POSITION PAPER

APS Neurofeedback & Psychology Interest Group

Neurofeedback for the Treatment of Attention-Deficit/Hyperactivity Disorder (ADHD)

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November 2011

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Abstract

Attention-deficit/hyperactivity disorder (ADHD) is a pervasive developmental disorder reported to affect between 2-20% of children. It is characterised by inappropriate levels of inattentiveness, impulsivity, and hyperactivity. The mainstream treatment for ADHD has been stimulant medication. Stimulants have short-term benefits for around 60% of children with ADHD, however, long-term benefits have not been demonstrated, and adverse side effects are often intolerable. Furthermore, there is little or no assessment in most practices of stimulant specificity.

Cognitive and behavioural treatments are also common, though generally subject to the pervasive view that medication should be the first line intervention. Neurofeedback therapy is also in widespread use for the treatment of ADHD but, in contrast to cognitive and behavioural interventions, has tended to be poorly understood by the mental health community.

This paper presents the position of the APS Neurofeedback & Psychology Interest Group on the efficacy and use of neurofeedback therapy (NFB) as a clinical intervention in ADHD. A review of relevant literature demonstrates that NFB meets Level 5 criteria of the American Psychological Association, as being both Efficacious and Specific as a primary treatment intervention in ADHD. It is shown to be as effective as stimulants in addressing symptoms in around 70%-80% of children with ADHD. It is recommended the Australian Psychological Society endorse NFB as a safe and effective intervention without adverse side effects in the treatment of ADHD. Evidence for this position is provided in Section 3 of this document. For the more general reader, Section 2 provides a general overview of ADHD, together with brief reviews of the main methods used in its treatment. Section 1 contains the Summary and Recommendations of this Position Paper.

Section 1: Summary and Recommendations

This paper provides basic information about the diagnosis and psychophysiological aetiology of attention deficit/hyperactivity disorder and the evidence for treatment of ADHD to include the modality of neurofeedback. There is significant evidence from multiple clinical studies around the world to show that neurofeedback therapy is an efficacious and specific treatment intervention in ADHD, with large effect sizes on impulsivity and inattention and medium effect sizes for hyperactivity. The following recommendations follow those of Sherlin et al (2010) in their position paper in neurofeedback for the treatment of ADHD:

1. Neurofeedback is a safe and efficacious treatment intervention for ADHD, meeting the rating of Level 5: Efficacious and Specific.

2. Neurofeedback in the treatment of ADHD has been shown to have long-term effects, lasting from 3 to 6 months. More research is required to investigate the effects after 3 to 5 years of treatment.

3. Neurofeedback appears to have similar effects to stimulant medication for inattention and impulsivity, but more controlled and randomized studies are required to further support this observation.

4. Additional research is required to investigate the working mechanism of neurofeedback.

5. Given that neurofeedback currently requires multiple treatment sessions, further research should be directed toward improving neurofeedback treatment to require fewer treatment sessions (e.g., LORETA neurofeedback, Independent Component Analysis [ICA] neurofeedback, Z-score neurofeedback).

6. Neurofeedback is efficacious when inattention and impulsivity are the main problems. When the main complaint is hyperactivity, medication is possibly a better choice given the limited success of neurofeedback in this domain. Controlled and randomized studies are required to further substantiate this claim.

7. No differences in neurofeedback efficacy have been found between medicated and non-medicated children; therefore, neurofeedback can be utilized in combination with a medication regimen.

8. Registered health care providers using neurofeedback should take necessary educational prerequisites to understand the methods and proper implementation of the modality and its appropriateness for the treatment of ADHD.

9. When appropriately trained in the planning, implementation, and monitoring of neurofeedback, the registered health care professional should consider including neurofeedback as a potential modality of treatment for ADHD.

Section 2: Attention Deficit Hyperactivity Disorder

Prevalence of ADHD

ADHD has been described as a relatively common behavioural disorder that substantially interferes with a child's ability to function normally at home and in school (American Psychiatric Association, 1994). The disorder is characterised by difficulties in a number of areas, including paying attention, sustaining mental effort, concentration, distractibility, forgetfulness, fidgetiness, poor impulse control and hyperactivity (American Psychiatric Association, 1994). It is generally accepted that the disorder occurs in 5 to 10% of children (American Psychiatric Association, 1994; Barkley, 1997b; Schneider & Tan, 1997). However, estimates of the occurrence of ADHD in the research literature range from 2 to 20% of school-age children (Cohen, Riccio, & Gonzalez, 1994). The increasing prevalence of ADHD over the past thirty years has prompted considerable research into its aetiology, and there have been several revisions of the classification of the disorder in subsequent issues of the American Psychiatric Association's Diagnostic and Statistical Manual for Mental Disorders over that period (American Psychiatric Association, 1980, 1987, 1994).

Despite an extensive body of research from various disciplines, there is little cross disciplinary dialogue in the literature that has elucidated the relationships between nutritional and metabolic anomalies, brain morphology, neurochemistry, neurophysiology and behavioural manifestations of the disorder. There has been no single aetiology proposed for the disorder and there has not been any laboratory tests found that can identify ADHD amongst the range of childhood behavioural disorders (Barkley, 1991). While ADHD generally continues to be viewed as a disorder which affects attention and/or hyperactivity and impulsive behaviours, theories of ADHD are beginning to focus more on poor inhibition and deficient self-regulation as being central to the disorder (Barkley, 2003). However, the diversity of the proposed causal factors and the range of core and associated behaviours suggest that ADHD may be a catch-all acronym for a range of underlying disorders with a wide range of behavioural manifestations (Goodman & Poillion, 1992).

Historical Perspective on ADHD

The first medical reference to symptoms of ADHD can be traced back to 1902, when an article by British Paediatrician, Sir George Frederick Still, appeared in the *Lancet* describing children who had attentional difficulties, were overactive and distractible (Still, 1902). Still ascribed their impaired "inhibitory volition" and "marked inability to concentrate and sustain attention" to "defects of moral control", which he felt was associated with neurological deficits (Still, 1902).

In the 1930s and 1940s, the concept of minimal brain dysfunction (MBD) was used to explain observations of a group of disorders in children which manifested primarily as disruptiveness, hyperactivity and impulsivity associated with poor attention span (Strause & Lehtinen, 1947). The terms 'Minimal Brain Damage' and

'Minimal Brain Dysfunction' were used in the 50s and 60s when disorders of attention and motor control were thought to result from central nervous system damage associated with birth trauma, infectious diseases or head injuries (Barkley, 1990). In the late 60s and early 70s, the focus of research was on the hyperactivity displayed by children. This was reflected in the terms "Hyperkinesis or Hyperactivity Syndrome" that were used to describe the disorder. In the 70s, the research focus switched to the attentional problems rather than hyperactivity as the core deficit and primary drive for the symptoms (Barkley, 1990; Whalen, 1989; Woods & Ploof, 1997).

The acknowledgment that it was possible for a child to have attentional difficulties without hyperactivity symptoms was first reflected in the third edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-III) (American Psychiatric Association, 1980), where the disorder was labeled "Attention Deficit Disorder" (ADD). The DSM-III diagnostic criteria differentiated between two classifications: ADD with hyperactivity and ADD without hyperactivity. Children diagnosed as having ADD without hyperactivity were described as withdrawn, passive, anxious and lethargic (Whalen & Henker, 1998) and seemed to have difficulties with short-term memory, processing speed and focused attention. On the other hand, children with hyperactivity seemed to have difficulties with sustained attention and behavioural disinhibition (Barkley, 1990). It was not until 1987 that the disorder was relabeled "Attention Deficit/Hyperactivity Disorder" in the DSM-IIIR, the revised third edition of the DSM-III (American Psychiatric Association, 1987). This diagnostic system is still currently used in the DSM-IV, the fourth edition of the DSM, which combines attention deficits with hyperactivity into a single classification that includes the combined symptoms of inattention, impulsivity and hyperactivity (American Psychiatric Association, 1994).

Prevalence of ADHD

Estimates of the occurrence of ADHD in the research literature range from 2 to 20% of school-age children (Cohen et al., 1994); the wide range of prevalence reported may vary depending on the diagnostic perspective employed by the clinician (Cohen et al., 1994). However, it is generally accepted that the disorder occurs in 5 to 10% of children (American Psychiatric Association, 1994; Barkley, 1997b; Schneider & Tan, 1997). Results of a US national survey indicated that the number of office-based visits documenting a diagnosis of ADHD increased from 947,208 in 1990, to 2,357,833 in 1995, and there was a 2.3-fold increase in the population-adjusted rate of office-based visits documenting a diagnosis of ADHD (Robison, Sclar, Skaer, & Galin, 1999). A Mental Health Survey in Australia in 2000 found that 11% of Australian children and adolescents met the criteria for ADHD and 23% had one of the childhood mental disorders surveyed: Depressive Disorder, Conduct Disorder and ADHD (Birleson et al., 2000; Sawyer et al., 2001). The majority of children with ADHD referred to mental health clinics are referred for assistance with aggression and other forms of misbehaviours, which are more common in boys, producing an apparently higher prevalence of boys with ADHD than girls (Brown, Madan Swain, & Baldwin, 1991). ADHD children without hyperactivity are frequently shy, socially withdrawn, and moderately unpopular. Consequently children with ADHD who are predominantly

inattentive are believed to be under-reported (Lahey & Carlson, 1991). Longitudinal studies have found that many of the symptoms of ADHD persist into adulthood, affecting work, social and familial situations (Weiss & Hechtman, 1993).

Studies into the distribution of ADHD have found that boys are 4 to 6 times more likely to be diagnosed than are girls. However, when hyperactivity is not included in the comparison, the difference between boys and girls is smaller (Brown et al., 1991; Strause & Lehtinen, 1947). In the 1990 US survey, mentioned above, the number of visits by girls diagnosed with ADHD rose 3.9-fold between 1990 and 1995 (Robison et al., 1999). In the Australian Twin Study, 2,391 twins, and sibling pairs from Australia, ages 3-18 were studied. The magnitude of familial influences was similar for boys and girls, although there were shared environmental influences on ADHD in girls but not in boys; and dominant genetic influences on ADHD in boys but not in girls (Rhee, Waldman, Hay, & Levy, 1999). Despite showing considerably less aggressive behaviours than boys, girls with ADHD tended to have more problems with mood, affect and emotions, tended to be more socially withdrawn and to show more internalising symptoms such as depression and anxiety (Barkley, 1990). A major long-term study of girls diagnosed with ADHD in elementary school, found that they were at greater risk for substance abuse, emotional problems and academic difficulties in adolescence than their non-ADHD counterparts (Hinshaw, Owens, Sami, & Fargeon, 2006). A 5-year long prospective study that followed girls with ADHD, along with a matched comparison sample found that the childhood-diagnosed ADHD group displayed moderate to large deficits in executive/attentional performance on childhood neuropsychological assessments and rapid naming tasks, relative to the comparison group at follow-up. Overall, the neuropsychological and executive deficits identified in childhood persisted for at least 5 years in girls with ADHD (Hinshaw, Carte, Fan, Jassy, & Owens, 2007).

