The effects of neurofeedback home training on typically developed and children with Attention Deficit Hyperactivity Disorder (ADHD): A feasibility study

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Declaration

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Abstract

Attention Deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental condition characterised by the inability to pay attention, inability to control impulsive behaviour and excessive hyperactivity, with prevalence rates in the UK at approximately 3 to 8% (National Institute of Clinical Excellence, 2018). The most common and accepted forms of treatment for ADHD are stimulant medication and behaviour therapy. However, stimulant medication only has a positive effect in approximately 65% of children (Johnston, Coghill, Matthews, & Steele, 2015) with as many as 15% of ADHD patients suffering from side effects including blunting of personality, headaches, lack of appetite (Fox, Tharp, & Fox, 2005). Consequently, a need has been identified for a new treatment with improved long-term effects (Arns, Heinrich, & Strehl, 2014). Evidence suggests that neurofeedback, a brainwave training programme, can normalise electroencephalogram (EEG) patterns and reduce inattentive and impulsive symptoms as a long-term strategy in ADHD (Vernon, Frick, & Gruzelier, 2004). There is a growing need for treatment, specifically neurofeedback, to be accessible at home (Vernon et al., 2004; Rutterford, Anderson, & Venables, 2008) but the effect of this is yet to be investigated. This was the main purpose of the present thesis.

The aim of this research was to test the feasibility of neurofeedback home training in both ADHD and typically developed population as well as stimulant medication in combination with neurofeedback home training in an ADHD sample. It was of particular interest to understand the effect of neurofeedback home training on personality, EEG measures, and neuropsychometric measures of inattention and impulsivity. The typically developed sample were randomly allocated to: (a) control group, sample size 15 participants (b) sensorimotor rhythm uptraining neurofeedback home training, sample size 16 participants, or (c) active control group, sample size 16 participants. The ADHD sample were randomly allocated to: (a) stimulant medication, sample size 19 participants, (b) stimulant medication and neurofeedback home training, sample size 8 participants, (c) stimulant medication and neurofeedback in clinic, sample size 4 participants, (d) neurofeedback home training, sample size 3 participants. The ADHD sample completed EEG informed neurofeedback. In both samples, 30 sessions of neurofeedback were completed. Dependent variables, which consisted of personality measures, concentration and impulsivity scales and EEG, were conducted pre- and post-intervention, then compared to assess the affect of the interventions.

The main results were that: (i) ADHD sample were significantly different to typically developed peers when rated by parents and on CPT, differences were found as expected on personality and EEG measures, but were not significant, (ii) stimulant medication significantly improved executive function, defiance, inattention, hyperactive and impulsive traits when rated by parents in an ADHD population, (iii) neurofeedback in clinic and home training did not significantly effect concentration, impulsivity, personality or EEG in a ADHD or typically developed sample. The work reported here calls into question the use of neurofeedback in the treatment of ADHD in a clinical setting. The present study made an original contribution to the neurofeedback field showing neurofeedback home training does not significantly affect concentration, personality of EEG, and contributes to existing knowledge about ADHD.

1 General Introduction

Our understanding of mental health difficulties and the impact it has on education, social functioning, relationships as well as the cost, has greatly expanded over the past few years. The UK has witnessed a shift in attitude from the government with investment taking place in transforming children's mental health services (Department of Education [DoE], 2017). The current government is committed to providing children and their families with the support they require. One condition specifically targeted by the Department of Health is Attention Deficit Hyperactivity Disorder (ADHD) of which over 132,000 children suffer severely in the UK (Department of Health [DoH], 2015).

As part of this thesis, a literature review was undertaken. Articles were searched for in the City, University of London, library catalogue in addition to ScienceDirect Literature search engine. Specific search terms included searching for articles regarding neurofeedback, ADHD, methylphenidate, Conners', personality and EEG. The criteria for including an article were that it was directly relevant to the thesis topic, was peer reviewed and the most recent article was discussed.

1.1 Introduction to Concentration and Impulsivity Difficulties

ADHD is a neurodevelopmental condition characterised by the inability to pay attention, inability to control impulsive behaviour and excessive hyperactivity. ADHD affects individuals of all ages, with prevalence rates as high as 14% among the general population, of which 70% of individuals continue to show symptoms into adulthood and affecting four times more males than females (Vernon, Frick, & Gruzelier, 2004). ADHD is a condition with a genetic contribution linked to abnormal levels of dopamine in the brain's neurotransmitters, a system involved in

the regulation of behavioural responses, which is exacerbated by environmental factors (Fuchs, Birbaumer, Lutzenberger, Gruzelier, & Kaiser, 2003).

Various management strategies are available to combat the deficits faced by individuals with ADHD, including psychoeducation for parents and children, Cognitive Behavioural Therapy (CBT), medication and neurofeedback (Halperin, & Healey, 2011; National Institute for Health and Care Excellence [NICE], 2018; Danforth, Harvey, Ulaszek, & McKee, 2006; Feingold, 1975; Barkley, 1998). Within this thesis, the most effective combination of treatments to improve symptoms of ADHD are investigated.

1.1.1 Diagnostic and Statistical Manual of Mental Health Disorders (DSM)

ADHD was first formally recognised in the second edition of the Diagnostic and Statistical Manual of Mental Health (American Psychiatric Association [APA], 1968), where the condition was described as hyperkinetic reaction of childhood. At this time, the conditions main symptom was excessive motor activity (APA, 1968).

The third edition of the DSM referred to the disorder as Attention Deficit Disorder, which consisted of two subtypes: with hyperactivity, and without hyperactivity (APA, 1980). The change in name was due to the belief that hyperactivity was not a common symptom of the disorder and to instead focus on inattention. The definition of the condition also expanded to examine the core symptoms, inattention and hyperactivity, independently of each other.

In the DSM-III-R (APA, 1987), the condition changed from Attention Deficit Disorder, with or without hyperactivity, to Attention Deficit Hyperactivity Disorder (ADHD) with a single diagnostic checklist. The change in name

emphasised the belief that the condition only existed with the presence of hyperactivity, reversing back to the original concept in the DSM-II (APA, 1968).

In 1994, following published research on the presence of ADHD without hyperactivity, the DSM-IV explained ADHD as consisting of the three subtypes that we know today: predominately inattentive, predominantly hyperactive-impulsive, and combined subtype (APA, 1994).

The Diagnostic and Statistical Manual of Mental Disorders, now the DSM-5, was most recently updated in 2013 where the condition continues to be referred to as ADHD (APA, 2013). Although the name has remained the same as the previous DSM-IV, there have been some minor changes in definition of the condition. In regard to the ADHD symptoms, additional examples of how symptoms may present themselves in adulthood have been given. The level of impairment that symptoms cause has been lowered to "reduce the quality of functioning" rather than being "clinically significant" (Epstein & Loren, 2013). The age of onset has been increased to twelve years old instead of seven, in addition to symptoms required for adult diagnosis, reflecting the growing understanding and acceptance of ADHD in adults (Barkley, 2003; Bresnham & Barry, 2002). These minor changes in the DSM-5 show that the previous DSM-IV has lasted well and remains current with the recent research (Epstein & Loren, 2013).

Internationally, there are strict criteria for diagnosis and guidance on how mental health conditions are managed. In accordance with the current DSM-5, in order for a diagnosis of ADHD to be made, symptoms need to be present for at least six months to the point that the severity of the symptoms are disruptive and inappropriate to the typical developmental and academic functioning. Symptoms

must be present before the age of twelve and seen in at least two settings, commonly home and school (APA, 2013).

However, some of the changes in the DSM-5 have not been positively received. As the DSM-5 requires fewer symptoms to fulfil a diagnosis, concerns have been raised regarding prevalence of ADHD, with an increase in diagnosis. Furthermore, suggestions have been made that lowering the age of onset may be less significant in diagnosis (Rigler et al., 2016).

In the DSM-5, the symptoms of ADHD are broken down into the following criteria:

Inattention:

- Often fails to give close attention to detail or makes careless mistakes in schoolwork, at work, or with other activities.
- Often has trouble holding attention on tasks or play activities.
- Often does not seem to listen when spoken to directly.
- Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g. loses focus, side-tracked).
- Often has trouble organising tasks and activities.
- Often avoids, dislikes, or is reluctant to do tasks that require mental effort over a long period of time (such as schoolwork or homework).
- Often loses things necessary for tasks and activities (e.g. school materials, pencils, books, tools, wallets, keys, paperwork, glasses, mobile phones).
- Is often easily distracted.
- Is often forgetful in daily activities.

Hyperactive/impulsive:

- Often fidgets with or taps hands or feet, or squirms in seat.
- Often leaves seat in situations when remaining seated is expected.
- Often runs about or climbs in situations where it is not appropriate (adolescents or adults may be limited to feeling restless).
- Often unable to play or take part in leisure activities quietly.
- Is often on the go acting as if driven by a motor.
- Often talks excessively.
- Often blurts out an answer before a question has been completed.
- Often has trouble waiting his/her turn.
- Often interrupts or intrudes on others (e.g. butts into conversations or games).

In any ADHD subtypes, six of the nine criteria must be met for a clinical diagnosis to be made (APA, 2013).

1.1.2 NICE Guidelines

In addition to the DSM-5, UK professionals also have the National Institute for Health and Care Excellence guidelines to adhere to (NICE, 2018). The most recent update of the NICE guidelines was in March 2018. The regular updates are to ensure that research findings are incorporated to provide the best care and treatment available for individuals with ADHD.

The NICE guidelines state that a diagnostic assessment should include an assessment of the individual's needs, any coexisting conditions considered, social and educational circumstances and a physical health examination. The NICE guidelines state that diagnosis should be made only by a specialist psychiatrist,

paediatrician, or other healthcare professional with expertise in ADHD. Rating scales can be valuable in contributing towards a clinical assessment, but diagnosis should not be made solely on the basis of such tool (NICE, 2018).

The most recent update of NICE guidelines has emphasised the importance of recognising ADHD in females. It notes that ADHD is thought to be under-recognised in females and consequently are less likely to be referred, are underdiagnosed and more likely to receive an incorrect diagnosis. Previous research has a lack of female presence, showing limited research considering gender differences. A possible reason for this is that historically, females have been excluded from research due to low recruitment numbers (Hasson & Fine, 2012).

1.2 Theories of Concentration and Impulsivity Difficulties

As previously discussed, concentration and impulsivity difficulties have been formally recognised in the UK since 1968 (APA, 1968) and there has been ongoing research to understand what causes these difficulties.

There are two main constructs underpinning ADHD: inattention and disinhibition. Inattention difficulties involve the inability to sustain attention while responding to tasks, to be able to follow instructions, and to resist distractions. Inattention difficulties are typically first seen ranging in age from 5 to 7. Disinhibition refers to a multidimensional construct whereby difficulties include inhibition to responses and heightened sensitivity to reward or excessive fear. Typically, difficulties with inhibition arise at the younger age of 3 to 4 years old (Barkley, 2003). These two constructs will now be discussed in more detail in relation to ADHD.

1.2.1 Barkley's Behavioural Response Inhibition Theory of ADHD

Barkley (1997) theorised that ADHD is a disorder based upon a deficit in inhibiting responses, the disinhibition construct. Specifically, Barkley stated that response inhibition consists of three processes. The first process is the ability to inhibit initial prepotent responses to a task. A prepotent response refers to a response with an immediate positive or negative reinforcement, exhibited as a reward seeking or avoidance behaviour. The second process is stopping a response that has already commenced. If someone has commenced a response but receives a signal that the response is ineffective, the behaviour needs to be interrupted and stopped. An example of stopping an ongoing response is the Wisconsin Card Sorting Task. The task involves changing responses to a more effective response when feedback has been provided. Children with ADHD often repeat the mistake they made, even when corrected. The third process is self-directed responses when a previously commenced response has been interrupted, also known as interference control. An example of interference control is the Stroop colour word task. Here, ADHD participants performed poorly when responding to the colour rather than reading the words. Barkley developed the theory in an attempt to create a unifying model of ADHD, based upon prior theories of neuropsychological brain functions, specifically creating a link between inhibition and executive functions (Barkley, 1997).

In turn, these three response inhibition processes exert control over 4 areas of executive functions: working memory, self-regulation, internalisation of speech, and reconstitution. These systems then have a direct downward effect, for example, behavioural inhibition effects working memory, which in turn effects motor control, fluency and syntax.

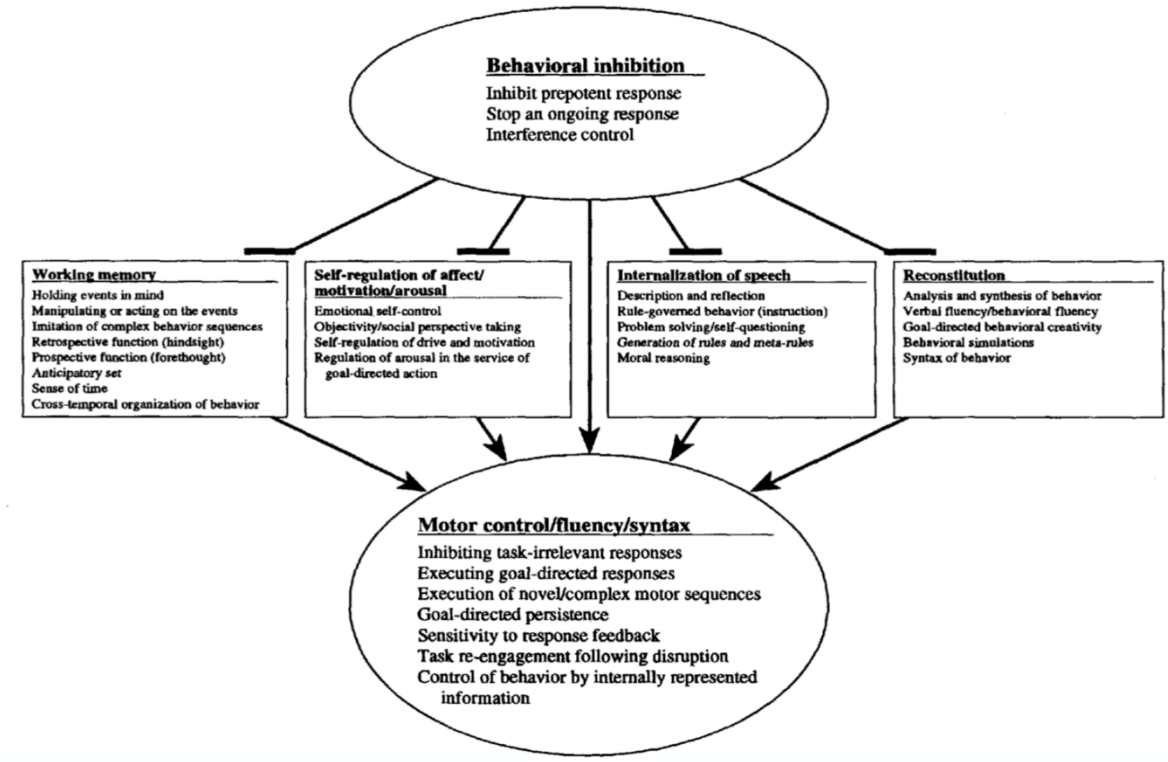

Figure 1. Behavioural Response Inhibition Theory of ADHD

Barkley supported that ADHD was caused by structural abnormalities in the prefrontal cortex and connections in the brain. Deficits in executive functions, the cognitive management system of the brain, overlap with ADHD symptoms (Barkley, 2003) with suggestion being made that ADHD is an impairment on the development of executive functions, specifically the ability to inhibit responses (Brown, 2006). In regards to ADHD inattentive subtype, Barkley (1997) stated that this is a distinct disorder and is not a subtype of ADHD as, in Barkley's opinion, it is not associated with executive functioning difficulties and has little in common with the other subtypes (Brown, 2006).

1.2.2 Brown's ADD Syndrome Model

Another theory to explain ADHD is the ADD Syndrome Model by Thomas Brown (2006). This theory states that ADD is caused by deficits in executive functions. The ADD Syndrome Model explains that there are six areas within

executive functions which cause impairment in ADD. These six clusters are as follows:

1. Activation: organizing, prioritising and activating for tasks.
    - Trouble initiating work, organising tasks, misunderstanding instructions.
2. Focus: focusing, sustaining and shifting attention to tasks.
    - Loses focus, forgets what was read, easily distracted.
3. Effort: regulating alertness, sustaining effort and processing speed.
    - Difficulty sleeping, loses interest quickly, difficulty completing tasks on time.
4. Emotion: managing frustration and modulating emotions.
    - Overreacts to frustration.
5. Memory: utilising working memory and accessing recall.
    - Forgets to do planned activities, poor recall.
6. Action: monitoring and self-regulation action.
    - Difficulty adjusting to situations, does tasks too fast.

These clusters within executive functions are continuously and unconsciously working together to manage daily tasks. An individual must self-regulate using attention and memory to move between tasks. Brown stated that the six clusters are dimensional and that we all have impairments within these clusters at times particularly when in different situations, but for individuals with ADHD the impairments are chronic and severe compared to individuals of the same age and developmental level causing impairment in most areas of life. However, impairments of executive function are not unique to ADHD but overlap with many other conditions (Brown, 2006).

Brown uses an analogy to describe the role of executive functions in ADD. "Imagine a symphony orchestra in which each musician plays his or her instrument very well. If there is no conductor to organise the orchestra, to signal the introduction of the woodwinds or the fading out of the strings, or to convey an overall interpretation of the music to all players, the orchestra will not produce good music. Symptoms of ADD can be compared to impairments, not in the musicians but in the conductor" (Brown, 2005, pp. 10).

The behavioural response inhibition theory and ADD syndrome model are based upon deficits in executive functions. However, the behavioural response inhibition theory only examines the combined subtype of ADHD and does not involve the impulsive-hyperactive or inattentive subtype. Additionally, the behavioural response inhibition theory states that the main executive functioning difficulty is behavioural inhibition, rather than multiple areas of executive functions, as the ADD syndrome model states (Brown, 2006).

In addition to these theories of ADHD based upon executive functions, there are other theories which will now be discussed.

1.2.3 Maturation Lag Model of ADHD

The Maturation Lag Model is based upon the notion that symptoms of ADHD are the result of a delay in typical brain development (Kinsbourne, 1973). The theory states that the behaviours of a child with ADHD is abnormal purely for their age and that they were eventually catch up with their peers. Consequently, as a child with ADHD matures and catches up with their peers overcoming the developmental delay, the symptoms may lessen. The Maturation Lag Model was developed through observations of an ADHD child and a younger child who displayed similar levels of hyperactivity, short attention span and lacked impulse

control (Burke & Edge, 2013). The theory predicts that the neurodevelopmental factors that inhibit the performance of a child with ADHD is similar to the typical limits of a younger child without the disorder. When examining brain activity, children with ADHD show cortical activity similar to children of a younger age without difficulties, namely an increase in slow wave theta activity and decrease in fast beta waves, evidence that an individual with ADHD is approximately 3 years behind a typically developed individual. A child with ADHD reaches maximum brain thickness at 10 years 6 months compared to 7 years 6 months in a healthy control. However, the lag in maturation differs across the brain, with the prefrontal cortex lagging by approximately 5 years, but faster maturing in other areas, such as the primary motor cortex. In a sample of ADHD children, maturation lag was present when examining brainwaves in 7% of individuals, mainly in the posterior regions of the brain (Burke & Edge, 2013).

One study examining the maturation lag model in ADHD was conducted by Berger, Slobodin, Aboud, Melamed, and Cassuto (2013). Five hundred and fifty nine children with ADHD and 365 healthy individuals underwent Continuous Performance Tests (CPT). Results showed improvement with age, but children with ADHD demonstrated impairments at all ages compared to the control sample. Specifically, ADHD children performed approximately 1 to 3 years younger than their age when compared to peers. This shows brain functions in individuals with ADHD develop slower than expected, supporting the maturation lag model.

1.2.4 Developmental Deviation Model of ADHD

The Developmental Deviation Model, also known as the Maturational Deviation Model, explains ADHD as the result of abnormal functioning in the Central Nervous System (CNS). The model states that the brain of individuals

with ADHD are unlikely to develop or mature to the expected level during their lifespan. The developmental deviation model was inspired by electroencephalogram (EEG) research which shows that 90% of individuals with ADHD have abnormal brainwave activity at all ages and do not mature with development, as is expected in a typical individual (Barry, Clarke, & Johnstone, 2003).

EEG patterns are created and regulated by our brainstem, thalamic and cortical processes, all of which use major neurotransmitters and in turn produce electrical activity at both multiple cortical and subcortical levels (Cantor & Chabot, 2009). These can then be recorded on the cortex via an EEG.

EEG abnormalities typically seem in ADHD include increased theta activity in the frontal regions, reduced alpha activity in parietal areas, and increased theta/beta and theta/alpha ratios. One study that supports the developmental deviation model was conducted by Burke and Edge (2013). An adult ADHD population with a mean age of 34 years old, underwent clinical interview, EEG and rating scales. Compared to a typically developed control group, results showed that ADHD behaviours are due to a neurodevelopmental lag, showing cortical activity similar to a younger child, specifically elevated relative theta activity as well as increased theta/beta ratio and theta/alpha ratio. However, the symptoms and abnormal EEG patterns were persistent in adulthood, supporting the developmental deviation model.

EEG abnormalities and the role it plays in an ADHD diagnosis will be discussed later in this thesis, with one of the research studies focusing on EEG differences between ADHD and typically developed individuals.

1.3 Causes of ADHD

The exact cause of ADHD is not known, but research indicates several contributing causes including genetic influences and environmental factors. Research from genetic and environmental factors suggests that neurodevelopmental factors are the major contributors to the cause of ADHD with evidence from family, twin, adoption and genetic studies (Faraone et al., 2005). However, there are other psychosocial factors that may contribute and will also be discussed (Barkley, 2003). Initially, genetic causes of ADHD will be focused on.

1.3.1 Genetics

Evidence suggests that ADHD has a genetic basis, although a single gene has not yet been pin-pointed as to the leading cause (Williams et al., 2010; Martinez et al., 2016; Rivero et al., 2015). One study published in the Lancet (Williams et al., 2010), examined genome analysis of copy number variants (CNV) in 366 children with ADHD and 1047 healthy controls, aged between 5 and 17 years old. CNV are associated with chromosomal deletions, with large rare CNVs being a risk factor for neurodevelopmental disorders. Results showed that 57 children across the 2 samples had large, rare CNV, with a significantly higher rate in the ADHD sample. Consequently, this is evidence that rare genetic variants may contribute to ADHD.

Several specific genes have been identified for their possible causation of ADHD, namely ADGRL3 and Cadherin-13. ADGRL3 encodes protein which regulates communication between brain cells; however, if this gene has a genetic variation, it can disrupt the regulation of communication (Martinez et al., 2016). Martinez et al. (2016) conducted family genetic analysis of 372 individuals with ADHD and 466 healthy controls. Individuals with ADHD showed a reduced

amount of ADGRL3 specifically in the thalamus, contributing to the cause of ADHD (Martinez et al., 2016).

Cadherin-13, also known as CDH13, is a cell adhesion molecule which has the potential to cause ADHD and other neurodevelopmental conditions. This gene is involved in inhibitory modulation of brain activity, a severe impairment in ADHD, and consequently vital for cognitive function and memory formation. Deficits in CDH13 can cause behavioural alterations, specifically behaviours associated with ADHD, with CDH13 deficits in mice shown to increase motor activity and memory deficits (Rivero et al., 2015).

Dopamine is a key cause of ADHD, both at the genetic level and chemicals within the brain. In regard to a dopamine gene, evidence suggests that a variation of the dopamine transporter gene, DAT1, which is responsible for stopping the dopamine signal, is involved in the cause of ADHD symptoms. The variation of DAT1 causes failure of dopamine cell response, specifically, individuals with ADHD are unable to respond to reinforcement in an appropriate way as the dopamine cell response does not work correctly (Tripp & Wickens, 2009). Another dopamine gene suspected in the cause of ADHD is the DRD4 dopamine receptor, although there is little evidence for this (Sharp, McQuillin, & Gurling, 2009).

In addition to a possible genetic predisposition to ADHD, neurotransmitters are a major contributor to causing impairment. Individuals with ADHD have underactive dopamine levels in the brain as well as the dopamine transporters not working efficiently (Sagvolden, Johansen, Aase, & Russell, 2005). Low levels of dopamine create difficultly sustaining attention which in turn leads to clumsiness, failure to inhibit responses, hyperactivity and poor executive functions. Stimulant

medication, one treatment option for individuals with ADHD, directly addresses underactive dopamine levels (Tripp & Wickens, 2009). The role of stimulant medication in the treatment of ADHD will be discussed in more detail later in this thesis.

1.3.1.1 Family Studies

ADHD is evident in families and it can be unclear if this is due to genetic or environmental factors. Consequently, studies on families, twins, and adoption, aid in separating these issues. Family studies demonstrate a strong genetic contribution to the development of ADHD (Kieling, Goncalves, Tannock, & Castellanos, 2008; Faraone et al., 2005). At least 25% of adults with hyperactive symptoms are a biological parent of a child with similar difficulties (Biedermann, Newcom, & Sprich, 1991). One family study conducted by Biedermann, Newcom, and Sprich (1991) found that 25% of first-degree relatives of a child with ADHD also had the condition compared to 5% in a control group. Consequently, if a child has ADHD, it is five times more likely that other members in the family are also at risk.

Twin studies are used in ADHD for various reasons including defining phenotype, defining gender differences, and examining the gene-environment interaction. Twin studies show heritability of ADHD at an approximate rate of 0.8 (Kieling et al., 2008) and heritability estimated at 0.76 from 20 extant twin studies. Through numerous studies, findings are clear that ADHD is contributed to by genetic factors although shared environment does show to be important (Faraone et al., 2005). One example is a study conducted by Levy, Hay, McStephen, Wood, and Walkdman (1997) where 1,938 families with twins and siblings, 4 to 12 years old, with one child who had ADHD, were examined. Findings showed ADHD to

have heritability levels of 0.75 to 0.91, a finding across twin, siblings and twin-siblings.

Only a few studies have examined the relationship between ADHD and adoption (Sprich et al., 2000). Sprich et al. (2000) found rates of ADHD to be significantly higher between biological relatives compared to adoptive relatives. Specifically, in an adopted ADHD sample, 6% of adoptive parents and 8% of adoptive siblings had symptoms of ADHD compared to 18% of biological parents and 31% of biological siblings. This evidence demonstrates that adoptive parents of children with ADHD were unlikely to have created the condition or raised the child in an inappropriate manner, but that the child's genetics predisposed the difficulties they experience. Interestingly, low levels of psychopathological difficulties have been observed among adoptive parents suggesting that they have the ability to implement behavioural strategies for any child placed in their care (Sprich et al., 2000).

1.3.2 Brain Abnormalities

Imaging studies have demonstrated brain abnormalities among individuals with ADHD, the most consistent finding being an overall reduction in brain size (Castellanos et al., 2002). A recent meta-analysis of function magnetic resonance imaging (fMRI) studies examining ADHD was conducted by Rubia (2018). Findings showed that individuals with ADHD have impairments in the right and left hemispheric dorsal, ventral and medial fronto-cingulo-striato-thalamic, creating widespread dysfunction. Specifically, individuals with ADHD are late in developing specific areas of the brain, including the fronto-striato-parietal and fronto-cerebellar networks, areas which are involved in motor response inhibition, working memory and sustained attention. Additionally, impairment has been

evident in orbital and ventromedial prefrontal and limbic areas, linked to emotion control. However, the majority of fMRI studies have used male participants with a diagnosis of ADHD combined subtype. Therefore, research needs to be conducted in females as well as the other subtypes of ADHD.

Evidence shows that individuals with ADHD have significantly smaller brain volume, specifically in the prefrontal cortex (Mostofsky, Cooper, Kates, Denckla, & Kaufmann, 2002). In individuals with ADHD, the caudate nucleus has a smaller volume and asymmetry differences. The basal ganglia has also been shown to be smaller in ADHD subjects compared to healthy controls (Krain & Castellanos, 2006). The basal ganglia is responsible for motor movements, procedural learning, routine behaviours, cognition and emotion. Prior to information reaching the basal ganglia, information passes through the caudate nucleus. One study looked specifically at the structure within the basal ganglia to find deficits in ADHD, namely the left globus pallidus as well as total globus pallidus were smaller in volume in a male ADHD population (Aylward et al., 1996). Abnormalities with the cerebellum have also been linked to ADHD; the cerebellum is important for cognitive functions such as language, attention and regulating a response to fear and pleasure. In typical individuals, the cerebellum is used to produce motor movements but also involved in activities such as attention shifting. Studies of individuals with ADHD have found that the cerebellum is up to 6% smaller in volume compared to healthy individuals, although it is unclear what effect this has on the functions of the cerebellum (Berquin et al., 1998).

In conclusion, the evidence suggests that individuals with ADHD have a brain which is structurally different to a healthy brain. Specifically, individuals with ADHD have decreased volume in the frontal lobe, particularly to the left side

of the lobe, that the basal ganglia, which is involved in the circuits, is smaller and a smaller cerebellum, which is involved with co-ordination and non-motor function (Krain & Castellanos, 2006).

1.3.3 Environmental and Psychosocial Factors

Although there is little evidence to suggest that social-environmental factors are the largest contributing factor to the cause of ADHD, they have a role to play in the development of the condition (Barkley, 2003). As we have seen, there is a wealth of evidence suggesting that ADHD is a result of our biology, however, our biology is further influenced by our environment. The severity of symptoms, type of comorbidities, and outcome of the disorder is related to varying degrees of environmental factors (Biedermann et al., 1996). Environmental risk factors include exposure to lead during childhood, prenatal smoking, alcohol, child rearing, family conflict, and marital difficulties (Tripp & Wickens, 2009).

1.3.3.1 Prenatal Risk Factors

Complications during pregnancy and birth may potentially have a detrimental effect on early brain development; there is a higher rate of birth and pregnancy complications in individuals with ADHD than healthy individuals. This early trauma can have long term effects, both in terms of cognition and behaviour, particularly if they occur during crucial times of development. In addition to pregnancy and birth complications, other risk factors include, if the mother has a low education level, if there is a long time between onset of labour and birth, presence of delivery complications and young mothers age at delivery. These factors combined account for 42% of the variance in ADHD. Younger maternal age at time of delivery is particularly important; this population are at greater risk

of complications during pregnancy and consequently the child is at a higher risk of having ADHD symptoms (Claycomb, Ryan, Miller, & Schnakenberg-Ott, 2004).

Another social-environmental factor that may contribute to ADHD are toxins. Elevated levels of lead in the body has been shown to have a small but significant relationship to ADHD symptoms (Needleman, Schell, Bellinger, Leviton, & Alfred, 1990). However, it has been shown that less than 38% of children who had high levels of lead exposure also had ADHD symptoms (Needleman et al., 1990). Smoking during pregnancy is a well-studied area in the ADHD field (Goodwin, Keyes, & Simuro, 2007). Approximately 10-20% of women smoke during pregnancy due to a variety of reasons (Goodwin et al., 2007). A review of previous research on smoking during pregnancy showed an association to develop disorders such as depression, addiction and ADHD. Maternal smoking shows the strongest association with ADHD whereas maternal alcohol consumption is less of a risk factor, but nonetheless a risk (O'Malley & Nansom, 2002).

1.3.3.1 Parenting Style

It has been previously suggested that a weak parenting style contributes towards the development of ADHD. Specifically, weak parenting can aggravate possible symptoms as well as contribute to developing other difficulties. As reported by parents of children with ADHD, there is often more family conflicts in the home, disorganisation, and less adherence to rules compared to typical home environments (Teixeira, de Freitas Marino, & Carreiro, 2015). These difficulties manifest itself in children showing more impulsivity, inattention and agitation. For example, Carlson, Jacobvitz, and Sroufe (1995) suggested that a large contributing factor of ADHD is parenting approach. Observations of mother and

son interactions were completed in a sample of 20 typically developed and 20 hyperactive boys aged 6 to 12 years old. Mothers of the hyperactive boys had more structure and control of the child's play, and the children were more active and less compliant. It has been noted that parents of hyperactive children tend to have overcritical and commanding parenting style, a result of psychological and physical overload (Barkley & Cunningham, 1979) which can exacerbate hyperactive and oppositional behaviour (Barkley, Fischer, Edelbrock, & Smallish, 1991). Keown and Woodward (2002) demonstrated the importance of family relations and functioning in the role of ADHD. Specifically, 33 children with pervasive hyperactivity and 33 typical boys were assessed at home with their mothers through interview, parental questionnaire and observations. Results showed higher rates of lax discipline, less efficient parental coping and lower rates of father-child communication among the ADHD population. This study demonstrated the importance of a father figure in behavioural development as well as parental coping. On the other hand, the challenging behaviour and temperament of a child with potential or diagnosed ADHD can affect the type of parenting as well as the stress placed on the parents (Cunningham, 2007). The parents have to attempt to manage impulsivity, poor choices, lack of friendships, low self-esteem, disruptive behaviour as well as their own social distress and negotiating with the health and education systems. As previously discussed, ADHD has a large genetic component (Faraone et al., 2005) and therefore parents may have ADHD symptoms, effecting their parenting skills.

1.4 Management of Concentration and Impulsivity Difficulties

A comprehensive treatment plan is required to manage individuals with a diagnosis of ADHD including psychological, behavioural and educational

strategies. The NICE guidelines recommend methods such as parent-training/education programmes, behavioural interventions from trained teachers, cognitive behavioural therapy, social skills classes, and possible medication (NICE, 2018). Some of these strategies, their strengths, weaknesses and guidance on implementing such strategies, will now be discussed.

1.4.1 NICE Guidelines on Treatment of ADHD

Recommended treatment by the NICE guidelines for an individual with ADHD depends upon the individual's age and severity of symptoms. For a pre-school child, parent training is recommended as the first line of treatment. For a school aged child or young person with moderate ADHD, parent training as well as group therapy, such as cognitive behavioural therapy or social skills training, are suggested. For a school aged child or young person with severe ADHD, drug treatment is the first line of treatment in conjunction with offering group-based parent training. For adolescents, individual psychological interventions are recommended; however, if an adolescent finds psychological treatments to be ineffective or the individual refuses to take part, drug treatment can be commenced (NICE, 2018).

1.4.2 Cognitive Training Programmes

Cognitive training programmes target deficits in cognitive domains that are causally linked to ADHD symptoms, such as executive function deficits (Halperin & Healey, 2011). Studies examining cognitive training programmes found improvements on working memory, inhibition, inattention, and non-verbal reasoning skills, in individuals with ADHD (Klingberg et al., 2005). One study that focused on working memory and attention shifting found little improvement on teacher rating scales. However, on parent rating scales, there was significant

improvement on inattentive symptoms and a small improvement on hyperactive and impulsive symptoms (Epstein & Tsal, 2010).

A specific cognitive training programme available for ADHD treatment is "Pay Attention!". This programme uses visual and auditory tasks to train different types of attention including sustained, selective, and divided attention. Research shows that through completing a course of Pay Attention! improvements in neurocognitive and academic behaviours are achieved. However, improvement was not noted in behavioural symptoms (Kerns & Thomson, 1999).

Another form of cognitive training specific for individuals with ADHD is the Cogmed Working Memory Training Program, also known as Cogmed. This training program is computer based, with beneficial outcomes after 25 sessions, specifically 5 sessions a week for 5 weeks. Through completing a course of Cogmed training, children with ADHD showed improvements in visual and verbal working memory in addition to nonverbal complex reasoning and response inhibition. The significant improvements were still evident at a three-month review (Klingberg et al., 2005). This intervention is also effective in adults with ADHD (Olesen, Westerberg, & Klingberg, 2004).

Sonuga-Barke et al. (2013) conducted a meta-analysis examining nonpharmacological interventions on ADHD. Six cognitive training trials were included, three focusing on attention and three focusing on working memory. In trials where observers were aware of the research and intervention, significant treatment effects were found. However, the significant treatment effects were lost when raters (e.g. teachers) were blinded to the intervention group.

1.4.3 Parent-Training Programmes

As noted in the NICE guidelines (NICE, 2018) parent training is an important part of a comprehensive treatment plan for children with ADHD. Parent training programmes teach effective communication with children in a stress-free way. When medical management is in place, parent training should still be completed due to medication effects wearing off and behavioural intervention consequently being implemented (Willis, 2003). Danforth, Harvey, Ulaszek, and McKee (2006) conducted parent training to parents of 45 children with ADHD. The training included an explanation of the features of ADHD and specific strategies to implement at home. Nine or ten families attended per group for approximately 90 minutes for 8 weeks. Upon completing the course, results showed reduced hyperactivity, aggression, and oppositional behaviour, and in addition it improved parenting behaviour and reduced parental stress. These benefits led to improved social skill abilities in children with ADHD (Hinshaw et al., 2002). In another study by Anastopoulos, Shelton, and DuPaul (1993) 34 children with ADHD along with their mothers took part in parent training. Nine sessions were completed including an overview of ADHD, general behaviour management principles, positive reinforcement, and punishment strategies. Results showed that parent training not only improved the child's behaviour but also enhanced family functioning, decreased parent stress and increased parent's self-esteem. Consequently, this led to an improved overall emotional climate in a family and strengthened relationships between parents (Barkley, 2003). Thus, if parent-training is completed and implemented effectively, there are benefits to the child and parent.

More recently Lange et al. (2018) examined parent training in improving core ADHD symptoms, specifically the New Forest Parenting Programme, in preschool children aged 3 to 7, with ADHD in a randomised control trial. Results showed positive improvement in ADHD symptoms as rated by parents. However, no effects were evident in direct observations or teacher rating, questioning the overall effectiveness of parent training.

A further type of parent-training programme is based on psychoeducation. This intervention has been developed by mental health care professionals and focus' on consumer outcomes as well as family outcomes through teaching on symptomology, its treatment, skills development and patient empowerment (Dixon et al., 2001). The intervention is informative using both psychotherapeutic and educational components and encourages families to recognise systems as well as the patient actively participating in treatment to increase compliance (Bauml, Frobose, Kraemer, Rentop, & Pitschel-Walz, 2006). However, the type of information provided can vary between providers (Dixon et al., 2001). Evidence suggests that psychoeducation and other educational programmes play a positive role in the intervention of ADHD in children and adolescents with studies showing reduced levels of relapse (Dixon et al., 2001). However, despite a systematic review of the evidence there is little research into the use of psychoeducation in the treatment of ADHD, an area that requires research (Dixon et al., 2001).

1.4.4 Teacher Training/Educational Strategies

Educational strategies also need to form part of a comprehensive treatment plan for children with ADHD (NICE, 2018). Interventions that take place in the home rarely translate to classroom situations and therefore, interventions within the school setting will be most effective for improving school performance

(Abramowitz & O'Leary, 1991). The most important aspect of educational strategies is for teachers to understand and have knowledge about the condition, how it affects the child in the classroom and basic strategies that can be put in place. Classroom strategies should be both proactive and reactive to create the most improvement. Specific strategies include seating the student at the front of the class next to a buddy and away from distractions, presenting instructions visually, chunking tasks into small manageable sections, and allowing the child movement breaks (DuPaul & Stoner, 2003).

Arcia, Frank, Sanchez-LaCay, and Fernand (2000) conducted semi structured interviews with 21 primary school teachers. Strategies implemented included: behavioural, use of rewards, positive praise; instructional, one to one instruction, peer tutoring; environmental, seating the child close to the teacher at the front of the class, with a buddy; and interpersonal strategies, the teacher talking with the child discussing appropriate behaviour. Results from the study showed that teachers lack understanding of ADHD and of classroom management and tended to employ reactive rather than proactive strategies. If a teacher lacks basic knowledge about ADHD, then classroom strategies generally have little impact on a child with ADHD (Arcia et al., 2000).

1.4.5 Medication

Drug treatment is the first line of recommended treatment for school aged children or young person with severe ADHD (NICE, 2018).

Stimulant medications are widely used to treat individuals diagnosed with ADHD to combat their symptoms. There are several types of stimulant medications including methylphenidate, dexamfetamine, amphetamine, and lisdexfetamine. Other non-stimulant medications used to treat ADHD include

atomoxetine, clonidine, and guanfacine, but are less effective and less tolerated than stimulant medication (NICE, 2008).

1.4.5.1 Neurochemistry of Stimulant Medications

Methylphenidate prevents the re-uptake of dopamine and norepinephrine in the brain (Shiels, Hawk, & Reynolds, 2009). The stimulant medication binds to the presynaptic neuron which increases the concentration of catecholamines in the extraneuronal space, and consequently improves postsynaptic catecholaminergic neurotransmission (Volkow et al., 2012). This explanation is coherent with the causes of ADHD as it is suggested that symptoms occur due to dysfunction in the neurotransmitters, specifically with low levels of dopamine (Sagvolden et al., 2005), which is involved in sending signals to the brain for reward processes as well as regulating behavioural processes (Shiels et al., 2009).

