurofeedback training in healthy subjects. *Clinical Neurophysiology* 2016;**127**(4):1931–7. [DOI: 10.1016/j.clinph.2016.01.004; PUBMED: 26971473

Fabiano 2009

Fabiano GA, Pelham WE Jr, Coles EK, Gnagy EM, Chronis-Tuscano A, O'Connor BC. A meta-analysis of behavioural treatments for attention-deficit/hyperactivity

disorder. *Clinical Psychology Review* 2009;**29**(2):129–40. [DOI: 10.1016/j.cpr.2008.11.001; PUBMED: 19131150

Faraone 2010

Faraone SV, Buitelaar J. Comparing the efficacy of stimulants for ADHD in children and adolescents using meta-analysis. *European Child & Adolescent Psychiatry* 2010;**19**(4):353–64. [DOI: 10.1007/s00787-009-0054-3; PUBMED: 19763664

Ferster 1957

Ferster CB, Skinner BF. *Schedules of Reinforcement*. New York (NY): Appleton-Century-Crofts, 1957.

Gaffan 1995

Gaffan EA, Tsaousis I, Kemp-Wheeler SM. Researcher allegiance and meta-analysis: the case of cognitive therapy for depression. *Journal of Consulting and Clinical Psychology* 1995;**63**(6):966–80. [DOI: 10.1037/0022-006X.63.6.966; PUBMED: 8543719

Ganguly 2013

Ganguly K, Poo MM. Activity-dependent neural plasticity from bench to bedside. *Neuron* 2013;**80**(3):729–41. [DOI: 10.1016/j.neuron.2013.10.028; PUBMED: 24183023

Garavan 1999

Garavan H, Ross TJ, Stein EA. Right hemispheric dominance of inhibitory control: an event-related functional MRI study. *Proceedings of the National Academy of Sciences of the United States of America* 1999;**96**(14): 8301–6. [PMC22229; PUBMED: 10393989]

Garvey 2016

Garvey M, Avenevoli S, Anderson K. The National Institute of Mental Health research domain criteria and clinical research in child and adolescent psychiatry. *Journal of the American Academy of Child & Adolescent Psychiatry* 2016;**55**(2):93–8. [DOI: 10.1016/j.jaac.2015.11.002; PMC4724376; PUBMED: 26802775

Gevensleben 2012

Gevenleben H, Holl B, Albrecht B, Schlamp D, Kratz O, Struder P, et al. Is neurofeedback an efficacious treatment for ADHD? A randomised controlled clinical trial. *Journal of Child Psychology and Psychiatry, and Allied Disciplines* 2009; **50**(7):780–9. [DOI: 10.1111/j.1469-7610.2008.02033.x; PUBMED: 19207632

Glahn 2016

Glahn DC, Knowles EEM, Pearlson GD. Genetics of cognitive control: implications for Nimh's research domain criteria initiative. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* 2016;**171**(1):111–20. [DOI: 10.1002/ajmg.b.32345

Golden 2002

Golden CJ, Freshwater SM. *Stroop Color and Word Test: Children's Version*. Wood Dale (IL): Stoelting Co, 2002.

Goldstein 2010

Goldstein S, Reynolds CR, editors. *Handbook of Neurodevelopmental and Genetic Disorders in Children*. 2nd Edition. New York (NY): The Guilford Press, 2010.

GRADEpro GDT 2015 [Computer program]

McMaster University (developed by Evidence Prime). GRADEpro GDT. Version accessed 10 May 2017. Hamilton (ON): McMaster University (developed by Evidence Prime), 2015.

Graham 2011

Graham J, Banaschewski T, Buitelaar J, Coghill D, Danckaerts M, Dittman RW, et al. European guidelines on managing adverse effects of medication for ADHD. *European Child & Adolescent Psychiatry* 2011;**20**(1):17–37. [DOI: 10.1007/s00787-010-0140-6; PMC3012210; PUBMED: 21042924

Guyatt 2006

Guyatt G, Gutterman D, Baumann MH, Addrizzo-Harris D, Hylek EM, Phillips B, et al. Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American College of Chest Physicians task force. *Chest* 2006;**129**(1):174–81. [DOI: 10.1378/chest.129.1.174; PUBMED: 16424429