Diagnostic Criteria and Subtypes

In the USA and Australia, the current criteria for diagnosing ADHD is that proposed in the Diagnostic and Statistical Manual for Mental Disorders, Fourth Edition (DSM-IV, American Psychiatric Association, 1994). The DSM-IV differentiates between three subtypes of ADHD: (a) A predominantly hyperactive impulsive type characterised by fidgetiness, squirming in or leaving assigned seat, excessive running or climbing, difficulty engaging in activities quietly and talking excessively, difficulty waiting in line or waiting for own turn, often interrupting others and blurting out answers; (b) A predominantly inattentive type characterised by distractibility, forgetfulness, difficulty sustaining attention or mental effort, difficulty following through on instructions, organising tasks or activities and paying close attention to details or schoolwork; and (c) A combined type with features of both previous subtypes.

The use of the DSM–IV's descriptive approach to ADHD subtyping may not be the most clinically useful definition of the disorder since this method does not enlighten treatment needs nor predict treatment response (Pelham, 2001). In addition, the DSM-IV subtypes do not consider the underlying neurophysiology that may be associated with the various subtypes. An adjunctive method of classifying ADHD

children may be according to anomalies in cortical field patterns, as measured by the electroencephalogram, which are more likely to reflect CNS anomalies (e.g. Loo & Barkley, 2005; Johnstone et al, 2005; Arns et al, 2008).

Symptoms Associated with Specific ADHD Subtypes

Children with ADHD have been reported as presenting with considerable variation in both the occurrence and in the severity of the symptoms displayed, with symptoms sometimes waxing and waning over time and varying between settings (Barkley, 1991; Cantwell, 1996). While most children display some inattention and hyperactivity at some time or another; children with ADHD have a persistent pattern of more severe inattention and/or hyperactivity and impulsivity (American Psychiatric Association, 1994). Some children appear unaffected in some circumstances when there is minimal demand for mental effort or self-control; but in demanding task conditions, their resources seem overwhelmed and they display inappropriate behaviours. Overall there are four core areas of concern in the behaviours of ADHD children: attention deficits, distractibility, impulse control (poor inhibition) and hyperactivity (American Psychiatric Association, 1994).

Attention Deficits and Distractibility

Children with ADHD are reported to experience difficulties sustaining attention mostly during tasks that they find boring, repetitive, or requiring mental effort. These deficits may not be observed when they are engaged in free play or in activities which they find enjoyable (American Psychiatric Association, 1994). However, the difficulties that they experience in sustaining mental effort may be responsible for their short attention span, their inability to concentrate for extended periods, their distractibility and selective attention (APA, 1994). The inattentiveness displayed by children with ADHD also results in careless mistakes, forgetfulness, poor organisational skills and appearing not to listen when spoken to (APA, 1994).

Impulse control

Children with ADHD are reported to be often impulsive, appearing to respond spontaneously to environmental stimuli and failing to first consider the impact and consequences of their actions (Farmer & Peterson, 1995). It was observed that they do not seem to think ahead, have poor organisational skills, and often expose themselves to high-risk situations. Children with ADHD suffer more injuries than controls, and it has been shown that they anticipate less severe consequences following risky behaviour and report fewer active methods of preventing injury than children without ADHD (Farmer & Peterson, 1995). They are often reported to blurt out answers to questions, jump queues and as having difficulties delaying gratification, preferring smaller immediate rewards and stimulation to larger delayed rewards (Schweitzer & Sulzer Azaroff, 1995).

Hyperactivity and fidgetiness

Most children display fidgetiness and over activity from time to time. However, children with ADHD exhibit restlessness, fidgetiness and age-inappropriate levels of motor activity which interferes with their daily lives (American Psychiatric Association, 1994). In some ADHD children it is not so much that they are overactive all the time, but rather that they seem unable to appropriately regulate their activity to match the situation (Barkley, 1997a).

Progress of the Disorder from Childhood to Adulthood

Many children with ADHD have difficulties with their academic performance at school. As many as 23% to 30% do not achieve the results that would be expected of children of their age and general intelligence (Frick & Lahey, 1991). Between 40% and 60% of children diagnosed with ADHD have repeated a grade at school by adolescence (Brown & Borden, 1986). Many are performing below grade level or have borderline academic performance. Children with ADHD typically have impaired concentration and attention. This results in poor self-organisation, poor self-regulation, and difficulty with time management, which in turn lead to the poor academic performance that is frequently observed (Searight, Nahlik, & Campbell, 1995).

Children with ADHD typically have difficulty forming and maintaining friendships with other children. They frequently misread social cues and as a result may act in an inappropriate manner (Barkley, 1990). Studies have indicated that the inattentive, disruptive, off-task, often provocative, immature behaviours of these children result in their peers being controlling and directive towards them during group tasks (Barkley, 1990). Because of combined difficulties and perceived failures in various life areas such as sport, academia, and social activities, children with ADHD frequently experience low self-esteem. While lack of self esteem can be clearly observed in some children with ADHD, in others it may be hidden behind a brash, and apparently confident exterior (Wallace, 1996).

Aggressive and antisocial behaviours such as fighting, stealing, and truancy are considered the most significant problems associated with ADHD. Estimates are that between 30% to 90% of children with ADHD exhibit conduct problems (Hinshaw, 1987). Apart from the immediate impact of conduct problems on the child's interactions with others, the presence of these problems has been shown to place children with ADHD at risk for drug or alcohol abuse, and for displaying other antisocial and delinquent behaviours as adolescents and young adults (Aylward, 1979; Mannuzza, Gittelmann-Klein, Konig, & Giampino, 1989).

Studies indicate that most children with ADHD continue to experience difficulties into adolescence (Weiss, 1990; G. Weiss & Hechtman, 1993). The changes and stresses to which all teenagers are subjected during this stage of their development, are frequently sufficient to lead to a re-emergence of symptoms which may have been controlled in childhood (Quinn, 1997). Several studies that have followed children with ADHD through their development have found that symptoms

that were predominant during childhood, such as hyperactivity, were no longer such a serious problem in adolescence (Weiss, 1990; Weiss & Hechtman, 1993). The studies have found that the hyperactivity often abates somewhat during teenage years, and that issues such as distractibility, restlessness and difficulty with relationships were reported to be the main source of problems (Quinn, 1997). In their clinical sample, Hart and colleagues found that, whilst hyperactivity declined with passing years, there were no age-related changes in inattentive behaviours (Hart, Lahey, Loeber, Applegate et al., 1995). Academic problems, where present, can continue at the same level over the transition from childhood to adolescence.

Academic difficulties can become evident at this stage as a result of general expectation at school that children have acquired a great deal of general knowledge during their primary school years which is immediately and automatically available to them (Levine, 1989). However, children with ADHD frequently do not have this fund of readily available knowledge, as a result of not having attended when the information was originally presented (Levine, 1989).

Many of those with ADHD continue to experience symptoms well into adulthood. While symptoms may not present during highly interesting or motivating tasks, attentional deficits are likely to show during tedious or uninteresting tasks. Without the structure provided by the school environment, or parents to assist with organising activities, the adult with ADHD may have difficulties meeting the organisational demands of everyday life (Weiss & Hechtman, 1993). Long-term studies that have followed children with ADHD treated with psycho-stimulant medication (methylphenidate or dexamphetamine) through to adulthood, have reported that these adults experience less stability and satisfaction in areas such as employment, educational achievement, interpersonal relationships and mental health (Weiss & Hechtman, 1993). For example, one study revealed that ratings of the social-skills and self-esteem of ADHD children, which were only slightly different between ADHD and non-ADHD early in the study, became progressively worse over time (Weiss & Hechtman, 1993). The measures showed that the social-skills and self-esteem of the ADHD group had deteriorated significantly by the 10- year follow-up, and much more by the 15 year follow-up. As adults, this group reported significantly higher levels of anxiety, depression and other psychiatric disorders (Weiss & Hechtman, 1993). Adults with ADHD frequently reported feelings of failure, frustration, underachievement and guilt (Green & Chee, 1997).

Co-morbidities and Differential Diagnosis of ADHD

Children with ADHD belong to a heterogeneous population with varying symptom range, severity and pervasiveness (Barkley, 1990). ADHD studies reflect variations in diagnostic criteria, measurement sampling and designs which confound diagnosis and study results (Cohen et al., 1994; Schacher, 1991). It is very difficult to find a group of children who exhibit purely ADHD symptoms, as co-morbidity with other disorders is common in ADHD (Castellanos, 1997), and children with emotional or learning problems can also appear to suffer from attention deficits (American Psychiatric Association, 1994; Cantwell, 1993). It has been estimated that 4% to 6% of

school-age children also suffer from some form of Learning Disorder. Although ADHD and Learning Disorders are thought of as distinct neuropsychiatric entities, there is considerable co-morbidity between the two disorders (American Psychiatric Association, 1994; Riccio & Jemison, 1998). As many as 20-30% of children with ADHD are estimated to have learning disabilities (Bender, 1997; Biederman, Newcorn, & Sprich, 1991; Rutter, 1982). Attempts to differentiate children with ADHD from normal controls or from psychiatric controls on measures of cognitive and/or neuropsychological function, neurotransmitter activity, genetic factors, and neuroanatomy have yielded inconsistent results (Barkley, Grodzinsky & DuPaul, 1992). Precise and accurate determination of the presence of ADHD versus Learning Disorders can be of critical importance for effective treatment to avoid the potentially devastating impact of these disorders on children and their families.

Up to 50% of children with ADHD may warrant a co-morbid diagnosis of Oppositional Defiant Disorder, Obsessive Compulsive Disorder or Conduct Disorder as a result of the presence of severe externalising behaviours (Bender, 1997; Biederman et al., 1991; Rutter, 1982). A further 25-35% may have co-morbid anxiety and 15% may have mood disorders with associated internalising symptoms (Bender, 1997; Biederman et al., 1991; Rutter, 1982).

Deficits in the ability to sustain attention may be common in children with other psychiatric disorders, making the task of differential diagnosis difficult (Swaab Barneveld et al., 2000). Sub-groups of children with ADHD delineated based on the disorder's co-morbidity with other disorders may have differing risk factors, clinical courses, and pharmacological responses. Thus, their proper identification and differentiation may lead to refinements in preventative and treatment strategies (Biederman et al., 1991).