1.4.5.2 History of Stimulant Medication

The first evidence of the use of stimulant medication was conducted by Bradley (1937). It demonstrated that benzedrine, when administered to children aged 5 to 14 who had behavioural difficulties, created academic improvement. The most widely used stimulant medication for the treatment of ADHD is methylphenidate which is recommended to be the first line of pharmacological treatment due to the large response rate and significant improvement in symptoms (NICE, 2018). In the United States, immediate release methylphenidate became commonly available in 1995. Immediate release preparations of methylphenidate have a rapid onset and an effect within 20 to 60 minutes. There is a peak plasma of concentration within approximately 1 to 2 hours after taking the tablet with the whole duration lasting approximately 4 hours (Hoffman & Lefkowitz, 1996). However, multiple doses may be needed which can be inconvenient and can lead

to missing of doses. Additionally, the overlap in doses can cause a fluctuation in symptoms. It was not until the mid 1990s that prescribing methylphenidate increased in the UK, along with a better understanding of ADHD and changes in regulatory frameworks. At the same time, once daily methylphenidate preparations became available, namely Concerta XL, Equasym XL and Medikinet XL. Once-daily preparations are effective for roughly 8 to 14 hours, depending on the preparation, 22-50% of the medication is released immediately and the remaining proportion released later, therefore continuing to be effective over a longer duration (Joint Formulary Committee, 2018; Loureiro-Vieira, Costa, de Lourdes Bastos, Carvalho, & Capela, 2017).

1.4.5.3 Benefits of Stimulant Medication in ADHD

It has been found that stimulant medication has a positive effect on ADHD symptoms in 65% to 77% of children with ADHD (Barkley, 1997; Johnston et al., 2015) while approximately 25% to 30% of ADHD children do not respond or tolerate stimulant medication (Elia, Borcherding, Rapport, & Keysor, 1991). Evidence shows that stimulant medication has positive effects on children of various ages including improvement in impulsiveness, disruptiveness, noncompliance, talking out of turn, restlessness, and aggression (Whalen, Henker, & Granger, 1990).

One study examined the effect of taking methylphenidate for 4 months in a child ADHD population. Ninety-one children participated, either in a methylphenidate or placebo condition. The methylphenidate condition showed improvement in symptoms and behaviour at school but not at home. Side effects of physiological symptoms and lack of weight gain were seen in the

methylphenidate condition, which were reported by parents but not teachers (Schachar, Tannock, Cunningham, & Corkham, 1997).

A meta-analysis was conducted reviewing 13 randomised control trials examining methylphenidate. It total, 882 participants with a diagnosis of ADHD, up to the age of 18 years old, participated. On parent ratings, there was a preference for long acting methylphenidate, due to improvements on hyperactive/impulsive behaviours. However, teacher ratings favoured short acting methylphenidate, specifically for hyperactivity (Punja et al., 2013).

A further study conducted a meta-analysis of all randomised controlled trials comparing methylphenidate to control conditions. In total, 5,111 child participants with a diagnosis of ADHD participated, with an average age of 9.7 years. Results showed 29% of participants experienced non-serious adverse effects, such as sleep problems and decreased appetite. When rated by teachers, an improvement in general behaviour was seen, although exact details were not discussed and parents reported an improvement in quality of life (Storebo et al., 2015).

Methylphenidate has been shown to improve performance on Continuous Performance Tests. Specifically, findings showed that the higher dose of methylphenidate taken, the fewer errors occurred, showing successful concentration. Additionally, the higher the cognitive ability of an individual, the higher the response rate to methylphenidate (Pearson et al., 2004). Continuous Performance Tests will be discussed in more detail later in this thesis, with one of the research studies using this as a measure of concentration.

In a questionnaire of 50 students aged 11 to 18 years old taking methylphenidate, they reported improvements in behaviour, social ability with

friends, parents and teachers and attention. However, they felt that methylphenidate did not show improvement in academic achievement. Additionally, the majority of students experienced some form of side effects, detail of which are discussed shortly (Moline & Frankenberger, 2001).

Cortese et al. (2018) conducted a systematic review on examining medication as an intervention for ADHD. One hundred and thirty-three double-blind randomised control trials were included. All medications consumed by children were more efficacious than placebo. It concluded, supporting NICE guidelines, that methylphenidate is the first choice of recommended short-term treatment for children and adolescents with ADHD.

1.4.5.4 Short Term Side Effects of Stimulant Medication

An unknown percentage of families do not wish to trial medication, despite the possible benefits, because of potential side effects (Arnold et al., 2013). Although stimulant medications are usually well tolerated, there is evidence of side effects. Short term side effects of methylphenidate include appetite suppression, sleep difficulties, headaches, stomach aches, blunting of personality, and irritability with frequency rates ranging between 5% to 15% (Monastra, 2008). In a study by Moline and Frankenberger (2001) 50 students aged 11 to 18 years old completed a questionnaire regarding their experiences of taking methylphenidate. Of the 50, 64% of participants reported some form of short-term side effect, specifically 48% experienced headaches, 54% experienced sleep difficulties, 57% experienced appetite suppression (particularly at lunchtime), and 40% experienced tics. It is very important that tics are closely monitored as it is known that methylphenidate can exacerbate these. Additionally, appetite suppression needs to be monitored as can lead to decrease in growth. However, in many cases the

positive effect of stimulant medication outweighs the negative side effects as these tend to wear off when the medication is not active within the body (NICE, 2018).

1.4.5.5 Long Term Side Effects of Stimulant Medication

Despite an increase in the understanding of mental health conditions and improvement in diagnostic criteria (APA, 2013), there are very few studies that have examined the long-term effect of stimulant medication, particularly on the long term effect of use during childhood and consequently the effect in adulthood (Loureiro-Vieira et al., 2017). Previous research suggests that methylphenidate can lead to heart rate difficulties and systolic blood pressure. One study investigated the effect of methylphenidate on blood pressure. In a sample of 125 adults with ADHD where methylphenidate was being taken, minor but statistically significant changes in heart rate and blood pressure were seen. In 10% of subjects, systolic or diastolic hypertension was caused as a result of taking methylphenidate (Wilens et al., 2005). Consequently, NICE supports monitoring on a 6-monthly basis (NICE, 2018).

Various interventions for ADHD have been discussed here including cognitive training, parent-training programmes, school strategies, and medication. All of these strategies have some benefit in the treatment of children with ADHD with stimulant medication having the highest effect rate at 73% to 77% (Barkley & Cunningham, 1979). However, stimulant medication only has an effect while in the individual's system. Consequently, with the development of new technologies, other interventions are becoming available with possible improved long-term effects.

1.4.6 Neurofeedback

In regard to ADHD, stimulant medication and behaviour interventions are the most widely used and accepted forms of treatment. However, a need has been identified for a new treatment with improved long-term effects (Arns, Heinrich, & Strehl, 2014).

Neurofeedback is a form of biofeedback whereby the person is being consciously made aware of their brain wave activity. "Neurofeedback is a very special discipline because it stands right at the landmark of brain and behaviour as it deals with all the complexities of brain function", (Romano-Micha, 2010, pp.80). Neurofeedback improves attention and behavioural control by an individual learning to regulate levels of cortical arousal in the brain via visual and/or auditory reinforcement (Monastra, 2005). Based upon the operant conditioning paradigm, developed by B. F. Skinner (1938) neurofeedback teaches modified behaviour through positive reinforcement, rewarding when desired behaviour is produced. In relation to neurofeedback, operant conditioning encourages specific amplitude and frequency of particular brain wave activity (Monastra, 2008).

A typical neurofeedback training session is approximately 45 to 60 minutes in duration where several short training periods take place. The average amount of sessions required to gain normalization of EEG patterns is 43 sessions, with total amount of sessions ranging from 34 to 50. A neurofeedback session is designed so that the patient demonstrates the undesired brainwave for 40% of the time and desired brainwave for 60% of the time, enabling the patient to learn what the desired brainwave feels like and how to maintain it through positive reinforcement (Monastra, Monastra, & George, 2002).

There are six different types of brain wave frequencies that form human's overall brain wave activity and therefore can be manipulated via neurofeedback. Brainwaves progress from the slow wave of delta, theta, alpha and SMR, to the fast wave of beta and gamma, each associated with varying behaviours (Demos, 2004). Further detail regarding EEG and specific roles of various brainwaves are discussed in a later chapter, where research focuses on the differences between typical and ADHD brainwave activity.

1.4.6.1 Neurofeedback and The Brain

Neurofeedback manipulates brainwave activity via neuronal plasticity. Neuronal plasticity, the ability for the human brain to continuously change structure and function, is evident during our childhood development but the brain remains malleable throughout a lifetime (Kolb, 1995; Raymont & Grafman, 2006). Neuromodulation and long-term potentiation (LTP) are two processes enabling brain plasticity to occur (Abarbanel, 1999). Neuromodulation is a neurotransmission whereby metabotropic receptors exert a great influence in electrophysiological properties of a cell. LTP is the increased synaptic transmission efficiency as the result of high-frequency synaptic activation (Andersen, 2004). LTP is the process enabling structural and biochemical changes to become long term changes (Bliss, Collingridge, & Morris, 2004). During neurofeedback, the relevant neural networks are modified through neuromodulation (Abarbanel, 1999) and these changes are long lasting through LTP (Abarbanel & Evans, 1999; Sterman & Egner, 2006).

Neurofeedback training exerts control over specific EEG parameters and consequently, associated functions. In the case of clinical applications, neurofeedback training aims to normalize electrophysiological imbalances

(Monastra et al., 2005). In healthy individuals, neurofeedback can improve performance in specific areas including sport, cognitive and artistic performance (Vernon, 2005). Neurofeedback can be used to treat a variety of disorders such as alcoholism and substance abuse, anxiety, ADHD, autism, depressive disorders, epilepsy, insomnia, post-traumatic stress disorder, and traumatic brain injury (Yucha & Montgomery, 2008).

1.4.6.2 Approaches to Neurofeedback

One group of neurofeedback practitioners use standard protocols to treat symptoms. Practitioners obtain a description of symptoms from the patient and interpret the symptoms and possible dysregulated brain wave patterns, a subjective approach which can lead to incorrect conclusions. Practitioners develop a neurofeedback protocol based upon the individual's symptoms rather than the underlying brain wave abnormalities (Romano-Micha, 2010).

An alternative approach to neurofeedback involves collecting and analysing EEG patterns in conjunction with the patient's symptoms to develop a personalised neurofeedback protocol. Arns, Drinkebury, and Kenemans (2012) investigated the effect of EEG-informed neurofeedback in a pilot study. Adults with ADHD underwent a 26 channel EEG recording, where the participant was exposed to a series of high and low-pitched tones. Participants were asked to press a button with their left and right index finger in response to the high-pitched tone, while keeping their eyes fixed on a red dot presented on a computer screen in front of them. The raw EEG data were then visually inspected to establish a neurofeedback protocol. Participants received at least one established protocol, Sensori-Motor Strip (SMR)/theta or theta/beta, and one protocol based on EEG findings. Personalised neurofeedback protocols based upon EEG findings

improved clinical outcomes, specifically on attention scales. Seventy-six percent of participants responded to personalised neurofeedback, 14% were non-responders, and a 10% drop out.

1.4.6.3 Neurofeedback Protocols in Healthy Participants

Evidence has shown that neurofeedback can be successfully applied in a healthy population (Egner & Gruzelier, 2001; Vernon et al., 2003; Vernon, 2005; Fritson, Wadkins, Gerdes, & Hof, 2007). For example, Egner and Gruzelier (2001) employed SMR training (increase of 12-15 Hz) at C4 and beta training (increase of 15-18 Hz) at C3 in a group of 22 healthy adults while simultaneously inhibiting theta (4-7 Hz) and high beta (22-30 Hz). After ten neurofeedback sessions, a significant reduction was seen on Continuous Performance Test commission errors but no change on omission errors. Also, SMR neurofeedback was highly positively correlated to commission error reduction. However, Vernon (2005) noted a lack of control group and absence of EEG changes.

In another healthy population study by Vernon et al. (2003) neurofeedback training was employed with two groups, a theta-group, up training theta (4-8 Hz), while inhibiting delta (0-4 Hz) and alpha activity (8-12 Hz); and an SMR-group, trained to enhance SMR (12-15 Hz) and simultaneously inhibiting theta and beta (18-22 Hz). Eight sessions were completed at the Cz location. Participants in the SMR-group showed improvements in the accuracy of attentional processing and semantic working memory tasks. It was hypothesized that the improvement produced by SMR training on working memory performance could be related to the fact that training in this frequency band may help to maintain the memory representation used in semantic working memory (Vernon et al., 2003).

Fritson, Wadkins, Gerdes, and Hof (2007), explored the effects of neurofeedback training on measures of response control and attention. Two groups of healthy participants, totalling 32 participants, were randomly assigned to a neurofeedback or control condition. In the neurofeedback condition, participants received SMR training (12-15 Hz), while inhibiting theta (4-7 Hz) and high beta (22-36 Hz), whereas the control condition received sham neurofeedback training (feedback based on a previously recorded EEG from another person). Participants attended a total of 20 twice-weekly sessions. No significant changes were found for measures of attention in either the control or treatment group. Although a control group was used, EEG measures were not reported.

1.4.6.4 Neurofeedback and ADHD

Neurofeedback was first used with individuals diagnosed with ADHD in 1976 whereby improvements were shown in distractibility and hyperactivity (Lubar & Shouse, 1976). These findings are considered to be the earliest clinical effects of neurofeedback on ADHD (Arns et al., 2014). A meta-analysis was conducted examining ADHD and neurofeedback studies with the main conclusions showing neurofeedback had a large effect size on improving impulsivity and inattention, and a medium effect size on improving hyperactivity (Arns, Ridder, Strehl, Breteler, & Ccoenen, 2009).

One standardised protocol for the treatment of ADHD is uptraining SMR, which has been shown to improve impulsivity and attention (Carmody, Radvanski, Wadhwani, Sabo, & Vergara, 2001; Fuchs, Birbaumer, Lutzenberger, Gruzelier, & Kaiser, 2003; Kaiser & Othmer, 2000; Rossiter, 2004; Rossiter & La Vaque, 1995), behavioural indices (Shouse & Lubar, 1979), changes in subcortical areas associated with response inhibition and selective attention (Beauregard &

Lévesque, 2006). Fuchs, Birbaumer, Lutzenberger, Gruzelier, and Kaiser (2003) conducted a study on 34 children with a diagnosis of ADHD, aged 8 to 12 years old, of which 22 children completed 3 months of neurofeedback sessions, 3 sessions a week with the same therapist at the same time of day. Children diagnosed with ADHD hyperactive-impulsive subtype received SMR uptraining at C4, whereas children diagnosed with ADHD inattentive subtype received beta uptraining at C3. Children diagnosed with ADHD combined subtype received a combination of these protocols. A further 12 children received stimulant medication. All conditions saw a reduction on ADHD symptoms. Specifically, all conditions had a reduction on Conners' Behaviour Rating Scale, as completed by parents and teacher, and on d2 Attention Endurance Test. Little change was seen on intelligence scales. This demonstrates the efficiency of neurofeedback in reducing ADHD symptoms using standard protocols, however, there was a small sample size.

Within the 2009 meta-analysis, several neurofeedback protocols were examined in the treatment of ADHD including: SMR increase with theta decrease and beta increase with theta decrease. The core locations that were targeted in training were Cz, C3 and C4, across the sensorimotor strip, with a small group of research targeting frontal regions. Analysis showed no difference on outcome measures such as parent and teacher ratings regardless of the type of protocol or location used (Arns et al., 2009).

One case study examined the effect of neurofeedback on an 11-year-old girl who met the criteria for ADHD combined subtype and ODD. She completed 31 neurofeedback sessions whereby theta was downtrained and beta was uptrainined at Fz, and coaching was also performed during the sessions. Upon

completing neurofeedback, the participant no longer met the criteria for ADHD, her concentration was improved on ADHD questionnaires, she was much calmer in her behaviour and was no longer hyperactive when described by her parents and teachers; she also no longer met the ODD criteria. However, it was unclear if the improvements were due to neurofeedback or coaching. The author felt that coaching did not take place at every session and it was not used as an intense therapy, inferring that neurofeedback created improvement (Winklemolen, 2011).

1.4.6.5 Control Group Studies

In neurofeedback studies, a variety of control groups can be used to prevent confounding variables from occurring. Possible confounding variables, if not controlled for, include failure to control treatment bias, combining neurofeedback with different strategies and accounting for therapist-patient interaction. Therefore, the benefit of a control group can ensure that these variables do not occur and that the outcomes are valid (Vernon et al., 2004).

The difficulty with "placebo or sham controlled double blind studies is that they violate fundamental ethical principles guiding human research in circumstances in which known standard treatments are available" (La Vaque & Rossiter, 2001, pp. 24). It was suggested that instead of comparing a relatively new treatment such as neurofeedback to a placebo or sham group, it should be compared to an already established treatment strategy, such as stimulant medication in the treatment of ADHD. Demonstrating that neurofeedback is statistically superior to a placebo condition is one of the conditions required for treatment to be considered "Efficacious and Specific", according to the guidelines for the evaluation of clinical efficacy of psychophysiological interventions (La

Vaque et al., 2002). This is difficult to achieve if research involving placebo conditions cannot take place.

Sham neurofeedback refers to conditions whereby an individual feels they are completing neurofeedback but are actually watching someone else's brain wave activity. A pilot double-blind sham-controlled randomised neurofeedback study was conducted by Arnold et al. (2013). Participants with a diagnosis of ADHD aged 6 to 12 years old, were randomly allocated to active neurofeedback twice a week, active neurofeedback 3 times a week or sham neurofeedback. Active neurofeedback consisted of rewarding a decrease in theta and alpha and an increase in beta. Sham neurofeedback appeared the same as the active group, but the feedback provided was random. Forty, 45-minute sessions were completed. Findings showed a large pre-post improvement on parent ratings but no difference between active and sham neurofeedback. Improvement on parent training plateaued at session 24. It was therefore recommended that neurofeedback is completed 3 times a week, which parents stated they preferred, for 30 sessions. Blinding to conditions did work and sham neurofeedback did not prevent recruitment for the study.

A further sham study was completed by Vollebregt et al. (2014) where 40 ADHD children completed EEG informed neurofeedback or sham neurofeedback for 30 sessions. Results showed no significant treatment effect on neurocognitive variables. The authors explained that existing literature fails to support any benefit of neurofeedback on neurocognitive functions which may be due to small sample sizes in this research field.

As shown here, there are only a few studies using sham neurofeedback in an ADHD population. This may be due to the ethical concerns and therefore other

strategies have been used such as comparing to an established treatment (La Vaque & Rossiter, 2001).

1.4.6.6 Randomised Control Trial (RCT) Studies

The first randomised controlled trial for individuals with ADHD using neurofeedback was conducted in 1996 (Linden, Habib, & Radojevic, 1996). Eighteen children with ADHD aged 5 to 15 years old were randomly allocated to 40, 45-minute beta increase neurofeedback while suppressing theta, or control condition. Findings showed improvements in cognitive measures, specifically on attention and IQ. Since then, more randomised control studies have been conducted, including a meta-analysis.

A large-scale study was conducted by Gevensleben et al. (2009) investigating the efficacy of neurofeedback as a treatment for ADHD. One hundred and two children diagnosed with ADHD, ranging in age from 8 to 12, were randomly assigned to 36 sessions of neurofeedback or computerised attention skills training. Neurofeedback consisted of theta/beta training and slow cortical potential training. Results showed parent and teacher ratings were superior for neurofeedback, demonstrating clinical efficacy for children who have a diagnosis of ADHD.

In a meta-analysis by Arns et al. (2009) several randomised control trials had been completed by this stage (Gevensleben et al., 2009; Holtmann et al., 2009; Leins et al., 2007; Levesque, Beauregard, & Mensour, 2006; Strehl et al., 2006). The meta-analysis of 15 studies showed neurofeedback had a large effect size on inattention and impulsivity and a medium effect size on hyperactivity in both parent and teacher ratings. Two of these studies (Gevensleben et al., 2009; Holtmann et al., 2009) were randomised control trials using computerised attention

skills training as a control condition, and improvement was greater in the neurofeedback conditions.

The most recent meta-analysis examining neurofeedback in the use of ADHD was conducted in 2016 (Cortese et al., 2016). Thirteen trials were included, with a total of 520 participants. Significant effects were found when rated by individuals who were least blinded to the interventions, compared to blinded raters who showed no significant effects. Consequently, this fails to support the use of neurofeedback as a treatment for ADHD.

1.4.6.7 Neurofeedback Compared to Stimulant Medication

An alternative way to examine the effectiveness of a new intervention is to compare to an already established treatment, such as stimulant medication (Arns et al. 2014). There are several studies which have examined neurofeedback compared to stimulant medication (Arns et al., 2009; Fuchs et al., 2003; Monastra et al., 2002; Rossiter, 2004; Rossiter & La Vaque, 1995; Duric, Assmus, & Elgen, 2012; Meisel, Servera, Garcia-Banda, Cardo, & Moreno, 2013). Here it was demonstrated that stimulant medication, namely methylphenidate, was not superior to neurofeedback. These studies will be discussed in more detail.

A neurofeedback RCT study has been conducted examining the effects of neurofeedback and methylphenidate. One hundred and twelve children with a diagnosis of ADHD ranging in age from 7 to 13 were involved. The three conditions consisted of neurofeedback, 30 sessions of theta/beta training at Cz for 10 weeks, physical activity semi-active control group, and methylphenidate. Pre to post EEG findings showed similar reduction in theta activity for individuals who received neurofeedback and methylphenidate. However, individuals who received neurofeedback showed greater overall reductions in ADHD symptoms as

measured by rating scales. Despite this success, improvement was not generalised to classroom behaviours (Janssen et al., 2016).

Meisel et al. (2013) also conducted a randomised control trial to evaluate the efficacy of neurofeedback compared to pharmacological intervention for the treatment of ADHD. Twenty three children diagnosed with ADHD aged between 7 and 14 years old were randomly allocated to 40 theta/beta neurofeedback training sessions or methylphenidate. At a 6 month follow up, similar improvements were reported by parents and teachers, but significant academic improvements were only shown in the neurofeedback condition. This is a significant finding for the first randomised control trial with a six month follow up comparing neurofeedback and methylphenidate in the treatment of ADHD.

Sonuga-Barke et al. (2013) conducted a meta-analysis examining nonpharmacological interventions on ADHD. Eight neurofeedback trials were included. Three trials implemented theta-beta training, one used slow cortical potential training, one used a combination of both of these, and one used personalised training. In trials where observers were aware of the research and intervention, significant treatment effects were found. However, the significant treatment effects were lost when raters (e.g. teachers) were blinded to the intervention group.

1.4.6.8 Neurofeedback in combination with medication

The studies mentioned above did not examine the effect of neurofeedback in combination with stimulant medication on EEG. Information regarding this interaction on the brain may aid in understanding the root cause of ADHD, as well as the best combination of treatments to normalise abnormal brainwaves seen in

ADHD. This is an area that the research presented in this thesis aims to examine. The evidence that is available will now be discussed.

Arns et al. (2009) conducted a meta-analysis examining neurofeedback in ADHD. Three studies compared neurofeedback to stimulant medication, totalling 12% of participants on medication. Findings suggest that the effects of neurofeedback are similar for medicated and unmedicated participants, but further research is required on the impact of stimulant medication on neurofeedback. Furthermore, the studies failed to report or examine any pre to post EEG difference, an area that requires further investigation.

Monastra et al. (2002) conducted neurofeedback on 100 children diagnosed with ADHD, with three conditions: neurofeedback only, neurofeedback with stimulant medication, and stimulant medication only. Following a series of neurofeedback sessions, all children had EEG patterns within one standard deviation of the mean. Measures were repeated 6, 12 and 24 months after the first treatment. At a one year follow up, when medication was present in the patient's system, there were no differences in EEG or behavioural measures between conditions. However, after a one-week washout of stimulant medication, all individuals who did not receive neurofeedback regressed on all measures whereas the neurofeedback group showed significant improvement on ADHD symptoms. Consequently, stimulant medication caused no enduring change as a standalone treatment whereas neurofeedback patients demonstrated long term improvements. Specifically, 80% of individuals who received neurofeedback and stimulant medication were able to reduce their medication dose by at least 50% (Monastra et al., 2002).

Looking specifically at randomised control trials, Duric et al. (2012) investigated 91 children diagnosed with ADHD ranging in age from 6 to 18. Participants were randomly allocated to one of three conditions; 30 participants completed neurofeedback focusing on enhancing cortical beta1activity and suppressing theta, 31 participants were in a methylphenidate control condition, and 30 participants received both neurofeedback and methylphenidate. Parents report on the Clinician's Manual for the Assessment of Disruptive Behaviour Disorders showed significant effects of treatment, but no significant difference between the treatment conditions. Consequently, neurofeedback was seen as effective as methylphenidate in treating ADHD symptoms (Duric et al., 2012).

1.4.6.9 Neurofeedback Setting

The majority of research concerning neurofeedback has taken place in clinical settings, however, there is evidence that neurofeedback is effective in other situations. Wadhwani, Radvanski, and Carmody (1998) investigated the effect of neurofeedback on a ten-year-old boy completing sessions at school. At C3, a beta protocol was used and at Cz a SMR uptraining protocol was used; the sessions lasted up to 30 minutes. Thirty-seven sessions took place over six months. The training was successful with improvement in national achievement tests and results in a school setting were similar to that of a laboratory setting. However, only one participant took part and the study therefore needs to be replicated with a greater number of participants.

More recently, Steiner et al. (2014) also examined the effect of neurofeedback at school. One hundred and four children aged between 7 to 11 years old with ADHD were allocated to neurofeedback, cognitive training, or a control group, while completing the interventions at school. Forty sessions of

neurofeedback were completed. At a 6 month follow up, neurofeedback participants maintained significant gains on Conners' Parent Rating Scale as well as having greater improvements compared to the cognitive training condition.

There is a current trend for individuals wishing to complete neurofeedback at home. This provides individuals with the flexibility of being able to complete neurofeedback when it is most convenient for them, in the comfort of their own home and as often as they wish. Despite this demand, the effectiveness of neurofeedback home training has not yet been examined (Vernon et al., 2004; Rutterford, Anderson, & Venables, 2008). This is an area which this thesis aims to address.

1.4.6.10 Side Effects

There are some potential negative side effects to neurofeedback. There is a very small possibility that neurofeedback could induce seizures, therefore it is absolutely vital that patients are closely monitored (Vernon et al., 2004). Monastra et al. (2002) reported transitional side effects when treated with both stimulant medication and neurofeedback, including irritability and moodiness. These side effects were overcome when the dose of stimulant medication was reduced. Other reported side effects include headaches and dizziness, both of which were overcome by having a rest or something to eat after a neurofeedback session (Monastra et al., 2005).

In conclusion, neurofeedback is a promising new treatment for ADHD. Neurofeedback has been shown to be an effective strategy in reducing ADHD symptoms, specifically impulsive and inattentive symptoms, particularly for patients who are unresponsive to pharmacological treatments such as stimulant medication or who have experienced side effects to medication (Monastra, 2005).

Protocols typically aim to increase SMR or increase beta waves across Cz, C3 and C4 and less often target frontal regions with sessions occurring on average twice a week for three to six months. However, there are still some unknowns about neurofeedback, including its effectiveness when used in the home (Vernon et al., 2004; Rutterford et al., 2008) and its effectiveness when combined with medication (Arns et al., 2009; Monastra et al., 2002; Duric et al., 2012) which this research aims to address.

1.5 Aims

Three research studies have been conducted as part of this thesis. Each study will be presented with a literature review regarding the specific area of focus with their own aims and hypothesis. After conducting the overall literature review, several areas have been identified that require further research which aim to be addressed in this thesis.

Neurofeedback has been shown to have a large effect size on inattention (0.8) and impulsivity (0.7) and a medium effect size (0.4) on hyperactivity (Arns et al., 2009). However, previous neurofeedback research has been conducted in clinical or school settings and not in the home (Vernon et al., 2004; Rutterford et al., 2008). Consequently, we aim to examine the effect of neurofeedback home training on a healthy and ADHD population, examining EEG, personality, and neuropsychological measures.

There have been limited previous research which has examined neurofeedback and stimulant medication. There have been several studies which have compared the two interventions, showing similar effect on ADHD symptoms (Arns et al., 2009; Fuchs et al., 2003; Monastra et al., 2002; Rossiter, 2004; Rossiter & La Vaque, 1995). The present study aims to examine the effect of

stimulant medication and neurofeedback combined on ADHD symptoms, specifically looking at EEG, personality, and neuropsychological measures.

Many previous neurofeedback studies have failed to examine the effect of treatment on EEG (Fritson, Wadkins, Gerdes, & Hof, 2007; Fuchs et al., 2003; Winklemolen, 2011) and therefore we lack understanding of the effect of neurofeedback on the brain. Consequently, in the present study, the aim is to examine the effect of neurofeedback home training on EEG in a healthy and ADHD sample.

Additionally, the present study is being conducted as a feasibility study, with the aim to examine if successful implementation of neurofeedback home training is possible. Consequently, the study aims to examine participant's ability and acceptability to complete the intervention at home in addition to the viability of recruitment.

The purpose of feasibility studies is to assess possible successful implementation of interventions. As described by the United Kingdom's National Institute for Health Research, Evaluation, Trials and Studies Coordination Centre (NETSCC, 2012) "feasibility studies are pieces of research done before a main study in order to answer the questions 'can this study be done?' used to estimate important parameters that need to design the main study" (Research Methods section). A feasibility study tests areas of a possible randomized control trial, whereas a pilot study conducts the whole piece of research on a smaller scale. Both studies are expected to have small sample sizes, consequently, this results in inadequate power statistics. Both feasibility and pilot studies are required to enable successful implementation of randomized control trials (Tickle-Degnen, 2013).

1.6 Significance of the Study

The proposed studies will focus on the application of neurofeedback home training, an area which has not been previously investigated (Vernon et al., 2004; Rutterford et al., 2008)

The outcomes of this research will produce unique, original findings. Specifically, it will examine the effect of neurofeedback and stimulant medication on personality, to our knowledge, an area that has not been previously investigated. If neurofeedback was deemed to alter personality, it may deter individuals from wishing to implement such intervention. There have been limited previous research which has examined neurofeedback and stimulant medication in an ADHD sample. There have been several studies which have compared the two interventions, showing similar effect on ADHD symptoms (Arns et al., 2009; Fuchs et al., 2003; Monastra et al., 2002; Rossiter, 2004; Rossiter & La Vaque, 1995) but the present study aims to examine the effect of stimulant medication in combination with neurofeedback on ADHD symptoms. This will examine what combination of treatments is the most effective in improving diagnosed inattention and impulsive behaviours. This is significant, as depending on the findings, the outcome may influence interventions within the ADHD field.

The research design and methods will ensure that the aims of the study are met in a controlled and rigorous manner, through the inclusion of a control group, randomized allocation to groups, and neuropsychological and psychophysiological measures (taken before and after neurofeedback training). This controlled methodology has often not been achieved in research concerning neurofeedback. Therefore, the present study will make an original contribution to the neurofeedback field and will add to existing knowledge.

2 General Methods

A quantitative approach was used to measure significant change in children's attention and impulsivity. Qualitative measures were a possibility; however, these scales are subjective and difficult to compare from pre- to post-measures as well as across parents and teachers. Consequently, a quantitative methodology was determined to be the most effective approach in this research design to enable direct comparisons across time points and settings.

2.1 Overview of Studies

The research within this thesis focused on the application of neurofeedback home training, an area which has not been previously investigated (Vernon et al., 2004; Rutterford et al., 2008).

Due to the vast number of dependent variables, the research was split into three studies: (1) focusing on personality, (2) focusing on neuropsychological measures, (3) focusing on EEG measures.

In all three studies, a between-participants design was used where pre-measures were initially taken, including quantitative EEG, Behavioural Inhibition System/Behavioural Activation System (BIS/BAS) personality scale, Conners' Parent and Teacher Rating Scale and Conners' Continuous Performance Test (CPT). Dependent on diagnosis, participants were randomly allocated to an intervention which was completed for 15 weeks, and then post-measures completed (repeated pre-measures).

2.2 Ethical Considerations

Informed consent was gained from the parents/guardians of participants. As participants were children and therefore may not have the capacity to provide informed consent, it was necessary to gain consent from parents. However,

children were verbally informed about the research and asked to give verbal consent and written assent, as, legally, children under 18 years cannot give consent for research. A written debrief was provided for parents and children with this verbally given to the participant as well.

High priority was placed on confidentiality. A numbering system was used to identify participants with only the researcher and supervisor team having access to this system. All information was kept in a locked cabinet and a password protected computer. As agreed with participants, parents and teachers were informed regarding involvement in the research, in addition to GPs and the Learning Assessment and Neurocare Centre Ltd consultant were advised of those in the ADHD sample. No one else was made aware of any individuals participation.

The questionnaires examined difficulties that the child may be experiencing or may have experienced in the past. This could have been upsetting to parents and had to be dealt with in a sensitive manner. If either the child or parents were emotionally or physically distressed, they were advised by the information sheet to contact the researcher who could arrange for them to receive coaching or counselling sessions. Furthermore, the neurofeedback home training group emailed the researcher after each session had taken place, enabling the researcher to keep in regular communication with the participant/family and monitor any distress. Ultimately, there were no requests made for coaching or counselling.

Participants were able to withdraw anytime up to 4 weeks after the post measures had been collected. After this point, data were analysed and consequently difficult to identify and withdraw from the dataset. There was no

penalty for withdrawing from this research. No participants withdrew at this stage. However, 11 participants withdrew during the intervention stage.

The ADHD patients Specialist Consultant Paediatrician at the Learning Assessment and Neurocare Centre Ltd was medically qualified and responsible for diagnosis as well as management of the patients with ADHD. He was responsible for prescribing, monitoring and titrating medication to the most appropriate dose if agreed to by the parents/patient. This was not the role of the researcher, who was observing and testing the effects of usual treatment variants.

For children with severe ADHD, the first line of recommended treatment as stated in the NICE guidelines is drug treatment, namely stimulant medication. In this research, medication was only recommended and prescribed by the Consultant if it was felt that this was within the NICE guidelines. The Learning Assessment and Neurocare Centre Ltd complies with the NICE guidelines for both diagnosis and management of individuals with ADHD.

Participants and/or their parents were charged a fee for the assessment and treatment they received at the Learning Assessment and Neurocare Centre Ltd. However, participants and/or their parents did not incur any additional fees for taking part in this research.

2.3 Design

A between participants design was used to ensure unbiased and clear results. Participants were randomly allocated to one of the following conditions, dependant on if they had a diagnosis or not:

- Neurofeedback home training (typically developed sample)
- No neurofeedback: control group (typically developed sample)
- Computer based activities: active control group (typically developed sample)

- Neurofeedback home training (ADHD sample)
- Neurofeedback home training and stimulant medication (ADHD sample)
- Stimulant medication only (ADHD sample)
- Neurofeedback in clinic and stimulant medication (ADHD sample)

The dependent variables obtained at the pre-and post-measure time points, before and after participating in their allocated condition, were:

- electrophysiological measures of EEG
- BIS/BAS personality scale with subscales of BIS, BAS drive, BAS fun seeking, and BAS reward responsiveness
- Conners' 3 Parent Rating Scale with subscales of learning difficulties, executive function, defiance, inattention, and hyperactive/impulsive
- Conners' 3 Teacher Rating Scale with subscales of learning difficulties, executive function, defiance, inattention, and hyperactive/impulsive
- Conners' Continuous Performance Test (CPT) with subscales of omissions, commissions, hit response time, hit response time standard error, variability, detectability, response style, preservations, response time block change, and response time block change standard error

2.3.1 Design Development

2.3.1.1 Design Development: Typically Developed Sample

Initially, the design of this sample was a between participants design, whereby the conditions consisted of neurofeedback home training and a control group of typically developed children.

Having undergone ethical review, the study was expanded to include a third condition, an active control group, where computer-based activities were

completed. The active control group was introduced to overcome potential confounding variables, specifically to see if sitting and watching a computer has an effect on brainwaves and behaviour.

2.3.1.2 Design Development: ADHD Sample

The design of the ADHD sample evolved considerably from the original concept to delivery. Initially, the study was a 2 x 2 between participants design where the conditions consisted of neurofeedback home training, neurofeedback home training and stimulant medication, stimulant medication only and a control group.

This research underwent various ethical reviews, including NHS. Please see Appendix A for detail regarding the ethical reviews. This was a lengthy and time-consuming process which took over 2 years to complete. There were several reasons as to why the process was so difficult and lengthy.

Firstly, it was advised that the research required NHS ethical review. Many questions were raised about the Centre that the research was being conducted at, as well as lack of clarity in the information sheets. However, 2 NHS ethical reviews were completed and alterations made, but were unsuccessful.

Secondly, it was advised by the University that after 2 unsuccessful NHS applications, NHS ethical review was not required, due to patients being private patients and not NHS patients, but instead be reviewed by the University ethics committee.

Feedback from the University ethics committee criticised the use of a waiting list control group in this sample as treatment that could potentially enhance the individuals' quality of life was being withheld. Consequently, the control group was withdrawn. To develop the design even further and to strengthen the

findings, it was suggested by the University ethics committee that two further conditions were added to the design: neurofeedback in clinic only and neurofeedback in clinic with stimulant medication. Incorporating these two conditions meant that comparisons could be drawn between neurofeedback home training and neurofeedback in clinic. These alterations were incorporated into the study and have made a stronger research study.

Thirdly, part way through the ethic review process, the research team (researcher and supervisor) changed University. The research proposal was submitted to the new University ethics committee. However, there was much confusion due to the lengthy process up to this point, why NHS approval was no longer required and why approval had not been granted.

Finally, the application was made explicitly clear that this research was not imposing any treatment that the patients were not already being offered as part of their care at the Centre. At this stage, after 2 years and 3 months, and seven ethical review applications, ethical approval was granted.

2.3.2 Participants

There were two samples of participants that took part: typically developed and ADHD participants.

2.3.2.1 Typically Developed Participants

Participants were a self-selected sample of volunteers who did not have any clinical diagnosis. Participants were children aged between 7 and 17 years old. The reasons for this age group was threefold. Firstly, normative databases for EEGs have been established from the age of 6 to 95 years old and therefore it would not be appropriate to use children below this age due to lack of data (Butnik, 2005). Secondly, children experience rapid brain development until the

age of 7, a process known as maturation. After this age, brain development slows, becomes less erratic and more consistent (Bresnaham & Barry, 2002). Thirdly, the norms for the Conners' Rating Scales are up to the age of 17. Prospective participants were approached via advertising the research on social media, local newspapers and websites. Local schools were contacted to distribute information leaflets but were not willing to do so.

Inclusion criteria:

- Child aged between 7 and 17 years old
- Child does not have any clinical diagnosis
- Child has not previously received any neurofeedback treatment
- Child is not and has not previously taken stimulant medication

Fifty healthy participants completed the pre-measures, 68% were male, 32% female, with a mean age of 10.98 years old. The ADHD sample had a higher male-to-female ratio. This was to be expected as ADHD occurs more in males.

2.3.2.2 ADHD Participants

The sample was self-selected and comprised of volunteers who had attended the Learning Assessment and Neurocare Centre Ltd for an assessment due to concerns regarding concentration. The Centre was an independent, multi-disciplinary lifespan clinic that specialised in the multi-professional assessment and management of children, adolescents, and adults, with complex neurodevelopmental difficulties, especially ADHD.

Prior to approaching a potential participant, the researcher discussed with the participant's consultant, their diagnosis and appropriate treatment. Participants were only informed of the research if both treatments involved in the present

study, neurofeedback and stimulant medication, were offered to them as part of their standard treatment at the Learning Assessment and Neurocare Centre Ltd.

Participants were children aged between 7 and 17 years old who had a main clinical diagnosis of ADHD (combined subtype). Individuals diagnosed with other psychiatric conditions were included as long as they had a main diagnosis of ADHD (combined subtype).

Inclusion criteria:

- Child aged between 7 and 17 years old
- Child who had a main clinical diagnosis of ADHD
- Child who had not previously received a course of methylphenidate or other stimulant medication
- Child who had not previously completed a course of neurofeedback

Forty-one ADHD participants completed the pre-measures; 85% were male, 15% were female, with a mean age of 11 years old. Eighty percent of the ADHD participants had pure ADHD with no additional diagnosed complications. The other twenty percent of participants had one comorbidity of either Autistic Spectrum Disorder (ASD), Developmental Co-ordination Disorder (DCD), Oppositional Defiant Disorder (ODD), or Dyslexia.

2.3.3 Intervention Conditions

Typically developed participants were randomly allocated to one of three conditions:

- Neurofeedback home training (typically developed sample)
- No neurofeedback: control group (typically developed sample)
- Computer based activities: active control group (typically developed sample)

Participants with a diagnosis of ADHD were allocated to one of the following conditions:

- Neurofeedback home training (ADHD sample)
- Neurofeedback home training and stimulant medication (ADHD sample)
- Stimulant medication only (ADHD sample)
- Neurofeedback in clinic and stimulant medication (ADHD sample)

Unfortunately, complete randomisation was not possible. Consideration needed to be made for individuals allocated to the neurofeedback in clinic condition, ensuring they live in close proximity to the Centre to access neurofeedback in clinic twice a week.