Happé 2006

Happé F, Booth R, Charlton R, Hughes C. Executive function deficits in autism spectrum disorders and attention-deficit/hyperactivity disorder: examining profiles across domains and ages. *Brain and Cognition* 2006;**61**(1): 25–39. [DOI: 10.1016/j.bandc.2006.03.004; PUBMED: 16682102

Happé 2014

Happé F, Frith U. Annual research review: towards a developmental neuroscience of atypical social cognition. *Journal of Child Psychology and Psychiatry* 2014;**55**(6): 553–77. [DOI: 10.1111/jcpp.12162

Hartman 2010

Hartman E, Houwen S, Scherder E, Visscher C. On the relationship between motor performance and executive functioning in children with intellectual disabilities. *Journal of Intellectual Disability Research* 2010;**54**(5):468–77. [DOI: 10.1111/j.1365-2788.2010.01284.x; PUBMED: 20537052

Heim 2012

Heim C, Binder EB. Current research trends in early life stress and depression: review of human studies on sensitive periods, gene-environment interactions and epigenetics. *Experimental Neurology* 2012;**233**(1):102–11. [DOI: 10.1016/j.expneurol.2011.10.032; PUBMED: 22101006

Heinrich 2007

Heinrich H, Gevensleben H, Strehl U. Annotation: neurofeedback - train your brain to train behaviour. *Journal of Child Psychology and Psychiatry, and Allied Disciplines* 2007; **48**(1):3–16. [DOI: 10.1111/j.1469-7610.2006.01665.x; PUBMED: 17244266

Higgins 2011

Higgins JPT, Deeks JJ, Altman DG, editor(s). Chapter 16: Special topics in statistics. In: Higgins JPT, Green S (editors). Cochrane Handbook for Systematic Reviews of Interventions. Version 5.1.0 (updated March 2011).

The Cochrane Collaboration, 2011. Available from handbook.cochrane.org.

Higgins 2017
Higgins JPT, Altman DG, Sterne JAC, editor(s). Chapter 8: Assessing risk of bias in included studies. In: Higgins JPT, Churchill R, Chandler J, Cumpston MS, editor (s). Cochrane Handbook for Systematic Reviews of Interventions Version 5.2.0 (updated June 2017). Cochrane, 2017. Available from www.training.cochrane.org/handbook.

Hooper 2008
Hooper SR, Hatton D, Sideris J, Sullivan K, Hammer J, Schaaf J, et al. Executive functions in young males with fragile X syndrome in comparison to mental age-matched controls: baseline findings from a longitudinal study. *Neuropsychology* 2008;**22**(1):36–47. [DOI: 10.1037/0894-4105.22.1.36; PUBMED: 18211154

Hsia 2014
Hsia Y, Wong AYS, Murphy DG, Simonoff E, Buitelaar JK, Wong IC. Psychopharmacological prescriptions for people with autism spectrum disorder (ASD): a multinational study. *Psychopharmacology* 2014;**231**(6):999–1009. [DOI: 10.1007/s00213-013-3263-x; PUBMED: 24005531

Hughes 2002
Hughes C, Graham A. Measuring executive functions in childhood: problems and solutions?. *Child and Adolescent Mental Health* 2002;**7**(3):131–42. [DOI: 10.1111/1475-3588.00024

Huster 2014
Huster RJ, Mokom ZN, Enriquez-Geppert S, Herrmann CS. Brain-computer interfaces for EEG neurofeedback: peculiarities and solutions. *International Journal of Psychophysiology* 2014;**91**(1):36–45. [DOI: 10.1016/j.ijpsycho.2013.08.011; PUBMED: 24012908

Insel 2010a
Insel TR, Wang PS. Rethinking mental illness. *JAMA* 2010;**303**(19):1970–1. [DOI: 10.1001/jama.2010.555; PUBMED: 20483974

Insel 2010b
Insel T, Cuthbert B, Garvey M, Heinssen R, Pine DS, Quinn K, et al. Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. *American Journal of Psychiatry* 2010;**167**(7):748–51. [DOI: 10.1176/appi.ajp.2010.09091379; PUBMED: 20595427

Jensen 2007
Jensen PS, Arnold LE, Swanson JM, Vitiello B, Abikoff HB, Greenhill LL, et al. 3-year follow-up of the NIMH MTA study. *Journal of the American Academy of Child & Adolescent Psychiatry* 2007;**46**(8):989–1002. [DOI: 10.1097/CHI.0b013e3180686d48; PUBMED: 17667478