Children with ADHD and co-morbid Obsessive Compulsive Disorder (OCD) may have obsessive thoughts and/or compulsions. A NIH study, found that twice as many boys than girls were diagnosed with OCD, and that boys had an earlier onset than girls, with an average age of onset of around 7 to 9 years of age as opposed to an age of onset of around 11 for girls (Swedo, Rapoport, Leonard, Lenane, & Cheslow, 1989). The less severely affected children and those attempting to hide their symptoms make this group more difficult to diagnose. The most common symptoms include: long unproductive hours doing homework; excessive erasing, sometimes to the point of tearing the paper; retracing over letters and words; re-reading paragraphs over and over; excessive laundry or toilet paper usage; insistence on using some clothes or towels only once; unusual bedtime rituals; unduly worrying about germs or about a small cut or pimple; exaggerated need for reassurance; rigid bedtime rituals and hoarding of useless objects (Leonard, Goldberger, Rapoport, Cheslow, & Swedo, 1990). These are not always obvious symptoms and OCD co-morbidity may be missed when the child also presents as hyperactive, oppositional and with behavioural problems (Leonard et al., 1990).

The diversity of symptoms and co-morbidities has led Goodman and Poillion to suggest that ADHD may be an acronym for a range of underlying factors (Goodman & Poillion, 1992). Following an extensive review of the literature, they found that there

had been 69 different characteristics attributed to children labeled ADHD along with 38 possible aetiologies suggested for the disorder (Goodman & Poillion, 1992). The psychological nature of these symptoms has led to the use of cognitive and behavioural interventions for this group, and this is the subject of the next section.

Behavioural and Cognitive Interventions for ADHD

There is a range of interventions that use cognitive-based strategies that focus on remediating deficiencies in thinking or cognitive processes (see Toplak et al, 2008). Those classified as cognitive-behavioural therapies (CBT) included strategy and metacognitive training. Cognitive studies included direct skills training of cognitive skills, such as working memory or attention. Behavioural only treatment includes clinical behavior therapy and contingency management and is grounded in learning theory, including principles of classical conditioning, operant conditioning, cognitive-behavioural theory and social learning theory (see Fabiano et al, 2009).

The Multimodal Treatment Study (MTS) of children with ADHD (MTA Cooperative Group, 1999) is probably regarded as the largest and most significant early efficacy study, addressing the treatment effects of both cognitive, behavioural and medication interventions in ADHD. The study has been very influential in persuading many that medication was the only viable option for treating children with ADHD, and that behavioural intervention strategies are not important for treating the core symptoms of ADHD (Hinshaw et al, 2006). It found that medication management significantly decreased ADHD symptoms compared to the behavior program alone, and that the combined medication and behavior treatment were not significantly better than the medication management or behavioural treatments alone.

Toplak et al (2008) provides a more recent evidence-based review of cognitive and cognitive-behavioural treatment approaches in ADHD examining studies published between March 1981 and May 2007. For cognitive-behavioural interventions, they demonstrated medium to large effect sizes on both cognitive and behavioural outcome measures. However, as in the MTS study, there was a significant confound with stimulant medication effects, once again rendering it difficult to evaluate the overall efficacy of cognitive-behavioural intervention.

Most of the cognitive training programs examined by Toplak et al (2008) used medication-free participants during treatment. Although based on a small set of studies, the positive impact of these programs on measures of cognitive outcome was quite evident, with medium to large effect sizes obtained where calculable. Effect sizes for these programs relative to behavioural rating measures were smaller, in the small to medium range.

Fabiano et al (2009) and Pelham and Fabiano (2008) recently conducted meta-analyses of behavioural treatments for ADHD. The Pelham and Fabiano (2008) study sought to extend and confirm findings from an earlier review (Pelham et al, 1998) and concluded behavioural parent training and behavioural classroom management were well-established treatments for children with ADHD. It also concluded that intensive

peer-focused behavioural interventions implemented in recreational settings were also effective. Fabiano et al (2009) were more cautious about the reliability of earlier findings for behavioural interventions. They argued there was considerable debate about the extent of the supportive evidence and therefore of the role of behavioural approaches in treatment. For this purpose, they undertook a comprehensive, quantitative study on the magnitude of the effectiveness of behavioural treatments for ADHD. Effect sizes were generally large, and consistently so across study methods and designs, and similar to the effect sizes reported in other meta-analyses of child treatment (Weisz & Weiss, 1989) and stimulant medication (Conners, 2002).

Overall, there would appear to be good evidence of efficacy in the use of cognitive-behavioural interventions in ADHD. Prior to this, however, the concomitant use of medication has been a source of contention and a basis for devaluing non-pharmacological approaches. Fabiano et al (2009) point out that influential treatment guidelines for ADHD in North America (e.g. AAP, 2001) have emphasized the first-line use of medication in ADHD treatment and chronic management, while de-emphasizing behavioural treatments, or casting them as a third-line or adjunctive treatments. The more recent work by Fabiano et al (2009) and Toplak et al (2008) suggest such guidelines and recommendations should be modified. Notably in this regard, the American Psychological Association has recommended that behavioural treatments be first-line interventions for ADHD (Brown et al., 2007).

Psychostimulant Treatment of ADHD

Psycho-stimulants (e.g. Methylphenidate and Dexamphetamine) are the most commonly used medical treatment for children with ADHD. The drug Methylphenidate is responsible for a high percentage of the psycho-stimulant medication market for ADHD (Bender, 1997). Dosage of stimulant medication varies from one individual to the next, but usually begins at the lowest recommended dose, and is usually given two or three times daily. Dosage is increased gradually to achieve a state of maximum symptom relief with minimal side effects. The effects of Methylphenidate can be observed 30 minutes after ingestion, and the peak efficiency is reached between one and three hours later, with the efficacy wearing off after four to six hours (Bender, 1997). The benefits of psycho-stimulant medication in the treatment of children with ADHD are firmly established. However, few studies have extended beyond 24 months (Greenhill, Halperin, & Abikoff, 1999).

According to Barkley, psychostimulant medications have been reported to reduce the problematic hyperactive symptoms of ADHD in approximately 60-70% of children with ADHD (Barkley, 1990). In a review of stimulant use in ADHD, the pooled results of the treatment of 5,899 children participating in 161 randomised controlled trials, found that 65-75% of children treated with psycho-stimulant medication showed clinical improvement, while the rates for clinical response from the placebo groups ranged from 4% to 30% (Greenhill et al., 1999). Research and clinical findings indicate that the ability to attend increases, social behaviours improve, and impulsivity decreases with the use of psycho-stimulants (Barkley, 1990). Frequently parents report not only that behaviours improve significantly, but also that relationships between the

child and the rest of the family, as well as with peers, improve markedly once the child is placed on medication (Bender, 1997).

In 1992, the National Institute of Mental Health and 6 teams of investigators began a multi-site clinical trial: "The Multimodal Treatment of Attention-Deficit Hyperactivity Disorder (MTA) Study" (Jensen et al., 2001). Five hundred and seventy nine children were randomly assigned to either one of four treatment conditions, each designed to reflect best-known practices within each treatment approach and each lasting 14 months. These were: (a) Routine community care; (b) Monthly medication management follow-up, following initial weekly titration; (c) Intensive behavioural treatment and (d) The combination of behavioural treatment and medication management.

This study reported that medication management combined with behavioural treatment was substantially superior to behavioural and community care interventions on their own for symptoms of ADHD. Results also suggested slight advantages of combined interventions over medical management, behavioural treatment and community care for social skills, academic performance, parent-child interactions, oppositional behaviours, anxiety and depression (Jensen et al., 2001). The MTA study results indicated that high quality medication management characterized by careful and adequate dosing, Methylphenidate administration three times daily, along with weekly initial titration and monthly follow-up visits, and communication with schools conveyed substantial benefits to those children that received Methylphenidate (Jensen et al., 2001). In addition, children with parent-defined co-morbid anxiety disorders, particularly those with overlapping disruptive disorder, showed preferences for the behavioural and combined interventions. Parental attitudes and disciplinary practices mediated improved response to the behavioural and combined interventions (Jensen et al., 2001). Reduced final doses of stimulant medications were achieved in the combined treatment group compared with the medication management group. The explanation offered for this was that behavioural therapy is a useful adjunct to medication and may reduce the overall consumption of stimulant drugs and their side effects (Jensen et al., 2001).

The superior effectiveness of optimally managed stimulant medication treatment over community care and behaviour intervention was demonstrated in the MTA study (Jensen et al., 2001). The study confirmed what previous smaller studies and reviews had already found, namely, that combined medication and intensive behaviour therapy offered the best treatment options for ADHD (Jensen et al., 2001). However the MTA study was not without its critics, as described in the next section (Greene & Ablon, 2001).

Concerns Relating to Psychostimulant Medication for ADHD

Greene and Ablon (2001) criticised the MTA study and raised the following concerns (a) whether the medication management and behavioural arms of the MTA were assessed to comparable degrees. Medication management was individually optimised with initial weekly titration of medication and monthly reviews and

assessment were carried out while on medications. On the other hand, behavioural interventions were not optimised based on individual needs, and post-treatment assessment was made months after termination of treatment. Hence it is questionable whether: (a) cognitive-behavioural interventions were incorporated to an adequate extent and tailored to individual needs, (b) whether core ADHD symptoms, attention, distractibility and impulse control, which were responsive to medication, were overemphasized relative to other important functional domains both as treatment targets and as outcome measures; and (c) whether parent and teacher characteristics warranted more emphasis than they were given in the research, as such an emphasis would represent parent- teacher reports of behavioural interventions more fairly (Greene & Ablon, 2001).

Pelham also criticised what he described as misinterpretations and premature reporting of findings of the MTA study, and bias towards drug prescription in the design of the MTA study (Pelham, 1999).

Pelham (1999) remarked that medication management was superior to behaviour treatment on parent and teacher ratings of inattention and teacher ratings of hyperactivity, but not on any of the other 16 measures. These included classroom observed behaviours, parent- and teacher-rated social skills, parent-rated parent-child relationships, peer sociometric ratings, and academic achievement. Hence, medication management was superior to behaviour treatment on only two of the 19 measures assessed, yet published results focused on attention and hyperactivity measures alone. In addition, Pelham made a strong case for methodological bias in favour of medication management on the basis that behaviour treatment was assessed 4-6 months after cessation of treatment while medication management was assessed in its acute and most active stage. In contrast to proponents of the study, he concluded that the MTA evidence suggest that: behavioural treatments are effective in the treatment of attention-deficit hyperactivity disorder, that combined treatments are usually superior to monotherapies, and that concurrent behavioural treatment allows for lower medication dosages (Pelham, 1999).

Three years following the end of the MTA study, 485 of the original 579 ADHD subjects (83.8%) now aged 10 to 13 years (mean 11.9 years), participated in the follow-up study. In contrast to the significant advantage of medication management and combined treatment over behaviour management and community care for ADHD symptoms found at 14 and 24 months, the treatment groups did not differ significantly on any measure at 36 months (Jensen et al., 2007).