2.3.3.1 Neurofeedback Home Training Condition in Typically Developed Sample

Sixteen participants were allocated to the neurofeedback home training condition. The mean age was 10.62 years old, 68.8% were male, 31.3% female.

During the initial meeting, the researcher explained that neurofeedback home training needed to be completed in a quiet environment, in the same room and same time of day, twice a week for 15 weeks.

Neurofeedback home training was administered using the PET Biofeedback system. The software used to digitize the signal and to design the training protocol was BioExplorer. A standard improving concentration protocol was used; an increase in SMR (12 - 16Hz) activity at a threshold of 70% across the sensorimotor strip at locations C3, Cz and C4.

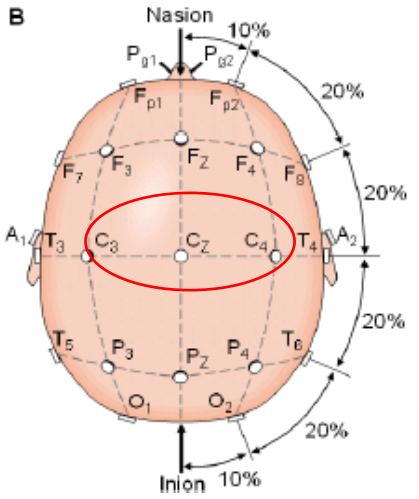

Figure 2. Typically developed sample neurofeedback home training placement.

The written neurofeedback instructions provided for the participant are shown in Appendix O. The scalp and mastoids were cleaned using NuPrep and then a conductive paste, namely Ten20 paste, was used on the electrode on the scalp. A referential montage was used, whereby a reference electrode was attached to the right mastoid and a ground electrode was attached to the left mastoid. The active electrode was first placed on C3, as per the 10-20 system. The 10-20 system is a standard EEG placement system with a minimum of 21 electrodes on the scalp allowing standardised placements of the electrodes. Each electrode name consists of a number and letter. The left side of the brain electrodes are odd and right sided electrodes are even. The letter refers to the brain region: F frontal, T temporal, C central, P parietal, and O occipital (Chong, Sahlem, & Bazil, 2007). At the C3 location, the participant completed 10 minutes of neurofeedback receiving Pacman as a visual and audio reinforcement. The participant then moved the electrode to Cz where the participant completed 10 minutes of neurofeedback receiving Boxes as a visual reinforcement. Finally, the

electrode was moved to C4 where the participant completed 10 minutes of neurofeedback watching Videos with both visual and audio reinforcement.

After every completed session, the participant sent the researcher the neurofeedback session as an attachment to an email for the researcher to review. This enabled the researcher to ensure that the neurofeedback home training sessions were being completed, being completed accurately, and to monitor the participants progress. After every two neurofeedback home training sessions, the researcher emailed the participant regarding their progress. This email took the following format:

- Telling the participant how the session was overall
- Quantitative information - rating the session out of 10 depending if the session had improved or not
- Qualitative information – giving information about the individuals concentration
- Signal quality from electrodes

Good signal

OK signal

*Press the electrode onto the scalp to ensure good connection

Bad signal

*Check the electrodes are correctly attached

- Providing the participant with encouragement

In this feedback email, the participant was also reminded to complete the neurofeedback sessions in the same room, at the same time of day, under the supervision of a parent.

2.3.3.2 Active Control Condition in Typically Developed Sample

Fifteen participants were allocated to the active control condition. The mean age was 10.63 years old, 60% were male, 40% female.

The active control condition completed 30 online computer game sessions, specifically 2 sessions a week for 15 weeks. When allocated to this condition, the participant was provided with written instructions which the researcher discussed with them. The researcher explained that the computer activities were required to be completed in a quiet environment, in the same room and same time of day every time. The written active control condition instructions provided for the participant are included in Appendix P.

The instructions explained that they were required to complete 30 minutes of BBC bitesize games. Thirty minutes of computer activities were used to ensure that the findings would be comparable to the neurofeedback group (whereby the participant was completing 30 minutes of active neurofeedback). The participant was provided with the website address appropriate for the participants age. The participant could then choose which activities they wished to complete.

After each session had been completed, the participant was required to email the researcher to notify them that the session had been completed. The researcher acknowledged this and reminded the participant to complete the sessions in the same room, at the same time of day, under the supervision of a parent.

2.3.3.3 Control Condition in Typically Developed Sample

Fifteen participants were allocated to the control condition. The mean age was 11.26 years old, 66.7% were male, 33.3% female.

The control group did not complete any additional tasks.

2.3.3.4 Neurofeedback Home Training Condition in ADHD Sample

Three participants were allocated to the neurofeedback home training condition. The mean age was 12.27 years old, and all participants were male

Neurofeedback home training for the ADHD sample was conducted in the same way as the typically developed sample, administered using the PET Biofeedback system. The software used to digitize the signal and to design the training protocol was BioExplorer. However, the ADHD sample used a QEEG informed protocol for all neurofeedback training. Consequently, the location and protocol were different for each participant. To determine the protocol, the raw EEG data were fed through a quantitative EEG database. A database is used to compare norms and can show any systematic changes in brainwave frequencies (Cantor & Chabot, 2009). In this research, the Neuroguide database was used, developed by Robert Thatcher. The database contains information from 625 individuals, with both eyes closed and eyes open conditions, and collects 943 variables including measures of power, coherence, phase, and power ratios (Johnstone & Gunkelman, 2010). Participants within the database range from two months old to 82.6 years old. In this research, absolute power was used and the 4 brainwave areas that were the furthest away from the norm (had the most standard deviations away from the norm) were used. The protocol had a reward threshold of 70%. The scalp and mastoids were cleaned using NuPrep and then a conductive paste, namely Ten20 paste, was used on the electrode on the scalp. A referential montage was used, whereby a reference electrode was attached to the right mastoid and a ground electrode was attached to the left mastoid. The active electrode was placed as per the 10-20 system.

2.3.3.5 Neurofeedback Home Training and Stimulant Medication Condition in ADHD Sample

Eight participants were allocated to the neurofeedback home training and stimulant medication condition. The mean age was 8.62 years old, 87.5% were male, 12.5% female.

As described above, neurofeedback home training for the ADHD sample was conducted in the same way as the typically developed sample. However, the ADHD sample used a QEEG informed protocol for all neurofeedback training. Additionally, participants allocated to this condition were prescribed a methylphenidate-based medication: namely Concerta XL, Medikinet XL, Equasym XL or Ritalin. Medication was taken daily at the dose prescribed by their consultant at the Learning Assessment and Neurocare Centre Ltd.

2.3.3.6 Stimulant Medication Condition in ADHD Sample

Nineteen participants were allocated to the stimulant medication condition. The mean age was 11.42 years old, 84.2% were male, 15.8% female.

Participants allocated to the stimulant medication condition were prescribed a methylphenidate-based medication: namely Concerta XL, Medikinet XL, Equasym XL or Ritalin. The Consultant Paediatrician at the Learning Assessment and Neurocare Centre Ltd was responsible for overseeing the participants on medication, ensuring they were on the most effective dose and were not experiencing any negative side effects. Stimulant medication was taken every day.

Table 1.

Detail of participants receiving medication

Drug Name	Frequency	Percent
No medication	10	25
Concerta	2	5
Concerta XL	10	25
Equasym XL	7	17
Medikinet XL	12	30

2.3.3.7 Neurofeedback Clinic Training and Stimulant Medication Condition in ADHD Sample

Four participants were allocated to the neurofeedback clinic training and stimulant medication condition. The mean age was 9.75 years old, all of which were male.

The researcher completed the neurofeedback session with the participant, setting up the equipment and providing verbal feedback about the session. The researcher ensured that the neurofeedback in clinic was completed in a quiet environment, in the same room and same time of day. The same equipment was used as the neurofeedback home training conditions. Additionally, participants allocated to this condition were prescribed a methylphenidate-based medication: namely Concerta XL, Medikinet XL, Equasym XL or Ritalin.

2.4 General Procedure

After discussing a potential participant with the Consultant and ensuring they fulfilled the inclusion criteria, the researcher approached the family via email or phone, providing them with the information sheet to consider. If the family wished to participate, the researcher, parent and child arranged a mutually agreed time to discuss the research and their involvement. This meeting took place at the Learning Assessment and Neurocare Centre Ltd who gave consent for this.

During the initial meeting, the study was explained in detail to the parent and child by the researcher. This meeting provided participants with the appropriate information sheets, consent and assent forms, information on what to do if they wished to withdraw and an opportunity to ask questions. The researcher ensured that the child met the inclusion criteria in terms of age and any clinical diagnosis. If parent and child were happy to proceed, the pre-measures were collected.

If the family consented to participate, the child completed the pre-measures, consisting of:

- EEG
- Conners' Continuous Performance Test
- BIS/BAS personality test

The child's parents and teachers completed the online version of the Conners' 3 Parent/Teacher Rating Scale which was emailed to them after the meeting. The standardised procedures proposed in the manuals of each test were followed to ensure the validity of the results. The tests were aimed at assessing concentration and impulsive abilities.

Following the pre-measures assessments, participants were randomly allocated to one of the conditions by the researcher. The conditions were previously described in Chapter 2.3.3.

If a participant was allocated to any neurofeedback home training condition, the researcher uploaded the appropriate software onto the participants laptop and provided them with a box of neurofeedback equipment, including PET BioExplorer system, NuPrep, 10-20 Paste, electrodes, batteries and a battery

charger. They were also given an interactive demonstration so that they were confident to use the equipment as well as written instructions.

After 15 consecutive weeks of receiving interventions, the measures were completed, namely EEG, Conners' Continuous Performance Test, BIS/BAS personality questionnaire and Conners' 3 Parent and Teacher Rating Scales, were repeated. These were again conducted at the Learning Assessment and Neurocare Centre Ltd. The participant was debriefed, provided with information regarding withdrawing from the study, who to contact if they had any concerns about the research or if they had concerns regarding their child, and were thanked for their time and effort. The child and parent were provided with debrief sheets and an opportunity to ask any questions.

2.5 Statistical Analysis

All statistical analysis was performed using the software SPSS 18.0. Significance level was set to $p < 0.05$. When referring to confidence intervals, all tables are set to 95% confidence intervals.

As many comparisons between variables, conditions and diagnosis were being performed, host hoc adjustments were necessary. The Bonferroni correction was used to reduce the chances of gaining type I error results. Specifically, if a null hypothesis is true, a significant difference would still be found at least once in every 20 trials. A Bonferroni correction consists of dividing the *p* value (0.05) by the number of comparisons (Perneger, 1998).

However, there is an argument that the Bonferroni correction can create more problems than solutions. Although the Bonferroni correction reduces the chance of type I error, it increases the likeness of type II errors. The Bonferroni correction is appropriate in some situations including repetition of the same tests in multiple

subsamples, when the null hypothesis has importance and when it is imperative to avoid a type I error (Armstong, 2014). Even in this situation, it is argued that describing the analysis, its significance and importance is the best way of dealing with multiple comparisons (Perneger, 1998).

In the present research, many comparisons between variables were made. Therefore, Bonferroni correction was based upon the amount of comparisons made within one specific measure. For example, the Conners' Parent Rating Scale consisted of 5 variables (learning difficulties, executive functions, defiance, inattention and hyperactive/impulsive). Consequently, with the Bonferroni correction, *p* value is set to 0.01 for the Conners' Parent Rating Scale. In the subsequent tables, *p* values will be left uncorrected, however, indication will be made as to which ones are significant after Bonferroni correction.

3 Study One: Effect of Neurofeedback on Personality

3.1 Introduction

Personality is defined as "the sum total of the behavioural and mental characteristics that are distinctive of an individual, and the personal qualities that make a person socially popular" (Colman, 2006, pp. 564). In accordance with the DSM-5 (APA, 2013) a personality trait is defined as "enduring patterns of perceiving, relating to, and thinking about the environment and oneself that are exhibited in a wide range of social and personal contexts" (APA, 2013, pp.686).

3.1.1 Personality and ADHD

There are many theories that attempt to explain personality including: psychodynamic, trait, social learning, and social cognitive theory (Gleitman, Fridluind, & Reisberg, 2004). Some of these theories will now be discussed in more detail in relation to ADHD.

3.1.1.1 Social Cognitive Theory

One approach to personality is the social-cognitive theory. Bandura is a key figure in the development of the social-cognitive theory which has two underlying principles: (1) the person, environment and behaviour influence one another and (2) our conscious cognitive capabilities dictate our ability to reflect on ourselves. The theory focuses on our ability to think and reflect on our past, present and future experiences as well as the capacity we have to influence our own development. Our thinking develops through interaction with the environment and our behaviours are learnt through observation, modelling, and positive reinforcement. Behaviours are likely to be strengthened when others are observed exhibiting the behaviour and particularly if the behaviour is rewarded (Gleitman et al., 2004).

Cailles, Bertot, Motte, Raynaud, and Abely (2014) specifically examined ADHD in relation to the social cognitive theory. In a sample of 30 children, 15 diagnosed with ADHD, 15 typically developed, aged 7 to 10 years old, assessment took place including examining theory of mind and executive function. Findings showed that individuals with ADHD experience problems with theory of mind, as well as inhibition control deficits.

Martin, Burns, and Collie (2017) also examined the social cognitive perspective in relation to ADHD. One hundred and sixty-four children with ADHD as well as 4658 healthy individuals, aged 11 to 13 years old, underwent a self-efficacy assessment as well as literacy and numeracy tests. Children with ADHD showed significantly stronger associations between self-efficacy, relational support and academic achievement.

3.1.1.2 The Psychodynamic Theory

The psychodynamic approach was developed from Freud's psychoanalytic theory. The theory states that our behaviours are a consequence of our unconscious influences. The theory states that our mind consists of three areas: conscious (how aware we are at any given moment), preconscious (what we could become aware of if we attended to it), and unconscious (part of the mind we are unaware of). Our personalities are formed in our mind, specifically in the unconscious and conscious areas, by conflicts that occurred during childhood. These conflicts have an impact on the development on the subsystems of personality: the id, ego and superego. Freud went on to explain that during childhood we go through stages of psychosexual development. How we progress through these stages and solve conflicts in each stage determines our personality later in life. Additionally, our personality involves several factors including:

instinctual drives, specifically food, sex and aggression, unconscious processes, and early childhood influences including our role models. Personality development undergoes rapid change during the first five years of life through the interplay of instinct and environment. Personality abnormalities and mental health difficulties are often tracked back to this crucial period of personality development (Freud, 1920). However, there has been much controversy surrounding this theory due to the lack of supporting evidence (Gleitman et al., 2004).

There is little literature available in regard to the psychodynamic approach to personality and ADHD. The approach describes hyperactivity as the result of avoiding emotional discomfort, therefore avoiding one's emotional turmoil. The theory states that the ADHD brain has difficulty balancing mental process as well as self-regulation. Furthermore, many relationships between others and a child with ADHD are negative. Consequently, as underlying emotions are not being dealt with, impulsive behaviour is exhibited along with a negative mindset and placing blame on others, known as learnt helplessness. As discussed earlier in this thesis, the lack of a father figure can contribute to the development of ADHD. From a psychodynamic perspective, lack of a father figure causes the child to represent the father and analyse any violence, which is exhibited as impulsive behaviour (Salomonsoon, 2017).

3.1.1.3 The Biological Trait Theory

The biological trait theory of personality was developed by Eysenck (1967) and assumes behaviour is determined by relatively stable traits. The theory explains extroversion as low levels of cortical arousal where individuals are impulsive but sociable, whereas introversion is characterised by high levels of cortical arousal and caution (Eysenck, 1967). This theory was further developed to

identify five major dimensions of personality, now known as the Big Five. This consists of: extroversion, neuroticism, agreeableness, conscientiousness, and openness to experience (Gleitman et al., 2004).

Shaw and Giambra (1993) examined the Big Five in relation to ADHD. Nine male and four female students with a diagnosis of ADHD completed questionnaires including the Sensation Seeking Scale and Conners Abbreviated Rating Scale. Results showed ADHD individuals to be hyper aroused and seeking high levels of sensation, due to boredom being stressful for them.

Furthermore, Braaten, and Rosen (1997) examined 68 students with ADHD compared to 59 control students. Several questionnaires were completed including Patient's Behaviour Checklist, Early Childhood Environment Rating Scale and Environmental Impact Statement. The ADHD population were positively associated with extraversion as well as neuroticism. High neurotic traits were associated with high emotional intensity and strong emotional reactions. Specifically, the ADHD sample were significantly lower on emotional reaction to punishment, showing that punishment is a less effective behavioural strategy in children with ADHD. Neuroticism, in the case of ADHD, can express itself through emotional liability. Specifically, neuroticism is a significant predictor of inattentive, hyperactive, and impulsive behaviours, unlike extraversion. The understanding behind the connection between ADHD and extraversion is that individuals with ADHD lack internal stimulation, and therefore compensate for this through disruptive behaviour patterns (Parker, Majeski, & Collin, 2004). This finding was from a sample of 587 students with a mean age of 19 years old who completed the Conners' Adult ADHD Rating Scale and the Personality Inventory. However, the other areas of personality as derived from the Big Five Theory,

including openness to experience, agreeableness and conscientiousness, has had little research conducted in relation to ADHD and are often ignored (Nigg, 2000).

3.1.1.4 The Biopsychosocial Model

The biopsychosocial model of personality has also explored ADHD. This model is based upon seven dimensions of personality. Three dimensions are based upon character: self-directed cooperativeness, self-transcendence and persistence, with a further four temperament dimensions: novelty-seeking, harm avoidance, reward dependence, and self-transcendence. In this theory novelty-seeking is the tendency to approach novel situations for rewards, and to experience relief from non-punishment. High novelty-seeking behaviours include impulsivity, quick-temper, and proneness to breaking rules. Harm avoidance behaviours include the tendency to inhibit or avoid responses to aversive cues, such as punishment and non-reward. High harm avoidance is associated with high anticipatory anxiety and fear. Reward dependence is the tendency to maintain responses that have been previously conditioned through rewards. High reward dependence is associated with being sociable and sensitive to social cues. Persistence is the tendency to maintain responses, despite frustration and fatigue. High persistence is associated with persevering and being ambitious. Self-directedness reflects the ability to control, regulate and adapt one's behaviour to a situation in order to achieve one's goals and values. Cooperation reflects identification with, and acceptance of, others. Self-transcendence reflects imaginativeness and spirituality (Gomez, Van Doorm, Watson, Gomez, & Stavropoulos, 2017).

Gomez et al. (2017) conducted a meta-analysis examining the biopsychosocial theory of personality in relation to ADHD. Fifteen studies were included, all of which consisted of ADHD samples compared to healthy controls.

Novelty-seeking and harm avoidance were positively associated with ADHD whereas reward dependence, persistence, self-directedness and cooperativeness were significantly negatively associated with ADHD. The personality dimensions were not affected by age. The significance of novelty-seeking and ADHD shows that these individuals tend to approach new situations for reward and for non-punishment. This system is associated with dopaminergic activity, of which there is a deficit in ADHD, as well as serotonin-transporters. Consequently, the significant result of ADHD and novelty-seeking in the present study suggests that ADHD is associated with dopamine and serotonin. The negative association between self-directedness and ADHD show a lack of ability to control, regulate and adapt behaviour.

3.1.1.5 Reinforcement Sensitivity Theory

Gray's (1972) personality theory, referred to as the Reinforcement Sensitivity Theory (RST) is based upon the biological basis of personality. The aim of the theory was to identify the brain-behavioural systems involved in the variation of human behaviour and to relate these systems to a measure of personality (Corr, 2008).

The theory was originally adapted from Eysenck's early theory of personality. However, Gray argued that the extraversion and introversion dimensions should be altered to create punishment sensitivity, presenting as anxiety, and reward sensitivity, presenting as impulsivity. Gray predicted that impulsively charged individuals are more sensitive to signals of reward whereas anxiously charged individuals are more sensitive to signals of punishment (Corr, 2004). Gray's RST of personality consists of three systems.

The first system is the Behavioural Approach System (BAS) which seeks reward and is responsible for feeling positive. This activation/impulsive system means that the individual seeks rewards and positive emotions which are sensitive to reward (Corr, 2008). Individuals who experience heightened BAS may have sociopathic personality and can be associated with disorders such as bipolar and conduct disorder (Carver & White, 1994). Within the brain, the BAS uses dopaminergic pathways particularly within the basal ganglia (Carver & White, 1994) in addition to the prefrontal cortex, the ventral tegmental area, the nucleus acumbens and the ventral striatum (Gomez & Corr, 2014).

The second system in the RST is the Behavioural Inhibition System (BIS) which is the aversive motivational system involved in resolving conflicts. Within the brain, the BIS uses the septohippocampal system, monoaminergic afferents from the brainstem, and the neocortical projection in the frontal lobe which consequently creates a feeling of anxiety (Carver & White, 1994). The BIS is sensitive to and influenced by punishment, nonreward and novelty and is responsible for negative emotions. Individuals who experience heightened BIS may experience difficulties linked to anxiety, particularly childhood anxiety disorders, and depressive disorders. These individuals are sensitive to fear and punishment and learn best from punishment (Jackson, 2003). On the other hand, individuals who experience underactive BIS and therefore have impaired inhibition to punishment, may experience Attention Deficit Hyperactivity Disorder symptoms (Carver & White, 1994).

The third system in the original RST is the Fight-Flight System (FFS) which motivates behaviours to avoid or escape certain stimuli such as fear. Brain areas that are activated and associated with this system include the periaqueductal

gray matter, medial hypothalamus, amygdale, anterior cingulated and prefrontal ventral stream. The FFS is sensitive to unconditioned aversive stimuli which provokes emotions such as panic (Corr, 2004).

The RST has largely been examined in an adult population. For the RST to be completely understood, it is important for personality to be examined across development as our behaviour changes, particularly during adolescence. For example, an increase in risk taking is seen in adolescence, consequently it is linked to an increase in reward responsiveness (Pagliaccio et al., 2016). Specifically, Urosevic, Collins, Muetzel, Lim, and Luciana (2012) examined 83 male and 101 female healthy individuals, aged 9 to 23 years old, who completed the BIS/BAS scale and MRI, which was repeated 2 years later. BAS reward responsiveness was seen to peak in mid to late adolescence and decline in adulthood. This change on BAS reward responsiveness is associated with developmental brain changes, specifically structural changes in brain regions and a decrease in brain volume. Similarly, a further study found a positive association between early personality development with age on the BIS/BAS scale, with a peak of BAS reward responsiveness in young adulthood followed by a decline. This finding is consistent with neuroimaging literature regarding brain development (Pagliaccio et al., 2016).

Furthermore, a female male difference was found, with adult females having higher BIS scores as well as higher BIS sensitivity with age. This could be due to females internalizing psychopathology, particularly during adolescence, and consequently altering personality in adulthood. Furthermore, females on average showed higher BAS reward responsiveness scores and males showed higher BAS

drive scores (Pagliaccio et al., 2016). However, this needs to be examined further in a child population.

There are limited studies that have examined the relationship between the RST and individuals with ADHD, but the findings that do exist are inconclusive (Barkley, DuPaul, & McMurray, 1990; Hundt, Kimbrel, Mitchell, & Nelson-Gray, 2008; Mitchell & Nelson-Gray, 2006; Gomez & Corr, 2010).

Overactive BAS is often seen in ADHD combined subtype and ADHD hyperactive impulsive subtype, giving more impulsive responses and an inability to switch attention. An overactive BAS system means that the individual is unable to resolve goal conflicts, is unable to sustain attention, and is engaged in task-irrelevant activities. In individuals with ADHD, this is consequently displayed as inattention and distractibility. The inattentive subtype of ADHD has a different association with the RST, specifically a negative relation to BAS resulting in low levels of anger and frustration but have higher anxiety levels (Barkley et al., 1990).

Hundt, Kimbrel, Mitchell, and Nelson-Gray (2008) examined 273 adults using the Sensitivity to Punishment and Sensitivity to Reward Questionnaires and ADHD Rating Scale. Results showed that high BAS was associated with hyperactive-impulsive symptoms of ADHD as well as alcohol abuse and psychopathy. Consequently, high BAS may be a factor towards externalising difficulties. Low BIS was also associated with hyperactive-impulsive symptoms, particularly when inattentive symptoms are not present. This shows that high BAS is a trait associated with external difficulties. In regard to inattentive ADHD, high BIS and low BAS are associated with these symptoms, with the possibility of being linked to anxiety and depression.

Mitchell and Nelson-Gray (2006) found all subtypes of ADHD to be associated with both BIS and BAS tendencies. Specifically, they examined 209 undergraduate psychology students, with a mean age of 18 years old. ADHD rating scale and Sensitivity to Punishment and Sensitivity to Reward Questionnaires were completed. BAS was significantly correlated with hyperactive-impulsive symptoms, whereas BIS was only moderate in comparison. Specifically, BAS scores were a predictor of hyperactive-impulsive symptoms whereas BIS scores were not. This finding supports that overactive BAS and behavioural control deficits are due to disinhibition in ADHD, although this is not supported for ADHD inattentive subtype.

In a study by Gomez and Corr (2010) the inattentive subtype of ADHD was examined. Two hundred and fourteen adults completed the BIS/BAS scale, the Sensitivity to Punishment and Sensitivity to Reward Questionnaire and ADHD ratings. Inattentive ADHD was positively associated with BIS and sensitivity to punishment, specifically BIS anxiety and BIS fear. ADHD combined subtype was positively associated with BAS reward responsiveness, BAS drive, positive emotionality, and high reward sensitivity.

In conclusion, these studies show that adult ADHD is positively associated with high BAS, which appear to lead to hyperactive-impulsive symptoms (Mitchell & Nelson-Gray, 2006; Barkley, DuPaul, & McMurray, 1990; Hundt et al., 2008). However, findings regarding ADHD inattentive subtype are less conclusive, but research suggests it is associated with BIS (Gomez & Corr, 2010). The research presented here regarding RST and ADHD have been conducted in adult populations. Consequently, this needs to be replicated in a child population to fully understand the development of personality in an ADHD sample.

3.1.2 Personality and EEG Patterns

Specific EEG patterns have been shown to be associated with personality traits. It has been suggested that, as a broad trend, extroverts show more low-arousal EEG activity such as alpha waves, when compared to introverts (Stenberg, 1992).

In a study of 41 students, aged 18 to 46, participants underwent 19 electrode EEG while exposure to pleasant, neutral and unpleasant images. Findings showed highly impulsive individuals to have higher levels of low arousal, specifically higher levels of slow theta activity (Stenberg, 1992).

In the biological theory of personality from which the RST is derived, Eysenck explained that differences in extroversion and neuroticism was due to differences in cortical arousal detected by EEG. However, little research has investigated this area. Of the research that is available, there are two key findings on the relationship between RST and EEG patterns. Firstly, higher levels of left frontal activity are related to high BAS scores. Secondly, high levels of activity in the right frontal areas of the brain are related to high BIS scores. Looking specifically at the BAS scale, EEG patterns include frontal asymmetry in alpha activity and posterior versus frontal theta activity which in turn is linked to extraversion (Wacker, Chavanon, & Stemmler, 2010). Furthermore, Gray and McNaughton (2000) understood that activation of BIS produces the theta rhythm.

To our knowledge, no previous research has examined the RST in relation to EEG in an ADHD sample. Although we know that ADHD presents with higher levels of BAS compared to typically developed peers (Mitchell & Nelson-Gray, 2006; Barkley et al., 1990; Hundt et al., 2008), it is not known how high levels of BAS affects the ADHD brain. Comparisons between a typically developed sample

and an ADHD sample will enable us to understand the effect ADHD has on the brain as well as on personality.

3.1.3 Personality and Neurofeedback

There is evidence to suggest that there are some treatments that can influence our personality, specifically neurofeedback (Peniston & Kulkosky, 1990), although how it affects personality is unclear.

Peniston and Kulkosky (1990) examined 20 people with alcohol dependency with 20 controls, using the Millon Clinical Multiaxial Inventory and Sixteen Personality Factor Questionnaire. Participants underwent medical treatment or alpha-beta neurofeedback. The neurofeedback condition saw a significant increase in personality warmth, abstract thinking, stability, conscientiousness, boldness, imaginativeness, and self-control, compared to the medical treatment who only saw am increase in concrete-thinking.

However, Raymond, Varney, Parkinson, and Gruzelier (2005) did not support Peniston and Kulkosky (1990) results. Raymond et al. (2005) allocated 12 medical students to active or sham neurofeedback. The neurofeedback consisted of eyes closed alpha frequency at Pz. When high levels of alpha were produced, a babbling brook sound was heard. Two sessions a week for 5 weeks were completed. Prior and post neurofeedback, the Personality Syndrome Questionnaire was completed. Results showed no significant difference on results between the active or sham neurofeedback. This could be due to a small number of neurofeedback sessions or that personality is too robust to change.

As seen here, there is very limited research examining the effect of neurofeedback on personality, with no research available in an ADHD or child

sample. Further research is required to understand the implications of neurofeedback on personality.

3.1.4 Personality and Stimulant Medication

An individual's response to particular medications can be affected by their personality. Response to stimulant medication, namely *d*-amphetamine, has been found to depend on the individual's personality traits. Corr and Kumari (2000) found that individuals low in psychoticism traits were more energetically aroused with reduced tense arousal when taking *d*-amphetamine. This is compared to individuals high in psychoticism who became lowered in energetic arousal and increased in tense arousal. In their study, it was found that other personality traits including novelty seeking and extraversion were not modified by *d*-amphetamine (Corr & Kumari, 2000).

There is limited knowledge and research about the relationship between ADHD and personality, specifically EEG measures and the effect of treatment on this relationship. Although there is research showing the benefits of treatment on ADHD symptoms, it is unclear as to if and how it affects an individual's personality. This gap in previous literature hopes to be addressed through the research conducted here, enabling effective management of the condition without altering the person's underlying personality.

3.2 Aims of the Study

The present study aimed to examine the effect of neurofeedback and stimulant medication on personality in a typically developed and ADHD sample. As discussed in the literature review, little is known about the effect of these treatments on personality, in a child population and in an ADHD population. If

neurofeedback was deemed to alter personality, it may deter individuals from wanting to implement such intervention.

3.3 Hypothesis

Based upon the previous research and the aims of the present study, the hypotheses for the pre-measures were as follows:

- The ADHD sample would score more highly on the BAS and lower on the BIS personality scale than the typically developed sample.

Based upon the previous research and the aims of the present study, the hypotheses for the post-measures were as follows:

- The ADHD sample who received neurofeedback home training and medication would have the most changed personality, with lowered BAS and increased BIS results across all samples.
- The typically developed sample, specifically the control and active control conditions, will notice no change on the BIS/BAS scale across time.

3.4 Method

For detail regarding participants and interventions, please see Chapter 2: General Methods.

3.4.1 Personality Measures

The present study used the BIS/BAS Personality Scale. The measures were conducted on two occasions, once during the initial meeting prior to any intervention, and 15 weeks later once the interventions had been completed.

The BIS/BAS Personality Scale consists of two scales, the Behavioural Inhibition Scale (BIS) and the Behavioural Activation Scale (BAS) and was devised by Carver and White (1994) based upon American undergraduate students. The BIS measures actions such as “I worry about making mistakes.” The BAS

scale measures reward responsiveness, for example, “When I get something I want, I feel excited and energised”, as well as drive, for example “I go out of my way to get things I want”, and fun seeking behaviours, for example “I crave excitement and new sensations”. The scale had been devised to be used in conjunction with the original RST. The BIS/BAS scale is presented as a questionnaire with 24 statements.

Participants were asked to rate how much they agree or disagree with each statement using a four-point scale and were told that there were no right or wrong answers. Participants took as long as they needed to complete the questionnaire.

3.5 Results

As the BIS/BAS scale was completed at 2 time points, pre-measures before any intervention and post-measures after any intervention, the results have been presented in this way.

3.5.1 Results from BIS/BAS Pre-measures

A multivariate analysis of variance (MANOVA) was conducted to examine the overall effect of diagnosis and gender on the BIS/BAS scale. A MANOVA is an extension of an ANOVA, examining the statistical differences of two or more dependent variables. Overall, there was no significant effect for gender F (5, 80) = 0.056, p<0.72; Wilk’s = 0.96, or diagnosis, F (5, 80) = 0.806, p<0.54; Wilk’s = 0.95.

T-tests were conducted to assess any statistical differences between the typically developed sample and ADHD sample (Table 2).

Table 2.

Descriptive statistics and T-test results of pre-measure dependent variables on independent variable, diagnosis

	Typically Developed Sample			ADHD Sample					
	N	Mean	SD	N	Mean	SD	t	df	Sig. (2-tailed)
BIS	48	14.92	3.19	39	14.87	2.86	0.74	85	0.94
BAS Drive	48	7.97	2.59	39	8.89	2.92	-1.53	85	0.12
BAS Fun Seeking	48	6.97	2.00	39	7.27	1.72	-0.74	85	0.46
BAS Reward Responsiveness	48	7.53	2.47	39	7.35	1.8	0.39	85	0.69

On the BIS/BAS scale, the scores between the ADHD and typically developed sample were not too dissimilar, specifically not significantly different.

A Pearson correlation coefficient was then computed to assess the relationship between the independent variables and age across the two samples.

Table 3.

Pearson Product Moment correlations for dependent variables and age on the typically developed sample

	N	Pearson Correlation	Strength of relationship	Sig
BIS	48	0.19	Weak positive	0.18
BAS Drive	48	0.15	Weak positive	0.29
BAS Fun Seeking	48	0.39	Moderate positive	0.006
BAS Reward Responsiveness	48	0.22	Weak positive	0.13

As shown above, age had a significantly positive correlation on BAS fun seeking in the typically developed sample.

Table 4.

Pearson Product Moment correlations for dependent variables and age on the ADHD sample

	N	Pearson Correlation	Strength of Relationship	Sig
BIS	39	0.28	Weak positive	0.07
BAS Drive	39	0.32	Moderate positive	0.04
BAS Fun Seeking	39	0.19	Weak positive	0.23
BAS Reward Responsiveness	39	0.43	Moderate positive	0.006

As shown above, age had a significantly positive correlation on BAS drive and BAS reward responsiveness in the ADHD sample.

3.5.2 Results from BIS/BAS Post-measures

A MANOVA was conducted to assess how diagnosis and gender combined affect the overall post measures. Overall, there was no significant effect of the BIS/BAS scale on post measures (gender F (4, 68) = 0.73, p<0.57; Wilk's = 0.95; diagnosis F (4, 68) = 1.24, p<0.31; Wilk's = 0.93).

T-tests were then conducted to assess any statistical differences between the typically developed sample and the ADHD sample across all of the dependent variables at the second-time point, post measures (Table 5).

Table 5.

T-test results of post measure dependent variables on independent variable, diagnosis

	Typically Developed Sample			ADHD Sample					
	N	Mean	SD	N	Mean	SD	t	df	Sig. (2-tailed)
BIS	41	15.83	3.43	34	16.5	4.14	0.73	73	0.46
BAS Drive	41	9.24	2.89	34	8.11	3.03	-1.64	73	0.11
BAS Fun Seeking	41	9.58	12.13	34	7.02	1.6	-1.21	74	0.22
BAS Reward Responsiveness	41	8.31	5.02	34	7.67	2.39	-0.67	74	0.51

A Pearson correlation coefficient was then computed to assess the relationship between the independent variables and age at the post measures time point (Table 6 and 7).

Table 6.

Pearson - product moment correlations for dependent variables and age on the typically developed sample for post measures

	N	Pearson Correlation	Strength of correlation	Sig
BIS	42	0.005	No relationship	0.97
BAS Drive	42	0.05	No relationship	0.72
BAS Fun Seeking	42	-0.03	No relationship	0.81
BAS Reward Responsiveness	42	-0.06	No relationship	0.69

Table 7.

Pearson - product moment correlations for dependent variables and age on the ADHD sample for post measures

	N	Pearson Correlation	Strength of correlation	Sig
BIS	34	-0.06	No relationship	0.71
BAS Drive	34	0.26	No relationship	0.12
BAS Fun Seeking	34	0.32	Weak positive	0.06
BAS Reward Responsiveness	34	0.52	Moderate positive	0.001

As Table 6 and 7 show, age had no correlation on the post measures in the typically developed sample, but had a significantly positive correlation on BAS reward responsiveness in the ADHD sample.

3.5.3 Results Comparing Pre to Post BIS/BAS Measures

A repeated MANOVA was then conducted to examine the overall effect of pre- to post BIS/BAS across all measures (Table 8). This indicated if there were any overall significant effects that therefore needed examining in more detail.

Table 8.

Repeated MANOVA, pre-to post across typically developed and ADHD sample

	Pre to Post			Gender			Diagnosis		
	Mean	F	Sig.	Mean	F	Sig.	Mean	F	Sig.
BIS	42.76	5.84	0.02	0.91	0.12	0.72	3.53	0.48	0.49
BAS Drive	0.201	0.06	0.81	3.48	0.99	0.32	0.22	0.06	0.79
BAS Fun Seeking	0.43	0.01	0.92	69.05	1.58	0.21	23.2	0.53	0.47
BAS Reward Responsiveness	111.42	0.11	0.76	2858	2.46	0.12	283.5	0.24	0.62

Table 8 shows across the two time points, there was no significant effect of gender and diagnosis on the ADHD sample. Furthermore, a repeated measures multivariate ANOVA with a Bonferroni correction was conducted to examine the significant difference between the BIS/BAS Scale and the study conditions. No significant differences were found.

A paired t-test was then conducted to compare the independent variables from the first time point, pre measures, to after any intervention, post measures.

Table 9.

Paired T-test results of pre-to post measure dependent variables

	Mean Difference	SD	t	df	Sig. (2-tailed)
BIS	-1.22	3.87	-2.74	75	0.009[a]
BAS Drive	-0.09	2.62	-0.31	75	0.75
BAS Fun Seeking	-1.21	9.28	-1.13	75	0.26
BAS Reward Responsiveness	-0.99	9.48	-0.92	75	0.35

[a] Significant after Bonferroni correction. Calculation of 0.05/4=0.012

As Table 9 shows, there was a significant main effect of diagnosis on the dependent variables on the BIS scale. This was examined further by looking at the two samples independently (Table 10 and 11).

Table 10.

Paired T-test results of pre-to post measure dependent variables on typically developed sample

	Mean Difference	SD	t	df	Sig. (2-tailed)
BIS	-0.85	3.28	-1.66	41	0.103
BAS Drive	-0.09	2.46	-0.25	41	0.79
BAS Fun Seeking	-2.27	12.35	-1.19	41	0.24
BAS Reward Responsiveness	-0.92	5.22	-1.15	41	0.25

Table 11.

Paired T-test results of pre to post measure dependent variables on ADHD sample

	Mean Difference	SD	t	df	Sig. (2-tailed)
BIS	-1.67	4.49	-2.17	33	0.03
BAS Drive	-0.08	2.84	-0.18	33	0.85
BAS Fun Seeking	0.11	1.68	0.407	33	0.68
BAS Reward Responsiveness	-0.17	1.86	-0.55	33	0.58

[a] Significant after Bonferroni correction. Calculation of 0.05/4=0.012

There were no significant differences from pre-to post on the typically developed BIS/BAS scale. However, there was a significant difference on pre-to post on BIS in ADHD conditions, although this was not significant once Bonferroni correction was applied.

Table 12 demonstrates the absolute mean change scores across several measures from pre to post intervention.

Table 12.

Descriptive statistics Absolute Mean Change Scores from pre to post measures

	T-test of dependent variables on diagnosis		Pearson Product correlation of age	
	Typically developed sample	ADHD sample	Typically developed sample	ADHD sample
BIS	0.91	1.63	-0.185	-0.34
BAS Drive	1.27	-0.78	-0.1	-0.06
BAS Fun Seeking	2.61	-0.25	-0.42	0.13
BAS Reward Responsiveness	0.78	0.32	-0.28	0.09

Paired t-tests were then conducted on each condition, comparing pre-to post measures on all the dependent variables (Table 13 to Table 19). This examined the main effect of intervention on the dependent variables.

Table 13.

Repeated measures t-test from pre-to-post-test differences in the various measures for the typically developed sample neurofeedback condition

	Neurofeedback							
	Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Confidence Interval Lower	95% Confidence Interval Upper
BIS	-0.77	3.75	-0.74	12	0.47	-0.21	-1.94	1.78
BAS Drive	0.54	2.11	0.92	12	0.37	0.26	-0.14	1.98
BAS Fun Seeking	-0.69	1.71	-1.47	12	0.17	0.17	-1.68	0.51
BAS Reward Responsiveness	-1.15	2.38	-1.75	12	0.11	0.11	-1.32	0.65

Table 14.