Kane 2003
Kane MJ, Engle RW. Working-memory capacity and the control of attention: the contributions of goal neglect, response competition, and task set to Stroop interference. *Journal of Experimental Psychology* 2003;**132**(1):47–70. [PUBMED: 12656297]

Klimesch 2006
Klimesch W, Hanslmayr S, Sauseng P, Gruber W, Brozinsky CJ, Kroll NE, et al. Oscillatory EEG correlates of episodic trace decay. *Cerebral Cortex* 2006;**16**(2):280–90. [DOI: 10.1093/cercor/bhi107; PUBMED: 15888605

Knoblauch 2012
Knoblauch A, Hauser F, Gewaltig MO, Körner E, Palm G. Does spike-timing-dependent synaptic plasticity couple or decouple neurons firing in synchrony?. *Frontiers in Computational Neuroscience* 2012;**6**(55):1–27. [DOI: 10.3389/fncom.2012.00055; PMC3424530; PUBMED: 22936909

Knox 2012
Knox A, Schneider A, Abucayan F, Hervey C, Tran C, Hessl D, et al. Feasibility, reliability, and clinical validity of the Test of Attentional Performance for Children (KiTAP) in fragile X syndrome (FXS). *Journal of Neurodevelopmental Disorders* 2012;**4**(1):2. [DOI: 10.1186/1866-1955-4-2; PMC3374289; PUBMED: 22958782

Knudsen 2004
Knudsen EI. Sensitive periods in the development of the brain and behavior. *Journal of Cognitive Neuroscience* 2004;**16**(8):1412–25. [DOI: 10.1162/0898929042304796; PUBMED: 15509387

Kouijzer 2009
Kouijzer MEJ, De Moor JMH, Gerrits BJL, Congedo M, van Schie HT. Neurofeedback improves executive functioning in children with autism spectrum disorders. *Research in Autism Spectrum Disorders* 2009;**3**(1):145–62. [DOI: dx.doi.org/10.1016/j.rasd.2008.05.001

Lansbergen 2011
Lansbergen MM, Van Dongen-Boomsma M, Buitelaar JK, Slaats-Willemse D. ADHD and EEG-neurofeedback: a double-blind randomized placebo-controlled feasibility study. *Journal of Neural Transmission* 2011;**118**(2):275–84. [DOI: 10.1007/s00702-010-0524-2; PMC3051071; PUBMED: 21165661

Lawrence 2015
Lawrence D, Johnson S, Hafekost J, De Haan KB, Sawyer M, Ainley J, et al. *The Mental Health of Children and Adolescents. Report on the Second Australian Child and Adolescent Survey of Mental Health and Wellbeing*. Canberra (AU): Department of Health, 2015. [www.health.gov.au/internet/main/publishing.nsf/Content/9DA8CA21306FE6EDCA257E2700016945/$File/child2.pdf]

Leckman 2010
Leckman JF, Yazgan MY. Editorial: developmental transitions to psychopathology: from genomics and epigenomics to social policy. *Journal of Child Psychology and Psychiatry* 2010;**51**(4):333–40. [DOI: 10.1111/j.1469-7610.2010.02226.x; PUBMED: 20180880

Leykin 2009
Leykin Y, DeRubeis RJ. Allegiance in psychotherapy outcome research: separating association from bias. *Clinical Psychology: Science and Practice* 2009;**16**(1):54–65. [DOI: 10.1111/j.1468-2850.2009.01143.x

Liberati 2009
Liberati A, Altman DG, Tetzlaff J, Mulrow C, Gøtzsche PC, Ioannidis JP, et al. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: explanations and elaboration. *Annals of Internal Medicine* 2009;**151**(4):W–65-W-94. [DOI: 10.7326/0003-4819-151-4-200908180-00136

Linden 1996
Linden M, Habib T, Radojevic V. A controlled study of the effects of EEG biofeedback on cognition and behavior of children with attention deficit disorder and learning disabilities. *Biofeedback and Self-regulation* 1996;**21**(1): 35–49. [PUBMED: 8833315]