Regardless of whether the ADHD participants changed their treatment use, all of the groups showed symptom improvement over baseline at the start of the study. None of the following initial factors moderated the ADHD children's 36-month treatment responses: (a) initial symptom severity, (b) gender (male), (c) existence of comorbidities, (d) whether they received public assistance, and (e) their parent's own ADHD psychopathology. However, these initial factors predicted worse outcomes over 36 months, regardless of original treatment assignment. The reviewers concluded that by 36 months, the NIH multimodal study revealed that the earlier advantages of having had 14 months of the medication treatment were no longer apparent. They suggested

that this may have resulted from age-related decline in ADHD symptoms, changes in medication management intensity, starting or stopping medications altogether, or other factors not yet evaluated (Jensen et al., 2007).

Swanson and fifteen co-authors conducted a comprehensive examination of 341 reviews of the effects of stimulant medication on children with attention deficit disorders (Swanson, McBurnett, Wigal, Pfiffner, & et al., 1993). Their review found that medication was ineffective for 25 to 40 percent of children with ADHD. A large proportion of those responding to Methylphenidate also showed improvements on a placebo. Across quantitative reviews, the average effect size for symptomatic improvement (0.83) was twice that for benefits on I.Q. and achievement measures (0.35). Amongst those that responded to stimulant medication, temporary management of over activity, inattention and impulsivity could be expected, as well as temporary improvement in compliance. Hyperactivity and aggression may be reduced, and consequently the amount of academic work completed may increase. However, contrary to the hopes of parents and practitioners, there was no evidence of significant long-term improvement in reading, athletic or game skills, proactive social skills, learning and achievement other than improved attending (Swanson et al., 1993). In other words, hyperactivity and attending may improve amongst the 60 to 75% of children who respond to psycho-stimulant medication, but their concentration, learning ability and cognitive skills may not. In the review it was also suggested that parents should not expect improvements in long-term adjustment, improved academic achievement, nor should they expect a reduction in antisocial behaviours or misconduct (Swanson et al., 1993).

Several side effects have been reported during treatment with psycho-stimulants. These include: decreased appetite, insomnia, dysphoria, headaches, weight loss, stomach and leg cramps and the onset or exacerbation of Tics or Tourettes Syndrome (American Psychiatric Association, 1994). Schachter and colleagues conducted a meta-analysis of randomized controlled studies of medication treatment for ADD/ADHD (Schachter, Pham, King, Langford, & Moher, 2001). They examined 62 randomized trials of stimulant medication, involving 2897 participants with a primary diagnosis of ADHD (with or without hyperactivity). They reported that the studies were of poor quality and had a strong publication bias. Interventions lasted an average of 3 weeks, with no trial lasting longer than 28 weeks. Each primary outcome (hyperactivity index) demonstrated a significant effect of Methylphenidate (effect size reported by teacher 0.78 and by parent 0.54). However, these apparent beneficial effects were tempered by a strong indication of publication bias, meaning that drug-company-funded studies, which failed to support the effectiveness of their product, or reported too many side effects, tended not to be submitted for publication. In addition, there was a lack of robustness in the findings, meaning that the findings varied greatly across studies, especially those involving core features of Attention Deficit Disorder (Schachter et al., 2001). They also concluded that the extension of this placebo-controlled effect beyond 4 weeks of treatment had not been demonstrated, and that the adverse event profile of Methylphenidate required further consideration (Schachter et al., 2001).

A comprehensive review from Oregon State University (The Drug Effectiveness Review Project, 2005) analyzed 2,287 studies involving all stimulant medications prescribed for ADHD. The group rejected 2,107 of the investigations as unreliable and reviewed the remaining 180 to reach their conclusions, published in a 731-page report. Their findings were that: (a) there was no evidence of long-term safety of drugs used to treat ADHD in young children or adolescents, (b) there was a lack of good quality evidence that ADHD drugs improve global academic performance, risky behaviours, social achievements and other measures, (c) there was little evidence of the safety of these drugs, and there were research findings which suggested that some ADHD drugs could stunt growth, (d) evidence that ADHD drugs help adults was not compelling, nor was there evidence that one drug was more tolerable than another, and (e) there was a poor understanding of the pharmacokinetics of these drugs. The review did not suggest that drugs used in the treatment of ADHD are unsafe or not helpful, only that sound scientific evidence is lacking to prove that they are safe and useful in the long term (McDonagh, Helfand, Chou, & Norris, 2005).

El-Zein and colleagues (2005) concluded that the lack of research on long-term effects of Methylphenidate use in humans warranted grave concerns and further investigation(El-Zein et al., 2005). They discovered that after 3 months, all of the ADHD children on Methylphenidate in their double blind placebo controlled study experienced chromosomal aberrations which could increase cancer risk (El-Zein et al., 2005). They likened the risk to that of the cytotoxic damage that has been found in adult methamphetamine users (Li, Hu, Chen, & Lin, 2003).

As previously stated, controversy is rising over the possibility of growth suppression while on stimulants, together with estimates that as many as 30% of children with ADHD do not respond to stimulant treatment or cannot tolerate the undesirable side effects (Daley, 2004). These concerns and reports of rising adverse cardiovascular events has prompted the FDA to direct manufacturers of all drugs used in the treatment of ADHD to develop patient Medication Guides to alert patients to possible cardiovascular risks and risks of adverse psychiatric symptoms associated with the medicines (FDA, 2006).

According to Daley (2004) children with ADHD are likely better served with multimodal treatment plans, that are individually tailored, including medication, parent/school counseling, and behavioural therapies (Daley, 2004). There is also a need for a treatment modality, which would provide permanent results and no side effects. Neurotherapy has no adverse side-effects and has shown promise as an effective emerging intervention for ADHD (Hirshberg, Chiu, & Frazier, 2005).

Section 3: Neurofeedback Therapy

The number of individuals diagnosed with ADHD together with the desire to avoid stimulant medication has brought increasing attention to neurofeedback treatment in recent years (Sherlin et al, 2010). But despite the increasing number of published studies reporting effective use of the modality, there is still populist controversy regarding the efficacy of neurofeedback for treating ADHD. This section will provide a brief history of neurofeedback, together with the rationale for its use, and will review the evidence for its level of efficacy in treating ADHD.

Neurofeedback is a type of operant conditioning in which an individual modifies the frequency, amplitude and other characteristics of his or her own EEG. It was demonstrated as early as 1941 that EEG rhythms can be classically conditioned, and that humans and animals can control their own EEG through feedback, and that the skills required to modulate EEG activity in the required direction are preserved over time. (Sherlin et al, 2010). With just three electrodes attached to the head, it is possible to record, analyse and give nearly instantaneous feedback based on the electrical activity of the brain.

Rationale for Neurofeedback in ADHD

There is a sound psychophysiological basis to the diagnosis of ADHD. It is clear from the substantial neuroimaging literature using hemodynamic methods such as cerebral blood flow, functional MRI and SPECT that electrical and chemical signalling systems mediate information processing and transfer within the brain. This is even clearer from the substantial literature on EEG, which provides a more instantaneous measure of brain function – due to its high temporal resolution of change in regional post-synaptic membrane currents once volume conducted from cortical pyramidal cells to the scalp surface. Further, EEG research on ADHD shows unequivocally that EEG measures can easily distinguish between children with ADHD and normal controls (e.g. Van der Stelt et al, 2001).

Much of this research indicates that children with ADHD often had excessive levels of slow-wave (theta and alpha) activity in the EEG and decreased levels of relative beta activity when compared to the EEG activity of normal controls (e.g. Barry et al, 2003a, 2003b). Sherlin et al (2010) note that the abnormalities seem to be more pronounced in children with the combined type of ADHD than the inattentive type. In addition, a large-scale multicentre study (Monastra et al, 1999) as well as a recent meta-analysis (Boutros et al, 2005) have concluded that excess theta is a robust population biomarker for ADHD, though more recent work (Arns et al, 2008) has shown at the individual level that this endophenotype applies only to 60—70% of such ADHD children. Arns et al (2008) also identified ADHD subgroupings that are better characterised by either slowed alpha peak frequency or low voltage EEG phenotypes.

Excess theta in ADHD has often been interpreted as a maturational lag due to the inverse correlation between theta power and cognitive development through out

the pre-teen and teen years (see Barry et al, 2003). Others have argued in terms of labile vigilance regulation or hypoarousal (see Sherlin et al, 2010) with the hypoarousal model explaining the normalising effects of stimulant medication in responders (see Barry et al, 2009). Indeed, Arns et al (2009) have shown that EEG profiles characterised by excess frontal theta and raised theta/beta ratios reflect those who obtain favourable treatment outcomes with stimulant medication. Notably this profile does not relate to any DSM subdiagnosis indicating that within behavioural homogenous groups such as ADHD, neurophysiological subgroups exist and respond differentially to treatment (see Sherlin et al, 2010). Ritalin does not have a clinically significant effect in 20-40% of children with ADHD. Arns et al (2008) note that ADHD children characterised by with excess frontal alpha rather than frontal theta excesses tend to respond to antidepressants rather than stimulants.

Such investigations provide sufficient evidence to conclude that not only is brain electrical activity reflective of the condition of ADHD, but its dysregulation contributes to the presence of the condition (see Sherlin et al, 2010). This leads inexorably to the view that operant conditioning to decrease dysregulation and later electrical activity is not only possible but stands as a potential treatment for the disorder (Abarbanel et al, 1995; Lubar, 1991; Nash, 2000; Sherlin et al, 2010).

Early Clinical Use of Neurofeedback

Neurofeedback therapy (also called EEG biofeedback) is an operant conditioning paradigm originally used in sleep studies by Sterman at UCLA to train cats to increase the frequency of occurrence of alpha spindles, or Sensorimotor Rhythm bursts (EEG alpha waves of 12-15Hz), from the sensorimotor cortex. Later it was found that these cats became resistant to chemically induced seizures, prompting Sterman to investigate the effectiveness of Neurofeedback in treating epileptics (Sterman, 1973). Unexpectedly, along with amelioration of seizure disorder, subjects reported better concentration and reduced hyperactivity (Sterman, 1973; 2000).

Early studies of operant conditioning of EEG indicated that animals could be trained to influence specific aspects of their EEG (Black, Young, & Batenchuk, 1970; Delgado, Johnston, Wallace, & Bradley, 1969, 1970; Hall, 1968; Lopes da Silva, 1991). Sterman et al (1972) trained cats by EEG operant conditioning, using food reward, to produce 11-15 Hz "alpha spindle" electrical activity over the sensorimotor cortex (Sterman et al., 1972). Sterman called this specific EEG activity, associated with behavioural stillness, the "Sensorimotor Rhythm (SMR)" (Sterman et al, 1974; Sterman et al, 1975; Sterman et al, 1972; Sterman, Howe, & Macdonald, 1970; Wyricka & Sterman, 1968). In a later study conducted for NASA, Sterman serendipitously discovered that the cats trained to increase SMR brain activity became resistant to chemically induced seizures (Sterman, LoPresti, & Fairchild, 1975). The conditioned parameter, an increase in the SMR activity being trained, had no intrinsic value for the animals and the associated raising of seizure threshold was an unexpected and unrelated finding. These early experiments resulting in changes in the EEG, increased behavioural stillness, and the raising of the seizure threshold suggested that organic changes may have occurred in the brains of the animals (Sterman et al., 1975).