Repeated measures t-test from pre-to-post-test differences in the various measures for the typically developed sample control condition

	Control							
	Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Confidence Interval Lower	95% Confidence Interval Upper
BIS	-1	3.38	-1.15	14	0.27	-0.31	-2.9	0.87
BAS Drive	-0.93	3.13	-1.16	14	0.27	-0.31	-2.7	0.79
BAS Fun Seeking	0.13	2.03	0.25	14	0.79	0.06	-1.11	1.25
BAS Reward Responsiveness	0.6	2.69	0.86	14	0.39	0.22	-0.9	1.58

Table 15.

Repeated measures t-test from pre-post-test differences in the various measures for the typically developed sample active control condition

	Active Control							
	Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Confidence Interval Lower	95% Confidence Interval Upper
BIS	-1.37	3.58	-1.43	13	0.18	-0.38	-3.43	0.69
BAS Drive	-0.08	2.01	-0.14	13	0.89	-0.04	-1.23	1.08
BAS Fun Seeking	-6.04	20.4	-1.15	14	0.27	-0.29	-17.3	5.27
BAS Reward Responsiveness	-6.17	20.5	-1.17	14	0.26	-0.29	-7.23	1.89

Table 16.

Repeated measures t-test from pre-post-test differences in the various measures for the ADHD sample medication condition

	Medication							
	Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Confidence Interval Lower	95% Confidence Interval Upper
BIS	-1.58	5.24	-1.31	18	0.21	-0.3	-4.11	0.94
BAS Drive	0.11	3.09	0.15	18	0.88	0.04	-1.38	1.59
BAS Fun Seeking	0.05	1.43	0.16	18	0.87	0.03	-0.63	74
BAS Reward Responsiveness	0.26	1.56	0.74	18	0.47	0.17	-1.02	0.71

Table 17.

Repeated measures t-test from pre-post-test differences in the various measures for the ADHD sample medication and neurofeedback home training condition

	Medication and Neurofeedback Home Training							
	Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Confidence Interval Lower	95% Confidence Interval Upper
BIS	-0.86	2.12	-1.07	6	0.32	-0.4	-2.81	1.09
BAS Drive	-0.43	2.37	-0.48	6	0.65	-0.2	-2.62	1.76
BAS Fun Seeking	-0.57	1.81	-0.83	6	0.44	-0.3	-2.24	1.11
BAS Reward Responsiveness	-0.29	2.29	-0.33	6	0.75	-0.1	-2.02	2.31

Table 18.

Repeated measures t-test from pre-post-test differences in the various measures for the ADHD sample medication and neurofeedback clinic training condition

	Medication and Neurofeedback Clinic Training							
	Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Confidence Interval Lower	95% Confidence Interval Upper
BIS	0	2.71	0	3	1	0	-0.129	4.31
BAS Drive	0.25	3.33	0.15	3	0.89	0.01	-5.09	5.51
BAS Fun Seeking	1.75	1.71	2.05	3	0.13	1.02	-0.96	4.46
BAS Reward Responsiveness	0.25	0.95	0.52	3	0.63	0.26	1.27	1.77

Table 19.

Repeated measures t-test from pre-post-test differences in the various measures for the ADHD sample medication and neurofeedback home training condition

	Neurofeedback Home Training							
	Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Confidence Interval Lower	95% Confidence Interval Upper
BIS	-4.11	4.36	-1.59	2	0.25	-0.9	-14.8	6.82
BAS Drive	0.33	2.31	0.25	2	0.83	0.14	-5.41	6.07
BAS Fun Seeking	0.67	2.08	0.55	2	0.63	0.32	-4.51	5.83
BAS Reward Responsiveness	3.12	6.08	0.85	2	0.48	0.49	0.88	3.46

As Table 13 to 19 show, there was no significant difference between conditions from pre to post measures. Although there were no significant changes, there were differences in absolute change scores.

Table 20.

Descriptive statistics absolute mean change scores and t-test from pre-post-test differences in the various measures for the typically developed sample under the difference conditions

	Neurofeedback			Control			Active Control		
	Pre Mean	Post Mean	Absolute Change Score	Pre Mean	Post Mean	Absolute Change Score	Pre Mean	Post Mean	Absolute Change Score
BIS	13.81	14.85	1.03	15.13	16.13	1.00	15.27	16.81	1.53
BAS Drive	8.38	8.15	-0.22	8.81	9.73	0.93	9.73	10.01	0.27
BAS Fun Seeking	6.75	7.62	0.87	7.87	7.73	-0.13	7.21	13.24	6.04
BAS Reward Responsiveness	6.44	7.08	0.64	8.33	8.01	-0.33	7.07	9.74	2.67

Table 20 shows that the largest and most amount of absolute change scores were in the active control condition, apart from the BAS drive which had the largest absolute change score in the control condition.

Table 21.

Descriptive statistics absolute mean change scores and t-test from pre-post-test differences in the various measures for the ADHD sample under the difference conditions

	Medication			Medication and Neurofeedback Home Training			Medication and Neurofeedback Clinic			Neurofeedback Home Training		
	Pre Mean	Post Mean	Absolute Change Score	Pre Mean	Post Mean	Absolute Change Score	Pre Mean	Post Mean	Absolute Change Score	Pre Mean	Post Mean	Absolute Change Score
BIS	14.84	16.42	1.58	14.88	15.71	0.84	16.25	16.25	0.00	14.00	18.00	4.00
BAS Drive	7.79	7.68	-0.11	8.50	8.57	0.07	8.50	8.25	-0.25	8.33	8.00	-0.33
BAS Fun Seeking	7.00	6.95	-0.05	7.00	7.71	0.71	7.50	5.75	-1.75	7.00	6.33	-0.67
BAS Reward Responsiveness	7.21	7.37	0.16	7.50	7.29	-0.21	8.25	8.00	-0.25	9.33	9.67	0.33

Table 21 shows the largest increase in absolute change was in the BIS scale. As expected, BAS drive decreased in the ADHD medication condition, medication and neurofeedback in clinic, and neurofeedback home training. On the other hand, in the neurofeedback home training and medication condition, there was a slight increase on the BAS drive scale.

3.6 Discussion

The present study investigated the difference in personality profiles between typically developed children and children with a diagnosis of ADHD. Comparisons took place between the two samples both pre and post interventions to examine the most effective strategy to improve inattention and impulsive behaviours. We shall look at the main findings from both prior and post intervention in turn in relation to previous literature.

3.6.1 Discussion Regarding Pre Measures

Based upon previous literature, it was hypothesized that the ADHD sample would show higher levels of the Behavioural Activation System and lower levels of the Behavioural Inhibition System (Barkley et al., 1990; Hundt, 2008). There was no significant difference on the BIS/BAS scales between the ADHD and typically developed sample. The research presented in this thesis showed the ADHD sample having higher levels of BAS drive and BAS fun seeking behaviours and less BIS than the typically developed sample, although this was not significantly different.

One possible explanation for the lack of significant difference between the samples is that the BIS/BAS questionnaire was not child friendly, particularly for younger individuals in the samples. For example, the wording of the BIS question "criticism or scolding hurts me quite a bit" may not be understood by a seven-year-old. The fact that there was a significant effect of age on the BIS/BAS scale may explain that the older children understood the questions and answered more appropriately.

Based upon the researcher's clinical experience, it is suggested that the ADHD population may not have been a typical sample due to recruitment taking

place from a private clinic setting. The clinic was based in an affluent area and participants had to pay to access the Centre, consequently being in a financial position to afford assessment. Furthermore, individuals accessing the Centre were typically previously turned away from the National Health Service due to not meeting their criteria level. This may account for there not being a significant difference between the two samples as expected.

The research in this thesis found a significant effect of age, specifically a moderate positive correlation between BAS fun seeking and age in the typically developed sample, and a moderate positive correlation between BAS drive, BAS reward responsiveness and age in the ADHD sample. This therefore supports that personality changes with age and supports Pagliaccio et al. (2016) who showed positive associations of BIS/BAS across early development, peaking in young adulthood and declining in later adulthood (Pagliaccio et al., 2016).

3.6.2 Discussion Regarding Post Measures

Based upon the previous research and the aims of the present study, the hypotheses for the post-measures were as follows:

- The ADHD sample who received neurofeedback home training and medication would have the most changed personality, with lowered BAS and increased BIS results across all samples.
- The typically developed sample, specifically the control and active control conditions, will notice no change on the BIS/BAS scale across time.

In the typically developed sample, the BIS/BAS scores increased, although not significantly, across time and interventions. This trend towards higher BIS/BAS scores over time albeit not statistically significant is in the same direction as findings by Urosevic et al. (2012). As the sample in the present study

was only to the age of 17, it did not find a decline in any scales which would have been expected when entering adulthood.

However, the finding of BIS/BAS scores increasing with age was not replicated in the ADHD sample. This could suggest that personality in an ADHD sample may not develop in the same way as a typically developed population. The developmental deviation and maturation lag theory would support this, showing that the ADHD brain is abnormal at all developmental stages (Kinsbourne, 1973; Barry, Clarke, & Johnstone, 2003).

As found in the pre measure results, the post measures also saw a significant effect of age on the ADHD sample on the BAS reward responsiveness scale. These findings support previous research where it is suggested that an increase in risk taking is seen in adolescence, consequently linked to an increase in reward responsiveness (Pagliaccio et al., 2016).

Somewhat of an unexpected finding was that the most changes in the typically developed sample was seen in the active control condition. Specifically, the active control condition in the typically developed sample saw a rise in BIS, BAS fun seeking and BAS reward responsiveness, although this was not a significant difference. It has been suggested that video gaming can alter neural plasticity as gaming requires an individual to process complex events in a specific sequence as well as to respond quickly and rapidly (Gong et al., 2015). This trend which though not significant may suggest that sitting and focusing on a computer-based programme could influence a child's personality, specifically seeking reward and positive feeling through a heightened BAS, but also a heightened BIS which can cause anxiety disorders (Carver & White, 1994). The research area of

computer use is growing due to the vast development of technology and its use in our everyday lives.

Whereas the active control condition in the typically developed sample saw an increase in many of the BIS/BAS subscales, the ADHD sample mainly saw a decrease. Based upon previous research, it was hypothesised that the ADHD sample who received neurofeedback home training and medication will have the most changes in personality by lowered BAS and increased BIS results across all samples compared pre to post measures. Results from the present study showed that the medication and neurofeedback clinic condition in the ADHD sample saw the largest change in absolute mean scores, with a decrease in BAS fun seeking and BAS reward responsiveness.

There have been very few studies which have examined the effect of stimulant medication on personality, with one study showing that individuals taking d-amphetamine became more energetic and aroused (Corr & Kumari, 2000). However, it is known that stimulant medication in an ADHD population can reduce impulsivity, disruptiveness, talking out of turn and restlessness (Whalen, Henker, & Granger, 1990). Similarly, there are few studies examining neurofeedback and personality. The evidence that is available showed an increase in conscientiousness, boldness and imaginativeness (Peniston & Kulkosky, 1990). When neurofeedback in clinic and stimulant medication were combined, a non-statistically significant trend was found showing a decrease in BAS fun seeking and BAS reward responsiveness. This was an expected finding in the neurofeedback home training and medication condition, but was discovered in the neurofeedback clinic training and medication condition. This suggests that the role of the clinician is more important than previously anticipated. This could be due

to several reasons. Firstly, the clinician can ensure that the equipment is set up and being used correctly. Secondly, the clinician is able to provide immediate verbal feedback and reinforcement to continue participation.

However, the expectation of increased BIS post intervention was not found in the neurofeedback home training and medication condition. Instead, this was found in the neurofeedback home training condition, in addition to a decrease in BAS drive.

Although there were no significant results within the present research, null results are important findings. Null findings can inform researchers of what should be examined differently in future research. Furthermore, the null results can inform policy and practice for implementing strategies (Miller-Halegoua, 2017). For example, the null results within this research would suggest that neurofeedback home training is not an effective strategy for treating the personality aspect of impulsivity in childhood ADHD.

This research suggests that neurofeedback may (or could) influence personality, although not significantly. In a typically developed child population, neurofeedback home training had very little effect on personality, with a slight increase in BIS, BAS fun seeking and BAS reward responsiveness. However, these changes were less than the other conditions in this sample. Participating in a computer-based learning activity saw the largest change in personality in a typically developed sample.

In the ADHD sample, the neurofeedback home training condition saw the largest changes in personality, specifically an increase in BIS and decrease in BAS drive. Although the findings were not significant, this is the first research to examine the effect of neurofeedback in a child population on personality. This

therefore supports Peniston and Kulkosky (1990) that neurofeedback can influence personality.

The lack of significant findings could be Type II error due to small sample sizes. Additionally, as the present study is a feasibility study with small samples, findings are treated as tentative for implications, and need to be replicated in a larger sample.

4 Study Two: Effects of Neurofeedback on Neuropsychometric Measures

4.1 Introduction

Currently, there is no single medical laboratory test to assess for ADHD (Hamed, Kauer, & Stevens 2015). There are several tools available, including rating scales, Continuous Performance Tests (CPT), and neuroimaging, that can be used to aid the diagnostic process for ADHD as well as evaluate the effectiveness of any treatment that has been implemented. Methods such as the CPT are important tools to overcome rater bias and ensure thorough and reliable assessments (Edwards et al., 2007). The role of CPTs and rating scales within the assessment and management of ADHD will be discussed in this chapter.

4.1.1 Continuous Performance Tests

The term CPT is used in conjunction with any performance test that measures sustained attention (Reynolds, Lowe, Moore, & Riccio, 1999). Typically, CPTs are a vigilance task of stimuli in quick succession on a computer screen for a fixed period of time, requiring the participant to be attentive and respond to specific stimuli (Edwards et al., 2007). CPTs measure the amount of omission errors (failure to identify a target stimuli), commission errors (identifying a non-target), and response time. CPTs are simple, fairly long and produce low levels of interest to measure the extent of the individual's ability to sustain attention (Preston et al., 2005) and are cognitively demanding (Ballard, 1996). CPTs are computer based tests ensuring a standardised procedure, reducing bias opinions from experimenters and professionals (McGee et al., 2000). Overall, literature suggests that CPTs are a useful tool and can screen individuals with various difficulties (Epstein, Conners, Sitarenios, & Erhardt, 2010; Conners, 2004). However, the underlying processes and what exactly CPTs measure is still

up for debate (Edwards et al., 2007, McGee et al., 2000). The role of CPTs and its use in an ADHD population will now be discussed in more detail.

4.1.2 Continuous Performance Tests and ADHD

CPTs are particularly useful in examining the main symptoms associated with ADHD and to measure improvement (Epstein et al., 2010). The key measures of CPTs, omission errors and commission errors, are direct measures of the key symptoms of ADHD. For example, high levels of omission errors in CPTs suggest that the individual is not responding to the stimuli or that they have a slow response. Usually, slow response time occurring with many errors reflects inattention whereas fast response time with lots of commission errors reflects impulsivity, both of which are the main symptoms of ADHD (Conners, 2004).

There are three CPTs available which are marketed for clinical use, namely Conners' Continuous Performance Test (CPT; Conners, 1995), Gordon Diagnostic System Vigilance Task (GDS: Gordon, 1988) and the Test of Variables of Attention (TOVA; Dupuy & Greenberg, 1993). These will be discussed in turn, in relation to their use in ADHD as well as the theory behind the tool.

4.1.2.1 Conners' CPT

The Conners' CPT is a computerised visual-motor task whereby participants hit the space bar when presented with any letter except for the letter "X" and therefore respond to both targets and non-targets (Conners, 2004). The task requires rapid letter identification skills, a potential confounding variable if the participant has a specific learning difficulty, and consequently makes it difficult to ascertain if results are due to inhibition deficits (McGee et al., 2000). Stimuli is presented every 2 or 4 seconds, taking approximately 14 minutes to complete and suitable for individuals over the age of 6 years old. The programme

contains a large database, consisting of 2,686 clinical and non-clinical individual's performances, 446 of which were school aged; approximately 50% of the population were male and 50% female. Forty-seven percent of the normative sample consisted of white ethnicity, 27% of black ethnicity, 4.6% Asian and 21.4% other. This specific programme, which is accessible to most clinicians, is standardised with norms for children up to the age of 17 (Conners, 1995). Research suggests that the Conners' CPT can successfully identify children with ADHD 52% of the time (Epstein et al., 2010).

The Conners' CPT is based upon Barkley's (1997) Behavioural Response Inhibition Theory of ADHD. Specifically, it assesses concentration for a duration of time but also the ability to inhibit responses to stimuli. Commission errors reflects impulsivity and omission errors reflects inattention, both of which are linked to abilities in executive functions (Barkley, 1997). Barkley (1997) stated that ADHD inattentive subtype is a distinct disorder rather than a subtype of ADHD. Consequently, the Conners' CPT may not be able to differentiate between subtypes, due to the differing presentation (Edwards et al., 2007). Commission errors were significantly related to 13 of the 18 diagnostic ADHD symptoms. A slowed response time over the duration of the CPT is related to four of the hyperactive-impulsive ADHD symptoms (Epstein et al., 2003). Usually, slow response time occurring with many errors reflects inattention whereas fast response time with lots of commission errors reflects impulsivity, both of which are the main symptoms of ADHD.

It has been suggested by McGee et al. (2000) that the Conners' CPT is able to identify individuals with ADHD, although is unable to distinguish between subtypes. McGee et al. (2000) came to this conclusion after conducting the

Conners' CPT, Auditory CPT and Conners' Rating Scale in 100 children aged 6 to 11 years old. Forty of the children had a diagnosis of ADHD, fourteen had reading disorders, a further 14 had ADHD and reading disorders, and 32 were controls. Conners' CPT was not correlated with age, showing appropriate age relative normalising. The only gender difference was that boys made more commission errors, but this was not a robust finding. There was no association found between Conners' CPT and parent teacher rating. However, Conners' CPT omission errors were moderately associated with teacher rating of hyperactivity. This demonstrates that Conners' CPT is sensitive to teachers rating of behaviour but only when there are high levels of behaviour disturbance.

No gender difference has been found in Conners' CPT results (Gianarris, Golden & Greene, 2001). Epstein et al. (2003) administered the Conners' CPT to 817 children, 21 of which met the diagnostic criteria for ADHD as well as interviewing the parents using the Child and Adolescent Psychiatric Assessment. The study found a relationship between neuropsychological task and ADHD. Specifically, CPT scales showed relationships with ADHD symptom clusters. However, CPT subscale mean hit response time related to ADHD as a whole, rather than symptom clusters. Overall, it showed that omission errors measured inattention and commission errors measured impulsivity.

There is now growing acceptance that ADHD is a disorder seen in adulthood and consequently the use of CPTs in an ADHD adult population needs to be understood (Epstein et al., 2010). One study examined the Conners' CPT in 95 adults. Thirty adults without a diagnosis, 26 with ADHD, 17 with a psychiatric disorder and 22 with various cognitive deficits completed the CPT. The ADHD group made more omission errors, had longer response times and greater

variability in responses. However, these differences were not significantly different to the other conditions (Advokat, Martino, Hill & Gouvier, 2007).

In another adult population, Epstein et al. (2010) examined sixty adults who were referred for an ADHD assessment using a semi-structured interview and underwent Conners' CPT. Thirty-nine participants fulfilled the diagnosis of ADHD inattentive subtype, 7 for ADHD hyperactive-impulsive subtype and 14 ADHD combined subtype. They were compared to 72 healthy controls. Findings showed that adults with ADHD showed significant impairment on Conners' CPT, consistent with findings among children with ADHD. Specifically, the ADHD adult sample showed increased omission and commission errors and decreased reaction times, supporting an impulsive presentation.

4.1.2.2 Test of Variable Attention

The Test of Variable Attention (TOVA) is a computer test that assesses attention and impulse control. It specifically measures four areas: response time variability, response time, impulse control (commission errors) and inattention (omission errors). The test comprises of a blank square with a smaller square placed inside, either at the top or bottom of the outer square. The inner square at the top of the outer square is the target. The TOVA presents 22.5% targets and 77.5% non-targets for half the test, and vice-versa for the remaining half of the test (Dupuy & Greenberg, 1993). The TOVA has standardised norms for 4 to 80 years old and takes 21 minutes to complete. The TOVA had mixed results in successfully identifying ADHD. It has found to misidentify as many as 35% of individuals without ADHD (Edwards et al., 2007). A study by Preston et al. (2005) used the TOVA to examine the ability of individuals with ADHD and possible subclinical levels of behaviour. One hundred and sixteen children with

ADHD and 51 children in a subclinical control group completed the SNAP parent and teacher questionnaires, TOVA CPT and a structured interview. It was concluded that the TOVA scores were unable to determine severity of symptoms and therefore its use was questioned. Consequently, a diagnosis should not be made primarily on the results of a CPT performance and this particular study felt that this CPT tool did little to aid a diagnosis. However, this tool may still be effective in a research capacity, but less accurate in identifying those at high risk with subclinical levels of symptoms of ADHD. Furthermore, it was noted that the TOVA is a difficult task for young children who are sometimes unable to complete the task (Preston et al., 2005).

4.1.2.3 Gordon Diagnostic System Vigilance Task

The final tool available marketed as a clinical tool to assess for ADHD is the Gordon Diagnostic System Vigilance Task (GDS; Gordon, 1986). This CPT consists of 10% targets and 90% non-targets. This is known as the rare target paradigm. The CPT is presented as two tasks. Initially, vigilance, where the participant presses when they see a specific sequence of numbers, and secondly, distractibility, where the same task is presented but with other numbers appearing elsewhere on the screen. Here, participants need the ability to accurately attend to changing stimuli and respond to targets which are infrequent. The GDS is understood to assess sustained attention. However, this tool lacks evidence in consistently identifying individuals with ADHD (Edwards et al., 2007).

Carlozzi and Horner (2007) investigated the use of the Gordon Diagnostic System Vigilance Task in an adult population and its use of measuring attention. The study failed to find any differences on the test of attention and non-attention, therefore questioning the use of this tool.

As previously discussed, ADHD is a condition which affects more males than females (Vernon et al., 2004). Therefore, gender is a variable that needs to be considered. In a meta-analysis by Hasson et al. (2012) a small but significant difference was found between genders on CPT commission errors but was not replicated in omission errors. Specifically, boys made more commission errors and females made more omission errors, although this latter result was not significant. This suggests that gender may influence inhibitory control, but also low numbers of girls are referred for assessment, and those that are, are typically due to inattentive rather than hyperactive concerns. Similarly, the difference between the male and female populations was also replicated in a typically developed child sample (Hasson & Fine, 2012).

Despite its use, CPTs do have limitations. For example, there are low correlations between CPT results and direct classroom observations, omission scores only moderately correlated to rating scales (Gordon, 1988), failure to find group differences (Schachar, Logan, Wachsmuth, & Chajczyk, 1988), lack of ecological validity (Barkley, 1991), and lacks the ability to differentiate between comorbidities and the subtypes of ADHD (Reynolds et al., 1999).

4.1.3 Rating Scales for ADHD

Another tool available to assist an assessment of ADHD are rating scales. Rating scales are often used in conjunction with parents and teachers to assess the extent of the individual's difficulties in various situations. Rating scales can be useful during an assessment for ADHD but are not diagnostic when used on their own (Fonseca et al., 2006). There are many rating scales available, a few of which will be discussed here.

4.1.3.1 Child Behaviour Checklist

One scale is the Child Behaviour Checklist (CBCL). This scale is for children aged 6 to 18 years old and comprises of a 3-point Likert rating scale, namely: not true, somewhat or sometimes true, or very true. The form is completed by parents and consists of 113 items. There are two overarching scales: internalizing problems and externalising problems. These scales are further broken down into the following eight areas: aggressive behaviour, anxious/depressed, attention problems, rule-breaking behaviour, somatic complaints, social problems, thought problems, withdrawn/depressed. The questionnaire scores a wide range of childhood behaviour difficulties including hyperactivity. Additionally, there is a version for teachers (CBCL-TRF), (Miller, Fee, & Netterville, 2004). Biedermann et al. (2001) examined the Child Behaviour Checklist in a longitudinal study in an ADHD population. One hundred and forty males with ADHD and a further one hundred and twenty healthy males, aged 6 to 17 years old, completed the CBCL and a clinical interview. This was completed again 4 years later. Results showed high levels of emotional functioning in the ADHD population, with stability of scores over time. Results support the use of the CBCL as a longitudinal measure in an ADHD sample as well as an effective instrument in the assessment of ADHD (Biedermann et al., 2001).

4.1.3.2 Conners' 3 Parent Rating Scale

An alternative rating scale is the Conners' 3 Parent Rating Scale. This tool comprises of 101 items on a 4-point scale: not true at all, just a little true, pretty much true, and very much true. It measures inattention, hyperactive/impulsivity, learning difficulties, executive function, defiance, and peer relations; each measure

has several statements for the parents to answer. The primary aim of the form is to gather information about the behaviours and feelings of the child. The Conners' Parent Rating Scale is designed to be brief, easy to score, and to administer (Conners, 2004) and said to be a reliable and valid tool in assessing neurobiological disorders but is unable to clearly define different disorders. The Conners' Parent Rating Scale is said to be a reliable and valid tool in assessing neurobiological disorders but is unable to clearly define different disorders (Gianarris, Golden, & Greene, 2001).

One study by Snyder et al. (2008) used the Conners' Parent Rating Scale (CPRS) in combination with EEGs to identify ADHD. One hundred and one males and fifty eight females, of which 97 were diagnosed with ADHD, aged 6 to 18 years old with attention and behaviour difficulties took part. Semi-structured interview, theta/beta ratio using a 19 lead electrode cap and Conners' Rating Scales were completed. Results showed that CPRS had between 47 – 58% accuracy at identifying ADHD compared to EEG that had an accuracy rate of 89%. The cause for the lower accuracy in CPRS was likely due to informant bias. There was low agreement between parent and teacher ratings, with a 64% agreement on the Conners' Rating Scale. Other studies found CPRS accuracy as high as 93% and 85% for teachers; however, other comorbidities were not correctly identified (Snyder et al., 2008; Conners 2008; Gianarris et al., 2001).

In addition to the Conners' 3 Parent Rating Scale, the Conners' 3 Teacher Rating Scale is also available; this is of similar format and purpose to the Parent Rating Scale. The Conners' 3 Teacher Rating Scale consists of 115 items rated on a 4-point scale measuring inattention, hyperactive/impulsivity, learning difficulties, executive function, defiance, and peer relations. The teacher's

perspective is important as difficulties need to be present in two settings in order for a diagnosis of ADHD to be made. (APA, 2013). Teachers are able to monitor behaviours during academic learning as well as unstructured peer interactions on the playground (Conners, 2008). In one study, one hundred and eighty four children aged 5 to 12 years old had parents complete the Conners' Parent Rating Scale and teachers complete the Conners' Teacher Rating Scale. Results showed that teachers report children with ADHD to have higher levels of behavioural difficulties compared to parent rating. Furthermore, the Conners' Teacher Rating Scale had higher levels of sensitivity, specificity and accuracy. Consequently, it was recommended that teacher and parent ratings were combined and used in conjunction with each other in an ADHD assessment (Tripp, Schaughency, & Clarke, 2006).

Sonuga-Barke et al. (2013) conducted a meta-analysis examining nonpharmacological interventions on ADHD. In both cognitive training and neurofeedback trials, where observers were aware of the research and intervention, significant treatment effects were found. However, the significant treatment effects were lost when raters were blinded to intervention. Here it was found that teacher completed measures were sensitive to change. Results suggested that individuals aware of the intervention may have inflated significance due to raters having an investment in implementing the strategy and therefore its success. For example, behavioural interventions are prone to rater bias, as the rater, the parents are implementing the intervention. On the other hand, the parents may be seeing improvements, but these results are not being generalised into other settings, such as school.

Interpreting observer results via rating scales needs to be done with caution. For example, in a less restrictive and demanding environment such as at home, symptoms may not be as evident or troublesome (Barkley, 2003). With CPRS, there is the possibility of informant bias which can significantly affect rating scale outcomes. This therefore implies that rating scales are useful within a clinical setting to help determine if ADHD is present or not, but needs to involve other elements, such as clinical interview, school observations or EEG (where available). However, having two individuals complete the scales, such as the Conners 3 Teacher Rating Scale, can help improve validity (Snyder et al., 2008).

4.1.4 Rating Scales, Continuous Performance Tests and Neurofeedback

So far, the presentation of individuals with ADHD on several CPTs and rating scales have been discussed. Another use of these tools is to evaluate the effectiveness of any treatment that has been implemented (Edwards et al., 2007). Evidence will be discussed here in regard to the effect that neurofeedback has on rating scales and continuous performance tests in both a healthy and ADHD sample.

In a healthy sample, Egner and Gruzelier (2001) employed SMR training (increase of 12-15 Hz) at C4 and beta training (increase of 15-18 Hz) at C3 in a group of 22 adults while simultaneously inhibiting theta (4-7 Hz) and high beta (22-30 Hz). After ten neurofeedback sessions, a significant reduction was seen on CPT commission errors but no change on omission errors. Also, SMR neurofeedback was highly positively correlated to commission error reduction.

In a further healthy sample, Vernon et al. (2003) examined 30 medical students aged 20 to 28 years old who completed neurofeedback, twice a week for 4 weeks. Two neurofeedback protocols were used, enhancing theta condition and

enhancing SMR condition. CPT and conceptual span task was completed before and after the neurofeedback training. Results showed that eight sessions of neurofeedback was able to change EEG activity in healthy individuals. Specifically, SMR activity showed greater improvement on CPT, showing an improvement in accuracy of attention. These two studies have demonstrated the use of CPT measuring improvement as a consequence of neurofeedback in an adult population and needs to be replicated in a child sample.

Fuchs et al. (2003) conducted a study of 34 children with a diagnosis of ADHD, aged 8 to 12 years old, of which 22 children completed 3 months of neurofeedback sessions, 3 sessions a week with the same therapist at the same time of day. Children diagnosed with ADHD hyperactive-impulsive subtype received SMR uptraining at C4 whereas children diagnosed with ADHD inattentive subtype received beta uptraining at C3. Children diagnosed with ADHD combined subtype received a combination of these protocols. A further 12 children received stimulant medication only. All conditions saw a reduction on ADHD symptoms. Specifically, all conditions had a reduction on Conners' Behaviour Rating Scale, as completed by parents and teachers, as well as a moderate effect size. This demonstrates the efficiency of neurofeedback in reducing ADHD symptoms as rated by parents and teachers, using standard protocols. However, this study had a small sample size and consequently needs replicating on a larger scale.

When CPT is used as an assessment tool, it needs to be used with caution. The Federal Drug Administration (FDA) promotes the use of new tools to aid the diagnosis of ADHD, clearly stating the tools are aids and are not diagnostic. FDA promotes approval for marketing of a tool, not promotion of best clinical practice (Arns et al., 2016).

4.1.5 Rating Scales, Continuous Performance Tests and Stimulant Medication

Methylphenidate has been shown to improve performance on Continuous Performance Tests. Specifically, findings showed that the higher dose of methylphenidate taken, the fewer errors occurred, showing successful concentration. Additionally, the higher the cognitive ability of an individual, the higher the response rate to methylphenidate (Pearson et al., 2004).

A meta-analysis was conducted reviewing 13 randomised control trials examining methylphenidate. It total, 882 participants with a diagnosis of ADHD, up to the age of 18 years old, were included. On parent ratings, there was a preference for long acting methylphenidate, due to improvements on hyperactive/impulsive behaviours. However, teacher ratings favoured short acting methylphenidate, specifically for hyperactivity (Punja et al., 2013).

4.2 Aims of the Study

The present study aimed to examine the differences between a healthy and ADHD child population when rated by teacher, parent and on a child concentration test. Furthermore, the aim is to determine the most successful intervention or combination of interventions that improves concentration in both a healthy and ADHD sample as rated by parents and teachers.

Specifically, there have been limited previous research which has examined neurofeedback and stimulant medication in an ADHD sample. There have been several studies which have compared the two interventions, showing similar effect on ADHD symptoms (Fuchs et al., 2003; Monastra et al., 2002; Rossiter, 2004; Rossiter & La Vaque, 1995). The present study aims to examine the effect of stimulant medication and neurofeedback on ADHD symptoms from a parent and teacher perspective.

Neurofeedback has been shown to have a large effect size on inattention and hyperactivity and a medium effect size on impulsivity in an ADHD sample (Arns et al., 2009). However, previous neurofeedback research has been conducted in clinic or school settings and not in the home (Vernon et al., 2004; Rutterford et al., 2008). Consequently, one aim of this thesis was to examine the effect of neurofeedback home training on a healthy and ADHD population, specifically in regard to parent and teachers' views.

4.3 Hypothesis

Based upon the previous research and the aims of the present study, the hypotheses for the pre-measures are as follows:

- The ADHD sample will have higher scores on omission and commission error subscales on the Continuous Performance Test than the typically developed sample.
- The ADHD sample will have a higher score on the inattention, hyperactive/impulsive and executive function subscales of the Conners' 3 Parent and Teacher Rating Scale than the typically developed sample.

Based upon previous research and the aims of the present study, the hypotheses for the post-measures are as follows:

- The ADHD neurofeedback home training condition will show greater improvement on Conners' CPT omission and commission subscales, as well as Conners' Parent/Teacher Rating Scales, specifically on the subscales of inattention, hyperactive/impulsive, and executive function, compared to the typically developed neurofeedback home training condition.

- The ADHD neurofeedback home training and medication condition will show greatest improvement across all conditions on Conners' CPT omission and commission subscales, as well as Conners' Parent/Teacher Rating Scales, specifically on the subscales of inattention, hyperactive/impulsive, and executive function.
- The typically developed sample who received neurofeedback home training will show greater improvement on Conners' CPT omission and commission subscales, as well as Conners' Parent/Teacher Rating Scales, specifically on the subscales of inattention, hyperactive/impulsive and executive function, compared to the typically developed sample control and active control group.
- No difference will be found between the typically developed sample control and active control group on Conners' CPT omission and commission subscales, as well as Conners' Parent/Teacher Rating Scales, specifically on the subscales of inattention, hyperactive/impulsive, and executive function, across time.

4.4 Method

For details regarding participants and interventions, please see Chapter Two: General Methods.

4.4.1 Neuropsychometric Measures

The study reported here used 3 neuropsychometric measures: Conners' Continuous Performance Test, Conners' 3 Parent Rating Scale and the Conners' 3 Teacher Rating Scale. The measures were conducted on two occasions, once during the initial meeting prior to any intervention, and 15 weeks later once the interventions had been completed.

4.4.1.1 Conners' Continuous Performance Test (CPT)

The Conners' CPT is a computerised visual-motor task whereby participants hit the space bar when presented with any letter except for the letter "X", therefore responding to both targets and non-targets (Conners, 2004). Stimuli is presented every 2 or 4 seconds, is suitable for individuals over the age of 6 years old and takes 14 minutes to complete. The CPT has a large database, consisting of 2,686 clinical and non-clinical performances. The Conners' CPT measures a range of variables. Please see Table 22 which identifies the variables and what they measure in relation to ADHD symptoms.

Conners' (1995), stated that when repeating the test, there is little practice effect, making it a useful tool to measure any possible benefit of interventions.

Table 22.

Conners' Continuous Performance Test Variables

Variable	Description	Measure
Omission	Failure to respond to target	Inattention
Commission	Response to non-target	Inattention and impulsivity
Conners CPT Hit Response Time	Average speed of correct responses	Inattention and impulsivity
Conners CPT Hit Response Time Std Error	Response speed consistency. High scores suggestive greater inconsistency.	Inattention
Conners CPT Variability	Amount of variability across the various sections.	Inattention
Conners CPT Dectectability	Ability to determine a target from a non-target based on distribution score.	Inattention
Conners CPT Response Style	Individuals response tendency. Higher score suggests more cautious approach.	
Conners CPT Preservations	Reaction time less than 100ms suggests individual anticipating stimulus rather than responding	Impulsivity
Conners CPT Response Time Block Change	Measures change in reaction times across the test. Higher scores show a slowing in reaction time.	Vigilance and alertness
Conners CPT Response Time Block Change Std Error	Measures change in reaction consistency across the test. Higher scores show a slowing in reaction time.	Vigilance and alertness

4.4.1.2 Conners' 3 Parent and Teacher Rating Scales

The Conners' 3 Parent Rating Scale is the most widely used scale among clinicians and researchers. The Conners' Rating Scale is designed to be brief, easy to score, and easy to administer. Its primary aim is to screen for any psychopathology, to be a diagnostic aid as well as a measure of general treatment outcome. Subscales include conduct disorder, anxiousness, restlessness, learning difficulties, psychosomatic, obsessive compulsive, anti-social and hyperactive behaviours. It measures inattention, hyperactive/impulsivity, learning difficulties, executive function, defiance, and peer relations; each measure has several statements for the parents to answer. These raw scores are combined to make a measure score which is then compared with a normative database of age and gender matched individuals. The normative database contains data for 50 boys and 50 girls from each age group, from 6 to 18 years old. Through the results, areas of difficulty can be identified.

The online full version of the Conners' 3 Parent and Teacher Rating Scales were used. Both parents and teachers were emailed the online questionnaire to complete; the questionnaire comprised of 109 items for parents and 115 items for teachers, using a 4-point scale: not true at all, just a little true, pretty much true, and very much true. The questionnaire was sent to the parent and teacher on the day that the initial meeting and last meeting took place with a covering email asking them to complete the questionnaire as soon as possible in one sitting. If the questionnaire had not been completed within one week, parents and teacher were reminded by email and phone to complete the questionnaire.

4.5 Results

As the neuropsychometric measures were completed at 2 time points, pre-measures before any intervention and post-measures after any intervention, the results have been presented in this way.

4.5.1 Results of neuropsychometric performance pre-measures

Table 23.

MANOVA of diagnosis and gender on pre-measures

	Gender					Clinical Diagnosis				
	Wilks Lambda	f	Hypothesis df	Error df	Sig.	Wilks Lambda	f	Hypothesis df	Error df	Sig.
Conners' 3 Parent Rating Scale	0.91	1.46	5.00	75.00	0.21	0.78	4.36	5.00	75.00	0.00[1a]
Conners' 3 Teacher Rating Scale	0.83	0.62	5.00	15.00	0.69	0.65	1.64	5.00	15.00	0.21
CPT	0.89	0.81	11.00	70.00	0.63	0.85	1.09	11.00	70.00	0.39

[a] Significant after Bonferroni correction. Calculation of 0.05/3=0.016

A multivariate analysis of variance (MANOVA) was conducted to examine the overall effect of diagnosis and gender on the BIS/BAS scale. A MANOVA is an extension of an ANOVA, examining the statistical differences of two or more dependent variables. As Table 18 shows, there was no significant effect for gender on the Conners' 3 Parent and Teacher Rating Scale or CPT, but there was a significant effect of clinical diagnosis on the Conners' 3 Parent Rating Scale, which remained significant after Bonferroni correction.

T-tests were conducted to assess further the significant difference between samples, Table 24.

Table 24.

Descriptive statistics and T-test results of pre-measure dependent variables on independent variable, diagnosis

		Typically Developed Sample			ADHD Sample					
		N	Mean	SD	N	Mean	SD	t	df	Sig. (2-tailed)
Conners' 3 Parent Rating Scale	Learning Difficulties	38.00	9.20	5.84	45.00	11.60	6.23	1.81	81.00	0.07
	Executive Functions	38.00	10.64	5.52	45.00	14.26	5.48	2.98	81.00	0.01 [a]
	Defiance	38.00	3.46	3.84	45.00	7.60	6.77	3.48	81.00	0.01 [a]
	Inattention	38.00	11.86	7.44	45.00	18.94	6.50	4.57	81.00	0.01 [a]
	Hyperactive/Impulsive	38.00	11.86	9.57	45.00	22.26	12.51	4.28	81.00	0.01 [a]
Conners' 3 Teacher Rating Scale	Learning Difficulties	15.00	3.13	3.81	9.00	8.00	6.36	2.35	22.00	0.02
	Executive Functions	15.00	7.00	4.70	9.00	11.37	4.20	2.19	21.00	0.03
	Defiance	15.00	3.60	5.51	9.00	5.00	5.91	0.58	22.00	0.56
	Inattention	15.00	8.00	7.32	9.00	16.25	7.38	2.56	21.00	0.01 [b]
	Hyperactive/Impulsive	15.00	9.26	11.18	9.00	7.12	6.89	-0.49	21.00	0.62
Conners' CPT	Omissions	36.00	15.31	17.08	48.00	20.05	20.43	1.16	82.00	0.25
	Commissions	36.00	22.15	7.81	48.00	24.05	7.91	1.10	82.00	0.27
	Hit Response Time	36.00	408.00	107.83	48.00	438.43	95.27	1.34	82.00	0.18
	Hit Response Time Std Error	36.00	10.99	7.50	48.00	14.45	9.22	1.89	82.00	0.06
	Variability	36.00	21.95	19.70	48.00	31.45	24.65	1.96	82.00	0.53
	Dectectability	36.00	0.36	0.37	48.00	0.31	0.40	-0.68	82.00	0.49
	Response Style	36.00	0.70	0.49	48.00	0.97	1.23	1.37	82.00	0.17
	Preservations	36.00	6.47	8.34	48.00	12.88	17.26	2.24	82.00	0.02
	Response Time Block Change	36.00	0.06	0.28	48.00	0.19	0.03	-0.76	82.00	0.44
	Block Change Std Error	36.00	0.11	0.29	48.00	0.09	0.10	-0.51	82.00	0.61

[a] Significant after Bonferroni correction. Conners' 3 Parent Rating Scale calculation of 0.05/5=0.01,
[b] Significant after Bonferroni correction. Conners' 3 Teacher Rating Scale calculation of 0.05/5=0.01,
[c] Significant after Bonferroni correction. Conners' CPT calculation of 0.05/10=0.005

Table 24 shows means and statistical effects for all the dependent variables. There was a significant main effect after Bonferroni correction of diagnosis on Conners' 3 Parent Rating Scale subscales executive functions, defiance, inattention and hyperactive/impulsive as well as inattention subscales of the Conners' 3 Teacher Rating Scale. As expected, the ADHD sample were rated significantly higher in the aforementioned scales.