Lubar 1995a
Lubar JF, Swartwood MO, Swartwood JN, O'Donnell PH. Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in TOVA scores, behavioral ratings, and WISC-R performance. *Biofeedback and Self-regulation* 1995;**20**(1): 83–99. [PUBMED: 7786929]

Lubar 1995b
Lubar J, Swartwood MO, Swartwood JN, Timmermann DL. Quantitative EEG and auditory event-related potentials in the evaluation of attention-deficit/ hyperactivity disorder: effects of methylphenidate and implications for neurofeedback training. *Journal of Psychoeducational Assessment* 1995;**ADHD Special Edition**: 143–60. [citeseerx.ist.psu.edu/viewdoc/download?doi= 10.1.1.501.6072&rep=rep1&type=pdf]

Luck 2014
Luck SJ. *An Introduction to the Event-Related Potential Technique*. 2nd Edition. Cambridge (MA): MIT Press, 2014.

Marx 2015
Marx AM, Ehlis AC, Furdea A, Holtmann M, Banaschewski T, Brandeis D, et al. Near-infrared spectroscopy (NIRS) neurofeedback as a treatment for children with attention deficit hyperactivity disorder (ADHD) - a pilot study. *Frontiers in Human Neuroscience* 2015;**8**(1038):1–13. [DOI: 10.3389/fnhum.2014.01038; PMC4285751; PUBMED: 25610390

Masten 2005
Masten AS, Roisman GI, Long JD, Burt KB, Obradovi

ć J, Riley JR, et al. Developmental cascades: linking academic achievement and externalizing and internalizing symptoms over 20 years. *Developmental Psychology* 2005; **41**(5):733–46. [DOI: 10.1037/0012-1649.41.5.733; PUBMED: 16173871

Maulik 2011
Maulik PK, Mascarenhas MN, Mathers CD, Dua T, Saxena S. Prevalence of intellectual disability: a meta-analysis of population-based studies. *Research in Developmental Disabilities* 2011;**32**(2):419–36. [DOI: 10.1016/ j.ridd.2010.12.018; PUBMED: 21236634

Miyake 2000
Miyake A, Friedman NP, Emerson MJ, Witzki AH, Howerter A, Wagner TD. The unity and diversity of executive functions and their contributions to complex "Frontal Lobes" tasks: a latent variable analysis. *Cognitive Psychology* 2000;**41**(1):49–100. [DOI: 10.1006/ cogp.1999.0734; PUBMED: 10945922

Moskowitz 2011
Moskowitz LJ, Carr EG, Durand VM. Behavioral intervention for problem behavior in children with fragile X syndrome. *American Journal on Intellectual and Developmental Disabilities* 2011;**116**(6):457–78. [DOI: 10.1352/1944-7558-116.6.457; PUBMED: 22126659

Murawski 2015
Murawski NJ, Moore EM, Thomas JD, Riley EP. Advances in diagnosis and treatment of fetal alcohol spectrum disorders: from animal models to human studies. *Alcohol Research* 2015;**37**(1):97–108. [PMC4476607; PUBMED: 26259091]

Murray 2008
Murray DW, Arnold LE, Swanson J, Wells K, Burns K, Jensen P, et al. A clinical review of outcomes of the multimodal treatment study of children with attention-deficit/hyperactivity disorder (MTA). *Current Psychiatry Reports* 2008;**10**(5):424–31. [DOI: 10.1007/ s11920-008-0068-4; PMC5524214; PUBMED: 18803917

Musall 2014
Musall S, von Pföstl V, Rauch A, Logothetis NK, Whittingstall K. Effects of neural synchrony on surface EEG. *Cerebral Cortex* 2014;**24**(4):1045–53. [DOI: 10.1093/cercor/bhs389; PUBMED: 23236202

Narzisi 2014
Narzisi A, Constanza C, Umberto B, Filippo M. Non-pharmacological treatments in autism spectrum disorders: an overview on early interventions for pre-schoolers. *Current Clinical Pharmacology* 2014;**9**(1):17–26. [PUBMED: 24050743]

Newport 2001
Newport EL, Bavelier D, Neville HJ. Critical thinking about critical periods: perspectives on a critical period for language acquisition. In: Dupoux E editor(s). *Language, Brain and Cognitive Development. Essays in Honour of Jacques Mehler*. Cambridge (MA): MIT Press, 2001: 481–502. [www.bcs.rochester.edu/people/newport/pdf/ Newport˙Bav˙Nev01.pdf]