Sterman et al (1972) initiated clinical trials using EEG operant conditioning in a 23-year-old female with a 7-year history of intractable tonic-clonic seizures of unknown origins at an average rate of two per month, unrelated to her menses. The patient's EEG indicated generalised 5-7 Hz excess theta activity, and the protocol used aimed to reduce theta and increase SMR (11-15 Hz) activity. After treatment at the rate of twice a week over 3 months, the seizures stopped and follow-up EEG indicated reduction in the excess theta and increase in SMR activity (Sterman & Friar, 1972). In two subsequent studies, this single-case study was extended to include more subjects, and, using the same protocol Sterman reported that seizure rates and EEG abnormalities significantly reduced in these patients as well (Sterman, 1973; M. B. Sterman & Friar, 1972; Sterman et al., 1974).

Several similar subsequent studies also found that EEG operant conditioning was associated with reduction in theta power, increase in SMR and seizure reduction (Finley, Smith, & Etherton, 1975; Kaplan, 1975; Seifert & Lubar, 1975; Upton & Longmire, 1975; Wyler, Ward, & Fetz, 1975). Finley (1976) conducted a blinded Sham feedback study on a male teenager with severe epilepsy, who after one year of SMR biofeedback training had decreased incidence of tonic seizures from eight per hour to less than 1 in 3 hours. SMR increased from 10% to 70%, and epileptiform discharges decreased from 45% to 15%. Blinded non-contingent feedback was introduced for a period of 7 weeks following which SMR decreased significantly (down 8%), and epileptiform discharges increased significantly (up 4%). Rate of seizures increased, but was not statistically significant over the preceding months of contingent feedback. Contingent feedback was reinstated following the 7-week sham feedback period and recovery of all variables to their former levels was observed. This blinded ABA design indicates that the gains were attributable to EEG operant conditioning (Finley, 1976).

Early experimentation to reduce theta and promote beta or Sensorimotor Rhythm (SMR) activity, mostly by Lubar at Tennessee University with hyperactive and inattentive children, found that these children could improve their symptoms considerably (Lubar, 1991; Lubar & Shouse, 1976; Lubar & Shouse, 1977). A number of studies have since demonstrated that Neurofeedback can be used to train the theta/beta ratio towards normal and that concurrently in around 70-80% of cases ADHD symptoms have been reported to have improved to the point where stimulant medication was no longer necessary (Arns et al., 2009; Fuchs, Birbaumer, Lutzenberger, Gruzelier, & Kaiser, 2003; Gevensleben et al., 2009; Lubar & Lubar, 1984; Rossiter, 2004a, 2004b; Rossiter & La Vaque, 1995; Tansey, 1990; Thompson & Thompson, 1998).

Neurofeedback in ADHD: Case and uncontrolled clinical studies

Many neurofeedback studies of ADHD have consisted of single-case, series of single-cases or very small, uncontrolled groups. The small sample size of the studies and the lack of larger placebo controlled groups attracted much criticism (e.g. Lohr, Meunier, Parker, & Kline, 2001; Loo & Barkley, 2005). The major criticism was that the results cannot be generalised to the ADHD population at large and conclusions cannot be drawn with regards to the specificity of the treatment effects, because other

associated confounding factors, may account for the treatment effects (Lohr et al., 2001; Loo & Barkley, 2005). This is clearly an appropriate caution. However, they do provide a useful background context to the many well controlled studies that have now been published, and which are reviewed later in this paper.

Early studies showing successful application of neurofeedback training to various forms of epilepsy in humans also found a coincidental decreases in hyperactivity together with subjectively reported increases in attentiveness, focus and concentration and reduced fidgetiness (Lubar & Bahler, 1976). In a subsequent study, Lubar and Shouse (1976) investigated the effects of Neurofeedback on ADHD in a single-case blind-crossover (ABA) study with an 8 year 11 months old hyperkinetic child who was on psycho-stimulant medication for attention deficits and hyperactivity. Following Neurofeedback treatment, (SMR enhancement and theta inhibit) two independent observers reported decreased oppositional and out of seat behaviours and increased cooperative behaviours. A concurrent increase in attentiveness and academic output was also reported. Reversed training (inhibit SMR and increase theta) over a four-week period was then provided, and the original undesirable behaviours returned. Training was reversed yet again (SMR enhancement and theta inhibit) and the child reportedly regained all previous losses, and school performance and behaviours again improved on all measures. The child was then taken off Methylphenidate, and reportedly continued to do well. Follow-up over several years showed that the gains were maintained (Lubar & Shouse, 1976).

Four hyperkinetic subjects from a cohort of 12 hyperkinetic children and 12 controls were selected for Neurofeedback on the basis that compared to the other subjects; they exhibited the worst classroom misconduct, combined with the lowest levels of SMR and general physiological arousal levels. In addition, their symptoms were reduced by the use of Methylphenidate, but not sufficiently enough to produce normalisation. The same Neurofeedback ABA blind crossover paradigm described in (Lubar & Shouse, 1976; Shouse & Lubar, 1978), with SMR enhancement and theta inhibit (Shouse & Lubar, 1979), was used on the four subjects.

Three of the four subjects in the study showed contingent increases in SMR which were correlated with reductions in classroom motor activity (Shouse & Lubar, 1979). The combination of EEG Neurofeedback treatment with medication treatment resulted in substantial improvements in tested behaviours that exceeded the effects of medication alone. They reported that a reduction in theta and an increase in SMR correlated with a significant decrease in hyperactivity. Attentiveness also improved, but to a lesser degree. Hyperactivity and attentiveness were significantly improved and/or reversed at every stage of the study design. In every case, when medication was withdrawn, the improvements were maintained with SMR enhancement and theta inhibit. A decrease in undesirable behaviours such as disruptive motor activities, self-stimulation, out of seat behaviours and oppositional behaviours, were observed. Additionally, an increase in desirable behaviours was observed, as was increased attention span and cooperation. Social behaviours such as self-initiated approaches to peers or teachers, and sustained interactions with them also improved (Shouse & Lubar, 1979).

Jackson and Eberley (1982) used Neurofeedback in a pilot study that aimed to decrease the percentage of time that alpha wave activity was being produced while engaged in an arithmetic task. The participants were five mentally retarded adults. Analysis of intra-subject and inter-subject data revealed an overall significant decrease in the total number of alpha bursts, and percentage of time in dominant alpha compared to baseline conditions (Jackson & Eberly, 1982). Decreases in Alpha activity have long been acknowledged as evidence of information processing and increased attention. The observed decreases in percentage of time in alpha, correlated with an increase in the percentage of problems completed correctly. This indicated an increase in facilitated attention following Neurofeedback (Jackson & Eberly, 1982). In addition, both an automated method of determining head turning and human direct observation confirmed a significant decrease in the number of distractible head-turning responses. This increased attention and reduced distractibility in a developmentally delayed population was observed following successful Neurofeedback down-training of alpha activity (Jackson & Eberly, 1982).

Working from a private clinical setting, Tansey and Bruner (1983) used a narrow band filter centered around 14 Hz for Neurofeedback on a boy aged 10 who had been diagnosed as perceptually impaired with developmental reading disorder, ocular instability and hyperactive behaviours. The boy, who was due to repeat special-education fourth grade, had been on Methylphenidate for a number of years. After three sessions of electromyographic (EMG) biofeedback, but prior to the start of Neurofeedback, the child's Paediatrician had stopped his medication because of his reduced hyperactivity. The learned reduction of EMG levels was accompanied by a reduction in hyperactivity level below that which had been achieved by past administration of Methylphenidate (Tansey & Bruner, 1983). Following the initial EMG training sessions, contingent amplitude and frequency modulated auditory feedback were used to teach the child to increase 14 Hz activity over the sensorimotor cortex. Following the EMG biofeedback training, the initially observed ADHD was no longer diagnosable. The learned increase in the amplitude of monitored SMR was accompanied by remediation of the developmental reading disorder and the ocular instability. These results remained unchanged, as ascertained by follow-ups conducted over a 24-month period subsequent to the termination of biofeedback training. Furthermore, the child's improvements in reading comprehension and behaviour over the summer period during which Neurofeedback took place, warranted the child being placed in normal fourth-grade (Tansey & Bruner, 1983).

Ten years after treatment had ceased, the boy's ongoing normal social and academic functioning were noted (Tansey, 1993). The boy had completed high school successfully, was attending college, had no attentional problems and remained stimulant medication-free. The boy's brainwave patterns were compared with those of 24 previously learning-disabled children, half of whom were classified as perceptually impaired prior to Neurofeedback treatment (Tansey, 1990, 1991), and his EEG measures appeared to have normalised. This confirmed the long-term stability of the outcomes of the 14-Hz Neurofeedback treatment (Tansey, 1993).

Lubar and Lubar (1984) conducted an experiment with six males with ADHD whose ages ranged between 10 and 19 years, to investigate whether Neurofeedback

improved school performance. The subjects were described as having varying degrees of learning difficulties, hyperactivity and attentional deficits. Neurofeedback treatment consisted of either increasing 12-15 Hz sensorimotor rhythm (SMR) or 16-20 Hz beta activity over the sensory motor strip, while suppressing theta activity. In this study, treatment was combined with academic training, including reading, arithmetic, and spatial tasks. Neurofeedback treatment was conducted twice a week for 10 to 27 weeks (Lubar & Lubar, 1984).

At the end of the experiment, all children were reported to have successfully increased their SMR or beta waves, and decreased slow-wave activity and muscle activity in their EEG; as determined by a post-hoc analysis of their brain electrical activity (Lubar & Lubar, 1984). All subjects demonstrated considerable improvement in their schoolwork in terms of grades or achievement test scores on the Metropolitan Achievement Test, the Peabody, the Stanford Achievement test, and the California Achievement Test. None of the subjects required medications for hyperactivity after the study. The Lubars (1984) concluded that individual results for each subject indicated that Neurofeedback treatment, if applied comprehensively, could be highly effective in helping children who experienced attention deficit disorders and difficulties with academic tasks (Lubar & Lubar, 1984).

Tansey (1984) used a Neurofeedback treatment regime, which attempted to redress pathological interhemispheric dysfunction in six learning disabled boys ranging in age between 10 years 2 months and 11 years 10 months. Neurofeedback treatment of the sensorimotor rhythm consisted of increasing 14 Hz burst patterns over the central romantic cortex and was conducted in weekly 30-min training sessions. The results replicated and extended earlier findings by Tansey and Bruner (1983) that operant conditioning of increases in amplitude of the sensorimotor rhythm had a positive effect on learning disability in a ten-year-old boy. In this study, the training appeared to increase bilateral sensorimotor transactions, resulting in substantive reduction and/or remediation in the learning disabilities of the participants (Tansey, 1984).