A Pearson correlation coefficient was then computed to assess the relationship between the independent variables and age.

Table 25.

Pearson Product Moment correlations for dependent variables and age on the typically developed sample

		N	Pearson Correlation	Strength of relationship	Sig
Conners' 3 Parent Rating Scale	Learning Difficulties	45.00	-0.23	Weak negative	0.11
	Executive Functions	45.00	0.13	Weak positive	0.36
	Defiance	45.00	-0.04	None	0.78
	Inattention	45.00	0.06	None	-0.28
	Hyperactive/Impulsive	45.00	-0.41	Moderate negative	0.01
Conners' 3 Teacher Rating Scale	Learning Difficulties	15.00	0.03	None	0.91
	Executive Functions	15.00	0.14	Weak positive	0.59
	Defiance	15.00	-0.22	Weak negative	0.41
	Inattention	15.00	0.05	None	0.84
	Hyperactive/Impulsive	15.00	-0.44	Moderate negative	0.10
Conners' CPT	Omissions	48.00	-0.47	Moderate negative	0.01
	Commissions	48.00	-0.18	Weak negative	0.21
	Hit Response Time	48.00	-0.51	Moderate negative	0.01
	Hit Response Time Std Error	48.00	-0.59	Moderate negative	0.01
	Variability	48.00	-0.49	Moderate negative	0.01
	Dectectability	48.00	0.21	Weak positive	0.13
	Response Style	48.00	-0.16	Weak negative	0.25
	Preservations	48.00	-0.46	Moderate negative	0.01
	Response Time Block Change	48.00	-0.11	Weak negative	0.44
	Block Change Std Error	48.00	-0.21	Weak negative	0.16

Table 26.

Pearson Product Moment correlations for dependent variables and age on the ADHD sample

		N	Pearson Correlation	Strength of Relationship	Sig
Conners' 3 Parent Rating Scale	Learning Difficulties	38.00	-0.15	Weak negative	0.36
	Executive Functions	38.00	0.38	Moderate positive	0.01
	Defiance	38.00	-0.32	Moderate negative	0.04
	Inattention	38.00	-0.11	Weak negative	0.52
	Hyperactive/Impulsive	38.00	-0.47	Moderate negative	0.01
Conners' 3 Teacher Rating Scale	Learning Difficulties	8.00	-0.82	Strong negative	0.01
	Executive Functions	8.00	0.31	Moderate positive	0.45
	Defiance	8.00	0.12	Weak positive	0.75
	Inattention	8.00	0.03	None	0.92
	Hyperactive/Impulsive	8.00	-0.32	Moderate negative	0.43
Conners' CPT	Omissions	36.00	-0.42	Moderate negative	0.01
	Commissions	36.00	-0.21	Weak negative	0.19
	Hit Response Time	36.00	-0.62	Moderate negative	0.01
	Hit Response Time Std Error	36.00	0.84	Strong positive	0.01
	Variability	36.00	-0.43	Moderate negative	0.01
	Dectectability	36.00	0.15	Weak positive	0.41
	Response Style	36.00	0.09	None	0.59
	Preservations	36.00	-0.22	Weak negative	0.19
	Response Time Block Change	36.00	-0.21	Weak negative	0.21
	Block Change Std Error	36.00	-0.03	None	0.83

As Table 25 and 26 show, age significantly correlated with Conners' 3 Parent Rating Scale hyperactive/impulsive subscale, Conners' CPT Omissions and Conners' CPT Hit response time in both the typically developed and ADHD sample. Also, age significantly correlated with Conners' CPT Hit Response Time Standard Error in both samples, although in different directions. Conners' CPT variability and preservations were both significantly correlated with age in the typically developed sample where as Conners' Teacher Learning Difficulties correlated with age in the ADHD sample. Consequently, age was included as a covariate in further analysis.

4.5.2 Results of neuropsychometric performance post-measures

The results from the post-measures, which were completed after the interventions, will now be discussed.

A MANOVA was conducted to assess how diagnosis and gender combined affect the overall post measures.

Table 27.

MANOVA examining post measures across diagnosis and gender

	Gender						Diagnosis				
	Absolute Mean Change Score	f	Hypothesis df	Error df	Sig.	Wilks Lambda	Absolute Mean Change Score	f	Hypothesis df	Error df	Sig.
Conners' 3 Parent Rating Scale	-0.07	2.32	5.00	60.00	0.05	0.71	-0.07	4.91	5.00	60.00	0.01
Conners' 3 Teacher Rating Scale	-0.77	6.16	5.00	2.00	0.15	0.01	-0.84	33.91	5.00	2.00	0.03
Conners' CPT	-0.05	0.94	11.00	56.00	0.51	0.65	-0.21	2.71	11.00	56.00	0.01

As Table 27 shows, gender had a significant effect on the Conners' Parent Rating Scale and diagnosis had a significant effect on three measures. Therefore, the post measures were examined in more detail. Due to the small sample in some of the conditions, it was not possible to conduct a post-hoc test.

T-tests were then conducted to examine if there were any significant difference between the samples after interventions.

Table 28.

T-test results of post measure dependent variables on independent variable, diagnosis

		Typically Developed Sample			ADHD Sample					
		N	Mean	SD	N	Mean	SD	t	df	Sig. (2-tailed)
Conners' 3 Parent Rating Scale	Learning Difficulties	38.00	8.31	5.35	30.00	10.43	5.28	1.62	66.00	0.11
	Executive Functions	38.00	9.84	4.66	30.00	12.16	5.21	1.93	66.00	0.05
	Defiance	38.00	2.84	3.22	30.00	5.93	6.29	2.62	66.00	0.01 [a]
	Inattention	38.00	9.05	5.69	30.00	15.40	6.11	4.41	66.00	0.01 [a]
	Hyperactive/Impulsive	38.00	9.42	7.67	30.00	18.86	8.40	4.83	66.00	0.01 [a]
Conners' 3 Teacher Rating Scale	Learning Difficulties	7.00	3.00	3.05	5.00	10.20	4.49	3.32	10.00	0.01 [b]
	Executive Functions	7.00	5.71	2.69	5.00	12.80	4.86	3.25	10.00	0.01 [b]
	Defiance	7.00	3.42	5.88	5.00	8.80	11.45	10.07	10.00	0.31
	Inattention	7.00	4.71	6.10	5.00	18.20	9.09	3.09	10.00	0.01
	Hyperactive/Impulsive	7.00	4.33	7.44	4.00	20.25	24.06	1.55	8.00	0.15
Conners' CPT	Omissions	43.00	14.65	14.32	27.00	24.22	26.46	1.96	68.00	0.05
	Commissions	43.00	23.06	7.41	27.00	20.66	6.09	-1.41	68.00	0.16
	Hit Response Time	43.00	398.61	66.17	27.00	470.69	80.21	4.08	68.00	0.01
	Hit Response Time Std Error	43.00	10.68	6.51	27.00	15.67	10.01	2.56	68.00	0.01
	Variability	43.00	20.71	17.30	27.00	33.91	31.08	2.28	68.00	0.02
	Dectectability	43.00	0.29	0.29	27.00	0.41	0.25	1.80	68.00	0.07
	Response Style	43.00	0.65	0.38	27.00	0.75	0.44	1.04	68.00	0.29
	Preservations	43.00	7.97	946.00	27.00	15.11	20.75	1.95	68.00	0.05
	Response Time Block Change	43.00	0.01	0.03	27.00	0.01	0.03	-1.02	68.00	0.31
	Block Change Std Error	43.00	0.06	0.09	27.00	0.04	0.09	-0.93	68.00	9.35

[a] Significant after Bonferroni correction. Conners' 3 Parent Rating Scale calculation of 0.05/5=0.01,

[b] Significant after Bonferroni correction. Conners' 3 Teacher Rating Scale calculation of 0.05/5=0.01

As Table 28 shows, there was a significant main effect after Bonferroni correction of diagnosis on some of the Conners' Parent subscales and some of the Conners' Teacher subscales.

Next, t-tests were conducted to examine if gender affected post-measure results (Table 29 and 30).

Table 29.

T-tests of post dependent variables with gender on typically developed sample

		Male			Female					
		N	Mean	SD	N	Mean	SD	t	df	Sig.
Conners' 3 Parent Rating Scale	Learning Difficulties	22.00	8.31	6.34	16.00	8.31	3.78	0.00	36.00	0.99
	Executive Functions	22.00	10.95	4.60	16.00	8.31	4.43	1.77	36.00	0.08
	Defiance	22.00	3.54	3.87	16.00	1.87	1.71	1.61	36.00	0.11
	Inattention	22.00	10.22	6.71	16.00	7.43	3.48	1.51	36.00	0.13
	Hyperactive/Impulsive	22.00	11.00	8.11	16.00	7.25	6.67	1.51	36.00	0.13
Conners' 3 Teacher Rating Scale	Learning Difficulties	4.00	3.00	9.84	3.00	3.00	3.00	0.00	5.00	1.00
	Executive Functions	4.00	6.75	3.30	3.00	4.33	0.57	1.22	5.00	0.27
	Defiance	4.00	6.00	6.97	3.00	0.00	0.00	1.45	5.00	0.21
	Inattention	4.00	6.50	7.85	3.00	2.33	2.08	0.87	5.00	0.42
	Hyperactive/Impulsive	4.00	8.00	9.84	3.00	0.66	1.15	1.28	5.00	0.26
Conners' CPT	Omissions	27.00	14.25	14.39	16.00	15.31	14.65	-0.23	41.00	0.81
	Commissions	27.00	23.77	7.83	16.00	21.87	6.71	0.81	41.00	0.42
	Hit Response Time	27.00	394.88	62.31	16.00	404.89	73.91	-0.47	41.00	0.63
	Hit Response Time Std Error	27.00	10.62	6.01	16.00	10.78	7.51	-0.07	41.00	0.93
	Variability	27.00	19.71	14.88	16.00	22.39	21.19	-0.48	41.00	0.62
	Dectectability	27.00	0.30	0.30	16.00	0.28	0.29	0.18	41.00	0.85
	Response Style	27.00	0.61	0.30	16.00	0.70	0.49	-0.68	41.00	0.49
	Preservations	27.00	8.18	8.95	16.00	7.62	10.55	0.18	41.00	0.85
	Response Time Block Change	27.00	0.01	0.02	16.00	0.02	0.04	-0.77	41.00	0.44
	Block Change Std Error	27.00	0.06	0.09	16.00	0.07	0.11	-0.18	41.00	0.85

[a] Significant after Bonferroni correction. Conners' 3 Parent Rating Scale calculation of 0.05/5=0.01,

[b] Significant after Bonferroni correction. Conners' 3 Teacher Rating Scale calculation of 0.05/5=0.01,

[c] Significant after Bonferroni correction. Conners' CPT calculation of 0.05/10=0.005

Table 30.

T-tests of post dependent variables with gender for ADHD sample

		Male			Female				
		N	Mean	SD	N	Mean	SD	t	df	Sig.
Conners' 3 Parent Rating Scale	Learning Difficulties	26.00	11.00	5.30	4.00	6.75	3.59	1.53	28.00	0.13
	Executive Functions	26.00	12.61	5.28	4.00	9.25	4.03	1.21	28.00	0.23
	Defiance	26.00	5.00	4.17	4.00	12.00	13.44	-2.21	29.00	0.03
	Inattention	26.00	16.15	6.21	4.00	10.50	1.73	1.78	28.00	0.08
	Hyperactive/Impulsive	26.00	20.34	7.77	4.00	9.25	6.02	2.71	298.00	0.01
Conners' 3 Teacher Rating Scale	Learning Difficulties	5.00	10.20	4.49	-	-	-	-	-	-
	Executive Functions	5.00	12.80	4.86	-	-	-	-	-	-
	Defiance	5.00	8.80	11.45	-	-	-	-	-	-
	Inattention	5.00	18.20	9.09	-	-	-	-	-	-
	Hyperactive/Impulsive	5.00	29.25	24.06	-	-	-	-	-	-
Conners' CPT	Omissions	24.00	26.87	26.89	3.00	3.00	5.19	1.51	25.00	0.14
	Commissions	24.00	21.37	5.72	3.00	15.00	7.21	1.77	25.00	0.08
	Hit Response Time	24.00	480.76	76.85	3.00	390.14	69.03	1.94	25.00	0.06
	Hit Response Time Std Error	24.00	16.78	9.95	3.00	6.72	5.41	1.70	25.00	0.10
	Variability	24.00	36.57	31.75	3.00	12.60	14.16	1.27	25.00	0.21
	Dectectability	24.00	0.40	0.26	3.00	0.52	0.17	-0.77	25.00	0.44
	Response Style	24.00	0.81	0.42	3.00	0.28	0.24	2.07	25.00	0.04
	Preservations	24.00	16.79	21.44	3.00	1.66	2.88	1.20	25.00	0.24
	Response Time Block Change	24.00	0.01	0.03	3.00	-0.01	0.02	0.97	25.00	0.34
	Block Change Std Error	24.00	0.05	0.08	3.00	-0.05	0.03	2.23	25.00	0.03

[a] Significant after Bonferroni correction. Conners' 3 Parent Rating Scale calculation of 0.05/5=0.01,
[b] Significant after Bonferroni correction. Conners' 3 Teacher Rating Scale calculation of 0.05/5=0.01,
[c] Significant after Bonferroni correction. Conners' CPT calculation of 0.05/10=0.005

Analysis showed that gender had no significant effect on neuropsychological measures after Bonferroni correction.

A Pearson correlation coefficient was then computed to assess the relationship between the independent variables and age at the post measures time point (Table 31 and 32).

Table 31.

Pearson - product moment correlations for dependent variables and age on the typically developed sample for post measures

		N	Pearson Correlation	Sig
Conners' 3 Parent Rating Scale	Learning Difficulties	38.00	-0.17	0.29
	Executive Functions	38.00	0.00	0.98
	Defiance	38.00	-0.07	0.66
	Inattention	38.00	-0.20	0.22
	Hyperactive/Impulsive	38.00	-0.45	0.01
Conners' 3 Teacher Rating Scale	Learning Difficulties	7.00	-0.61	0.13
	Executive Functions	7.00	-0.37	0.40
	Defiance	7.00	-0.23	0.61
	Inattention	7.00	-0.36	0.48
	Hyperactive/Impulsive	7.00	-0.35	0.48
Conners' CPT	Omissions	43.00	-0.43	0.01
	Commissions	43.00	-0.38	0.01
	Hit Response Time	43.00	-0.56	0.01
	Hit Response Time Std Error	43.00	-0.54	0.01
	Variability	43.00	-0.46	0.01
	Dectectability	43.00	0.39	0.02
	Response Style	43.00	0.00	0.15
	Preservations	43.00	0.00	0.01
	Response Time Block Change	43.00	-0.26	0.08
	Block Change Std Error	43.00	-0.20	0.19

Table 32.

Pearson - product moment correlations for dependent variables and age on the ADHD sample for post measures

		N	Pearson Correlation	Sig
Conners' 3 Parent Rating Scale	Learning Difficulties	30.00	-0.14	0.44
	Executive Functions	30.00	0.41	0.02
	Defiance	30.00	-0.15	0.42
	Inattention	30.00	0.15	0.41
	Hyperactive/Impulsive	30.00	-0.12	0.51
Conners' 3 Teacher Rating Scale	Learning Difficulties	5.00	-0.68	0.19
	Executive Functions	5.00	0.04	0.94
	Defiance	5.00	-0.11	0.86
	Inattention	5.00	-0.25	0.67
	Hyperactive/Impulsive	5.00	-0.14	0.85
Conners' CPT	Omissions	27.00	-0.31	0.11
	Commissions	27.00	-0.34	0.07
	Hit Response Time	27.00	-0.04	0.02
	Hit Response Time Std Error	27.00	-0.32	0.11
	Variability	27.00	-0.29	0.13
	Dectectability	27.00	0.19	0.33
	Response Style	27.00	-0.37	0.05
	Preservations	27.00	-0.27	0.17
	Response Time Block Change	27.00	-0.48	0.01
	Block Change Std Error	27.00	-0.08	0.66

As Table 31 shows, age had a significant correlation of Conners' 3 Parent Rating hyperactive subscale, and several of the Conners' CPT subscales, within the typically developed sample. Whereas Table 32 shows age had a significant correlation on Conners' 3 Parent Rating executive functions subscale, and several of the Conners' CPT subscales, within the ADHD sample. In neither sample did age correlate with Conners' 3 Parent Teacher Rating.

4.5.3 Results Comparing Pre to Post of Neuropsychometric Performance Measures

Table 33.

Repeated MANOVA, pre-to post across typically developed and ADHD sample

		Pre to Post			Gender					
		Mean	f	Sig.	Mean	f	Sig.	Mean	f	Sig.
Conners' 3 Parent Rating Scale	Learning Difficulties	4.98	1.42	0.24	0.33	0.09	0.76	0.73	0.21	0.65
	Executive Functions	4.71	0.76	0.39	7.94	1.28	0.26	27.93	4.50	0.04
	Defiance	31.91	4.99	0.03	6.90	1.08	0.30	15.92	2.49	0.12
	Inattention	51.28	4.12	0.05	0.02	0.00	0.97	17.80	1.43	0.24
	Hyperactive/Impulsive	112.18	5.45	0.02	0.86	0.04	0.84	55.98	2.72	0.10
Conners' 3 Teacher Rating Scale	Learning Difficulties	0.01	0.01	0.93	5.54	5.29	0.07	0.35	0.34	0.59
	Executive Functions	0.56	0.21	0.68	1.15	0.41	0.55	7.34	2.55	0.17
	Defiance	0.22	0.02	0.88	39.18	4.23	0.09	0.41	0.04	0.84
	Inattention	0.67	0.29	0.61	0.00	0.00	0.97	3.71	1.63	0.26
	Hyperactive/Impulsive	1.57	0.03	0.86	183.96	3.98	0.11	0.35	0.01	0.93
Conners' CPT	Omissions	60.68	0.54	0.47	36.92	0.33	0.57	175.35	1.55	0.22
	Commissions	0.54	0.02	0.89	20.19	0.71	0.41	136.13	4.73	0.03
	Hit Response Time	1.82	0.00	0.98	1740.71	0.47	0.49	2893.58	0.78	0.38
	Hit Response Time Std Error	1.43	0.07	0.79	7.07	0.34	0.56	7.15	0.34	0.56
	Variability	10.96	0.06	0.81	7.67	0.04	0.84	81.13	0.42	0.52
	Dectectability	0.01	0.08	0.78	0.01	0.08	0.78	0.18	1.85	0.18
	Response Style	0.57	1.12	0.29	0.00	0.01	0.92	0.33	0.63	0.43
	Preservations	114.58	1.39	0.24	0.98	0.01	0.91	23.77	0.29	0.59
	Response Time Block Change	10.95	1.39	0.24	0.09	0.01	0.91	2.39	0.29	0.59
	Block Change Std Error	0.05	1.24	0.27	0.01	0.31	0.58	0.01	0.20	0.66

[a] Significant after Bonferroni correction. Conners' 3 Parent Rating Scale calculation of 0.05/5=0.01,

[b] Significant after Bonferroni correction. Conners' 3 Teacher Rating Scale calculation of 0.05/5=0.01,

[c] Significant after Bonferroni correction. Conners' CPT calculation of 0.05/10=0.005

A paired t-test was then conducted to compare the independent variables from the first time point, pre measures, to after any intervention, post measures

Table 34.

Paired T-test results of pre-to post measure dependent variables

		Mean	SD	t	df	Sig. (2-tailed)
Conners' 3 Parent Rating Scale	Learning Difficulties	0.98	2.61	3.05	65.00	0.01[a]
	Executive Functions	1.36	3.61	3.06	65.00	0.01[a]
	Defiance	1.51	3.67	3.34	65.00	0.01[a]
	Inattention	3.18	5.00	5.16	65.00	0.01[a]
	Hyperactive/Impulsive	3.50	6.61	4.30	65.00	0.01[a]
Conners' 3 Teacher Rating Scale	Learning Difficulties	0.54	2.25	0.80	10.00	0.44
	Executive Functions	-0.45	3.07	-0.49	10.00	0.63
	Defiance	0.90	4.80	0.62	10.00	0.54
	Inattention	1.45	2.94	1.63	10.00	0.13
	Hyperactive/Impulsive	0.66	11.50	0.17	8.00	0.86
Conners' CPT	Omissions	-2.57	15.01	-1.41	67.00	0.16
	Commissions	0.19	7.68	-1.41	67.00	0.16
	Hit Response Time	-5.64	85.34	-0.54	67.00	0.58
	Hit Response Time Std Error	-0.48	6.34	-0.62	67.00	0.53
	Variability	-0.70	19.35	-0.29	67.00	0.76
	Dectectability	0.03	0.43	0.54	67.00	0.58
	Response Style	0.12	1.01	0.98	67.00	0.32
	Preservations	-2.70	12.64	-1.76	67.00	0.08
	Response Time Block Change	0.03	0.24	1.14	67.00	0.25
	Block Change Std Error	0.05	0.27	1.52	67.00	0.13

[a] Significant after Bonferroni correction. Conners' 3 Parent Rating Scale calculation of 0.05/5=0.01,
[b] Significant after Bonferroni correction. Conners' 3 Teacher Rating Scale calculation of 0.05/5=0.01,
[c] Significant after Bonferroni correction. Conners' CPT calculation of 0.05/10=0.005

As Table 34 shows, there was a significant main effect of diagnosis on the dependent variables on all of the Conners' Parent subscales.

Furthermore, a repeated measures multivariate ANOVA with a Bonferroni correction was conducted to examine the significant difference between the neuropsychometric post measures and the study conditions. No significant differences were found.

Table 35.

Paired T-test results of pre-to post measure dependent variables on typically developed sample

		Mean	SD	t	df	Sig. (2-tailed)
Conners' 3 Parent Rating Scale	Learning Difficulties	1.13	2.37	2.91	36.00	0.01[a]
	Executive Functions	0.62	2.89	1.31	36.00	0.19
	Defiance	0.70	2.81	1.51	36.00	0.13
	Inattention	2.45	4.83	3.09	36.00	0.01[a]
	Hyperactive/Impulsive	2.24	4.83	3.09	36.00	0.01[a]
Conners' 3 Teacher Rating Scale	Learning Difficulties	0.00	2.64	0.00	6.00	1.00
	Executive Functions	0.42	2.99	0.37	6.00	0.71
	Defiance	1.71	5.28	0.89	6.00	0.42
	Inattention	2.00	2.58	0.85	6.00	0.08
	Hyperactive/Impulsive	3.49	12.78	0.67	6.00	0.53
Conners' CPT	Omissions	-0.36	10.78	-0.23	40.00	0.81
	Commissions	-1.37	7.51	-1.12	40.00	0.26
	Hit Response Time	3.96	93.57	0.27	40.00	0.78
	Hit Response Time Std Error	0.02	5.73	0.03	40.00	0.97
	Variability	0.74	17.14	0.27	40.00	0.78
	Dectectability	0.08	0.43	1.28	40.00	0.21
	Response Style	0.04	0.49	0.56	40.00	0.57
	Preservations	-1.92	6.14	-2.01	40.00	0.05
	Response Time Block Change	0.04	0.31	0.99	40.00	0.32
	Block Change Std Error	0.05	0.33	1.11	40.00	0.27

[a] Significant after Bonferroni correction. Conners' 3 Parent Rating Scale calculation of 0.05/5=0.01,
[b] Significant after Bonferroni correction. Conners' 3 Teacher Rating Scale calculation of 0.05/5=0.01,
[c] Significant after Bonferroni correction. Conners' CPT calculation of 0.05/10=0.005

Table 36.

Paired T-test results of pre to post measure dependent variables on ADHD sample

		Mean	SD	t	df	Sig. (2-tailed)
Conners' 3 Parent Rating Scale	Learning Difficulties	0.79	2.93	1.45	28.00	0.15
	Executive Functions	2.31	4.22	2.94	28.00	0.0[1a]
	Defiance	2.55	4.38	3.13	28.00	0.0[1a]
	Inattention	4.10	5.14	4.29	28.00	0.01[a]
	Hyperactive/Impulsive	1.51	7.78	3.52	28.00	0.01[b]
Conners' 3 Teacher Rating Scale	Learning Difficulties	1.49	0.99	2.99	3.00	0.05
	Executive Functions	-1.99	2.94	-1.35	3.00	0.26
	Defiance	-0.49	4.12	-0.24	3.00	0.82
	Inattention	0.49	3.69	0.27	3.00	0.79
	Hyperactive/Impulsive	-4.99	6.92	-1.25	3.00	0.33
Conners' CPT	Omissions	-5.92	20.12	-1.53	26.00	0.13
	Commissions	2.48	7.51	1.71	26.00	0.09
	Hit Response Time	-20.24	79.22	-1.49	26.00	0.14
	Hit Response Time Std Error	-1.24	7.22	-0.86	26.00	0.37
	Variability	-2.88	22.47	-0.66	26.00	0.51
	Dectectability	-0.05	0.43	-0.71	26.00	0.47
	Response Style	0.23	1.49	0.92	26.00	0.41
	Preservations	-3.88	18.75	-1.07	26.00	0.29
	Response Time Block Change	0.01	0.04	1.39	26.00	0.17
	Block Change Std Error	0.03	0.13	1.47	26.00	0.15

[a] Significant after Bonferroni correction Conners' 3 Parent Rating Scale calculation of 0.05/5=0.01,
[b] Significant after Bonferroni correction Conners' 3 Teacher Rating Scale calculation of 0.05/5=0.01

As Table 35 and 36 show, both samples showed a significant main effect of change from pre-to-post measure on several of the Conners' 3 Parent Rating Scale. There was no significant difference between pre-to post measures in either samples for the Conners' Teacher Rating Scales.

A Pearson correlation coefficient was then computed to assess the relationship between the pre and post measures and the independent variables (Table 37).

Table 37.

Pearson correlation coefficient for pre to post measures of age on dependent variable of diagnosis

		Pearson Correlation	Sig.	Strength of relationship
Conners' 3 Parent Rating Scale	Learning Difficulties	0.90	0.01	Strong positive
	Executive Functions	0.78	0.01	Strong positive
	Defiance	0.79	0.01	Strong positive
	Inattention	0.78	0.01	Strong positive
	Hyperactive/Impulsive	0.84	0.01	Strong positive
Conners' 3 Teacher Rating Scale	Learning Difficulties	0.95	0.01	Strong positive
	Executive Functions	0.14	0.71	Very weak positive
	Defiance	0.92	0.01	Strong positive
	Inattention	0.79	0.01	Strong positive
	Hyperactive/Impulsive	0.70	0.01	Strong positive
Conners' CPT	Omissions	0.68	0.01	Strong positive
	Commissions	0.49	0.01	Moderate positive
	Hit Response Time	0.55	0.01	Moderate positive
	Hit Response Time Std Error	0.70	0.01	Strong positive
	Variability	0.68	0.01	Strong positive
	Dectectability	0.25	0.03	Weak positive
	Response Style	0.69	0.57	Strong positive
	Preservations	0.59	0.01	Moderate positive
	Response Time Block Change	-0.03	0.80	None
	Block Change Std Error	0.01	0.91	None

As Table 37 shows, age had a significant correlation on all but 4 subscales when comparing pre to post results across all samples.

Table 38.
Descriptive statistics Absolute change mean scores from pre to post measures

		T-test of diagnosis		T-test of gender Typically developed sample		T-test of gender ADHD sample		Pearson Product correlation of age	
		Typically developed sample	ADHD sample	Male	Female	Male	Female	Typically developed sample	ADHD sample
Conners' 3 Parent Rating Scale	Learning Difficulties	-0.89	-1.17	-0.89	-0.87	-0.87	-12.41	0.06	0.01
	Executive Functions	-0.81	-2.11	-0.56	-0.75	-1.64	-5.08	-0.126	0.03
	Defiance	-0.62	-1.67	-0.56	-0.44	-1.93	0.84	-0.03	0.17
	Inattention	-2.81	-3.54	-3.33	-1.38	-2.72	-8.83	-0.261	0.256
	Hyperactive/Impulsive	-2.44	-3.41	-1.93	-2.68	-3.25	-5.916	40.55	0.35
Conners' 3 Teacher Rating Scale	Learning Difficulties	-0.13	2.21	-0.51	0.61	2.21	-	-0.64	0.14
	Executive Functions	-1.29	1.43	-1.35	-0.47	1.43	-	-0.51	-0.27
	Defiance	-0.18	3.81	2.51	-3.81	3.81	-	21.77	-0.23
	Inattention	-3.29	1.95	-2.4	-3.87	1.95	-	-0.41	-0.28
	Hyperactive/Impulsive	-4.93	13.13	-0.9	-1899.3	22.13	-	0.09	0.18
Conners' CPT	Omissions	-5.41	8.91	-1.59	1.06	6.78	-21.00	0.04	0.11
	Commissions	-0.99	-1.49	0.91	1.19	-2.69	-9.00	-0.2	-0.13
	Hit Response Time	-39.82	62.69	-9.08	-10.96	44.93	-58.21	-0.05	0.58
	Hit Response Time Std Error	-3.77	4.68	-0.39	-0.16	2.14	-6.52	0.05	-1.16
	Variability	-10.74	11.96	-1.81	-0.44	4.39	-14.31	0.03	0.14
	Dectectability	-0.02	0.05	-0.04	-0.14	0.064	0.41	0.18	0.04
	Response Style	-0.32	0.05	-0.05	-0.078	-0.16	-0.73	0.16	-0.46
	Preservations	-4.91	8.64	1.00	2.56	2.83	-4.54	0.46	-0.05
	Response Time Block Change	-0.18	-0.05	-0.06	0.01	-0.002	-0.03	-0.15	-0.27
	Block Change Std Error	-0.03	-0.07	-0.07	-0.01	-0.04	-0.11	0.004	-0.05

Paired t-tests were then conducted on each condition, comparing pre-to post measures on all the dependent variables (Table 39 to Table 45). This examined the main effect of intervention on the dependent variables.

Table 39.
Repeated measures T-test from pre-to post measure dependent variables on typically developed neurofeedback condition

		Neurofeedback							
		Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval
Conners' 3 Parent Rating Scale	Learning Difficulties	1.73	2.83	2.02	10.00	0.07	0.61	-0.17	3.62
	Executive Functions	1.55	2.66	1.93	10.00	0.08	0.58	-0.24	3.33
	Defiance	1.64	4.25	1.28	10.00	0.23	0.39	-1.21	4.49
	Inattention	4.18	7.11	1.95	10.00	0.08	0.59	-0.59	8.95
	Hyperactive/Impulsive	4.36	6.98	2.07	10.00	0.06	0.62	-0.32	9.04
Conners' 3 Teacher Rating Scale	Learning Difficulties	-4.33	7.51	-1.00	2.00	0.42	-0.58		
	Executive Functions	0.33	0.58	1.00	2.00	0.42	0.57	-5.85	6.85
	Defiance	-1.67	3.79	-0.76	2.00	0.53	-0.44		
	Inattention	-2.00	3.46	-1.00	2.00	0.42	-0.58	-6.85	5.85
	Hyperactive/Impulsive	-2.38	12.68	-0.68	2.00	0.51	-0.19		
Conners' CPT	Omissions	-2.38	9.22	-0.93	12.00	0.37	-0.26	-11.37	3.04
	Commissions	-33.95	147.93	-0.83	12.00	0.42	-0.23	-25.66	9.19
	Hit Response Time	-2.36	7.31	-1.16	12.00	0.27	-0.32	-135.25	60.36
	Hit Response Time Std Error	-4.79	18.82	-0.92	12.00	0.38	-0.25	-7.55	1.85
	Variability	0.03	0.42	0.30	12.00	0.77	0.07	-18.32	5.02
	Dectectability	-0.13	0.35	-0.36	12.00	0.20	-0.37	-0.24	0.31
	Response Style	-1.38	4.63	-1.08	12.00	0.30	-0.30	-0.35	0.11
	Preservations	-0.43	1.43	-1.08	12.00	0.30	-0.30	-4.66	1.32
	Response Time Block Change	0.16	0.56	1.02	12.00	0.33	0.29	-0.21	0.54
	Block Change Std Error	0.18	0.57	1.15	12.00	0.27	0.32	0.17	0.55

Table 40.

Repeated measures T-test from pre-to post measure dependent variables on typically developed control condition

		Control							
		Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval
Conners' 3 Parent Rating Scale	Learning Difficulties	0.80	1.78	14.00	0.10	0.45	1.00	-0.18	1.78
	Executive Functions	-0.60	2.72	-0.85	14.00	0.42	-0.22	-2.10	0.91
	Defiance	0.00	1.85	0.00	14.00	1.00	0.00	-1.02	1.02
	Inattention	0.47	3.25	0.56	14.00	0.59	0.14	-1.33	2.26
	Hyperactive/Impulsive	0.00	3.51	0.00	14.00	1.00	0.00	-1.94	1.94
Conners' 3 Teacher Rating Scale	Learning Difficulties	5.25	16.13	0.65	3.00	0.56	0.33	-5.31	3.31
	Executive Functions	-1.00	2.71	-0.74	3.00	0.51	-0.37	-4.43	2.43
	Defiance	-1.00	2.16	-0.93	3.00	0.42	-0.46	-9.32	13.82
	Inattention	2.25	7.27	0.62	3.00	0.58	0.31	0.71	3.29
	Hyperactive/Impulsive	1.60	8.42	0.74	14.00	0.47	0.19	-20.42	30.92
Conners' CPT	Omissions	-0.33	6.29	-0.21	14.00	0.84	-0.05	-3.06	6.36
	Commissions	12.25	39.27	1.21	14.00	0.25	0.31	-10.33	8.65
	Hit Response Time	1.09	4.06	1.04	14.00	0.31	0.27	-9.51	33.99
	Hit Response Time Std Error	2.20	14.07	0.60	14.00	0.56	0.16	-1.15	3.34
	Variability	0.13	0.46	1.12	14.00	0.28	0.28	-5.59	9.98
	Dectectability	0.14	0.46	1.15	14.00	0.27	0.30	-0.12	0.38
	Response Style	-1.60	6.49	-0.95	14.00	0.36	-0.25	-0.11	0.39
	Preservations	-0.49	2.01	-0.95	14.00	0.36	-0.24	-5.19	1.99
	Response Time Block Change	0.00	0.04	-0.18	4.00	0.86	0.00	-0.02	0.02
	Block Change Std Error	0.00	0.12	-0.11	14.00	0.91	0.00	-0.06	0.06

Table 41.

Repeated measures T-test from pre-to post measure dependent variables on typically developed active control condition

		Active Control							
		Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval
Conners' 3 Parent Rating Scale	Learning Difficulties	1.00	2.68	1.24	10.00	0.24	0.37	-0.80	2.80
	Executive Functions	1.36	298.00	1.52	10.00	0.16	0.46	-0.63	3.36
	Defiance	0.73	2.00	1.20	10.00	0.26	0.37	-0.61	2.07
	Inattention	3.45	2.91	3.94	10.00	0.01	1.19	1.49	5.41
	Hyperactive/Impulsive	3.16	4.64	2.27	10.00	0.05	0.69	0.06	6.30
Conners' 3 Teacher Rating Scale	Learning Difficulties	-	-	-	-	-	-	-	-
	Executive Functions	-	-	-	-	-	-	-	-
	Defiance	-	-	-	-	-	-	-	-
	Inattention	-	-	-	-	-	-	-	-
	Hyperactive/Impulsive	-	-	-	-	-	-	-	-
Conners' CPT	Omissions	-1.00	7.24	-0.52	13.00	0.61	-0.14	-5.21	6.78
	Commissions	30.60	52.97	2.16	13.00	0.05	0.58	-15.07	8.72
	Hit Response Time	1.35	5.12	0.99	13.00	0.34	0.26	0.00	61.17
	Hit Response Time Std Error	5.52	18.11	1.14	13.00	0.27	0.30	-1.60	4.31
	Variability	0.08	0.42	0.70	13.00	0.50	0.19	-4.93	15.97
	Dectectability	0.09	0.62	0.53	13.00	0.61	0.15	-0.16	0.32
	Response Style	-2.50	7.18	-1.30	13.00	0.22	-0.35	-0.26	0.44
	Preservations	-0.77	2.22	-1.30	13.00	0.22	-0.35	-6.64	1.64
	Response Time Block Change	0.01	0.03	0.81	13.00	0.43	0.33	-0.01	0.02
	Block Change Std Error	0.02	0.10	0.77	13.00	0.46	0.20	-0.03	0.08

None of the interventions had a large effect size on CPRS, CTRS or CPT.

Table 42.

Repeated measures T-test from pre-to post measure dependent variables on ADHD medication condition

		Medication							
		Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval
Conners' 3 Parent Rating Scale	Executive Functions	3.00	4.27	2.81	15.00	0.01	0.70	0.72	5.27
	Defiance	2.88	4.38	2.63	15.00	0.02	0.66	0.54	5.21
	Inattention	5.44	4.86	4.48	15.00	0.01	1.12	2.84	8.02
	Hyperactive/Impulsive	6.38	8.12	3.14	15.00	0.00	0.79	2.04	10.70
Conners' 3 Teacher Rating Scale	Learning Difficulties	1.67	1.15	2.50	2.00	0.13	1.45	-1.20	4.53
	Executive Functions	-0.67	1.53	-0.76	2.00	0.53	-0.44	-4.46	3.12
	Defiance	1.33	2.31	1.00	2.00	0.42	0.58	-4.40	7.07
Conners' CPT	Omissions	-6.13	22.95	-1.04	14.00	0.32	-0.27	-18.84	6.57
	Commissions	0.30	8.07	0.10	14.00	0.92	0.02	-11.83	13.04
	Hit Response Time	-27.15	82.22	-1.28	14.00	0.22	-0.33	-72.68	18.38
	Hit Response Time Std Error	-2.00	7.43	-1.04	14.00	0.31	-0.27	-6.11	2.11
	Variability	-0.45	19.05	-0.09	14.00	0.93	-0.02	-12.44	28.91
	Dectectability	0.09	0.47	0.75	14.00	0.47	0.19	-0.16	0.35
	Response Style	0.27	1.95	0.54	14.00	0.69	0.14	-0.81	1.35
	Preservations	-6.47	10.70	-2.34	14.00	0.03	-0.60	2.76	-12.39
	Response Time Block Change	0.01	0.03	1.53	14.00	0.15	0.33	-0.01	0.03
	Block Change Std Error	0.05	0.12	1.50	14.00	0.15	0.42	-0.01	0.11

Table 43.

Repeated measures T-test from pre-to post measure dependent variables on ADHD medication and neurofeedback home training condition

		Medication and Neurofeedback Home Training							
		Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval
Conners' 3 Parent Rating Scale	Executive Functions	2.00	4.73	1.04	5.00	0.35	0.42	-2.96	6.96
	Defiance	2.17	4.26	1.25	5.00	0.26	0.51	-2.31	6.63
	Inattention	3.83	5.34	1.76	5.00	0.14	0.72	-1.77	9.44
	Hyperactive/Impulsive	3.50	6.35	1.35	5.00	0.23	0.55	-3.16	10.16
Conners' 3 Teacher Rating Scale	Learning Difficulties	-	-	-	-	-	-	-	-
	Executive Functions	-	-	-	-	-	-	-	-
	Defiance	-	-	-	-	-	-	-	-
Conners' CPT	Omissions	1.50	10.95	0.34	5.00	0.75	0.14	-9.99	12.99
	Commissions	5.83	7.68	1.86	5.00	0.12	0.70	-6.17	38.58
	Hit Response Time	12.93	4.66	0.73	5.00	0.50	0.30	-32.82	58.67
	Hit Response Time Std Error	3.53	6.87	1.30	5.00	0.24	0.53	-3.57	10.84
	Variability	8.25	19.79	1.02	5.00	0.35	0.42	-12.44	28.91
	Dectectability	-0.24	0.34	-1.71	5.00	0.15	-0.71	-0.60	0.12
	Response Style	0.04	0.71	0.13	5.00	0.90	0.06	-0.71	0.78
	Preservations	0.33	20.74	1.11	5.00	0.32	0.45	-12.43	31.10
	Response Time Block Change	0.01	0.03	0.68	5.00	0.53	0.33	-0.02	0.03
	Block Change Std Error	-0.03	0.17	-0.39	5.00	0.71	-0.18	-0.20	0.14

Table 44.