Nunez 2000
Nunez PL. Toward a quantitative description of larger-scale neocortical dynamic function and EEG. *Behavioral and Brain Sciences* 2000;**23**(3):371–98. [PUBMED: 11301576]

Oosterlaan 2005
Oosterlaan J, Scheres A, Sergeant JA. Which executive functioning deficits are associated with AD/HD, ODD/ CD and comorbid AD/HD+ODD/CD?. *Journal of*

Abnormal Child Psychology 2005;**33**(1):69–85. [PUBMED: 15759592]

Ozonoff 1998
Ozonoff S, Cathcart K. Effectiveness of a home program intervention for young children with autism. *Journal of Autism and Developmental Disorders* 1998;**28**(1):25–32. [PUBMED: 9546299]

Padmanabhan 2015
Padmanabhan A, Garver K, O'Hearn K, Nawarawong N, Liu R, Minshew N, et al. Developmental changes in brain function underlying inhibitory control in autism spectrum disorders. *Autism Research* 2015;**8**(2):123–35. [DOI: 10.1002/aur.1398; PMC4944206 ; PUBMED: 25382787

Pelham 1999
Pelham WE, Gnagy EM, Chronis AM, Burrows-MacLean L, Fabiano GA, Onyango AN, et al. A comparison of morning-only and morning/late afternoon Adderall to morning-only, twice-daily, and three times-daily methylphenidate in children with attention-deficit/hyperactivity disorder. *Pediatrics* 1999;**104**(6):1300–11. [PUBMED: 10585981]

Pennington 1996
Pennington BF, Ozonoff S. Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry* 1996;**37**(1):51–87. [PUBMED: 8655658]

Perani 2003
Perani D, Abutalebi J, Paulesu E, Brambati S, Scifo P, Cappa SF, et al. The role of age of acquisition and language usage in early, high-proficient bilinguals: an fMRI study during verbal fluency. *Human Brain Mapping* 2003;**19**(3):170–82. [DOI: 10.1002/hbm.10110; PUBMED: 12811733

Perreau-Linck 2010
Perreau-Linck E, Lessard N, Lévesque J, Beauregard M. Effects of neurofeedback training on inhibitory capacities in ADHD children: a single-blind, randomized, placebo-controlled study. *Journal of Neurotherapy* 2010;**14**(3): 229–42. [DOI: 10.1080/10874208.2010.501514

Polich 2007
Polich J. Updating P300: an integrative theory of P3a and P3b. *Clinical Neurophysiology* 2007;**118**(10):2128–48. [DOI: 10.1016/j.clinph.2007.04.019; PMC2715154; PUBMED: 17573239

Reichow 2011
Reichow B. Overview of meta-analyses on early intensive behavioral intervention for young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders* 2012;**42**(4):512–20. [DOI: 10.1007/s10803-011-1218-9; PUBMED: 21404083

Reid 2015
Reid N, Dawe S, Shelton D, Harnett P, Warner J, Armstrong E, et al. Systematic review of fetal alcohol spectrum disorder interventions across the life span. *Alcoholism, Clinical and Experimental Research* 2015;**39**(12):2283–95. [DOI: 10.1111/acer.12903; PUBMED: 26578111

Review Manager 2014 [Computer program]
Nordic Cochrane Centre, The Cochrane Collaboration. Review Manager 5 (RevMan 5). Version 5.3. Copenhagen: Nordic Cochrane Centre, The Cochrane Collaboration, 2014.

Roozen 2016
Roozen S, Peters GJ, Kok G, Townend D, Nijhuis J, Curfs L. Worldwide prevalence of fetal alcohol spectrum disorders: a systematic literature review including meta-analysis. *Alcoholism, Clinical and Experimental Research* 2016;**40**(1): 18–32. [DOI: 10.1111/acer.12939; PUBMED: 26727519

Ros 2010
Ros T, Munneke MA, Ruge D, Gruzelier JH, Rothwell JC. Endogenous control of waking brain rhythms induces neuroplasticity in humans. *European Journal of Neuroscience* 2010;**31**(4):770–8. [DOI: 10.1111/j.1460-9568.2010.07100.x; PUBMED: 20384819

Ros 2014
Ros T, Baars BJ, Lanius RA, Vuilleumier P. Tuning pathological brain oscillations with neurofeedback: a systems neuroscience framework. *Frontiers in Human Neuroscience* 2014;**8**:1008. [DOI: 10.3389/fnhum.2014.01008; PMC4270171; PUBMED: 25566028

Rutter 2010
Rutter M, Bishop D, Pine D, Scott S, Stevenson J, Taylor E, et al. *Rutter's Child and Adolescent Psychiatry*. 5th Edition. Chicester (UK): Wiley-Blackwell, 2010.