Tansey (1985) observed discrete brainwave frequencies during Neurofeedback, a procedure that he thought might be reflective of the brain's functional neurophysiology. Eight boys, ranging in age between 7 years 11 months and 15 years 3 months, were provided with long-term SMR biofeedback training until their learning disabilities were remediated. Concurrently, five narrow frequency bands of brainwave activity, (5 Hz, 7 Hz, 10 Hz, 12 Hz and 14 Hz), were simultaneously recorded from one active electrode equidistant from reference and ground. These individual frequency recordings were intended to provide a glimpse of the brain's global response. It was thought that these measures would reflect the dynamic and synergistic processes involved in neural activation of the sensorimotor sub-networks during SMR training. In subjects with a Full Scale I.Q. between 76 and 85 there was a tendency for slow-wave activity to decrease as fast-wave activity increased. For subjects with a Full Scale I.Q. between 102 and 116, there were increased amplitudes over most of the five bands. However, the increased amplitudes were much less at the slower frequencies. In the four subjects who had either a significant Verbal>Performance I.Q, or Performance>Verbal I.Q, there was a 40% greater increase in the lower of the Verbal

or Performance I.Q. scores. This also indicated that the SMR training protocol used resulted in changes towards normalisation in functional interhemispheric asymmetry associated with the learning disabilities (Tansey, 1985).

Tansey (1985) administered 33 sessions of SMR training to a 14-year-old girl with a long history of absence seizures that were occurring at the rate of 4-5 per hour, sudden rages, spatial disorientation, attention deficits and academic difficulties. Biofeedback consisted of rewarding increases in 14-Hz neural discharges over the central rolandic cortex. Increases in SMR from operant conditioning resulted in a total cessation of the girl's absence seizures. Her sudden rages, spatial disorientation, attending and academic functioning also improved substantially (Tansey, 1985)

Tansey (1990) assessed the effectiveness of an SMR Neurofeedback treatment protocol in 24 children with learning disabilities, aged between 7 years 4 months and 15 years 6 months. All subjects had high theta/beta power ratios prior to treatment. Following SMR training, slow-wave activity decreased in overall power, 14-Hz SMR power activity increased and theta/beta power ratios decreased significantly. In addition WISC-R profiles normalised along with a significant remediation of the learning disorders (Tansey, 1990). In a follow-up paper, Tansey (1991) reported that 22 of the 24 subjects manifested increases in full-scale I.Q. scores on the WISC-R of at least 15 points, with the remaining two obtaining an increase of 13 and 14 points respectively. Tansey (1991) suggested that these results are consistent with an increase in bi-hemispheric skills, and complementary verbal-expressive and visuomotor abilities, which are prerequisite for successful learning, for the acquisition of reading and for the integration of higher-order learning (Tansey, 1990, 1991).

In three separate experiments, Lubar and colleagues (1995b) assessed the effectiveness of Neurofeedback treatment for 42 ADHD children and adolescents, aged 8-19 years, on both objective and subjective measures. In the first experiment, using 19 subjects, those who successfully decreased theta activity showed significant improvements in the Test of Variables of Attention (TOVA). In the second experiment, using 13 subjects, significant improvements in parent evaluations on the Attention Deficit Disorders Evaluation Scale (ADDES) were obtained following Neurofeedback treatment. In the last experiment, significant increases in Wechsler Intelligence Scale for Children-Revised (WISC-R) scores were obtained following Neurofeedback treatment. Lubar and colleagues (1995b) suggested that the findings of these three studies indicate that Neurofeedback treatment can be an appropriate and effective treatment for children with ADHD, as it significantly increased their cognitive skills, and decreased their attention deficits (Lubar, Swartwood, Swartwood, & O'Donnell, 1995b).

Thompson and Thompson (1998) reviewed the evaluation and treatment charts of 111 consecutive clients diagnosed with ADHD in their clinic. The clients, 98 children, aged between 5 and 17 years, and 13 adults aged between 18 and 63 years, attended 40 Neurofeedback sessions of 50 min duration, combined with the teaching of metacognitive strategies. Reward during Neurofeedback treatment was contingent mostly on suppressing slow-wave theta (4-7 Hz) activity and occasionally alpha (9-11 Hz) whichever was the most deviantly elevated, as well as increasing fast-wave activity (15-18 Hz) for most clients. However, clients with impulsivity and hyperactivity were

initially trained on a protocol that required them to increase SMR (13-15) Hz (Thompson and Thompson, 1998).

Metacognitive strategies relating to academic tasks were taught when feedback criteria were met, indicating that the clients were able to focus and hence were most receptive to learning. The treatment outcomes indicated that although 30% of the children were taking Methylphenidate at intake, only 6% remained on stimulant medications by the end of treatment. Significant improvements were found in ADHD symptoms, on intelligence scales, and in academic performance. The average gain for the Full Scale I.Q. equivalent score was 12 points. A decrease in the theta/beta EEG ratio was also observed. The positive outcomes of decreased symptoms of ADHD, combined with the academic and intellectual functioning improvements suggest that Neurofeedback combined with teaching of metacognitive strategies as an adjunct are a useful combination in the treatment of ADHD (Thompson and Thompson, 1998).

Most Neurofeedback treatment has been carried out in University or clinical settings. However, in a study by Boyd and Campbell (1998), six middle school students diagnosed with ADHD, aged between 13 and 15 years, underwent SMR biofeedback training in a school setting. Prior to and post-training, they were evaluated with the WISC-III Digit Span subtest and the TOVA, following a 72-hour medication-free period. Five of the subjects received 20 sessions of SMR biofeedback and one received nine sessions. Five out of the six subjects improved on their WISC-III Digit Span performance and their TOVA inattention and impulsivity scores. These results supported previously reported findings that Neurofeedback can be effective in the treatment of ADHD, and, in addition, demonstrated that Neurofeedback can be used in a school setting (Boyd & Campbell, 1998). Neurofeedback has also been conducted in home setting.

Rossiter (1998) reported on self-administered Neurofeedback for ADHD patients conducted in their own home. The first ten sessions were used to train the adult patients or parents of younger children on how to use the Neurofeedback equipment which consisted of inexpensive, easy to operate, 1 or 2 channel Personal Optimisation Devices (POD) manufactured by Lexicor Medical Corporation. The remaining 50 sessions were conducted at the patients' homes. Further therapist involvement was to monitor treatment and to make changes in the treatment protocol as necessary. Results from the initial six patients, aged 7- 45 years, were reported. Prior to Neurofeedback treatment, 13 of 24 standardised TOVA measures (attention, impulsivity, reaction time, and variability in the reaction time) were below average at baseline. After 30 Neurofeedback sessions, only five TOVA variables remained below average. Rossiter (1998) concluded that home based self-administered Neurofeedback may be an effective alternative to therapist-directed treatment for many ADHD patients and can be delivered at a substantially lower cost to clients (Rossiter, 1998).

Egner and Gruzelier (2001) recruited healthy volunteers to help assess the relationship between specific Neurofeedback treatment and electrocortical measures associated with the attentional system. Results indicated that the operant conditioning enhancement of SMR (12-15 Hz) component was associated with a reduction in commission errors and improved perceptual sensitivity on a Continuous Performance Task. However, they found the opposite relation for low beta (15-18 Hz) enhancement,

when SMR effects were controlled. Nonetheless, both 12-15 Hz and 15-18 Hz enhancements were associated with significant increases in P300 event-related potential (ERP) amplitudes in an auditory oddball task. They concluded that the results could be interpreted as stemming from band-specific effects of EEG operant conditioning on perceptual and motor aspects of attentional measures (Egner & Gruzelier, 2001)

Vernon and colleagues (2003) postulated that given the association between theta activity (4-7 Hz) and working memory performance, and between SMR activity (12-15 Hz) and attentional processing, enhancement of either of these frequencies might specifically influence particular aspects of cognitive performance. They investigated the possibility by training healthy individuals to either increase SMR or theta activity, and compared their performances to those of a non-Neurofeedback control-group. The results revealed that after eight sessions of Neurofeedback, participants in the SMR-group were able to enhance their SMR activity selectively, as indexed by increased SMR/theta and SMR/beta ratios. In contrast, those trained to enhance theta activity had no remarkable changes in their EEG. Additionally, the SMR group exhibited a clear and significant improvement in cued recall performance when using a semantic working memory task. The SMR group also exhibited (to a lesser extent) improved accuracy of focused attentional processing when using a 2-sequence Continuous Performance Task. Vernon and colleagues (2003) concluded that normal healthy individuals could learn to increase specific components of their brain electrical activity. This may be associated with the enhancement of brain connectivity responsible for cued recollection, which might facilitate semantic processing in a working memory task and to a lesser extent focused attention. The control group, who were required to increase theta activity showed no such improvements (Vernon et al., 2003).

One hundred children, aged 6 to 19, with a diagnosis of ADHD of either inattentive or combined subtypes, participated in a one-year, multimodal, outpatient program that included Methylphenidate, parent counseling, and a standardised academic support plan at school. Fifty-one of the participants also received Neurofeedback. Post-treatment assessments were conducted both on and off stimulant medication. When tested on Methylphenidate, participants demonstrated significant improvements on the TOVA and Attention Deficit Disorder Evaluation Scales (ADDES). When tested while off Methylphenidate, only participants who received Neurofeedback treatment sustained these gains and had significant reductions in theta/beta power ratios at Cz. Parenting style exerted a significant moderating effect on behavioural symptoms at home but not at school (Monastra et al., 2002).

Kropotov and colleagues (2005) involved eighty-six children with ADHD, aged 9 to 14, in a study during which event-related potentials (ERPs) were recorded in auditory GO/NO-GO task before and after 15 to 22 sessions of Neurofeedback. Each session consisted of 20 minutes of enhancing the ratio of the EEG power in the 15 to 18 Hz band to the EEG power in the rest of spectrum, and 7–10 min of enhancing of the ratio of the EEG power in 12 to 15 Hz band to the EEG power in the rest of spectrum. Bipolar electrode-placement at C3-Fz was used for enhancing power in the first protocol and C4-Pz in the second protocol. Based on quality of performance

during training sessions, the patients were divided into two groups: good performers and bad performers. For GO and NO-GO cues, good performers showed increases in the amplitude of positive ERP components within 180–420 ms. However, no statistically significant differences between pre- and post-training ERPs were observed for bad performers. The ERP differences between post- and pre-treatment conditions for good performers were distributed over fronto-central areas, and appeared to reflect an activation of frontal cortical areas associated with beta training (Kropotov et al., 2005).