Repeated measures T-test from pre-to post measure dependent variables on ADHD medication and neurofeedback in clinic condition

		Medication and Neurofeedback Clinic Training							
		Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval
Conners' 3 Parent Rating Scale	Executive Functions	2.50	4.65	1.07	3.00	0.36	0.54	-4.91	9.91
	Defiance	3.00	6.06	0.99	3.00	0.39	0.50	-6.63	12.63
	Inattention	3.40	5.92	1.18	3.00	0.32	0.59	-5.91	12.91
	Hyperactive/Impulsive	5.75	10.53	1.09	3.00	0.35	0.55	-11.01	22/.508
Conners' 3 Teacher Rating Scale	Learning Difficulties	-	-	-	-	-	-	-	-
	Executive Functions	-	-	-	-	-	-	-	-
	Defiance	-	-	-	-	-	-	-	-
Conners' CPT	Omissions	-20.75	14.97	-2.77	3.00	0.07	-1.39	-44.57	3.07
	Commissions	3.75	4.03	1.86	3.00	0.16	0.93	-7.39	28.22
	Hit Response Time	-27.73	48.23	1.15	3.00	0.33	-0.57	-104.48	49.01
	Hit Response Time Std Error	-5.40	471.00	-2.29	3.00	0.11	-1.15	-12.89	2.09
	Variability	-26.56	24.43	-2.17	3.00	0.12	-1.09	-65.42	12.31
	Dectectability	-0.22	0.20	-2.15	3.00	0.12	-1.10	-0.54	0.11
	Response Style	0.57	0.73	1.57	3.00	0.21	0.78	-0.58	1.72
	Preservations	-4.75	27.97	-0.34	3.00	0.76	-0.17	-49.25	39.75
	Response Time Block Change	-0.02	0.07	-0.45	3.00	0.68	-0.29	-0.12	0.09
	Block Change Std Error	0.05	0.16	0.62	3.00	0.58	0.31	-206.00	0.31

Table 45.

Repeated measures T-test from pre-to post measure dependent variables on ADHD neurofeedback in clinic condition

		Neurofeedback Clinic Training							
		Mean	SD	t	d	Sig. (2-tailed)	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval
Conners' 3 Parent Rating Scale	Executive Functions	-1.00	2.00	-0.87	2.00	0.48	-0.50	-5.96	3.96
	Defiance	1.00	4.36	0.40	2.00	0.73	0.23	-9.82	11.82
	Inattention	-1.67	2.08	-1.39	2.00	0.30	-0.80	-6.83	3.50
	Hyperactive/Impulsive	0.67	6.03	0.19	2.00	0.87	0.11	-14.04	15.64
Conners' 3 Teacher Rating Scale	Learning Difficulties	-	-	-	-	-	-	-	-
	Executive Functions	-	-	-	-	-	-	-	-
	Defiance		-	-	-	-	-	-	-
Conners' CPT	Omissions	-	-	-	-	-	-	-	-
	Commissions	-	-	-	-	-	-	-	-
	Hit Response Time	-	-	-	-	-	-	-	-
	Hit Response Time Std Error	-	-	-	-	-	-	-	-
	Variability	-	-	-	-	-	-	-	-
	Dectectability	-	-	-	-	-	-	-	-
	Response Style	-	-	-	-	-	-	-	-
	Preservations	-	-	-	-	-	-	-	-
	Response Time Block Change	-	-	-	-	-	-	-	-
	Block Change Std Error	-	-	-	-	-	-	-	-

Table 46.

Descriptive statistical and absolute change scores of dependent variables on typically developed sample conditions

		Neurofeedback			Control			Active Control		
		Pre Mean	Post Mean	Absolute Change Score	Pre Mean	Post Mean	Absolute Change Score	Pre Mean	Post Mean	Absolute Change Score
Conners' 3 Parent Rating Scale	Learning Difficulties	10.33	9.00	-1.33	7.87	7.07	-0.80	10.25	9.25	-1.00
	Executive Functions	12.53	11.36	-1.17	8.27	8.87	0.60	11.83	9.67	-2.17
	Defiance	4.60	3.36	-1.24	3.07	3.07	0.00	3.08	2.08	-1.00
	Inattention	15.47	11.00	-4.47	8.53	8.07	-0.47	12.67	8.50	-4.17
	Hyperactive/Impulsive	13.80	10.36	-3.44	8.73	8.73	0.00	12.92	9.42	-3.50
Conners' 3 Teacher Rating Scale	Learning Difficulties	5.60	6.33	0.73	1.80	3.25	1.45	4.25	-	-
	Executive Functions	6.60	8.67	2.07	4.80	5.75	0.95	10.75	-	-
	Defiance	1.40	2.67	1.27	3.40	2.00	-1.40	6.75	-	-
	Inattention	8.40	10.33	1.93	4.60	3.00	-1.60	16.50	-	-
	Hyperactive/Impulsive	2.60	7.67	5.07	10.00	6.00	-4.00	19.50	-	-
Conners' CPT	Omissions	14.67	18.43	3.76	15.47	13.87	-1.60	12.21	12.73	0.52
	Commissions	21.53	23.14	1.61	22.80	23.13	0.33	21.79	22.60	0.81
	Hit Response Time	379.62	411.73	32.11	410.79	398.55	-12.25	427.99	398.76	-29.23
	Hit Response Time Std Error	9.29	11.57	2.28	11.98	10.89	-1.09	11.31	10.06	-1.24
	Variability	19.09	23.73	4.64	24.37	22.17	-2.20	21.96	17.07	-4.89
	Dectectability	0.37	0.34	-0.02	0.39	0.25	-0.13	0.37	0.30	-0.06
	Response Style	0.57	0.75	0.18	0.69	0.55	-0.14	0.75	0.68	-0.07
	Preservations	4.00	5.57	1.57	7.20	8.80	1.60	6.71	8.93	2.22
	Response Time Block Change	0.14	0.01	-0.13	0.03	0.03	0.00	0.01	0.00	-0.01
	Block Change Std Error	0.19	0.05	-0.14	0.11	0.11	0.00	0.05	0.04	-0.01

When examining the effect size of the interventions in the ADHD sample, medication has a large effect size on CPRS executive function, hyperactive/impulsive. Medication and neurofeedback home training has a large effect size on CPRS inattention, and CPT commissions. Medication and neurofeedback in clinic has a large effect size on CPT response style and a very large effect size on CPT commissions.

The neurofeedback condition saw an increase, worsening of results, in absolute change on many of the Conners' Teachers Rating Scale and Conners' CPT scales.

Table 47.

Descriptive statistics and absolute change mean scores of dependent variables on ADHD sample conditions

		Medication			Medication and Neurofeedback Home Training			Medication and Neurofeedback Clinic			Neurofeedback Home Training		
		Pre Mean	Post Mean	Absolute Change Score	Pre Mean	Post Mean	Absolute Change Score	Pre Mean	Post Mean	Absolute Change Score	Pre Mean	Post Mean	Absolute Change Score
Conners' 3 Parent Rating Scale	Learning Difficulties	10.83	9.53	-1.30	11.43	9.83	-1.60	14.50	14.75	0.25	10.00	11.00	1.00
	Executive Functions	14.50	11.65	-2.85	12.43	10.00	-2.43	14.25	11.75	-2.50	19.00	20.00	1.00
	Defiance	7.11	4.35	-2.76	11.43	10.17	-1.26	9.00	6.00	-3.00	7.33	6.33	-1.00
	Inattention	19.00	14.29	-4.71	17.57	12.67	-4.90	20.75	17.25	-3.50	23.00	24.67	1.67
	Hyperactive/Impulsive	22.78	16.88	-5.90	24.00	20.33	-3.67	24.00	18.25	-5.75	28.67	28.00	
Conners' 3 Teacher Rating Scale	Learning Difficulties	7.33	7.67	0.33	-	-	-	-	-	-	-	28.00	-
	Executive Functions	10.80	9.67	-1.13	-	-	-	-	-	-	-	54.00	-
	Defiance	5.83	2.67	-3.17	-	-	-	-	-	-	-	12.00	-
	Inattention	14.00	12.00	-2.00	-	-	-	-	-	-	-	16.00	-
	Hyperactive/Impulsive	8.80	3.00	-5.80	-	-	-	-	-	-		28.00	-
Conners' CPT	Omissions	13.53	19.33	5.80	26.57	13.17	-13.40	28.50	49.25	20.75	27.00	61.00	-
	Commissions	21.24	20.13	-1.10	25.00	20.00	-5.00	28.25	24.50	-3.75	32.00	20.00	-
	Hit Response Time	424.61	454.21	29.60	480.53	441.93	-38.61	488.49	516.22	27.73	366.70	595.55	-
	Hit Response Time Std Error	11.62	13.66	2.04	17.35	10.14	-7.21	19.99	25.39	5.40	19.88	38.97	-
	Variability	24.73	25.61	0.88	36.99	20.90	-16.09	39.05	65.61	26.56	51.34	113.32	-
	Dectectability	0.44	0.40	-0.04	0.26	0.47	0.22	0.05	0.27	0.22	0.00	0.85	-
	Response Style	0.97	0.71	-0.26	0.82	0.75	-0.07	1.44	0.87	-0.57	1.07	0.75	-
	Preservations	5.18	11.27	6.09	17.00	4.33	-12.67	27.00	31.75	4.75	43.67	85.00	-
	Response Time Block Change	0.02	0.00	-0.02	0.01	0.01	0.00	0.03	0.04	0.02	0.05	-0.01	-
	Block Change Std Error	0.11	0.05	-0.05	0.03	0.07	0.04	0.09	0.04	-0.05	0.15	-0.05	-

4.6 Discussion

The present study investigated the differences of neuropsychological profiles between typically developed children and children with a diagnosis of ADHD. Comparisons took place pre and post interventions to understand the effect of neurofeedback home training on concentration and impulsive behaviours. The main findings from prior and post interventions in turn, in relation to previous research, will now be discussed.

4.6.1 Discussion Regarding Pre Measures

In line with previous literature, it was hypothesised that there would be a significant difference between the ADHD and typically developed sample on the Conners' 3 Parent Rating Scale (Snyder et al., 2008), with the ADHD sample showing significantly impaired results on the Conners' 3 Parent and Teacher Rating Scales.

The research presented in this thesis replicated this finding; the ADHD sample were significantly different from the typically developed sample, with the ADHD sample showing higher impairment on the executive functions, defiance, inattention, hyperactive/impulsive Conners' 3 Parent Rating Subscales and learning difficulties, executive functions, inattention and hyperactive/impulsive Conners' 3 Teacher Rating Subscales. This finding replicates previous research on the different presentation between typically developed individuals and individuals with a diagnosis of ADHD, as rated by parents and teachers (Conners, 2008; Snyder et al., 2008; Tripp et al., 2006). Additionally, no effect was found for gender which supports previous research (Gianarris et al., 2001).

It is interesting that both executive functions, inattention and hyperactive/impulsive subscales on the Conners' 3 Parent and Teacher Rating Scales were significantly different in the ADHD sample compared to the typically developed

sample. Difficulties in inattention are part of executive function, requiring activation, focus and effort (Brown, 2006). Similarly, impulsivity requires executive function input by being able to inhibit a response (Barkley, 1997). This evidence supports Barkley and Brown's theories of ADHD, that the condition consists of impairments in executive functions, which overlap with inattention and impulsivity (Barkley, 2003).

Based upon previous research, it was hypothesised that the ADHD sample would have higher scores, specifically commission and omission errors, on the CPT than the typically developed sample (Preston et al., 2000; McGee et al., 2000; Epstein et al., 2003; Advokat et al., 2007). This research found a significant difference between the ADHD and typically developed sample on the preservations subscale, although this was not significant after Bonferroni correction. Although this was not the omission and commissions subscales as expected, the ADHD sample did show more impairment on these scales, but not significantly. This provides very limited support for previous literature that the CPTs are able to identify individuals with ADHD traits because only one of the 10 CPT items showed a statistically significant difference (Preston et al., 2005, McGee et al., 2000; Epstein et al., 2003; Advokat et al., 2007). Furthermore, this finding links other variables of CPT to the theories of ADHD. Although commission and omission errors are typically referred to (McGee et al., 2000; Epstein et al., 2003; Advokat et al., 2007), the research in this thesis suggests that other CPT measures, such as preservations, is an indicator of inattention. As both Barkley (1997) and Brown (2006) state, ADHD is caused by deficits in executive function, which includes the ability to sustain and shift attention.

The reason for the lack of significant difference on the commission and omission errors could be due to the self-selecting nature of participants. Specifically, based upon the researcher's clinical experience, it is suggested that the ADHD

population may not have been a typical sample due to recruitment taking place from a private clinic setting. The clinic was based in a wealthy area and participants had to pay to access the Centre, consequently being in a financial position to afford assessment. Furthermore, individuals accessing the Centre were typically previously turned away from the National Health Service due to not meeting their criteria level. This may account for there not being a significant difference between the two samples as expected.

McGee et al. (2000) found that Conners' CPT was not correlated with age, showing appropriate age relative norms. This was not a finding that was replicated in our study. Instead, age had an effect on CPT performance, specifically a moderate negative relationship with CPT results, across both the ADHD and typically developed samples. This would suggest that older children attend better or understand the task to complete it more effectively. In practice, two seven-year-old children in the ADHD sample were unable to complete the task due to finding it difficult and older children persevering to complete it.

In regard to gender, McGee et al. (2000) found that males with a diagnosis of ADHD made more commission errors, although this was not a robust finding. Hasson et al. (2012) also found males with ADHD made more commission errors than omission errors and replicated this in a typically developed sample. This finding was not produced in our study. As mentioned previously, this could be due to the self-selecting nature of participants and consequently skewed the data.

4.6.2 Discussion Regarding Post Measures

Based upon previous research, it was hypothesised that the typically developed sample who received neurofeedback home training would show greater improvement on neuropsychometric scales compared to the other conditions in that sample. Previous

research has shown that neurofeedback can reduce CPT scores in typically developed adults (Vernon et al., 2003; Egner & Gruzelier, 2001). As hypothesized, results in our study showed that the typically developed sample neurofeedback home training condition saw the most amount of absolute change mean scores across the typically developed sample, although not a statistically significant change. Specifically, all of the Conners' 3 Parent Rating subscales reduced, with the largest decreases across conditions in learning difficulties, defiance, inattention and hyperactive/impulsivity subscales.

Vernon et al. (2003) examined adults completing neurofeedback, with a theta enhancing condition and a SMR enhancing condition. Results showed SMR neurofeedback to improve CPT results, specifically attention. Results in the present study showed neurofeedback created a worsening on CPT but an improvement on parent rating. This evidence therefore conflicts with Vernon et al. (2003) findings. Previous research has shown potential negative side effects of neurofeedback, including inducing seizures (Vernon et al., 2004), irritability and moodiness (Monastra et al., 2002) with the present study showing neurofeedback home training potentially worsening concentration. This could support Cortese et al. (2016) where evidence failed to support the use of neurofeedback as a treatment for ADHD in addition to Sonuga-Barke et al. (2013) where significant treatment effects of neurofeedback were non-existent when participants were blinded to intervention.

However, many of the Conners' 3 Teacher Rating subscales and CPT subscales in the neurofeedback home training condition in the typically developed sample increased, showing a worsening of concentration. Specifically, an increase on executive function, defiance, inattention, and hyperactive/impulsive subscales of the Conners'3 Teacher Rating Scale, and an increase in CPT omissions, commissions, hit

response time, hit response time standard error and response style. This supports Kaner (2011) and McGee et al. (2000) who found poor agreement on difficulties between rater's responses. Every individual will interpret behaviours differently depending on rater's familiarity with the behaviour and situation of the observed behaviour (De Log Reyes & Kazdin, 2005). The worsening of inattention as rated by teachers and CPT but improvement when rated by parents supports Snyder et al. (2008) who found that rating scales had an accuracy rate of 47-58% in identifying ADHD, likely to be due to informant bias. The findings of positive outcome from parent's report but not teacher report suggests possible parental bias due to subconscious bias by parents based on cognitive dissonance. As parents have invested time and energy into participating in the research, they may be more likely to expect, and hope for, a positive outcome. This supports Cortese et al. (2016) and Sonuga-Barke et al. (2013) where it was also found significant effects of nonpharmacological treatments when rated by individuals not blinded to the intervention, compared to blinded raters who showed non-significant effects. This therefore highlights that the CPRS is a very subjective scale (Gianarris, Golden, & Greene, 2001) which is a weakness of this tool, and highlights the importance of other measures, such as a teacher rating scale or EEG. Additionally, it fails to support the use of neurofeedback as a treatment for ADHD as measured by parent, teacher and CPT.

Unexpectedly, the active control condition in the typically developed sample saw a significant effect from pre to post measures in the CPRS subscales of inattention, hyperactive/impulsive and CPT commission subscales. This was an unexpected finding as it was hypothesized that there would be no differences on Conners' CPT omission and commission subscales, as well as Conners' Parent/Teacher Rating Scales in the typically developed sample control and active control conditions. However,

literature shows that video gaming can alter brainwave activity as well as attention skills. Specifically, a more complex video game that is played, the faster brainwaves are produced to aid in extra concentration to successfully complete the game (Bakaoukas, Coada, & Liarokapis, 2015). The evidence in this research supports this, with significant improvement in attention, as rated by parents, and on a less subjective measure, the CPT.

As expected, the ADHD sample saw larger absolute change mean scores in the CPRS and CTRS compared to the typically developed sample. Specifically, in the ADHD sample, most absolute change mean scores on CPT were seen in the medication and neurofeedback home training condition, followed by the medication and neurofeedback in clinic condition. When examining neurofeedback and stimulant medication independently in an ADHD sample, it is well established that stimulant medication is an effective treatment, having a positive effect in approximately 65% to 77% of children (Barkley, 1997; Johnston et al., 2015) and improves CPT performance (Pearson et al., 2004). Additionally, it is known that neurofeedback, when completed within a clinic, improves clinical outcomes with a large effect size on inattention and impulsivity (Arns et al., 2012). As stated by Arns et al. (2009) further research is required to understand the impact of medication of neurofeedback. In an ADHD sample, there were more and larger absolute change mean scores when medication was combined with neurofeedback in clinic than medication in isolation. This suggests that for the best outcome for medication, it could be combined with neurofeedback, either in clinic or home training. Unfortunately, due to the small sample size, the study was unable to assess the use of neurofeedback home training only and therefore it is not possible to make comparisons to the other conditions. The lack of significant findings could be Type II error due to small sample sizes. Additionally, as the present study is a

feasibility study with small samples, findings are treated as tentative for implications, and need to be replicated in a larger sample.

Fuchs et al. (2003) conducted enhancing SMR neurofeedback on ADHD children for 36 sessions. A reduction on Conners' Behaviour Rating Scale, as completed by parents and teacher, as well as a moderate effect size was found. In our study, when neurofeedback home training in an ADHD sample was completed, no moderate effect sizes were found. However, when neurofeedback home training was combined with stimulant medication, a moderate effect size was found on CPRS subscales including defiance, inattention, and hyperactive/impulsive. Similarly, when neurofeedback in clinic was combined with stimulant medication, a moderate effect size was found on all CPRS subscales. However, there was not a significant difference from pre-to-post measures or between conditions. Unfortunately, the finding was not replicated in teachers due to the small sample size. Consequently, future research could replicate this research with a larger sample to examine if the improvements seen by parents is transferred to a school setting. The findings in the present research show that neurofeedback in clinic or home training does not statistically significantly affect concentration. Therefore, neurofeedback as a treatment for ADHD may not be as beneficial in the treatment of ADHD as previously thought, as suggested by a recent meta-analysis (Cortese et al., 2016).

The present research is evidence to support the use of stimulant medication, with only this condition in the ADHD sample finding significant effects from pre to post measures. Specifically, stimulant medication found a significant effect on Conners' 3 Parent Rating Scale executive functions, defiance, inattention, and hyperactive/impulsive subscales. This replicates findings from the meta-analysis, which found improvements on hyperactive/impulsive behaviours as rated by parents,

when long acting methylphenidate was used (Punja et al., 2013). Additionally, the significant finding of stimulant medication improving executive functions, defiance, inattention, and hyperactive/impulsive subscales, supports the theories and causes of ADHD. It is understood that ADHD is caused by deficits in executive functions (Barkley, 1997; Brown, 2006) as well as the role of dopamine (Tripp & Wickens, 2009) with our finding suggesting that stimulant medication directly effects these deficits. The lack of significant findings could be Type II error due to small sample sizes. Additionally, as the present study is a feasibility study with small samples, findings are treated as tentative for implications, and need to be replicated in a larger sample. Alternatively, neurofeedback may not be as effective as a treatment for ADHD, supporting Cortese et al. (2016).

5 Study Three: Effect of Neurofeedback on Electroencephalograms

5.1 Introduction

As previously discussed, neurofeedback has been shown to improve concentration and impulsivity in an ADHD sample (Lubar & Shouse, 1976; Arns et al., 2014; Arns et al., 2009; Carmody et al., 2001; Fuchs et al., 2003; Kaiser & Othmer, 2000; Rossiter, 2004; Rossiter & La Vaque, 1995). Neurofeedback works by an individual learning to regulate levels of cortical arousal in the brain via visual and/or auditory reinforcement (Monastra, 2005), specifically via operant conditioning (Skinner, 1938). However, the extent to which neurofeedback manipulates EEG is unclear with limited studies examining this (Lansbergen, Arns, Dongen-Boomsma, Spronk, & Buitelaar, 2011).

Electroencephalograms (EEGs) collect electrical data from the cerebral cortex, specifically the electrical activity of neurons and information sent between areas of the brain, created by the central nervous system (Demos, 2004). Postsynaptic changes are reflected in EEGs, tracking rapid changes in brain functioning. EEGs are digitally recorded and quantitatively analysed to inspect specific frequency and amplitude of electrical brain activity. Brainwaves progress from the slow wave of delta, theta, alpha and SMR, to the fast wave of beta and gamma, each associated with varying behaviours (Demos, 2004).

5.1.1 EEG Development

Throughout childhood, as humans physically develop, our brain develops, and EEG patterns change. During our first year of life, EEG patterns evolve through interactions with physical items in our surroundings. Specifically, there is an increase in slow theta waves, 3-4 Hz at 3 months to 5Hz at 5 months, increasing further to 6-7Hz at 12 months of age. The pace of EEG development slows during the second year

of childhood, with theta rhythms developing while delta becomes more prominent. At approximately 3-4 years old, EEG patterns are established in the alpha range, continuing to evolve when eyes are closed; while theta is becoming more noticeable. During the ages of 4 to 6 years old, a large brain growth occurs, increasing coherence in the frontal regions and left frontal-occipital coupling (Barry, Clarke, McCarthy, & Selikowitz, 2002). A further EEG growth spurt occurs during 8 to 10 years old which involves increasing fronto-temporal connections in the right hemisphere. A final growth spurt occurs at 11 to 14 years old, during which time synapses within the brain are either being formed or eliminated, a process known as synaptic pruning (Barry et al., 2002). The first age-related resting state EEG study was conducted by Matousek and Petersen (1973), showing slow wave activity, namely delta and theta, were dominant up to the age of 4. After this time, delta and theta decrease with an increase in age, with the rate of higher frequency waves, namely alpha and beta, increasing.

In summary, in typically developed children, brain activity changes and develops with age; a process known as maturation. Specifically, as a child gets older, there is a decrease in slow wave theta levels and an increase in beta levels (Bresnahan & Barry, 2002).

With regards to EEG development and gender differences, findings show a maturational lag in males (Matousek & Petersen, 1973). However, this gender difference disappears during adolescents. Matousek and Petersen (1973) suggested that EEG differences showed early maturation in girls. In 1980, Matthis, Scheffner, Benninger, Lipinski, and Stolzis sampled 285 healthy individuals aged 4 to 11 years old. A 12 channel eyes closed resting state EEG was conducted. Findings showed that girls, compared to boys, have higher levels of relative theta power and lower levels of fast alpha frequencies at the age of 6. However, by age 11, girls have surpassed boys

in alpha in occipital regions, but girls have lower levels of alpha in frontal regions compared to boys.

In a study of 40 healthy adults aged between 20 and 26, an 18 channel EEG with eyes closed resting state showed females to have a higher EEG amplitude. Specifically, females had significantly higher amplitude of theta, alpha and beta bands at 16 electrode locations: FP1, FP2, F7, F8, F3, Fz, F4, C3, C4, P3, Pz, P4, T5, T6, O1 and O2. The underlying reason for the male female EEG difference is not known, although thought that menstrual phase, smaller average women head size and skull thickness does not have an impact. Consequently, age and gender matching is important when comparing samples (Wada, Takizawa, Zheng-Yan, & Yamaguchi, 1994).

Many neurodevelopmental conditions have been linked to differences in EEG patterns (Chabot, Michele, & Prichep, 2005; Cantor & Chabot, 2009). Specific brainwaves associated with concentration and impulsivity difficulties will now be discussed.

5.1.2 EEG and ADHD

There is a large body of research that examines evidence of EEG patterns in individuals with Attention Deficit Hyperactivity Disorder (Monastra, 2008; Chabot et al., 2005; Chabot & Serfontein, 1996; Barry, Johnstone, & Clarke, 2003; Fonseca et al., 2008; Cantor & Chabot, 2009; Bresnahan & Barry, 2002; Clarke, Barry, McCarthy, & Selikowitz,1998). As research demonstrates, EEGs have a high rate of specificity and sensitivity in identifying individuals with ADHD (Fonseca et al., 2008; Chabot & Serfontein, 1996). In a sample of 30 children with ADHD and 30 controls, aged 8 to 11 years old, an eyes closed resting state EEG was recorded from 21 electrodes. EEG had a rate of 83.3% sensitivity and specificity in correctly identifying ADHD (Fonseca

et al., 2008). Another study showed quantitative EEGs to be 93.7% sensitive and had 88% specificity to distinguish healthy children from children with a diagnosis with ADHD. This was concluded from a study of 439 children with ADHD and 310 healthy children, aged between 6 and 17 years old who underwent an EEG eyes closed procedure for 20 minutes with 19 electrodes. The specific EEG markers found in an ADHD population included theta/alpha excess with normal alpha mean frequency in frontal regions, and a second marker of theta/alpha excess in addition to decreased alpha mean frequency (Chabot & Serfontein, 1996).

5.1.2.1 EEG and ADHD in Children

Chabot et al. (2005) conducted a meta-analysis of EEG data of children and adolescents with ADHD. With the development of technology and more EEG recording locations across the scalp, EEG activity in resting eyes closed conditions of children with ADHD are characterised by increased delta and theta and decreased alpha, specifically in the occipital region.

The main EEG pattern in children with ADHD is an excess of theta waves in approximately 84 – 94% of individuals. One study with such findings was conducted by Chabot and Serfontein (1996), whereby 439 children with ADHD and 310 healthy children, aged between 6 and 17 years old, underwent an eyes closed resting state EEG. Findings showed the ADHD population to have theta/alpha excess in the frontal regions with normal alpha mean frequency, or theta/alpha excess across the posterior and/or midline regions with decreased alpha mean frequency. Furthermore, Fonseca et al. (2008) compared 30 children with ADHD to a healthy control sample during a resting EEG with eyes open. Results showed ADHD participants had more absolute theta power, and diffuse increase in delta power across all but occipital regions, compared to healthy children. Another example is by Chabot and Serfontein (1996).

A sample of 310 healthy children and 407 clinical children, 40% ADD, 43% ADHD, aged 6-12 years old, underwent a resting state eyes closed EEG using 19 electrodes. Children with ADHD had increased theta with decreased alpha mean frequency in frontal regions.

Absolute and relative power provide reliable measures of quantifiable changes in EEG. Absolute power refers to averaging the amplitude of every wave in a given bandwidth whereas relative power refers to dividing the absolute amplitude of one frequency by the sum of the absolute amplitudes of all the calculated frequency bands (Barry et al., 2003).

As discussed in the previous section, power levels have been shown to be abnormal in individuals with ADHD (Chabot & Serfontein, 1996; Lubar, 1995). Usually their EEG profiles are characterised by increased theta, both absolute and relative power, in the frontal lobe with slight increase in alpha relative power (Chabot & Serfontein, 1996). Lubar (1995) found that the power of theta compared to beta was different among children with ADHD, with the greatest difference being at the 10-20 electrode locations of Cz and frontal regions, F3 and F4. Absolute and relative power has been shown to be the most reliable EEG measures to demonstrate differences between ADHD and healthy controls, with many clinicians using this information to aid diagnosis due to its overall classification accuracy of 83.1% (Magee, Clarke, Barry, McCarthy, & Selikowitz, 2005). The overall pattern in absolute and relative power research shows that individuals with ADHD exhibit increased slow wave activity in the frontal lobe, with an increase in absolute theta and relative theta, a pattern which has been replicated in males and females (Clarke, Barry, Bond, McCarthy, & Selikowitz, 2002).

Another EEG measurement is coherence. Coherence focuses on a particular bandwidth and measures the cross-spectral power between two electrodes (John, Prichep, & Easton, 1987) providing information regarding coupling of the brain activity across two electrodes (Barry et al., 2003). Coherence provides information regarding the degree of connectivity between structures underlying a pair of electrodes (Dupuy, Clarke, Barry, McCarthy, & Selikowitz, 2008).

There is some evidence to suggest abnormal coherence levels in individuals with ADHD, as demonstrated in the following literature. Looking specifically at interhemispheric coherence, Montagu (1975) found that hyperactive children had higher levels of intrahemispheric coherence in 2Hz, 4Hz, 6Hz, and 8Hz compared to control groups.

Barry et al. (2002) found increased intrahemispheric coherences at short-medium inter-electrode distances but found reduced intrahemispheric coherences at longer electrode distances. The sample consisted of 40 female children diagnosed with ADHD combined subtype, 40 ADHD inattentive subtype, and 40 control, all of which were aged 8 to 12 years old and completed a resting state eyes closed EEG. Dupuy et al. (2010) demonstrated elevated frontal interhemispheric coherences in the theta band and that individuals with combined ADHD were reported to have increased laterality over short-medium interhemispheric distances. This sample consisted of males aged between 8 to 12 years old diagnosed with ADHD and a control group, completing an EEG resting eyes closed measure. The evidence of children with ADHD having elevated interhemispheric coherence suggests underdeveloped long axonal connections compared to control groups.

Ratio is an EEG feature which refers to the relationship between brain frequencies compared to normal controls. Initial findings showed that individuals with

ADHD had an increased theta and increased beta activity, therefore creating an abnormal theta/beta ratio compared to healthy peers (Lubar, 1991).

Clarke et al. (2002) conducted eyes closed resting state EEG in 20 participants with ADHD and 20 healthy individuals, aged 8 to 12 years old. Here it was found that individuals with ADHD had higher theta/alpha ratio compared to the control group. Arns et al. (2012) conducted a meta-analysis on theta/beta data during eyes open EEG. EEG recordings took place at Cz, with a sample of 1253 children with and 517 without ADHD, aged 6 to 18 years old. Analysis found a decrease in theta/beta ratio across the years in the ADHD sample, whereas an increase in theta/beta ratio was seen in the healthy children. It was concluded that excessive theta/beta ratio was not a reliable measure of ADHD.

5.1.2.2 EEG and ADHD in Adults

Bresnahan and Barry (2002) conducted research in adults with ADHD. The sample consisted of 50 adults diagnosed with ADHD, 50 adults with ADHD symptoms but not diagnosed, and 50 controls. EEG was completed in a resting state while eyes where fixated on a screen, using a 17 electrode cap. Findings showed the ADHD group to have higher levels of absolute delta than the other two conditions, as well as more absolute and relative theta power, particularly at Cz.

Straub et al. (2015) conducted a similar study, examining 33 adults with ADHD compared to 35 matched controls in an eyes closed resting state. Analysis showed that the ADHD participants had significantly lower levels of arousal and less EEG vigilance as measured by EEG and the vigilance algorithm, compared to the controls. This was seen as a predictor of ADHD, as hyperactivity and sensation seeking is due to an unstable regulation of brain arousal.

The typical EEG pattern seen in ADHD is an excess of slow theta wave activity in the frontal regions of the brain (Monastra, 2008; Chabot et al., 2005; Chabot & Serfontein, 1996; Barry et al., 2003; Fonseca et al., 2008; Cantor & Chabot, 2009; Bresnahan & Barry, 2002; Clarke et al., 1998). In typically developed children, brain activity changes and develops with age; a process known as maturation. Specifically, as a child gets older, there is a decrease in slow wave theta levels and an increase in beta levels (Bresnahan & Barry, 2002). The pattern seen in individuals with ADHD is similar to that seen in a child during development, indicating possible developmental delay in the maturation process in an individual with ADHD. The research in adults with ADHD discussed here (Bresnahan & Barry, 2002; Struab et al., 2015) suggests that brain activity in ADHD populations do change with age, however they do not catch up with their counterparts.

5.1.3 ADHD Subtypes and EEG Findings

As previously discussed, according to the current DSM-5, there are three diagnostic subtypes of ADHD: ADHD inattentive, ADHD hyperactive impulsive and ADHD combined subtype (APA, 2013).

Research has found subtle differences in EEG abnormalities across the different ADHD subtypes. Studies focusing on ADHD inattentive subtype concluded that their EEGs are characterised by generalised high levels of theta, particularly high in frontal regions, and generalised low levels of beta across the brain (Chabot et al., 2005). Barry et al. (2003) agreed with such findings, with a meta-analysis of EEG and ADHD research showing an increase in absolute and relative theta in the frontal regions in children with ADHD inattentive subtype. Dupuy, Clarke, Barry, McCarthy, and Selikowitz (2010) found increased intrahemispheric beta coherence; higher levels of beta connection between structures underlying the pair of recording electrodes within

one hemisphere, specific to the combined subtype of ADHD. This sample consisted of males aged between 8 to 12 years old diagnosed with ADHD and a control group, completing an EEG resting eyes closed measure.

However, findings by Clarke et al. (1998) disagreed with this. Clarke et al. (1998) conducted EEG in a resting eyes closed state using 21 electrodes with 60 children aged 8 to 12 years old. Twenty participants were diagnosed ADHD combined subtype, 20 were ADHD inattentive subtype and 20 were a control. ADHD combined participants had greater levels of absolute and relative theta over all regions. This was also the case in the ADHD inattentive subtype sample but less severe than ADHD combined subtype. Barry et al. (2002) found similar results. EEG was conducted in an eyes closed resting state on children aged 8 to 12 years old, 40 diagnosed ADHD combined subtype, 40 diagnosed ADHD inattentive subtype and 40 controls. Findings showed individuals with ADHD inattentive and combined subtypes had the same abnormalities as each other, specifically high levels of interhemispheric coherences for delta and theta bands, but with the inattentive subtype being less deviant.

5.1.4 EEG and Neurofeedback in ADHD

Evidence suggests that using neurofeedback as a strategy for individuals diagnosed with ADHD can normalise EEG patterns as well as reduce inattentive and impulsive symptoms as a long-term strategy (Vernon et al., 2004). However, there are very few studies that have looked at changes in EEG oscillations after neurofeedback training (Lansbergen, Arns, Dongen-Boomsma, Spronk, & Buitelaar, 2011). Monastra et al. (2002) examined EEG changes after neurofeedback in an ADHD population. Here, 100 children aged 6 to 19 years old with a diagnosis of ADHD, underwent neuropsychological and EEG measures. EEG was completed while participants underwent a performance task, such as reading, listening, drawing and recorded

activity between 4 to 8Hz and 13 to 21Hz. Fifty participants received EEG neurofeedback downtraining theta and uptraining beta at Cz and Fz, with an average of 43 sessions. Measures were completed a year after the initial measures. Results showed a significant reduction in cortical slowing, which was only found in participants who received neurofeedback.

A neurofeedback RCT study has been conducted examining the effects of neurofeedback and methylphenidate. One hundred and twelve children with a diagnosis of ADHD ranging in age from 7 to 13 were involved. The three conditions consisted of neurofeedback, 30 sessions of theta/beta training at Cz for 10 weeks, physical activity semi-active control group, and methylphenidate. Pre to post EEG findings showed similar reduction in theta activity for individuals who received neurofeedback and methylphenidate. However, individuals who received neurofeedback, with a theta/beta training protocol, showed greater overall reductions in ADHD symptoms as measured by rating scales. Despite this success, improvement was not generalised to classroom behaviours (Janssen et al., 2016).

Although there is evidence on neuropsychological measures that neurofeedback improves ADHD symptoms (Carmody et al., 2001; Fuchs et al., 2003; Kaiser & Othmer, 2000; Rossiter, 2004; Rossiter & La Vaque, 1995), very little is known as to how EEG is affected. Further research is required to understand the full affect neurofeedback has on underlying EEG in an ADHD population. This is a vast area that needs to be examined further, an issue that aims to be addressed in this research by conducting EEGs prior and post neurofeedback treatment.

5.1.5 EEG and Stimulant Medication

There are several studies that have examined the effect of stimulant medication, in the treatment of ADHD, has on EEG. Clarke et al. (2002) completed pre and post

eyes closed resting state EEG of 20 males with ADHD inattentive subtype and 20 controls, aged 8 to 13 years old. An EEG recording was conducted following a 6-month stimulant medication trial. Findings showed that medication produced changes in EEG towards normalization, with reduction in absolute and relative theta, theta/alpha and theta/beta ratios and increase in relative alpha and beta. Similarly, Chabot, Orgil, Crawford, Harris, and Serfontein (1999) conducted an eyes closed resting EEG during an initial assessment, and repeated 10 months later following stimulant medication treatment which the child had taken on the day of repeat testing. This was conducted in a sample of 130 ADHD diagnosed children, aged 6 to 16 years old. Results showed that 56.9% of the children had normalized EEG at the repeat testing, 33.8% were unchanged, and 9.3% had an increase in EEG abnormality.

Clarke et al. (2005) completed an eyes closed resting state EEG, on 40 males aged 8 to 13 years old, 20 diagnosed with ADHD combined subtype. The EEG was repeated after a 6-month medication trial and 1 hour after medication had been taken, but failed to identify any changes in coherence due to stimulant medication. Dupuy et al. (2008) conducted a similar study in females, 20 females diagnosed ADHD combined subtype, 20 females diagnosed ADHD inattentive subtype and 20 control, all aged between 7 and 12 years old. EEG eyes closed resting state was recorded pre and post a medication trial. Again, there was no significant difference on coherence between the two EEGs in the ADHD conditions.

As recently discussed, although there is little research, it is shown that neurofeedback can normalise EEG in ADHD (Vernon et al., 2004), and that stimulant medication in isolation can also normalise EEG in ADHD. However, it is not known what effect neurofeedback and stimulant medication have in combination on EEG in ADHD. Information regarding this interaction on the brain may aid in understanding

the root cause of ADHD, as well as the best combination of treatments to normalise abnormal brainwaves seen in ADHD. This is an area that the research presented in this thesis aims to examine.

5.2 Aims of the Study

The present study firstly aims to examine EEG differences between an ADHD and typically developed sample.

Secondly, the study aims to examine the effect of neurofeedback home training on EEG, in both an ADHD and typically developed sample. This is an area that has not been previously investigated (Vernon et al., 2004; Rutterford et al., 2008). As seen in the literature review, although there have been studies which have examined the effect of neurofeedback on ADHD symptoms (Lubar & Shouse, 1976; Arns et al., 2014; Arns et al., 2009; Carmody et al., 2001; Fuchs et al., 2003; Kaiser & Othmer, 2000; Rossiter, 2004), there are few studies which have examined the effect on brainwaves (Vernon et al., 2004; Lansbergen et al., 2011, Monastra et al., 2002).

Thirdly, the study aims to examine the effect of stimulant medication in combination with neurofeedback home training on EEG. It has been shown that neurofeedback can normalise EEG in ADHD (Vernon et al., 2004), and that stimulant medication in isolation can also normalise EEG in ADHD (Clarke et al., 2002; Chabot et al., 1999). However, it is not known what effect neurofeedback and stimulant medication have in combination on EEG in ADHD. Information regarding this interaction on the brain may aid an understanding of the root cause of ADHD, as well as the best combination of treatments to normalise abnormal brainwaves seen in ADHD.

5.3 Hypothesis

Based on the previous research and the aims of the present study, the hypotheses for the pre-measures are as follows:

- The ADHD sample will show more slow brain waves, specifically theta, SMR and alpha, across C3, Cz, and C4 than the typically developed sample.

Based upon the previous research and the aims of the present study, the hypotheses for the post-measures are as follows:

- The ADHD sample neurofeedback home training and medication condition will show the greatest increase in theta, alpha and SMR activity, across the two samples.
- Across the typically developed sample, the neurofeedback home training condition will see the greatest change in theta, alpha and SMR activity.
- No change will be seen in theta, SMR or alpha waves in the control or active control conditions in the typically developed sample.