Sauseng 2008
Sauseng P, Klimesch W. What does phase information of oscillatory brain activity tell us about cognitive processes?. *Neuroscience & Biobehavioral Reviews* 2008;**32**(5):1001–13. [DOI: 10.1016/j.neubiorev.2008.03.014; PUBMED: 18499256

Sauseng 2010
Sauseng P, Griesmayr B, Freunberger R, Klimesch W. Control mechanisms in working memory: a possible function of EEG theta oscillations. *Neuroscience & Biobehavioral Reviews* 2010;**34**(7):1015–22. [DOI: 10.1016/j.neubiorev.2009.12.006; PUBMED: 20006645

Scharnowski 2015
Scharnowski F, Veit R, Zopf R, Studer P, Bock S, Diedrichsen J, et al. Manipulating motor performance and memory through real-time fMRI neurofeedback. *Biological Psychology* 2015;**108**(88):85–97. [DOI: 10.1016/j.biopsycho.2015.03.009; PMC4433098; PUBMED: 25796342

Scheifes 2013
Scheifes A, de Jong D, Stolker JJ, Nijman HL, Egberts TC, Heerdink ER. Prevalence and characteristics of psychotropic drug use in institutionalized children and adolescents with mild intellectual disability. *Research in Developmental Disabilities* 2013;**34**(10):3159–67. [DOI: 10.1016/j.ridd.2013.06.009; PUBMED: 23886758

Schmidt 1996
Schmidt M. *Rey Auditory and Verbal Learning Test: A Handbook*. Los Angeles (LA): Western Psychological Services, 1996.

Schünemann 2006
Schünemann HJ, Jaeschke R, Cook DJ, Bria WF, El-Solh AA, Ernst A, et al. An official ATS statement: grading the quality of evidence and strength of recommendations in ATS guidelines and recommendations. *American Journal of Respiratory and Critical Care Medicine* 2006;**174**(5): 605–14. [DOI: 10.1164/rccm.200602-197ST; PUBMED: 16931644

Sergeant 2000
Sergeant J. The cognitive-energetic model: an empirical approach to attention-deficit hyperactivity disorder. *Neuroscience & Biobehavioral Reviews* 2000;**24**(1):7–12. [PUBMED: 10654654]

Shing 2010
Shing YL, Lindenberger U, Diamond A, Li SC, Davidson MC. Memory maintenance and inhibitory control differentiate from early childhood to adolescence. *Developmental Neuropsychology* 2010;**35**(6):679–97. [DOI: 10.1080/87565641.2010.508546; NIHMSID: NIHMS255032; PMC2999360

Siegel 2010
Siegel DJ. *Mindsight: The New Science of Personal Transformation*. New York (NY): Bantam Books, 2010.

Skinner 1958
Skinner BF. Teaching machines. *Science* 1958;**128** (3330):969–77. [apps.fischlerschool.nova.edu/toolbox/instructionalproducts/edd8124/fall11/1958–Skinner–TeachingMachines.pdf]

Sonuga-Barke 2010
Sonuga-Barke EJ, Halperin JM. Developmental phenotypes and causal pathways in attention deficit/hyperactivity disorder: potential targets for early intervention?. *Journal of Child Psychology and Psychiatry* 2010;**51**(4):368–89. [DOI: 10.1111/j.1469-7610.2009.02195.x; PUBMED: 20015192

Sonuga-Barke 2013
Sonuga-Barke EJ, Brandeis D, Cortese S, Daley D, Ferrin M, Holtmann M, et al. Nonpharmacological interventions for ADHD: systematic review and meta-analyses of randomized controlled trials of dietary and psychological treatments. *American Journal of Psychiatry* 2013;**170** (3):275–89. [DOI: 10.1176/appi.ajp.2012.12070991; PUBMED: 23360949