Strehl et al (2006) investigated the effects of self-regulation of slow cortical potentials for children with attention-deficit/hyperactivity disorder. They reported electroencephalographic data from the training and the 6-month follow-up on twenty-three children with attention-deficit/hyperactivity disorder aged between 8 and 13 years, as well as changes in their behavior and cognition. Measurement before and after the trials showed that children with attention-deficit/hyperactivity disorder learn to regulate negative slow cortical potentials. After training, significant improvement in behavior, attention, and IQ score was observed. The behavior ratings included DSM criteria, number of problems, and social behavior at school. The cognitive variables were assessed with the Wechsler Intelligence Scale for Children and with a computerized test battery that measures several components of attention. All changes proved to be stable at 6 months' follow-up after the end of training.

Neurofeedback in ADHD: controlled experimental studies

Much of the early criticism of Neurofeedback have been that there were no controls to demonstrate that the treatment effects did not result from placebo effects, or the extensive attention received during treatment, the high expectations of parents or merely from attending to the computer task. These concerns were addressed in the following studies.

Linden and colleagues (1996) confirmed the findings of Lubar and colleagues (1995b) in a controlled study, which investigated the effects of Neurofeedback on cognition and behaviour with 18 ADHD children, aged between 5 and 15 years. For the experimental group, training consisted of enhancing beta-activity and suppressing theta-activity. The control group received no Neurofeedback training. The Neurofeedback group demonstrated increases on the Kaufman Brief Intelligence Test and reduced inattentive behaviours on the Behaviour Rating Scale, compared to the controls (Linden et al., 1996).

Following traumatic brain injury, reports of Attention Deficit Disorder are commonplace (Duff, 2004; Hirshberg et al., 2005; Max et al., 1998). Adults diagnosed with mild traumatic brain injury (mTBI) or ADHD were treated with Neurofeedback and cognitive retraining for their attention deficits (Tinius & Tinius, 2001). The waiting-period control group consisted of subjects who did not receive Neurofeedback Training.

Psychological and neuropsychological tests were completed at pre- and post-treatment and compared to the control group who were also tested on two occasions

with an interval matching that of the training period (Tinius & Tinius, 2001). Using the IVA, a computer administered Continuous Performance Task; significant improvements were found on attention and response accuracy in both the mTBI and ADHD groups compared to the control group. A self-report showed a significant decline in symptoms in the mTBI and ADHD groups but not in the control group. However, errors on a problem-solving task decreased only in the mTBI group. The combination of Neurofeedback and the cognitive retraining protocol used in this study resulted in significant improvement in the sustained attention of individuals diagnosed with mTBI and ADHD after twenty treatment sessions compared to controls (Tinius & Tinius, 2001).

Carmody and colleagues (2001) conducted a Neurofeedback study, on site, in an elementary school with 16 unmedicated schoolchildren with ADHD or attention deficits. Eight children, aged between 8 and 10, were assigned to the experimental group and completed 35 to 47 sessions of Neurofeedback Training over a six-month period. Four participants in the experimental group were diagnosed with ADHD and four were not. The other eight children were assigned to a waiting list control group matched to the experimental group on age, grade, teacher, and diagnosis. Results, as assessed by the TOVA, indicated that the experimental group reduced the number of errors of commission and anticipatory errors made, indicating a reduction in impulsivity. Teacher reports indicated improvements in attention but no changes in impulsivity and hyperactivity. No changes were observed in the waiting-period group control (Carmody, Radvanski, Wadhwani, Sabo, & Vergara, 2001).

Fernandez and colleagues (2003) postulated that since children with Learning Disabilities have higher values of theta EEG absolute and relative power than normal children, and that minimal alpha absolute power is necessary for adequate performance, then training a decrease in the theta/alpha power ratio may reduce learning difficulties. TOVA, WISC-III and EEG were administered to ten children with Learning Difficulties and with higher than normal theta/alpha power ratios. They were then divided into two groups, each with similar socioeconomic status, I.Q. and TOVA values. The five children in the experimental group received Neurofeedback to promote a reduction in theta/alpha ratio, at a rate of two half-hour sessions per week for 10 weeks, at a site with the highest theta/alpha power ratio. Non-contingent (sham) reinforcement was given to the control group. TOVA, WISC-III and EEG measures were obtained at the end of the 20 sessions. WISC-III performance improved and EEG absolute power decreased in all clinical bands only in the experimental group. Children in the waiting-period control group showed only a decrease in relative power in the delta band. Thus, results indicated improvements in cognitive performance and EEG changes towards normalisation in the experimental group only (Fernandez et al., 2003).

Heinrich et al (2004) examined the behavioural and neurophysiological effects of slow cortical potential training using a wait list control group. Twenty two children with ADHD between 7-13 years, half of whom were on medication, completed 25 sessions of 50 minutes duration over a three week period. They found a 25% decrease in ADHD ratings after training, together with Continuous Performance Test improvement with a decrease in impulsivity and an increase in the amplitude of the contingent negative variation.

Levesque et al (2005) followed up the finding that individuals with ADHD have shown abnormal functioning of the anterior cingulate cortex (ACC) during tasks involving selective attention. They conducted an fMRI study to measure the effect of Neurofeedback on the neural substrates of selective attention in children with AD/HD. Twenty ADHD children not taking any psychostimulant and without co-morbidity participated in a between groups design comparing neurofeedback (EXP) with no treatment (CON). Subjects from both groups were scanned one week before the beginning neurofeedback (Time 1) and one week after the end of this training (Time 2) while they performed a Counting Stroop task. At Time 1, for both groups, the Counting Stroop task was associated with significant loci of activation in the left superior parietal lobule and no activation was noted in the ACC. At Time 2, for both groups, the Counting Stroop task was still associated with significant activation of the left superior parietal lobule. This time, however, for the EXP group only there was a significant activation of the right ACC, suggesting that in ADHD children, neurofeedback has the capacity to normalize the functioning of the ACC, the key neural substrate of selective attention.

Gevensleben and colleagues (2009) evaluated the clinical efficacy of neurofeedback therapy in children with ADHD in a multi-site randomised controlled study using a computerised attention skills training (AST) software as the control condition. One hundred and two children with ADHD, aged 8 to 12 years, (diagnosed by a Child and Adolescent Psychiatrist and Psychologist as meeting DSM-IV criteria for ADHD), were randomly assigned to one of two groups with no mean differences in pre-treatment demographic, psychological or clinical variables (Gevensleben et al., 2009). Children with co-morbid disorders, other than Conduct Disorder, Anxiety, Depression, Tic disorder and Dyslexia, were excluded from the study. None of the participants had gross neurological or other organic disorders; all were drug-free and without psychotherapy for at least 6 weeks prior to the start of training and 87 of the participants were drug-naive. Prior to training, several behaviour rating scales, including the German ADHD rating scale (FBB-HKS) the Strength and Difficulties Questionnaire (SDQ) were completed by parents and teachers, and these were repeated at an intermediate point and post-training. To control for parental expectations and satisfaction with the treatment, placebo evaluation scales were used. Training for each group consisted of two three to four week blocks of 18 sessions each (conducted as nine double sessions of about 50 minutes per session, separated by a short break. Two to three double sessions per week were used, to accommodate for the weekly schedule of the families. Pre-training assessment was conducted during the week prior to training, while intermediate and post-training assessments were done about one week after the last session of the training blocks. Neurofeedback Training consisted of a block of theta/beta training, and of a block of Slow Cortical Potential (SCP) training in a balanced order. On completion of the study, improvements in parent and teacher ratings were superior in the Neurofeedback group compared to those in the control group. The effect size was 0.60 for the parent rated scale, and 0.64 for the teacher-rated scale for the FBB-HKS total score of primary outcome measure of hyperactivity, impulsivity and attention. Comparable effects were obtained for theta/beta training and the SCP training protocols. Statistics revealed a trend towards better improvements in the FBB-HKS total score, when theta/beta training preceded

SCP training (F (1,50) = 3.00; $p < 0.1$). Parental attitudes towards either treatment groups or controls did not differ (Gevensleben et al., 2009).

Gevensleben and colleagues (2009) argued that since parents of the Neurofeedback groups and the control group did not differ in expectations or in satisfaction with treatment, parental expectation factors should not have influenced the outcome. Thus, they concluded that non-specific factors did not account for the clinical outcome and that the superiority of the combined NF training indicates clinical efficacy of NF in children with ADHD. They recommended that future studies should investigate how to optimise the benefits of Neurofeedback as a treatment for ADHD, and study the specificity of effects, resulting from SCP training or theta/beta training (Gevensleben et al., 2009).

Neurofeedback in ADHD: comparisons with stimulant medication

Lubar and colleagues (1995a) measured the QEEG of 23 individuals with ADHD. They examined theta/beta ratios of the ADHD participants, both with and without medication, and found no significant effect of stimulant medication on the theta/beta ratios in the QEEG at all 19 sites evaluated. They concluded that Methylphenidate had very little effect on theta/beta ratios (Lubar et al., 1995a). Methylphenidate and other stimulant medications used to enhance attention produce state-dependent effects. This means that the medication works while it is in the system but that there is virtually no long-term carry-over to the non-dependent state. On the other hand, Neurofeedback works not only while doing training, but has a carry-over effect that lasts for a very long time, perhaps even a whole lifetime (Lubar et al, 1997).

Rossiter and La Vaque (1995) compared the effects of Neurofeedback to stimulant medication in reducing ADHD symptoms. The study compared the effects of a medical treatment program, to 20 sessions of Neurofeedback. Each group had 23 participants who were matched by age, I.Q, gender and diagnosis. The Test of Variables of Attention (TOVA) was administered pre- and post-treatment. Both groups improved significantly on TOVA measures of inattention, impulsivity, reaction time, and variability in the reaction time, and did not differ from each other on TOVA change scores. Rossiter et al (1995) suggested that Neurofeedback was an effective alternative to stimulants and may be the treatment of choice when medication is ineffective, or produces unacceptable side effects, or when compliance to medication is a problem (Rossiter & La Vaque, 1995). Following criticisms of their statistical methodology, Rossiter (2004a) re-analyzed the data to control the experiment-wise alpha level for multiple comparisons. All planned comparisons for which significant differences were predicted met their adjusted alpha levels for significance with the experiment-wise α = .05. Equivalence/non-inferiority testing indicated that the proportion of the Neurofeedback group significantly improved, was non-inferior, but not-equivalent to that of the medication group (Rossiter, 2004a).