5.4 Methods

The present study followed the general methods previously set out in this thesis. For details regarding participants and interventions, see Chapter Two: General Methods.

5.4.1 EEG Measures

The EEG was acquired with a sampling rate of 500Hz and amplified before being converted into a digital format, using the Deymed TruScan system including TruScan Acquisition software. The EEG was recorded from 19 electrode sites (FP1, FP2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T3, T4, T5, T6, Fz, Cz, Pz) in line with the International 10-20 System using an electrode cap and fixed in place by an elastic strap around the participant's chest. The prefrontal electrodes, namely FP1 and FP2, were additionally secured with disposable sponge disks. Approximately 30 minutes

was spent fitting the electrodes with gel (which conducts electricity from the scalp to the electrode) being injected through the electrodes with a blunt syringe. The EEG referenced to linked ears, using an additional electrode on the left and right mastoid. Impedance readings were kept below 5 kΩ.

The EEG was recorded in two resting conditions: eyes open and eyes closed. Each condition was replicated 5 times in an alternating way, each of them lasting 1 minute. The EEG recording was completed while resting as it is a reliable measure that differentiates between ADHD and typically developed individuals as well as sensitive to any changes (Barry et al., 2003).

This process was completed on 2 occasions with all participants, once at the beginning prior to any intervention, and again 15 weeks later after an intervention had taken place.

5.5 Analysis

Power EEG data from the eyes closed resting condition was analysed using Brain Vision Analyser. Unfortunately, data collection did not include the use of electrooculogram (EOG), electrical noise generated by eye movement. Due to this, analysis was unable to remove EOG artefact from the eyes open data, and therefore was unable to analyse this data. This was overcome by using eyes closed data.

To convert the oscillatory activity in the EEG into a measured form, data was segmented into 300000ms bins. Artefact correction took place, then a Fast Fourier Transform (FFT) was conducted to transform the data from time to frequency domain to express the frequency of each brainwave.

5.6 Results

The EEG recording was completed at 2 time points, pre-measures before any intervention and post-measures after intervention, and specific bandwidths were

examined, alpha, SMR and theta. Results are presented looking at each bandwidth in turn, firstly at pre-measures, secondly at post-measures, and finally comparing pre and post measures.

5.6.1 Results from Alpha Bandwidths (8-12 Hz)

5.6.1.1 Alpha Pre Measures

Firstly, descriptive statistics are provided for the data obtained on alpha in both samples. The ADHD sample had a higher mean amplitude of alpha at Cz and C4 compared to the typically developed sample, and the typically developed sample had a higher mean amplitude of alpha at Cz than the ADHD sample.

Table 48.

Descriptive statistics of alpha pre-measures

		N	Mean	Std. Deviation	Std. Error Mean
Alpha C3	ADHD sample	39	6.65	6.82	1.09
	Typically developed sample	47	8.97	12.63	1.84
Alpha Cz	ADHD sample	39	7.37	7.92	1.27
	Typically developed sample	47	6.38	4.05	0.59
Alpha C4	ADHD sample	39	7.01	7.02	1.12
	Typically developed sample	47	6.33	3.53	0.52

Next, a multivariate ANOVA was conducted to examine any effect of diagnosis and gender of total alpha scores. However, neither gender F (3, 80) =0.0.3, p<0.82; Wilk's = 0.98, or diagnosis, F (3, 80) =0.85, p<0.46; Wilk's = 0.96, had a significant effect on alpha.

Then, a t-test was conducted to examine any significant difference of alpha between the two samples: ADHD and typically developed, but again differences were not significant; alpha C3 (ADHD (M = 6.64, SD = 6.82) and typically developed (M = 8.97, SD = 12.62) samples; t(84) = -1.03, p = 0.306), alpha Cz (ADHD (M =7 .38, SD = 7.91) and typically developed (M = 6.38, SD = 4.04) samples; t(84) = 0.74, p =

0.45), and C4 (ADHD (M = 7.008, SD = 7.02) and typically developed (M = 6.33, SD = 3.53) samples; t(84) = 0.57, p = 0.56).

5.6.1.2 Alpha Post Measures

Descriptive statistics are provided for the data obtained on alpha in both samples at the second data collection, post measures. The means were very similar across all electrode locations in both samples.

Table 49.

Descriptive statistics of alpha post-measures

		N	Mean	Std. Deviation	Std. Error Mean
Alpha C3	ADHD sample	32.00	6.19	2.14	0.38
	Typically developed sample	40.00	6.38	3.86	0.61
Alpha Cz	ADHD sample	32.00	6.75	3.42	0.61
	Typically developed sample	40.00	6.47	5.31	0.84
Alpha C4	ADHD sample	32.00	6.79	3.07	0.54
	Typically developed sample	40.00	6.57	3.75	0.59

Next, a multivariate ANOVA was conducted to examine any effect of diagnosis and gender of total alpha scores, but neither gender, F (3, 66) = 0.42, p<0.98; Wilk's = 0.99, or diagnosis, F (3, 66) = 0.19, p<0.89; Wilk's = 0.99, had a significant effect on alpha.

Then, a t-test was conducted to examine any significant difference of alpha between the two samples: ADHD and typically developed. Alpha at C3 showed no significant difference between the ADHD (M = 6.19, SD = 2.14) and typically developed (M = 6.38, SD = 3.86) samples; t(70) = -0.24, p = 0.09, there was no significant difference at electrode Cz (ADHD (M = 6.74, SD = 3.42) and typically developed (M = 6.46, SD = 5.31) samples; t(70) = 0.25, p = 0.38, and C4 (ADHD (M = 6.79, SD = 3.07) and typically developed (M = 6.57, SD = 3.74) samples; t(70) = 0.26, p = 0.28).

5.6.1.3 Alpha Comparison Pre to Post Measures

A paired t-test was conducted to examine any significant difference of alpha from pre to post measures across samples, but again differences were not significant (Alpha C3 pre (M = 8.57, SD = 11.409) and post (M = 6.27, SD = 3.22) measures; t(69) = 1.604, p = 0.11; alpha Cz pre (M = 7.24, SD = 6.66) and post (M = 6.59, SD = 5.82) measures; t(69) = 0.77, p = 0.43; alpha C4 pre (M = 7.02, SD = 5.82) and post (M = 6.67, SD = 3.49) measures; t(69) = 0.46, p = 0.64).

An absolute change score was calculated on mean scores across conditions in both samples, to see the extent on any change.

Table 50.

Absolute change scores for alpha on typically developed sample conditions

	Neurofeedback						Control						Active Control					
	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95%Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval
Alpha C3	6.14	6.4	0.29	0.06	-3.408	3.74	9.71	5.15	-4.56	0.32	-6.23	17.51	7.96	7.35	-0.61	0.11	-1.89	2.84
Alpha Cz	6.04	6.2	0.15	0.06	-3.44	3.56	5.12	4.85	-0.27	0.76	-2.17	2.89	8.54	8.06	-0.48	0.05	-2.84	3.35
Alpha C4	5.89	6.9	0.97	-0.11	-4.73	3.109	5.31	5.28	-0.03	0.82	-1.98	2.45	8.201	7.43	-0.7707	0.18	-1.34	2.58

In the typically developed sample, the largest absolute mean change was seen at alpha C3 in the control condition with a reduction in amplitude

Table 51.

Absolute change scores for alpha on ADHD sample conditions

	Medication						Medication and Neurofeedback Home Training						Medication and Neurofeedback Clinic						Neurofeedback Home Training					
	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval
Alpha C3	7.403	5.51	-1.893	0.2	-2.63	6.408	5.64	7.37	1.73	-0.61	-3.96	1.03	7.41	8.02	0.62	-0.12	-8.56	7.33	5.04	3.52	-1.52	-	-	-
Alpha Cz	7.46	5.92	-1.54	0.18	-2.51	5.58	5.99	9.504	3.514	0.84	-8.52	0.92	13.83	6.91	-6.92	0.41	-20.26	34.08	5.11	3.15	-1.96	-	-	-
Alpha C4	7.903	5.87	-2.033	0.21	-2.69		6.43	9.49	3.06	-0.55	-9.83	3.07	8.19	7.44	-0.75	-0.15	-7.05	8.54	4.49	3.32	-1.17	-	-	-

In the ADHD sample, the largest absolute mean change after intervention was seen at alpha Cz in the medication and neurofeedback in clinic condition with a reduction in alpha amplitude compared to the other ADHD conditions. The medication condition and neurofeedback home training condition saw a reduction in absolute mean change in alpha at all electrode locations, whereas the medication and neurofeedback home training condition saw an increase in absolute mean change in alpha at all electrode locations.

5.6.2 Results from Theta Bandwidths (4-7 Hz)

5.6.2.1 Theta Pre Measures

Firstly, descriptive statistics are provided for the data obtained on theta in both samples. The ADHD sample had a higher mean amplitude of theta at Cz and C4 compared to the typically developed sample, and the typically developed sample had a higher mean amplitude of alpha at Cz than the ADHD sample.

Table 52.

Descriptive statistics of theta pre-measures

		N	Mean	Std. Deviation	Std. Error Mean
Theta C3	ADHD sample	40.00	8.47	15.22	2.41
	Typically developed sample	47.00	9.49	12.82	1.87
Theta Cz	ADHD sample	40.00	9.02	13.89	2.20
	Typically developed sample	47.00	8.00	8.75	1.28
Theta C4	ADHD sample	40.00	8.74	15.01	2.37
	Typically developed sample	47.00	7.63	7.45	1.09

Next, a multivariate ANOVA was conducted to examine any effect of diagnosis and gender of total theta scores. Neither gender F (3, 81) = 0.16, p<0.91; Wilk's = 0.99, or diagnosis F (3, 81) = 0.69, p<0.5; Wilk's = 0.97, had a significant effect on theta.

Then, a t-test was conducted to examine any significant difference of theta between the two samples: ADHD and typically developed.

There was no significant difference of theta pre-measures between the ADHD and typically developed sample (theta C3 ADHD (M = 8.46, SD = 15.21) and typically developed (M = 9.49, SD = 12.82) samples; t(85) = -0.34, p = 0.73; theta Cz ADHD (M = 9.02, SD = 13.88) and typically developed (M = 7.99, SD = 8.75) samples; t(85) = 0.41, p = 0.67; and theta C4 ADHD (M = 8.73, SD = 15.01) and typically developed (M = 7.63, SD = 7.44) samples; t(85) = 0.44, p = 0.65).

5.6.2.2 Theta Post Measures

Descriptive statistics are provided for the data obtained on theta in both samples. The ADHD sample had a higher mean amplitude of theta at C4 than the typically developed sample, and the typically developed sample had a higher mean amplitude of theta at C3 and Cz than the ADHD sample.

Table 53.

Descriptive statistics of theta post-measures

		N	Mean	Std. Deviation	Std. Error Mean
Theta C3	ADHD sample	32.00	6.64	2.66	0.47
	Typically developed sample	40.00	6.84	3.91	0.62
Theta Cz	ADHD sample	32.00	7.72	4.78	0.84
	Typically developed sample	40.00	8.09	7.65	1.21
Theta C4	ADHD sample	32.00	7.19	4.56	0.81
	Typically developed sample	40.00	6.74	3.96	0.63

Next, a multivariate ANOVA was conducted to examine any effect of diagnosis and gender of total theta scores. Neither gender F (3, 66) = 2.22, p<0.88; Wilk's = 0.99, or diagnosis F (3, 66) = 0.407, p<0.74; Wilk's = 0.98, had a significant effect on theta.

Then, a t-test was conducted to examine any significant difference of theta between the two samples: ADHD and typically developed.

There was no significant difference of theta post-measures between the ADHD and typically developed sample on any of the electrodes; theta C3 ADHD (M = 6.64,

SD = 2.65) and typically developed (M = 6.83, SD = 3.91) samples; t(70) = -0.23, p = 0.81; theta Cz ADHD (M = 7.72, SD = 4.77) and typically developed (M = 8.09, SD = 7.65) samples; t(70) = -0.23, p = 0.81; and theta C4 ADHD (M = 7.18, SD = 4.56) and typically developed (M = 6.74, SD = 3.96) samples; t(70) = 0.44, p = 0.66).

5.6.2.3 Theta Comparison Pre to Post Measures

There was no significant difference of theta from pre to post measures (theta C3 pre (M = 9.97, SD = 15.33) and post (M = 6.76, SD = 3.42) measures; t(69) = 1.69, p = 0.09; theta Cz pre (M = 9.28, SD = 12.504) and post (M = 7.97, SD = 6.56) measures; t(69) = 0.75, p = 0.45; theta C4 pre (M = 8.88, SD = 12.69) and post (M = 6.95, SD = 4.26) measures; t(69) = 1.207, p = 0.23).

Next, a between subjects repeated multivariate ANOVA was conducted to examine any effect of diagnosis and gender of total theta scores across samples. Neither gender F (2, 65) = 0.22, p<0.79; Wilk's = 0.99, or diagnosis, F (2, 65) = 0.04, p<0.95; Wilk's = 0.99, had a significant effect on theta.

An absolute change score was calculated on mean scores across conditions in both samples, to see the extent on any change.

Table 54.

Absolute change scores for theta on typically developed sample conditions

	Neurofeedback						Control						Active Control					
	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval
Theta C3	8.24	8.12	-0.12	0.11	-6.61	8.91	5.65	6.43	0.78	-0.15	-3.13	1.91	11.55	6.15	-5.4	0.37	-2.93	13.47
Theta Cz	8.26	9.98	1.72	-0.05	-11.6	9.85	5.62	6.29	0.67	-0.15	-2.77	1.71	11.42	8.23	-3.19	0.17	-6.91	12.63
Theta C4	8.24	7.81	-0.43	0.11	-6.62	9.19	5.28	6.09	0.81	-0.17	-3.11	1.81	10.66	6.43	-4.23	0.37	-2.38	10.58

In the typically developed sample, the largest absolute mean change was seen at theta in the active control condition with a reduction in amplitude at C3, Cz and C4. On the other hand, the control condition saw an increase of theta at all three electrode locations.

Table 55.

Absolute change scores for theta on ADHD sample conditions

	Medication						Medication and Neurofeedback Home Training						Medication and Neurofeedback Clinic						Neurofeedback Home Training					
	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval
Theta C3	5.42	6.29	0.87	-0.41	-1.89	0.14	6.44	7.82	1.38	-0.61	-2.81	0.73	32.8	6.98	-25.8	0.58	-45.5	97.2	5.33	3.48	-1.85	-	-	-
Theta Cz	5.71	7.11	1.39	-0.6	-2.49	-0.28	6.73	10.7	3.95	-0.55	-12.11	3.82	36.5	6.71	-29.8	0.81	-28.5	88.1	6.23	2.92	-3.31	-	-	-
Theta C4	5.83	6.39	0.56	-0.21	-1.79	0.68	7.51	9.66	2.16	-0.31	-9.78	5.42	32.3	7.49	-24.8	0.54	-47.8	97.4	5.32	3.47	-1.85	-	-	-

In the ADHD sample, the largest absolute mean change was seen at theta in the medication and neurofeedback clinic condition.

5.6.3 Results from SMR Bandwidths (12-15 Hz)

5.6.3.1 SMR Pre Measures

Descriptive statistics are provided for the data obtained on SMR in both samples. The typically developed sample had a higher amplitude of SMR at all three electrode locations.

Table 56.

Descriptive statistics of SMR pre-measures

		N	Mean	Std. Deviation	Std. Error Mean
SMR C3	ADHD sample	40	2.49	4.29	0.68
	Typically developed sample	47	3.62	4.93	0.72
SMR Cz	ADHD sample	40	2.19	2.84	0.45
	Typically developed sample	47	3.08	4.02	0.59
SMR C4	ADHD sample	40	2.43	4.33	0.68
	Typically developed sample	47	2.82	3.25	0.47

Next, a multivariate ANOVA was conducted to examine any effect of diagnosis and gender of total SMR scores. Neither gender F (3, 81) = 0.49, p<0.68; Wilk's = 0.98, or diagnosis F (3, 81) = 1.24, p<0.31; Wilk's = 0.95, had a significant effect on SMR.

Then, a t-test was conducted to examine any significant difference of SMR between the two samples: ADHD and typically developed. There was no significant difference of SMR pre-measures between the ADHD and typically developed sample (SMR C3 ADHD (M = 2.49, SD = 4.29) and typically developed (M = 3.62, SD = 4.92) samples; t(85) = -1.13, p = 0.26; SMR Cz ADHD (M = 2.18, SD = 2.83) and typically developed (M = 3.08, SD = 4.02) samples; t(85) = -1.17, p = 0.24; SMR C4 ADHD (M

= 2.43, SD = 4.32) and typically developed (M = 2.82, SD = 3.25) samples; t(85) = -0.47, p = 0.63).

5.6.3.2 SMR Post Measures

Descriptive statistics are provided for the data obtained on SMR in both samples. The typically developed sample had a higher amplitude of SMR at all three electrode locations.

Table 57.

Descriptive statistics of SMR post-measures

		N	Mean	Std. Deviation	Std. Error Mean
SMR C3	ADHD sample	32	1.960	0.797	0.141
	Typically developed sample	40	2.03	1.21	0.19
SMR Cz	ADHD sample	32	2.00	1.02	0.18
	Typically developed sample	40	2.24	1.93	0.31
SMR C4	ADHD sample	32	2.05	1.31	0.23
	Typically developed sample	40	2.23	1.15	0.18

Next, a multivariate ANOVA was conducted to examine any effect of diagnosis and gender of total SMR scores. Neither gender F (3, 66) = 0.24, p<0.86; Wilk's = 0.98, or diagnosis F (3, 66) = 0.53, p<0.66; Wilk's = 0.97, had a significant effect on SMR.

Then, a t-test was conducted to examine any significant difference of SMR between the two samples: ADHD and typically developed. There was no significant difference of SMR post-measures between the ADHD and typically developed sample (SMR C3 ADHD (M = 1,96, SD = 0.76) and typically developed (M = 2.02, SD = 1.207) samples; t(70) = -0.26, p = 0.79; SMR Cz ADHD (M = 2.003, SD = 1.01) and typically developed (M = 2.23, SD = 1.93) samples; t(70) = -0,62, p = 0.53; SMR C4 ADHD (M = 2.05, SD = 1.31) and typically developed (M = 2.23, SD = 1.14) samples; t(70) = -0.62, p = 0.53).

5.6.3.3 SMR Comparison Pre to Post Measures

There was a significant difference of SMR from pre to post measures on SMR C3 (SMR C3 pre (M = 3.37, SD = 5.15) and post (M = 2.02, SD = 1.04) measures; t(69) = 2.16, p = 0.03) although this was not significant after Bonferroni correction (calculation of 0.05/3 = *p* value of 0.016). Consequently, this was replicated in the separate samples to see where the significance lay.

The typically developed showed significant change in SMR at electrode C3 from pre to post measures (SMR C3 pre (M = 3.93, SD = 5.36) and post (M = 2.05, SD = 1.209) measures; t(38) = 2.13, p = 0.04) although this was not significant after Bonferroni correction (calculation of 0.05/3 = *p* value of 0.016). .

Next, a between subjects repeated multivariate ANOVA was conducted to examine any effect of diagnosis and gender of total SMR scores across samples. Neither gender F (2, 65) = 0.503, p<0.607; Wilk's = 0.98, or diagnosis, F (2, 65) =0 .88, p<0.42; Wilk's = 0.97, had a significant effect on SMR.

An absolute change score was calculated on mean scores across conditions in both samples, to see the extent on any change.

Table 58.

Absolute change scores for SMR on typically developed sample conditions

	Neurofeedback						Control						Active Control					
	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval
SMR C3	2.72	2.27	-0.45	0.27	-0.68	1.73	3.38	1.54	-1.84	0.37	-1.55	5.89	3.91	2.22	-1.69	0.29	-1.59	4.78
SMR Cz	2.56	2.33	-0.23	0.07	-1.04	1.39	2.005	1.72	-0.285	0.22	-0.602	1.24	5.22	2.64	-2.58	0.42	-0.93	5.87
SMR C4	2.69	2.59	-0.1	0.05	-1.09	1.37	1.97	1.98	-0.01	0.01	-0.77	0.809	4.19	2.19	-2.00	0.34	-1.33	5.18

In the typically developed sample, the largest absolute mean change was seen at SMR Cz and C4 in the active control condition with a reduction in amplitude.

Table 59.

Absolute change scores for SMR on ADHD sample conditions

	Medication						Medication and Neurofeedback Home Training						Medication and Neurofeedback Clinic						Neurofeedback Home Training					
	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval	Pre Mean	Post Mean	Absolute Change Score	Cohen's d	95% Lower Confidence Interval	95% Upper Confidence Interval
SMR C3	3.09	1.6	-1.46	0.24	-1.49	4.406	1.6	2.4	0.79	-0.88	-2.23	0.19	2.41	2.4	0.01	0	-3.53	3.52	1.9	1.23	-0.7	-	-	-
SMR Cz	2.29	1.7	-0.62	0.18	-1.04	2.28	1.5	2.76	1.22	-1.43	-2.49	-0.38	3.59	1.8	-1.8	0.36	-6.02	9.57	1.6	1.01	-0.6	-	-	-
SMR C4	3.04	1.6	-1.47	0.23	-1.54	4.501	1.8	3.19	1.42	-0.87	-3.78	0.34	2.47	2.1	-0.4	0.18	-3.24	4.08	1.4	0.97	-0.4	-	-	-

In the ADHD sample, the largest absolute mean change was seen at SMR C3 and C4 in the medication condition with a reduction in amplitude, and SMR Cz in the medication and neurofeedback in clinic condition.

5.7 Discussion

The present study investigated the difference in EEG profiles, specifically alpha, theta and SMR activity over the central motor strip at C3, C4 and Cz, between typically developed children and children with a diagnosis of ADHD. Comparisons between the two samples took place pre and post interventions. We shall look at the main findings from both prior and post interventions in turn.

5.7.1 Discussion Regarding Pre Measures

Based upon previous research, it was hypothesised that there would be a greater amount of alpha, theta and SMR waves, across C3, Cz and C4 in the ADHD sample (Barry et al., 2003; Fonseca et al., 2008). Findings from this research showed no significant difference between alpha, theta or SMR across these electrodes between the typically developed and ADHD sample, before interventions took place.

Unfortunately, due to a design flaw, the present study was unable to use eyes open data as EOG data were not collected and therefore eye blinks were unable to be screened for.

The research presented in this thesis did not find an effect of gender in either sample or EEG bandwidths. This was contrary to Matthis et al. (1980), who found healthy girls had higher levels of relative theta power and lower levels of fast alpha frequencies at the age of 6, compared to boys. However, by age 11, girls had surpassed boys in alpha in occipital regions but deficiencies in alpha frontal regions. The reason for lack of evidence in this research could be due to the small sample size, particularly a small sample of females in the ADHD sample (typically developed sample were 68% male, 32% female, whereas the ADHD sample were 85% male and 15% female).

One possible reason for the lack of significant difference on the EEG measures could be due to the self-selecting nature of participants. Specifically, based upon the

researcher's clinical experience, it is suggested that the ADHD population may not have been a typical sample due to recruitment taking place from a private clinic setting. The clinic was based in a wealthy area and participants had to pay to access the Centre, consequently being in a financial position to afford assessment. Furthermore, individuals accessing the Centre were typically previously turned away from the National Health Service due to not meeting their criteria level. This may account for there not being a significant difference between the two samples as expected.

5.7.2 Discussion Regarding Post Measures

Based upon previous research regarding the use of neurofeedback, it was hypothesised that the ADHD sample would show the greatest differences on EEG results, particularly across the C3, Cz, and C4 electrodes, compared to the typically developed sample (Egner & Gruzelier, 2001; Vernon et al., 2003; Vernon, 2005; Fritson et al., 2007; Carmody et al., 2001; Fuchs et al., 2003; Kaiser & Othmer, 2000; Rossiter, 2004; Rossiter & La Vaque, 1995). It was unexpected to find that there were no significant difference on alpha, theta and SMR bandwidths between the two samples after completion of the interventions.

A possible reason for the lack of significant difference is that in the ADHD conditions, there was a small sample per condition that may have skewed the results. The small sample size was due to recruitment difficulties, specifically in the neurofeedback conditions. With regard to the neurofeedback home training conditions, the research had limited access to neurofeedback equipment only enabling 3 neurofeedback participants at any one time. Participation in the research was a long process with each participant completing the intervention at home for almost 4 months. These two difficulties combined meant that, particularly for the neurofeedback home training conditions, recruitment was limited. Additionally, recruitment for the

neurofeedback in clinic condition was poor as individuals needed to live in close proximity to the Centre to access neurofeedback. This limited the target population.

Alternatively, it may be that the interventions, specifically neurofeedback, did not have the impact on the brainwaves as was expected (Egner & Gruzelier, 2001; Vernon et al., 2003).

The typically developed sample saw the largest amount of absolute change mean scores in the SMR bandwidth when comparing pre to post measures. This was expected, as the typically developed sample received SMR uptraining in the neurofeedback home training condition. However, the largest absolute change mean scores were in the active control condition, not the neurofeedback home training condition. Furthermore, the active control group saw the largest amount of absolute change scores across alpha, theta and SMR. Specifically, the active control condition saw a decrease in all theta readings as well as alpha at Cz, SMR at Cz and C4. This suggests that sitting and focusing on a computer-based programme can alter brainwaves, specifically reduce slow brainwave activity. Computer use is a growing area due to the vast development of technology and its use in our everyday lives. Literature shows that video gaming can alter brainwave activity as well as attention skills. Specifically, a more complex video game that is played, the faster brainwaves are produced to aid in extra concentration to successfully complete the game (Bakaoukas et al., 2015). It has been suggested that video gaming can alter neural plasticity because video games require an individual to process complex events in a specific sequence as well as to respond quickly and rapidly (Gong et al., 2016). The findings in this research supports the ever-changing neural plasticity of children and how brain activity can be altered by participating in a computer-based activity. Future

research involving gaming should include some form of brain activity measures, to understand the impact it is having on the brain.

In regard to the ADHD sample, most absolute mean change scores on EEG measures were seen in the medication and neurofeedback in clinic condition, although this was not statistically significant. As an outcome of the combined interventions, alpha at Cz and SMR at Cz were within the same range as the typically developed sample. This trend suggests that neurofeedback may alter brainwave activity. However, this was only achieved when neurofeedback took place in clinic and in conjunction with stimulant medication, suggesting the role of the clinician in the neurofeedback process may impact on the success of the intervention. The role of the clinician in neurofeedback has not been previously considered and is an area that requires further investigation (Vernon et al., 2004; Rutterford et al., 2008). On the other end of the scale, the condition with the least amount of changes was the neurofeedback home training condition. Again, this supports that the role of the clinician is important in administering neurofeedback but also the possibility that the home may not be a very controlled environment to conduct such treatment.

Previous research of neurofeedback in an ADHD child population showed a reduction in cortical slowing following downtraining theta and increasing beta at Cz and Fz (Monastra et al., 2002). The present study found similar results, with the neurofeedback home training condition showing the largest trend of reduction in theta compared to neurofeedback home training and medication.

When considering the effect of medication on the brain, Clarke et al. (2002) found that medication produced changes in EEG towards normalization, with reduction in absolute and relative theta. The present results did not support this, with the

stimulant medication only condition showing an increase in theta and a decrease in alpha.

Furthermore, when examining neurofeedback compared to medication, previous EEG findings showed similar reduction in theta activity for both conditions, with the neurofeedback condition showing greater overall reductions in ADHD symptoms as measured by rating scales. However, the present study was unable to replicate these findings. Specifically, medication saw an increase in theta, and neurofeedback home training having a decrease in theta, with the latter finding similar to that of Janssen et al. (2016).

Both of these findings are unexpected. The reasons for these results are unclear, whether it is due to small sample size or the nature of the samples. As previously discussed, the typically developed sample may not have been a true typical sample due to self-selecting participation. Although a lack of diagnosis was screened for, possible undiagnosed difficulties were not. Similarly, the ADHD sample were recruited from a private Centre. The Centre was based in a wealthy area and participants had to pay to access the Centre, consequently being in a financial position to afford assessment. Furthermore, individuals accessing the Centre were typically previously turned away from the National Health Service due to not meeting their criteria level. This may account for there not being a significant difference between the two samples as expected. Furthermore, the lack of significant findings could be Type II error due to small sample sizes. Additionally, as the present study is a feasibility study with small samples, findings are treated as tentative for implications, and need to be replicated in a larger sample.

In summary, the results from the electroencephalogram and neurofeedback home training study revealed no significant effect in either ADHD or typically developed

sample. The ADHD neurofeedback home training condition saw the least EEG changes and the medication only and medication combined with neurofeedback in clinic saw the largest improvements in EEG.

6 General Discussion

This thesis examined the effectiveness of neurofeedback home training on concentration and impulsivity difficulties in children. Data were collected from a typical and ADHD child population. Analysis examined differences between the two populations as well as the effect of neurofeedback home training on EEG measures, personality, concentration, and impulsivity as measured by neuropsychological measures.

The main findings were:

Pre measures:

- The ADHD sample were significantly affected on all Conners' 3 Parent Rating subscales compared to the typically developed sample.
- The ADHD sample were significantly more impaired on CPT preservations than the typically developed sample, although not significant after Bonferroni correction.
- There were no significant differences on personality or EEG measures between the 2 samples.

Post measures:

- Within the typically developed sample, the active control condition had the most amount of absolute mean change, with an increase in SMR at C4 and C3, alpha at Cz and theta at Cz, C3 and C4, as well as an increase in BIS, BAS fun and BAS reward but was not statistically significant.
- The typically developed neurofeedback home training condition saw the most improvement in the Conners' 3 Parent Rating Scale with a reduction in symptoms, specifically on learning difficulties, defiance, inattention and hyperactive/impulsivity, but teachers saw an increase in

difficulties, specifically executive functions, defiance, inattention and hyperactive/impulsive but was not statistically significant.

- Within the ADHD sample, the medication condition had a statistically significant improvement in the executive function, defiance, inattention and hyperactive/impulsive subscales of the Conners' 3 Parent Rating Scale.
- The ADHD medication and neurofeedback in clinic condition saw the most improvement on EEG measures, including theta at Cz, C3 and C4, alpha at Cz and SMR at Cz but was not statistically significant.
- In the ADHD sample, the neurofeedback home training condition had the largest change on personality, with an increase in BIS and decrease in BAS drive but was not statistically significant.
- The ADHD neurofeedback home training and medication condition saw the most change with an improvement on CPT, including commission, hit response time, and preservations but was not statistically significant.

These findings are discussed below. This chapter considers the findings in relation to theories of ADHD. Finally, the implications of this research for clinical applications and future research are outlined.

6.1 Summary of Experimental Findings

6.1.1 Summary of experimental findings comparing ADHD and typically developed samples

Prior to interventions being conducted, comparisons took place between the two populations and the way they present on neuropsychological, personality and brainwave measures.

The present study found that the ADHD sample were significantly affected on all Conners' 3 Parent Rating subscales, 4 of the Conners' 3 Teacher Rating subscales, as well as more impaired on CPT hit response time standard error and preservations, compared to the typically developed sample. Significant impairment on parental and teacher ratings replicates previous research, including Biedermann et al., 2001; Snyder et al., 2008; Conners, 2008; Tripp et al., 2006. This research also found a significant difference, although not significant after Bonferroni correction, between the ADHD and typically developed sample on the CPT preservations scores, showing impulsivity. Although significant difference on omission and commissions subscales was not found as expected (Gianarris et al., 2001; Epstein et al., 2003; Epstein et al., 2010; Advokat et al., 2007), the ADHD sample did show more impairment on these scales and showed inattention and impulsive abnormalities on other scales. Combining the results from the parental and teacher rating scales, and from the CPT, gives results that support previous theories of ADHD. Executive functions, inattention, hyperactivity and impulsivity were significantly different in the ADHD sample compared to the typically developed sample when rated by parents, teachers and on CPT subscales. This supports Barkley's Behavioural Response Inhibition Theory, where ADHD is explained as a deficit in a specific area of executive function, namely inhibition response, which overlaps with inattention and impulsivity (Barkley, 1997). All of these areas, namely executive functions, inattention and impulsivity, were significantly different between ADHD and typically developed sample when rated by parents, teachers and CPT subscales, in this research. Similarly, the evidence in this thesis supports Brown's ADD syndrome model, whereby ADD is explained by deficits in all 6 areas of executive functions, which includes focus and effort (Brown, 2006).

Contrary to the parental and teacher results which showed significant differences, the personality and EEG measures between the two samples showed differences, but they were not significant. This was a surprising result, with previous literature showing that individuals with ADHD have higher levels of slow brainwave activity, specifically theta (Chabot & Serfontein, 1996; Fonseca et al., 2008; Bresnahan & Barry, 2002) and an overactive BAS, consequently seeking rewards and positive emotions, with low BIS showing an impaired inhibition to punishment (Barkley et al., 1990; Hundt et al., 2008; Gomez & Corr, 2010).

Carver and White (1994) explained the Behavioural Activation System uses dopaminergic pathways within the brain, particularly within the basal ganglia. As the current study and previous literature has shown, individuals with ADHD have increased BAS (Barkley et al., 1990; Hundt et al., 2008). ADHD is linked to abnormal levels of dopamine in the brain's neurotransmitters (Fuchs et al., 2003). Specifically, there is evidence that individuals with ADHD have a variation in the dopamine transporter gene, causing deficits in the dopamine cell response (Tripp & Wickens, 2009). Consequently, the present study supports Carver and White (1994) that individuals with ADHD have higher levels of BAS, and therefore suggests that dopamine contributes to the cause of ADHD. Furthermore, this suggests that dopamine pathways not only cause ADHD, but also effects our personality.

As discussed in previous chapters, one theory of ADHD is the developmental deviation model. Typical development of the Central Nervous System includes synapses within the brain either being formed or eliminated (Barry et al., 2002), changes which are measured by EEG (Demos, 2004). The developmental deviation model would suggest that the ADHD brain is unlikely to mature to the expected level. In this research, prior to any interventions taking place, there was no significant

difference on EEG measures between the two samples. Consequently, this does not support the developmental deviation model of ADHD or Burke and Edge (2013) who found evidence of abnormal EEG patterns at all ages. The reason for this is not clear, but could be due to the self-selecting nature of the participants. This is discussed further in the methodological issues and technical limitations section of this thesis. Alternatively, EEG measures in an ADHD and typically developed population may not be as different as previously expected.

6.1.2 Summary of experimental findings of the typically developed sample

Neurofeedback has been shown to have a positive effect on typically developed children (Egner & Gruzelier, 2001; Vernon et al., 2003; Vernon, 2005; Fritson et al., 2007). However, much of the evidence came from a clinical setting and overlooked the use of neurofeedback being conducted in the family home under the remote guidance of a therapist (Vernon et al., 2004; Rutterford et al., 2008).

Across all 3 studies within this research, attempts were made to establish whether neurofeedback home training has a positive effect on improving concentration and impulsivity in a typically developed sample. Specifically, study one examined the effect on personality, study two examined the effect on neuropsychological measures, specifically attention and impulsivity, and study three examined the effect on brainwaves.

The research did not find a statistically significant effect of neurofeedback home training or clinic training on concentration, impulsivity, personality or EEG. The neurofeedback home training condition saw an increase in symptoms when rated by teachers. Snyder et al., (2008) found that there was an informant bias and low agreement between parent and teacher ratings. As Snyder et al. (2008) suggests and this research confirms, rating scales are useful within a clinical setting to help determine if

ADHD is present or not, but needs to involve other elements of assessment. Furthermore, interpreting observer results via rating scales needs to be done with caution. Every individual will interpret behaviours differently depending on rater's familiarity with the behaviour and situation of the observed behaviour. For example, in a less restrictive and demanding environment such as at home, symptoms may not be as evident or troublesome (Barkley, 2003). This concept is supported by Cortese et al. (2016) and Sonuga-Barke et al. (2013) where significant effects of nonpharmacological treatments when rated by individuals not blinded to intervention, compared to blinded raters who showed non-significant effects. Specifically, possible parental bias due to subconscious bias by parents based on cognitive dissonance. The use of such subjective measures then become questionable in its use of assessment and monitoring progress, and therefore suggest the possibility of an independent observer who does not know the individual, may be more reliable.

This research found the active control group had the largest change on EEG, specifically increased SMR at C3 and C4, alpha at Cz and theta at Cz, C3 and C4, as well as the largest mean absolute change scores, although not statistically significant, on personality, with an increase in BIS, BAS fun and BAS reward. Research by Gong et al. (2015) shows that video gaming can alter brainwaves via neural plasticity due to gaming requiring the ability to process complex events in a specific sequence as well as to respond quickly and rapidly. The research presented here has produced a unique finding, comparing neurofeedback home training to a computer-based activity, with the trend in results suggesting that sitting and focusing on a computer-based programme can influence our personality, specifically seeking reward and positive feelings (Carver & White, 1994). Furthermore, completing a computer-based programme can alter brainwaves. Literature shows that video gaming can alter brainwave activity as well as

attention skills. Specifically, a more complex video game that is played, the faster brainwaves are produced to aid in extra concentration to successfully complete the game (Bakaoukas et al., 2015). Within today's society and the ever-growing use of modern technology, a computer-based treatment would be very beneficial and accessible to many individuals. Computer based treatment in the treatment of neurodevelopmental conditions is an area which needs greater understanding.

6.1.3 Summary of Experimental Findings of the ADHD Sample

Neurofeedback has been shown to have a positive effect on individuals with a diagnosis of ADHD, improving impulsivity and attention (Carmody et al., 2001; Fuchs et al., 2003; Kaiser & Othmer, 2000; Rossiter, 2004; Rossiter & La Vaque, 1995). However, much of the evidence took place in a clinical setting and overlooked the use of neurofeedback being conducted in the family home under the remote guidance of a therapist (Vernon et al., 2004; Rutterford et al., 2008). Similarly, medication is an established intervention in the treatment of ADHD (Barkley, 1997; Whalen et al., 1990; Schachar et al., 1997; Punja et al., 2013; Storebo et al., 2015; Pearson et al., 2004) but there are few studies examining the combination of neurofeedback and stimulant medication in the treatment of ADHD (Monastra et al., 2002; Monastra et al., 2004; Duric et al., 2012).

Across all 3 of the studies, attempts were made to establish whether neurofeedback home training has a positive effect on improving concentration and impulsivity. Specifically, study one examined the effect on personality, study two examined the effect on neuropsychological measures, specifically attention and impulsivity, and study three examined the effect on brainwaves.

Neurofeedback produced no statistically significant effects on personality, concentration and impulsivity, or EEG. The lack of significant findings could be Type

II error due to small sample sizes. Additionally, as the present study is a feasibility study with small samples, findings are treated as tentative for implications, and need to be replicated in a larger sample. Although there were few significant results within the present research, null results are important findings. Null findings can inform researchers of what should be examined differently in future research. Furthermore, the null results can inform policy and practice for implementing strategies (Miller-Halegoua, 2017). For example, the null results within this research would suggest that neurofeedback home training is not an effective strategy for treating childhood ADHD.

Previous research has shown potential negative side effects to neurofeedback, including inducing seizures (Vernon et al., 2004), irritability and moodiness (Monastra et al., 2002) with the present study showing neurofeedback home training potentially worsening concentration. This could support Cortese et al. (2016) where evidence failed to support the use of neurofeedback as a treatment for ADHD in addition to Sonuga-Barke et al. (2013) where significant treatment effects of neurofeedback were non-existent when participants were blinded to intervention.

6.2 Methodological Issues and Technical Limitations

The research in this thesis underwent many ethical reviews which ensured a very robust design; however, several methodological and technical limitations were experienced.

Neurofeedback home training is an up and coming technology, with, to the researcher's knowledge, this being the first study to examine its effect. Consequently, the technology is yet to be developed and refined to be sufficiently user friendly for someone who is not experienced in conducting neurofeedback. Because of this, problems were experienced by participants setting up and completing neurofeedback at home, creating a dropout rate of 10%. Difficulties included the software not being

compatible with Mac computers, electrodes not being applied correctly and consequently not giving a clear signal, and batteries not lasting. However, in many situations, these difficulties were overcome, through the loan of laptops and provision of new equipment and advice for the participants. It is hoped that with the development of technology and as the use of neurofeedback grows, the equipment will become more robust and user friendly for individuals who are not experienced in the field.

The research was initially comprised through ill advice regarding the collection of electrooculography (EOG) data, the omission of which only became evident at the analysis stage. In particular, this made eyes open data unusable as eye blinks and twitches could not be screened for. However, this was overcome by using eyes closed data only. For future research, it is recommended that the EEG is conducted with an EOG measure in place and eyes open data collected and analysed, then compared to eyes closed data.