St. Clair-Thompson 2006
St. Clair-Thompson HL, Gathercole SE. Executive functions and achievements in school: shifting, updating, inhibition and working memory. *Quarterly Journal of Experimental Psychology* 2006;**59**(4):745–59. [DOI: 10.1080/17470210500162854; PUBMED: 16707360

Stephenson 2008
Stephenson J, Wheldall K. Miracle takes a little longer: science, commercialisation, cures and the Dore Program. *Australasian Journal of Special Education* 2008;**32**(1): 67–82. [ISSN:1833–6914; hdl.handle.net/1959.14/77782; mq–rm–2007010351; mq:7875]

Vance 2010
Vance DE, Roberson AJ, McGuinness TM, Fazeli PL. How neuroplasticity and cognitive reserve project cognitive functioning. *Journal of Psychosocial Nursing and Mental Health Services* 2010;**48**(4):23–30. [DOI: 10.3928/02793695-20100302-01; PUBMED: 20349891

Visser 2014
Visser SN, Danielson ML, Bitsko RH, Holbrook JR, Kogan MD, Ghandour RM, et al. Trends in the parent-report of health care provider-diagnosed and medicated attention-deficit/hyperactivity disorder: United States, 2003-2011. *Journal of the American Academy of Child and Adolescent Psychiatry* 2014;**53**(1):34–46. [DOI: 10.1016/j.jaac.2013.09.001; NIHMS699115; PMC4473855

Wachs 2014
Wachs TD, Georgrieff M, Cusick S, McEwen BS. Issues in the timing of integrated early interventions: contributions from nutrition, neuroscience, and psychological research. *Annals of the New York Academy of Sciences* 2014;**1308**: 89–106. [DOI: 10.1111/nyas.12314; PMC4075015; PUBMED: 24354763

Walters 2016
Walters S, Loades M, Russell A. A systematic review of effective modifications to cognitive behavioural therapy for young people with autism spectrum disorders. *Review Journal of Autism and Developmental Disorders* 2016;**3**(2): 137–53. [DOI: 10.1007/s40489-016-0072-2

Weber-Fox 1996
Weber-Fox CM, Neville HJ. Maturational constraints on functional specializations for language processing: ERP and behavioral evidence in bilingual speakers. *Journal of Cognitive Neuroscience* 1996;**8**(3):231–56. [DOI: 10.1162/jocn.1996.8.3.231; PUBMED: 23968150

Weston 2016
Weston L, Hodgekins J, Langdon PE. Effectiveness of cognitive behavioural therapy with people who have autistic spectrum disorders: a systematic review and meta-analysis. Clinical Psychology Review 2016; Vol. 49:41–54. [DOI: org/10.1016/j.cpr.2016.08.001; PUBMED: 27592496

WHO 1993
World Health Organization. *The ICD-10 Classification of Mental and Behavioural Disorders: Diagnostic Criteria for Research*. Geneva (CH): World Health Organization, 1993.

WHO 2013
World Health Organization. Meeting Report. Autism Spectrum Disorders & Other Developmental Disorders: From Raising Awareness to Building Capacity. apps.who.int/iris/bitstream/10665/103312/1/9789241506618˙eng.pdf (accessed 22 September 2017). [ISBN 978 92 4 150661 8; NLM Classification: WS 350.8.P4]

WHO 2016a
World Health Organization. Autism Spectrum Disorders. www.who.int/mediacentre/factsheets/autism-spectrum-disorders/en/ (accessed 10 November 2016).

WHO 2016b
World Health Organization. Genes and Human Disease. www.who.int/genomics/public/geneticdiseases/en/index1.html (accessed 10 November 2016).

Youings 2000
Youings SA, Murray A, Dennis N, Ennis S, Lewis C, McKechnie N, et al. FRAXA and FRAXE: the results of a five year survey. *Journal of Medical Genetics* 2000;**37**(6): 415–21. [DOI: 10.1136/jmg.37.6.415; PMC1734610; PUBMED: 10851251

Zatorre 2013
Zatorre RJ. Predispositions and plasticity in music and speech learning: neural correlates and implications. *Science* 2013;**342**(6158):585–9. [DOI: 10.1126/science.1238414

* *Indicates the major publication for the study*

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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