Fuchs (1998) compared the effectiveness of Neurofeedback to stimulant medication in 22 children, 8 to 12 years of age, from a Social-Pediatric Hospital, with a primary diagnosis of ADHD. Half of the participants were assigned to a Neurofeedback

experimental group and the other 11 were assigned to a control group matched in age and sex. The Neurofeedback group received thirty 45-minutes sessions of Neurofeedback to enhance SMR and/or beta activity and suppress theta activity, over a period of 10 weeks. The control group of children with ADHD was optimally medicated with Methylphenidate. No other psychological treatment or medication was administered to either group. Both groups were administered a test battery consisting of: an I.Q. test (HAWIK-R), a TOVA, a paper-pencil-test (d2) and the IOWA Conners Behaviour Rating Scales (parent and teacher version), pre- and post-treatment. Results indicated that the children in both the Methylphenidate and Neurofeedback conditions showed comparable and significant improvements in attention and concentration abilities in the objective (d2 and TOVA), and subjective (Conners Behaviour Rating Scales) measurements. Performance I.Q. scores also improved significantly in both groups. Fuchs (1998) reported that while Methylphenidate and Neurofeedback had comparable treatment effectiveness, the gains from Neurofeedback were expected to be permanent, while the gains from medication were expected to be dependent on the continuation of medication treatment (Fuchs, 1998).

One hundred children diagnosed with ADHD, either inattentive or combined subtype, aged 6-19 years, participated in a one-year, multimodal, outpatient program that included Methylphenidate, parent counseling and academic support at school (Monastra et al., 2002). In addition, 51 of the children also received neurofeedback. Pre- and post-treatment assessments were conducted both while on and off stimulant therapy. Significant improvements were noted on the TOVA and the Attention Deficit Disorders Evaluation Scale (ADDES) (Adesman, 1991), while participants were on Methylphenidate. However, only those who had received neurofeedback sustained these gains when tested while off Methylphenidate. Only children who had received neurofeedback had reductions in theta/beta power ratio at the Vertex, and these changes were statistically significant. ADDES behavioural measures indicated that parenting style exerted a significant moderating effect on the expression of behavioural symptoms at home and not at school (Monastra et al., 2002). Arns and colleagues (2009) cautioned that the results from the Monastra et al (2002) study needed to be interpreted with care, as the study only included subjects with an elevated theta/beta ratio, thereby potentially selecting for those subjects with ADHD who would benefit most from Neurofeedback treatment. The subjects in that study might therefore not have been representative of the general ADHD population (Arns et al., 2009).

Fuchs and colleagues (2003) selected 34 children, aged 8-12 years with a diagnosis of ADHD to participate in a study comparing the effects of a 3-month Neurofeedback treatment program to Methylphenidate (Fuchs et al., 2003). The children were from families of heterogeneous socioeconomic backgrounds with diagnoses made by two independent clinicians: either a Child Neurologist or a Pediatrician together with a Child and Adolescent Clinical Psychologist. None of the children had received any kind of treatment for their ADHD prior to selection for participation in the study. Twenty-two of the participants were assigned to the Neurofeedback group and 12 to the Methylphenidate group according to their parents' preferences. Neurofeedback training consisted of rewarding SMR (12-15 Hz) and

beta activity (15-18 Hz). Behavioural measures were assessed using the German version of the IOWA-Conners Behaviour Rating Scale, and were completed by each child's teacher and both parents prior to, and after Neurofeedback. Teachers were blinded to the choice of treatment group while parents were not (Fuchs et al., 2003). Both Neurofeedback and Methylphenidate were associated with improvements on all subscales of the TOVA, and on the speed and accuracy measures of the "d2" Attention Endurance Test (Fuchs et al., 2003). Furthermore, ADHD behaviours were significantly reduced in both groups, as rated by teachers and parents on the IOWA-Conners Behaviour Rating Scale. The authors concluded that Neurofeedback was as effective in improving the behavioural problems of children with ADHD as stimulant medication (Fuchs et al., 2003). Further evidence of the effectiveness of Neurofeedback, when compared with Methylphenidate came from Rossiter (2004a, 2000b), who replicated an earlier study (Rossiter & La Vaque, 1995).

The replication used a larger sample of children with ADHD, with a wider age range, improved statistical analysis and more comprehensive behavioural data. Thirty-one patients who chose Methylphenidate treatment were matched with 31 patients who chose Neurofeedback treatment. Of the Neurofeedback patients, 14 received training in the clinic while the remaining 17 received training in their own home. This study design is one described by Kazdin (2003) as an "effectiveness research design" whereby patients choose assignment to either the Neurofeedback, experimental group, or the Methylphenidate, active treatment control group (Kazdin & Nock, 2003). The Methylphenidate dose was titrated for optimum effect using the TOVA. Both groups showed statistically and clinically significant improvements on TOVA measures of attention, impulse control, processing speed, and variability in reaction time. Clinically significant gains were made by the Neurofeedback and Methylphenidate groups based on the percentage of patients showing significant improvement over baseline (84% in each). There were large effect sizes for Neurofeedback (1.01-1.71) and Medication (0.80-1.80), and the percentage of individual TOVA scores showing significant improvement (Neurofeedback: 55%, Methylphenidate: 56%). Post-treatment mean scores for both the Neurofeedback and the Methylphenidate groups fell within the average range of functioning. Both groups had clinically significant improvement in behaviours based on their large effect size. The Neurofeedback group had effect size (1.15–1.75) on the Behaviour Assessment System for Children (BASC) and (1.59) for the Brown ADD Scale. There were no statistically significant differences in the TOVA gain scores between the Neurofeedback and Methylphenidate groups, and the proportion of patients in the Neurofeedback group that significantly improved behaviourally was equivalent to that of the medication group. Confidence interval and non-equivalence null-hypothesis testing confirmed that the Neurofeedback program produced patient outcomes equivalent to those obtained with Methylphenidate (Rossiter, 2004a,2004b).

Studies Indicating Levels of Efficacy of Neurofeedback

The preceding review has outlined many of the case and controlled group studies conducted over the past thirty years reporting the effects of EEG biofeedback in ADHD. They generally report improved attention and behavioural control, increased cortical activation on quantitative electroencephalographic examination, and gains on

tests of intelligence and academic achievement in response to this type of treatment. Monastra et al (2005) critically examined the empirical evidence of the studies available at that time, applying the efficacy guidelines jointly established by the Association for Applied Psychophysiology and Biofeedback (AAPB) and the International Society for Neuronal Regulation (ISNR). On the basis of these scientific principles, they concluded at the time that EEG biofeedback was determined to be "probably efficacious" for the treatment of ADHD. Sherlin et al (2010) note that the main concerns were the lack of well-controlled, randomized studies; the small group sizes; and the lack of proof that the EEG feedback is solely responsible for the clinical benefit and not nonspecific factors such as the additional time spent with a therapist or ''cognitive training.'' In 2006, Holtmann and Stadtler concluded that EEG biofeedback has gained promising empirical support in recent years, but there was still a need for more empirically and methodologically sound evaluation studies.

Cohen and Jackson (2006) reported a meta-analysis of neurofeedback therapy in ADHD and acquired brain injury (ABI) at the National Conference of the APS College of Clinical Neuropsychologists. Twenty two papers met inclusion criteria, covering 26 studies and including over 1400 clients with ADHD. Study inclusion criteria were that (1) they be written in English, (2) were published in a peer reviewed journal between 1990 and 2004, (3) used a control group or used a within-subjects design, (4) reported at least one test or rating scale analyzing attention or other aspects of cognition, (5) used neurofeedback therapy, (6) incorporated statistical analysis sufficient for the derivation of at least one effect size from a dependent variable, and (7) reported original results not reported elsewhere. The study found that 59% to 67% of all effect sizes in relation to cognitive and behavioural measures were either Medium or Large.

Since 2006, new research has been published investigating the clinical efficacy of neurofeedback for the treatment of ADHD. The most influential publication has been the meta-analysis on neurofeedback in ADHD by Arns et al. (2009), which assessed efficacy against the standards published by the American Psychological Association (APA). The APA deem five levels of clinical efficacy, ranging from Level 1 for those interventions whose support derives from anecdotal evidence or non-peer reviewed case studies ("Not Empirically Supported"), to Level 5 for those interventions that are shown to be statistically superior to credible placebo therapies or to actual treatments and as shown in two or more independent studies ("Efficacious and Specific"). Intervening levels reflect interventions that are Possibly Efficacious (Level 2), Probably Efficacious (Level 3) and Efficacious (Level 4). The meta-analysis conducted by Arns et al (2009) concluded that neurofeedback for ADHD met Level 5 standards as Efficacious and Specific and that neurofeedback had large effect sizes (large clinically relevant effect) on the domains of impulsivity and inattention and a medium (medium clinically relevant) effect size on hyperactivity.

The Arns et al (2009) meta-analysis included 15 studies and 1,194 patients with ADHD. Six studies employed randomization allocation of subjects and three studies compared neurofeedback with stimulant medication, the then accepted "gold standard" in the treatment of ADHD. Study inclusion criteria were stricter than those employed by Cohen and Jackson (2006) in that they required treated clients to have a primary diagnosis of ADHD/ADD and either (1) controlled between subject designs

using a passive (e.g. waiting list) or active (e.g. stimulant medication, biofeedback, cognitive training) control groups) either randomised or not or (2) prospective within subject design studies or (3) retrospective within subject design studies with a large enough sample to provide a reliable representation of daily practice (N>500). Neurofeedback was also required to be provided in a standardised manner with no more than two treatment protocols. Further standardised pre- and post assessment means and standard deviations for at least one of the following domains: Hyperactivity, Inattentiveness or CPT commission errors. When means were not available they were requested from the authors. These differences in inclusion criteria meant that there was minimal overlap between the studies included in the two meta-analyses, with only three studies incorporated by Cohen and Jackson (2006) included in Arns et al (2009) meta-analysis. Further, the Arns et al (2009) study included twelve studies from the period 2003 to 2009 not included in the Cohen and Jackson (2006) study, either because they were missed by the latter study or were published later.

The pre- and post-assessment measures collected from the studies included in the Arns et al (2009) meta-analysis were: (1) Hyperactivity assessed with a DSM rating scale such as the Conners (CPRS-R); ADDES-Home, BASC, SNAP, FBB-HKS (parents) or DSM-IV Rating Scale (Lath and Schlottke); (2) Inattention: assessed with an inattention rating scale such as FBB-HKS, Conners (CPRS-R, BASC, ADDES-Home, SNAP/Iowa-Conners) or DSM-IV Rating Scale; (3) Impulsivity: commission errors on a CPT such as a TOVA, IVA (auditory prudence measure) or a G0-NoGo test. These measures were used as treatment end points.

Two randomized controlled trials, both published in 2009, were the key studies on the basis of which the efficacy rating could be lifted to Level 5: the randomized multicenter study from Gevensleben, Holl, Albrecht, Vogel, et al. (2009) and the randomized study from Holtmann et al. (2009). Both of these studies used sound methodological designs, employed randomization, and implemented semiactive control groups, which can be considered a credible placebo control. The study by Gevensleben, Holl, Albrecht, Schlamp, et al. (2009) consisted of a large sample size (N=94). Therefore, in line with the APA guidelines, neurofeedback was shown to be superior to a credible placebo control, which was demonstrated in two independent research settings, thereby meeting Level 5.

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