During the final data collection phase of the research, the Centre from where ADHD participants were being recruited, closed its main Centre in Horsham with resultant redundancy of staff, including the researcher of this thesis; instead, patients were seen in the London and Manchester Centres. This made data collection for the final phase difficult despite permission being granted for remote access to the patient records after redundancy. Furthermore, data collection for the neurofeedback in clinic condition was no longer possible as there was no clinic for this to take place. Therefore, the data collection phase was ended sooner than anticipated, this being part of the reason why a larger sample size was not achieved.

The small ADHD sample size was due to recruitment difficulties. For the neurofeedback home training conditions, the research had limited access to neurofeedback equipment only enabling 3 neurofeedback home training participants at

any one time. Participation in the research was a long process with each participant completing the intervention at home for almost 4 months. Access to 3 home training kits and long participation meant that, particularly for the neurofeedback home training conditions, recruitment was limited. Additionally, recruitment for the neurofeedback in clinic condition was poor as individuals needed to live in close proximity to the Centre to access neurofeedback. This limited the target population and the number of appropriate participants.

As previously discussed, the typically developed sample may not have been a true typical sample due to self-selecting participation. Although a lack of diagnosis was screened for, possible undiagnosed difficulties were not. Similarly, the ADHD population may not have been a typical sample due to recruitment taking place from a private clinic setting in an affluent area with participants being in a financial position to pay to access the Centre. Furthermore, individuals accessing the Centre were typically previously turned away from the National Health Service due to not meeting their criteria threshold. This may have contributed to there not being a significant difference between the two samples as had been expected.

6.3 Implications for Future Research

It is acknowledged that some of the work in this thesis should be viewed with consideration of several limitations; it is hoped that future research would be able to address the highlighted issues.

Firstly, regarding the small sample size, particularly in the ADHD neurofeedback conditions; with an effect size of 0.50 and the usual recommended power of 0.80, the ideal sample size needed would have been 64 participants per condition. Unfortunately, after 3 years of data collection, the sample size was not achieved due to limited access to neurofeedback equipment, difficulties with the neurofeedback equipment and proximity

of participants to the Centre. Future research would need to address these difficulties to achieve a larger sample and provide more robust statistical data.

With regard to the typically developed sample; it is possible that the sample had not developed as typically as expected, either academically or socially. The present study did not examine this but assumed typical development based upon a lack of formal diagnosis. A recommendation for future studies would be to include a measure of intelligence to control the sample.

A further suggestion for future research is that a personality scale which is more child friendly, be used. For example, the wording of the BIS question "criticism or scolding hurts me quite a bit" may not be understood by seven-year olds. The fact that there was a significant effect of age on the BIS/BAS scale may be explained by the fact that the older children understood the questions and answered appropriately.

The present study was conducted as a feasibility study, with the aim to examine if successful implementation of neurofeedback home training is possible. Consequently, the present study showed that neurofeedback home training can be conducted within the home after participants have been provided with a demonstration and visual information. Furthermore, recruitment, although sample sizes, was possible through a private Centre. Therefore, to valid the findings of the present research, it would be beneficial to complete within a larger sample.

6.4 Conclusion

In conclusion, the work within this thesis expands on literature in regard to neurofeedback, specifically within the ADHD field, and investigates the effect of neurofeedback being conducted at home; to the researcher's knowledge, this is the first study to examine neurofeedback home training. The research presented within this thesis

has been an extension of an existing body of research focused on concentration and impulsivity in typical individuals and individuals with a diagnosis of ADHD.

The main results were that: (i) ADHD sample were significantly different to typically developed peers when rated by parents and on CPT, differences were found as expected on personality and EEG measures, but these were not significant, (ii) stimulant medication significantly improves executive function, defiance, inattention, hyperactive and impulsive traits when rated by parents in an ADHD population, (iii) neurofeedback in clinic and home training does not significantly effect concentration, impulsivity, personality or EEG in a ADHD or typically developed sample. In order to fully understand the use of neurofeedback home training, further research would benefit from replication of this model on a larger scale with the research's identified limitations being addressed.

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8 Appendix

Appendix A: Ethical process

17th January 2012	Application submitted to and reviewed by NRES Committee South East Coast Research Ethics Committee
31st January 2012	Received unfavourable opinion outcome letter. The committee asked for the following information/alterations: • An explanation of what type of clinic the study would be taking place in. • A definition of Neurofeedback. • Clarification of how the recruitment process will be randomised. • Clarification as to whether the NICE guidelines would be followed. • Separate consent forms for over 12 years old. • Details on the dosage of the treatment. • Clarification on the sample size of the study. • The word 'expected' in the second paragraph on the debrief sheet should be changed to 'may'. • Changes to the recruitment structure. It was considered that the randomised method needed to be changed, as the randomisation method proposed may be seen as biased. • Clarification of what Neurofeedback is in the participant information sheet. • The paediatricians involved in the study should be named and their involvement made more prominent.
5th April 2012	The following changes were made and application resubmitted and reviewed by NRS Committee South East Coast Research and Ethics Committee. • The recruitment structure was changed so that the intervention strategies would be randomly allocated to participants, rather than self selection.

	• Definition of neurofeedback was inserted in the Participant Information Sheet. • The involvement of Specialist Consultant Paediatrician was explained in more detail on the Participant Information Sheet. • The 2008 NICE guidelines were discussed in more detail and an explanation is given as to how diagnosis and management of individuals with ADHD is conducted at the Centre in line with these guidelines. • A more detailed description of the Learning Assessment and Neurocare Centre was provided. • A separate Research Participant Consent Form for 12 to 17 years old participants was included. • Information was inserted in the Participant Information Sheet to clarify what forms part of the patients assessment at the Centre, what information from the assessment would be used for research and what additional information would be collected for research purpose. • Information was inserted in the Participant Information Sheet regarding the risks and disadvantages of taking part.
12th April 2012	Received unfavourable opinion outcome letter. The committee asked for further information/alterations and made the following comments: • Clarity required on why an external independent peer review had not been sought for the study. • Concerned that the control group of participants would not be offered any treatment for 15 weeks if they agreed to take part. • Members commented that the paediatrician would not have the expertise to make a full assessment especially in mental health terms.

	• Concern that the submission indicated that the study drugs would not cause any harm and did not have any long-term effects, however, members did not agree with this. • Clarity on whether participants who were distressed or upset for any reason as a result of taking part would be referred back to their GPs. • Members commented that there was a small chance that the completed questionnaires may reveal problems that had not been picked up earlier which members felt needed to be addressed. • Information sheets were still quite difficult to understand especially for the target population. • It was pointed out that information sheets and consent forms for parents and teachers to take part in the study in their own right was also required in addition to the information sheet submitted. • Consent forms required for older children, assent forms for young children and consent forms for parent/guarding. • Members queried whether there was any particular reason why the study was not being registered on a national database (A50 of the REC form). • Queried whether the neurofeedback sessions would be done at home, and wanted to know, if there was outcome measure for this intervention. • Members queried whether the private patients had to pay for their treatment for 26 weeks.
17th April 2012	Meeting with UEA University's Research, Enterprise and Engagement office stating NHS ethical approval was no longer required.
June 2012	The following changes were made in response to the NHS ethics latest comments and application submitted to UEA FMH Ethics Committee. • External independent peer review completed.

	• The NICE guidelines do not recommend neurofeedback as a form of treatment for ADHD. However, there is a wealth of evidence that neurofeedback is an effect treatment for ADHD. • It was clarified that a complete assessment is completed at the Learning Assessment and Neurocare Centre by Consultant Paediatrician who adheres to the DSM-IV and NICE guidelines. The doctor is qualified and experienced to diagnosis and manage ADHD and other neurodevelopmental conditions. • A discussion on the long-term effects of stimulant medication was added to the Participant Information Sheet. • Information has been inserted in the Information Sheet to point out that there is the possibility that the participant and/or family will experience distress due to the sensitive issues that are being discussed in the questionnaire. • Responses to additional questionnaires conducted specifically for this research, after completing the initial assessment at the Learning Assessment and Neurocare Centre would be passed to the Specialist Consultant Paediatrician. This will ensure that if any further concerns are discovered, the correct specialist would be aware and able to take appropriate action. • Child/participants will be provided with a written information sheet and assent form to complete. Parents will also have an information sheet and consent form to complete. A teachers consent form was devised. • The child/participant will be provided with their own information sheet and assent form to understand and complete. • The research will be registered on a national database via journals.

	• The outcome measure for neurofeedback home training would be the post measures QEEG, Conners' rating scales for parents and teachers and the CPT. • The conflict of interests would be overcome by a second supervisor on the supervisor team who is completely independent of the Learning Assessment and Neurocare Centre. • Participants would be required to pay the cost of their treatment at the Learning Assessment and Neurocare. They would not be paying any additional fees to take part in this research.
27th September 2012	Reviewed by UEA Faculty Research Ethics Committee
October 2012	Received unfavourable outcome letters. Issues included: • Concern regarding the fourth, control condition. The committee felt that the condition should be dropped. • Concern regarding the information sheets. Nature of neurofeedback and exactly what participants are consenting to needs to be clear. • Need clear consent from the owners of the clinic. • The ethics committee stated that ideally NRES approval could be gained. • Altering the design of the project.
12th December 2012	Following changes were made and application resubmitted to UEA FMH Ethics Committee (however, the review was delayed due to administrative reasons). • The control arm of the study was removed. • The information sheets were re-written. Diagrams were inserted to help illustrate the procedure. • UEA advised that NRES approval was not required.

	• Participants would only be asked to take part if both treatments involved in this study, neurofeedback and stimulant medication, were offered to them as part of their standard treatment as deemed appropriate by Consultant Paediatrician at the Learning Assessment and Neurocare Centre. If a participant decided to take part, they would randomly allocated by the researcher to a treatment condition. • Two further conditions have been added to the project; neurofeedback clinic training and neurofeedback clinic training with stimulant medication. This would result in the project having 5 conditions; 1 neurofeedback home training, 2 neurofeedback home training and stimulant medication, 3 stimulant medication, 4 neurofeedback clinic training, 5 neurofeedback clinic training and stimulant medication.
September 2013	The researcher transferred to City University London
1st October 2013	Reviewed by City University Senate Research Ethics Committee
14th October 2013	Received unfavourable outcome letter
10th December 2013	Reviewed by City University Senate Research Ethics Committee
18th December 2013	Received outcome letter, unable to reach a decision. Issues included: • Unclear why UEA ethics approval was not granted. • Committee wanted further information regarding why the NRES approval was not necessary. • The study should be registered as a clinical trial. • Allocation of groups was not randomised. • There needs to be a statistical review to clarify what effect size the study is looking at. • The intervention will be provided for 30 sessions, below sub-optimal level of 43 sessions.

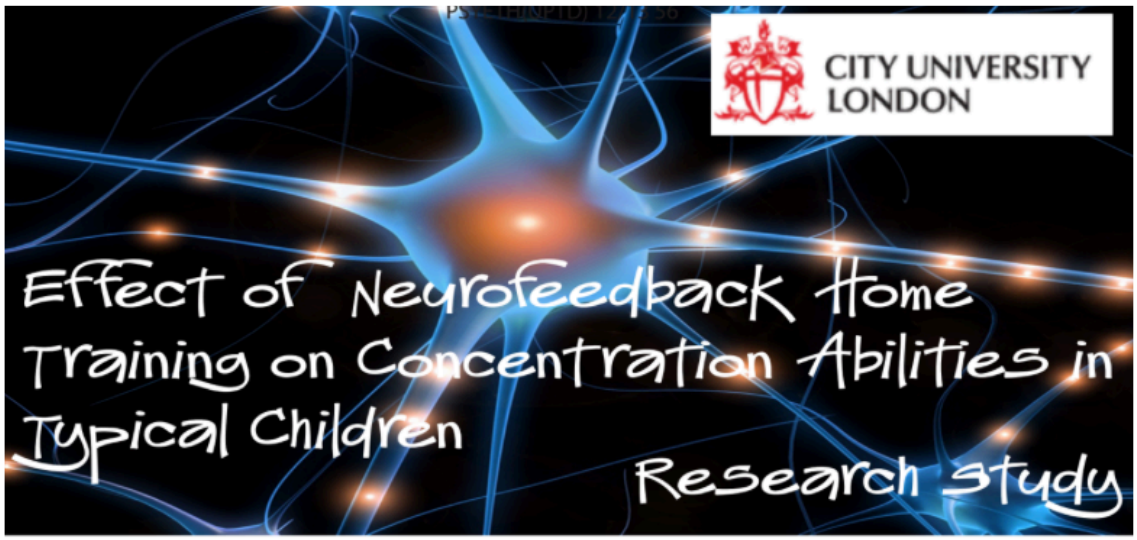
	• Issue of side effects, risk of seizure unattended at home. • A full trail should not proceed until results of the current (sample one) study is available.
January 2014	Application submitted and reviewed by the Learning Assessment and Neurocare Centre Ethical Committee and received a favourable opinion
January 2014	The following changes and clarifications were made and application resubmitted to City University Senate Research Ethics Committee. • It would be the parent's decision to approach the Centre, and this would not be a formal referral; they could do this independently of their NHS GP. The Centre would provide the GP with patient information (with their consent) as a courtesy, and not as an official report. Therefore, as these were private, and not NHS patients, NRES approval was not needed. This was the decision of the ethics committee of the Faculty of Medicine and Health Sciences. • This is not a formal clinical trial; it is an initial experimental feasibility study. It is in no way intended to provide data of direct clinical significance. Rather it is intended to provide scientific knowledge of an experimental nature that could then be used to inform early-stage clinical trials. Such a study is entirely appropriate for a PhD project. • Allocation to groups is randomised, and only patients willing to participate in the study would be eligible. The Consultant will only be deciding from a medical perspective if appropriate for the patient to receive neurofeedback and/or stimulant medication. • The main statistical information to be derived from this study is estimation of the effect size of the different treatments. The nature of this study is thus exploratory and these important statistical matters will form a crucial part of the thesis. As this research is part of a PhD there are necessary resource limitations as regards the number of

	sessions and number of participants; however, the data would be novel and would be the first of their kind and, as such, will represent new, and perhaps important, information. • There has only been one previous research study that has referred to the possibility of neurofeedback inducing a seizure (Vernon et al., 2004), and there is very little evidence for this. There have been no incidences of seizures when neurofeedback is conducted in the clinic. • The first study, titled "Examining the effect of neurofeedback home training on typical individuals' concentration abilities" does have some early findings available. A paired t-test assessed the difference between pre and post measures of concentration and impulsivity across the conditions. Parents' rating on concentration and impulsivity were improved in the neurofeedback home training condition ($t = 3.00$, df = 3, $p = 0.058$) compared to no change in the control conditions ($t = 0.555$, df = 8, $p = 0.594$). The measures and interventions that would be collected for this study are already in use at the Learning Assessment and Neurocare Centre. This includes the use of quantitative EEGs, Conners CPT, neurofeedback home training, in clinic training and in combination with stimulant medication. This research would not be looking into the effect of a new treatment but of a treatment that is already established and in use. The research would be collecting the data that is already being produced and using it in a research perspective. It is, therefore, known that the procedures outlined in this research study are effective.
February 2014	Email received from Research Ethics Committee asking for clarification on following issues: • What is the involvement of the Centre and consultant paediatrician

	• The approval letter from the Centre mentions that many of the procedures intended to be used in the study are already being conducted at the Centre. The Committee would like to know explicitly which procedures are in use already, and which ones are not and how these will be supported. • Proposed alterations to wording in Information Sheet.
February 2014	Following changes were made and application resubmitted to City University Senate Research Ethics Committee. • The Centre undertakes a quantitative EEG and Conners CPT on all child patients as part of their initial diagnostic paediatric assessments. The Centre provides a range of management options for patients including all conditions which will be involved in this study; namely, patients receiving neurofeedback at home with remote supervision, patients receiving neurofeedback at home with remote supervision with stimulant medication monitored by the consultant paediatrician, patients receiving medication only supervised by the consultant paediatrician, patients receiving neurofeedback in the clinic, patients receiving neurofeedback in the clinic with stimulant medication supervised by consultant paediatrician. • The research should not have been submitted to the NRES in the first place, so this was probably the result of a misunderstanding of the NRES on the part of the University of East Anglia (UEA). They returned it to UEA. We believe that what changed was UEA's understanding of these rules, which in an event did not involve private patients. This set of events may have reflected UEA's over-caution of this proposal because it involved children.

	• The title of the study was changed to "*A feasibility study to investigate the effect of neurofeedback and stimulant medication on children with Attention Deficit Hyperactivity Disorder (ADHD)"*
March 2014	Received a favourable outcome

Appendix B: Typically developed sample information sheet for parents



You are being invited to take part in a research study based in West Sussex, conducted by research student Hannah Wachnianin with City University London. Before you decide if you wish to be involved, it is important for you to understand why the research is being done and what it will involve. You may wish to discuss this research with your family. It is up to you to decide if you want to join this study.

Who Is organising This study?

This research is organised by the Department of Psychology, City University London. The researcher is Hannah Wachnianin, PhD student, who is also funding this study. The research supervisors are [redacted]

What Are the Aims of the study?

This study intends to examine whether neurofeedback home training aids in improving concentration abilities and impuslive behaviour at school and home in children. Neurofeedback is a form of brain wave training, where the individual is stimulated to reduce or enhance activity of particular brainwaves via visual and audio reinforcement. The aim of neurofeedback is to improve attention and behavioural control by teaching the individual to regulate levels of cortical arousal in the brain. This study also aims to see if neurofeedback affects a child's response to positive rewards.

What Will the participant Have To Do?

You and your child will be asked to meet with the researcher at the Learning Assessment and Neurocare Centre in Horsham. Your child will complete a QEEG, a non-invasive brain wave reading. This is where the child wears a cap that contains 19 electrodes (see picture) and a further electrode on each ear lobe. The QEEG cap and electrodes will be fitted to the participants. This is a non-invasive procedure in whchi a cap is placed on the participants head and QEEG gel is injected into the holes in the cap. Static bands are placed on each wrist to create a safe electric circuit. Once this has been completed, the recording will take place, which takes 10 minutes, whereby the child needs to sit as still as possible but also relaxed. In total, the QEEG procedure takes approximately an hour.

After the QEEG, the child will then complete a computer concentration test as well as a personality questionnaire which will take 15 minutes. Additionally, you and your child's teacher will complete an electronic questionnaire. You will then be randomly allocated to a group, receiving either neurofeedback home training, computer game training or nothing. The neurofeedback home training group will be shown how to use the neurofeedback equipment by the researcher which will take an hour. The software will be installed by the researcher onto the participants laptop. You will be provided with written instructions on how to set up and use the equipment during a neurofeedback session at home, which the researcher will go through and explain. If you are allocated to the neurofeedback home training group, this will be completed by your child twice a week for 15 weeks at home. The neurofeedback received will be using a peak performance training protocol specifically aimed at improving concentration abilities. Each neurofeedback session will consist of three 10 minute games; pacman with picture and sound, boxes with pictures, and videos with pictures and sound. The neurofeedback sessions will be completed by the child/participant and will be aided by their parents. After each session has been completed, you will be required to email this to the researcher to ensure that each session has been completed and been completed correctly. The computer game training group will complete computer based games for 30 minutes twice a week at home for 15 weeks.

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After 15 weeks of receiving neurofeedback, computer game training or no treatment, the same information will be collected again; QEEG, computer concentration test and questionnaire for the child in addition to parent and teacher rating scales.

Who Can Take Part?

Children between the age of 7 and 17 who do not have any formal clinical diagnosis can take part. If you decide to take part you will be asked to sign a consent form and your child will be asked to complete an assent form.

Who Can Not Take Part?

Children that are aged 6 years old or below, or above 18 years old or children who have any formal diagnosis (including ADHD, Autism or Dyslexia) may not be involved. Furthermore, individuals who have had a previous course of neurofeedback treatment will not be able to take part or if the child is taking a medication for any mental health conditions.

What Are The Possible Disadvantages And Risks of Taking Part?

There are some potential negative side effects. There is a very small, and highly unlikely possibility that neurofeedback could induce seizures. Therefore, the sessions will be carefully monitored as each neurofeedback session will be emailed to and reviewed by the researcher. Other potential side effects include feeling anxious or having difficulty sleeping, headaches and dizziness, all of which are overcome by having a rest after receiving neurofeedback or something to eat. If you are concerned at any stage about side effects, please contact [redacted] or [redacted] or your family doctor. If you as a parent or your child becomes distressed while being involved in this research, please contact the researcher **Hannah Wachnianin on** [redacted] or your family doctor.

This study may be inconvenient to participants as it is a commitment, particularly if the individual is allocated to the neurofeedback home training group which will take place twice a week for 15 weeks. To overcome the practical inconveniences, as much as possible the pre and post measures will be collected outside of school hours, i.e. evenings, weekends or school holidays. The most convenient time for a participant to be involved will be determined in conjunction with the participant and family to ensure that it does not coincide with exams or holidays abroad.

What Are The Possible Benefits of Taking Part?

Neurofeedback has been shown to enhance peak performance and consequently may help your child's academic performance.

What If Something Goes Wrong?

If you are concerned by taking part in this research project or you wish to complain about any aspect of the way you have been approached or treated during the course of this study, please contact [redacted]

[redacted]

If you wish to withdraw your data from this study you are able to do so until four weeks after the post measures have been collected without giving a reason.

This project has been approved by the Research and Ethics Committee of the Department of Psychology of City University London (project number PSYETH(UPTD) 12/13 56). If you have any comments, concerns or observations about the conduct of the study or your experiences as a participant, please contact the Secretary to the Committee [redacted] quoting the above project number, on [redacted] or [redacted]

What Happens to the Information I Provide?

Participation in this study guarantees confidentiality of the information you provide. No one apart from the researcher and research supervisor will have any access to the information you provide. Your name and any other identifying information will be stored separately from your data in a securely locked filing cabinet. Data will be stored in a securely locked room for as long as is required by the Data Protection Act, and then they will be destroyed by our confidential shredding service. The data collected for this study will be used for a student project. Once the data is analysed, a report of the findings may be submitted for publication and data cannot be withdrawn. Only broad trends will be reported and it will not be possible to identify any individuals. A summary of the results will be available from the researcher on request.

What If Relevant New Information Becomes Available?

Sometimes we receive new information about neurofeedback during the course of research taking place. If this happens, your researcher will contact you and discuss whether you would like to continue in the study. If you decide not to carry on, your researcher will make arrangements for your care to continue. If you decide to continue in this study, you will be asked to sign an agreement outlining the discussion.

Contact for Further Information

If you are interested in taking part in this research, or would like any further information or have any queries about this study please contact the researcher:

[redacted]

Appendix C: ADHD sample information sheet for parents

You are being invited to take part in a research study being conducted by research student Hannah Wachnianin with the City University, London. Before you decide if you wish to be involved, it is important for you to understand why the research is being done and what it will involve. Our researcher will go through this information sheet with you and answer any questions you have. Talk to others about the study if you wish. It is up to you if you wish to join this study.

What are the aims of the study?

This study intends to find if neurofeedback and stimulant medication (methylphenidate) has a positive effect on children's ADHD symptoms when used together. Additionally, the study aims to examine if neurofeedback is effective when conducted in the home setting.

Neurofeedback is a form of brain wave training, where the individual is stimulated to reduce or enhance activity of particular brainwaves via visual and audio reinforcement. The aim of neurofeedback is to improve attention and behavioural control by teaching the patient to regulate levels of cortical arousal in the brain.

During neurofeedback three electrodes are used; one behind each ear and one on the child's head. The child is then required to sit still and concentrate on watching a computer game such as Pacman. When they are concentrating well, the computer game will work, if the child is not concentrating or is moving, the computer game will stop.

Stimulant medication prevents the re-uptake of dopamine and norepinephrine on the brain and has been found to have a positive effect on ADHD symptoms on 73% of children with ADHD.

Independently, neurofeedback and stimulant medication have been shown to be effective for individuals with ADHD, however, it is unclear if they are effective when combined.

Do I have to take part?

It is your choice whether you wish to take part in the study. We will explain the study to you so that you fully understand what it is about. If you agree to take part, we will then ask you to sign a consent form. You are free to withdraw at any time, without giving a reason. This will not affect the standard of care you receive.

Who can take part?

Children and young adults:

- Aged between 7 and 17
- Who have a main clinical diagnosis of ADHD combined subtype
- Who have not previously received a course of neurofeedback
- Who are not taking a medication other than methylphenidate
- Whose Consultant Paediatrician, Dr Kewley, considers that both neurofeedback and stimulant medication are suitable for the individual to receive and would be offered as a standard part of their treatment package at the Centre.
- who have access to a windows laptop

You cannot take part if the child or young adult:

- Is 6 or younger or 18 or older
- Does not have a diagnosis of ADHD combined subtype
- Is on medication for other neurodevelopmental conditions
- Has previously received neurofeedback

You will not need to do anything over and above the assessment and treatment process that you will receive at the Learning Assessment and Neurocare Centre.

What will we have to do if we take part?

Involvement in this study is split into three phases as follows.

Phase 1 Assessment

During the assessment phase, you and your child will meet with the researcher for 2 hours. Initially, the researcher will discuss with you in detail regarding the research, explaining what will be involved, how to withdraw, gain your consent and your child's assent. Your child will then complete a QEEG. This is a non-invasive brain wave reading, where the child wears a cap that contains 19 electrodes and a further electrode on each ear lobe. The electrodes are then filled with a conductive gel to enable the information the brain produces to be collected. Once this has been prepared, the recording will take place. This process takes 10 minutes, whereby the child needs to be relaxed and sit as still as possible. In total, the QEEG procedure takes approximately an hour.

After this, your child will complete a computer concentration test and a quick paper questionnaire in addition to parent and teacher completing an electronic questionnaire.

Phase 2 Treatment

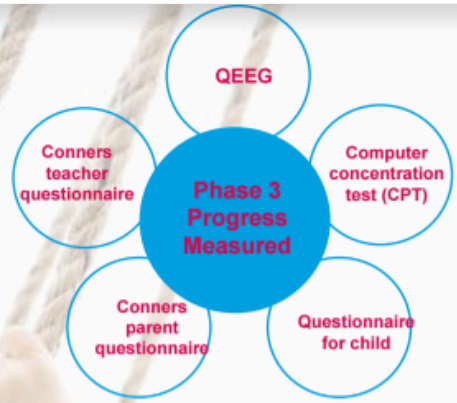
The researcher will randomly allocate you to one of the following treatment strategies, all of which will be carried out over 15 weeks:

Phase 2
Treatment allocated, one of either;

Neurofeedback at home	If allocated **neurofeedback at home**, you will meet the researcher for an hour to demonstrate how to use the neurofeedback equipment as well as providing you with written instructions. Neurofeedback will be carried out twice a week at home by the child with the help of a parent. A neurofeedback session is approximately 45 minutes long. After completing each session, you will email the results to the researcher.
Neurofeedback at home and medication	If you are allocated **neurofeedback at home and medication,** you will meet the researcher for an hour to demonstrate how to use the neurofeedback equipment as well as providing you with written instructions. Neurofeedback will be completed twice a week at home by the child with the help of a parent. A neurofeedback session is approximately 45 minutes long. After completing each session, you will email the results to the researcher. This group will also take stimulant medication every day.
Medication	If only stimulant **medication** is allocated, this will be taken every day.
Neurofeedback at clinic	If you are allocated to this group, **neurofeedback** will be carried out by the researcher twice a week at the **clinic**. A neurofeedback session is approximately 45 minutes long.
Neurofeedback at clinic and medication	If you are allocated to this group, **neurofeedback** will be carried out by the researcher twice a week at the **clinic**. A neurofeedback session is approximately 45 minutes long. Stimulant **medication** will also be taken every day.

Phase 3 Progress measured

After 15 weeks of receiving the treatment, the same information will be collected again by the researcher at the Learning Assessment and Neurocare Centre; qEEG, computer concentration test and questionnaire for the child in addition to parent and teacher electronic questionnaire. This meeting will take approximately 2 hours. At this point, if the child has been taking medication, they will not take medication for 24 hours before collecting this information.



What are the possible side effects of any treatment recieved when taking part?

There are potential risks with taking stimulant medication. These risks are only short-term and include headaches, sleep difficulties, appetite suppression and blunting of personality. There have been very few studies that have reported on the long term effect
of taking stimulant medication, but these have concluded that there are no long term negative side effects.

The patients Specialist Consultant Paediatrician at the Learning Assessment and Neurocare Centre, Dr G D Kewley, is medically qualified and will be responsible for the diagnosis and management of the patient. He will be responsible for prescribing, monitoring and titrating medication, if they are allocated to a stimulant medication group. This will not be the role of the researcher, who will be observing and testing the effects of usual treatment variants on patients within each category. If, during this study, you suffer from any of these side effects or have any other medical concerns, please contact your Specialist Consultant [redacted] or [redacted] as soon as possible to discuss.

There are some potential negative side effects to neurofeedback. There is a very small, and highly unlikely possibility that neurofeedback could induce seizures. Therefore, the sessions will be carefully monitored as each neurofeedback session will be emailed to and reviewed by the researcher. Other potential side effects include feeling anxious or having difficulty sleeping, headaches and dizziness, all of which are overcome by having something to eat or a rest after receiving neurofeedback. If you are concerned at any stage about side effects, please contact [redacted] or [redacted] or your family doctor. If, during or after a neurofeedback session, the child experiences black outs, confusion, loss of consciousness, convulsion or difficulty talking, you should contact an ambulance immediately.

If you as a parent or your child becomes distressed while being involved in this research, please contact the researcher Hannah Wachnianin on [redacted] or your family doctor.

What are the possible benefits of taking part?

We hope that the treatments will help your child with their ADHD symptoms. However, this cannot be guaranteed. The information we get from this study may help us to better treat patients with ADHD in the future.

What happens when the reasearch study stops

Your child will continue to be managed by the Specialist Consultant Paediatrician [redacted] at the Learning Assessment and Neurocare Centre. You will be able to continue accessing these treatments with the Centre once the research has finished.

What if relevant new information becomes available?

Sometimes we get new information about the treatment being studied. If this happens, your researcher will contact you and discuss whether you would like to continue in the study. If you decide not to carry on in the research, your researcher will make arrangements for your care to continue directly with the Learning Assessment and Neurocare Centre. If you decide to continue in this study, you will be asked to sign an agreement outlining the discussion.

What happens if i dont want to carry on in the research?

You can withdraw from the research any time up until four weeks after the second meeting with the researcher when the treatment plan has been completed. If you do so, all information taken from you will be destroyed. After this time, the data will be unable to be withdrawn. If you wish to withdraw from this research, please contact research Hannah Wachnianin [redacted] or

[redacted] This will not affect the care that you receive by the researcher or the Learning Assessment and Neurocare Centre.

What if something goes wrong?

If you have a concern about any aspect of this study, you should speak to the researcher who will do their best to answer any questions. Hannah Wachnianin can be contacted on [redacted] or [redacted] If you remain unhappy and wish to complain formally, please contact supervisors of the research [redacted] on [redacted] or [redacted]

Will taking part in the study be confidential?

Participation in this study guarantees confidentiality of the information you provide. No one apart from the researcher and research supervisor will have any access to the information you provide. Your name and any other identifying information will be stored separately from your data in a securely locked filing cabinet. Data will be stored in a securely locked room for as long as is required by the Data Protection Act, and then they will be destroyed by our confidential shredding service. The data collected for this study will be used for a student project. When this research is published, only trends will be identified and not individuals data.

If you decide to take part in this research, with your consent, your GP will be notified that you are taking part as will your child's teacher.

What will happen to the results of the reseach study?

Once the data is analysed, a report of the findings may be submitted for publication. Only broad trends will be reported and it will not be possible to identify any individuals. A summary of the results will be available from the researcher on request.

Who is organising and funding the study?

This research is organised by the Department of Psychology at the City University, London. The researcher is Hannah Wachnianin, PhD student, who is also funding this study. The research supervisors are [redacted]

Who has reviewed the study?

This research has been reviewed by the City University London Ethics Committee where it was given a favourable opinion.

I would like to take part or further information

If you require any further information, have any queries about this study or would like to take part, please contact the researcher:

[redacted]

Appendix D: Typically developed sample child information sheet

[redacted]

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CITY UNIVERSITY LONDON

What Is Research?

Research is a way we try to find out the answers to questions. We want to see how neurofeedback at home may help children in improving their concentration and behaviour. Neurofeedback is a form of brain training, where you will be asked to concentrate to make a computer program work correctly. This may help you to improve your attention by learning to control your brain waves.

Do I Have To Take Part?

No. It is up to you. We will ask for your assent and then ask if you would sign a form. We will give you a copy of this information and your signed form to keep. You are free to stop taking part at any time during the research without giving a reason, until four weeks after the second time you have met the researcher. If you decide to stop, this will not affect the care you receive.

What Will I Be Asked To Do?

First you will meet the reseracher at a Centre in Horsham. You will be asked to have a QEEG which will involve you wearing an unusual coloured hat. This is where you will wear a cap which contains 19 electrodes (see picture) and a further electrode on each ear lobe. The electrodes are then filled with a conductive gel to enable the information the brain produces to be collected. Static bands are also placed on each wrist. Once this has been completed, the recording will take place, which takes 10 minutes, whereby you will need to sit as still as possible but also relaxed. In total, the qEEG procedure takes approximately an hour. You will do some questionnaires and a computer concentration test. You will then to told which treatment you will start to receive of either neurofeedback (which is a brain training programme you will do at home), computer game training that will be completed at home, or nothing.

[redacted]

If you are given neurofeedback, you and your parents will be shown how to do this by the researcher. If you are given the computer game training, you will complete 30 minutes of computer games twice a week at home for 15 weeks, again with the help from your parents. If you are in the group that receives nothing, you will not do anything for 15 weeks. After 15 weeks, you will meet the researcher again at the Centre for a couple of hours. Here you will have another QEEG, complete more questionnaires and complete another computer concentration test.

Will the Neurofeedback Upset Me?

Neurofeedback may make you feel worried, tried, give you headaches or you may have difficulty sleeping. If you feel any of these, or are worried about anything, tell your parents or contact the researcher on [redacted] You will not get into trouble.

Will Joining in Help Me?

We cannot promise the study will help you but the information we get might help treat young people with better medicines in the future.

What Will Happen When the Research Project Stops?

You will no longer receive neurofeedback once this study is completed. However, if you would like to continue to receive neurofeedback, the researcher will put you in contact with someone to arrange this.

Will Anyone Else Know I'm Doing This?

Your parents and teacher will be told that you are taking part in this study. All the information you give us will be kept safe in a locked cabinet that only the researcher and medical professional can access. Once the study has been completed, the information will be destroyed.

What If I Don't Want To Do The Research Anymore?

If at any time you don't want to do the research anymore, just tell your parents or the researcher on [redacted] They will not be cross with you.

What If Something Goes Wrong?

If something goes wrong or you are worried, talk to your parents or the researcher on [redacted]

Who is Organising This Study?

This research is organised by the Department of Psychology, City University London. The researcher is Hannah Wachnianin, PhD student, who is also funding this study. The research supervisors are [redacted] and [redacted]

Contact for Further Information

If you would like any more information or have any questions, please contact the researcher:
Hannah Wachnianin
[redacted]

Appendix E: ADHD sample child information sheet 7-11 year olds

Learning Assessment & Neurocare Centre Ltd

CITY UNIVERSITY LONDON

How Does Medication and Neurofeedback Make You Feel? Participant Information Sheet 7 - 11 Years

My name is Hannah and I am studying to be a psychologist. I am doing a project for my course and would like some help.

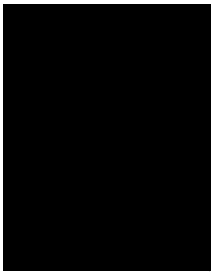
I would really like to know about how medication and neurofeedback makes you feel. Neurofeedback is a computer game that you play with your brain. Games include Pacman and videos.

Do I have to take part?

You can say yes or no. It is up to you whether you take part. You can talk with your parents as well as Hannah to see if you would like to take part in the project.

If you would like to take part but change your mind, you can stop taking part at any time.

What will happen to me if I take part?



You will meet with Hannah and will do 3 things:

1. You will be asked to wear an odd hat which has electrodes in it (see picture) and a further electrode on each ear. Hannah will then spend some time putting get into each electrode. When this is finished, you will need to sit as still as possible but also relaxed for 1 minute at a time. You will sit still for 10 minutes all together.

2. You will do some questionnaires which Hannah will help you with

3. You will do a computer concentration test.

You will then be told which of the following groups you are put in for 15 weeks:

☺ You might take some medicine everyday.
☺ You might take some medicine everyday and do Neurofeedback at home with your parents.
☺ You might only do Neurofeedback at home with your parents.
☺ You might do Neurofeedback at the clinic with Hannah.
☺ You might do Neurofeedback at the clinic with Hannah and take some medicine everyday.

After 15 weeks of doing this, you will meet Hannah again where you will wear the odd hat again, answer more questions and complete another computer concentration test.

Will taking part upset me?

Taking part should not upset you. If you do feel upset or do not feel yourself, it is important you tell your parents.

Will anyone else know I'm doing this?

Your parents will know you are taking part as will [redacted] your family GP and your teacher.

Appendix F: ADHD sample child information sheet 12 - 17 year olds

Learning Assessment & Neurocare Centre Ltd

CITY UNIVERSITY LONDON

How Medication and Neurofeedback Effects You

Participant Information Sheet

We are asking if you would join in a research project to find out how medication and neurofeedback effects you. Before you decide if you want to join in, it's important to understand why the research is being done and what it will involve for you. So please consider this leaflet carefully. Talk to your family, friends, doctor if you want to.

Why are we doing this research?

We want to see if medicine treats ADHD better than neurofeedback or if they work well together. Knowing this will help other children to concentrate better.

What is neurofeedback?

Neurofeedback is a form of brain training, where you will be asked to concentrate to make a computer program work correctly. This may help you to improve your attention by learning to control your brain waves. During neurofeedback three electrodes are used; one behind each ear and one your head. You need to sit still and concentrate on watching a computer game such as Pacman. When you are concentrating well, the computer game will work, if you are not concentrating or moving, the computer game will stop.

Why have I been invited to take part?

You have been invited to join our study because you have ADHD and are one of [redacted] patients. At least 100 children will be taking part in this study.

Do I have to take part?

No. It is up to you. We will ask if you would like to take part and then ask if you would sign a form. We will give you a copy of this information and your signed form to keep.

You are free to stop taking part at any time during the research without giving a reason. If you decide to stop, this will not effect the care you receive.

What will happen to me if I take part?

You will be asked to have a QEEG which will involve you wearing an unusual coloured hat. This is where you will wear a cap which contains 19 electrodes (see picture) and a further electrode on each ear lobe. The electrodes are then filled with a conductive gel to enable the information the brain produces to be collected. Static bands are also placed on each wrist. Once this has been completed, the recording will take place, which take s 10 minutes, whereby you will need to sit as still as possible but also relaxed. In total, the qEEG procedure takes approximately an hour. You will do some questionnaires and a computer concentration test. You will then be told which treatment you will start to receive of either medication, neurofeedback (which is a brain training programme) at home or at the clinic, or both.

[redacted]

You will have this treatment for 15 weeks, either taking medication everyday, or twice a week having neurofeedback at home with the help of your parents or at the clinic with the help of the researcher. A neurofeedback sessions takes about 45 minutes. After 15 of treatment, you will come to the Centre again and meet the researcher for a couple of hours. Here you will have another qEEG, complete more questions and complete another computer concentration test.

Will the neurofeedback or medication upset me?

Sometimes you may have headaches, sleep difficulties or not feel yourself. If you feel any of these, tell your parents or ask to speak to your Specialist Consultant Paediatrician, [redacted] who can be contacted on [redacted] Your doctor will be able to help stop you feeling like this.

What are the possible benefits of taking part?

We cannot promise the study will help you but the information we get might help treat young people with ADHD with better medicines in the future.

What happens when the research stops?

You will continue having the same treatment and will continue receiving care from your Specialist Consultant Paediatrician [redacted] at the Learning Assessment and Neurocare Centre.

Will anyone else know I'm doing this?

We will keep your information in confidence. This means we will only tell those who have a need or right to know. Wherever possible, we will only send out information that has your name and address removed. Your parents will know you are taking part, as will [redacted] your family GP and your teacher.

What happens to the information I provide?

All the information you give us will be kept safe in a locked cabinet that only the researcher and medical professional can access. Once the study has been finished, the information will be got rid of.

Who is organising and funding the research?

This research is organised by the Department of Psychology at City University, London. The researcher is Hannah Wachniapin, PhD student, who is also funding this study. The research supervisors are [redacted] and [redacted] This research has been reviewed by the Ethics Committee at City University, London who have given a favourable opinion.

Further information and contact details

If you require any further information or have any queries about this study please contact the researcher:

[redacted]

Appendix G: Typically developed sample assent form

PSYETH(UPTD) 12/13 56

CITY UNIVERSITY LONDON

Participant Identification Number

"Effect of Neurofeedback Home Training on Children"

Research Study

Assent Form For Child

Child (or if unable, parent on their behalf) /young person to circle all they agree with:

Has somebody else explained this project to you?	Yes/No
Do you understand what this project is about?	Yes/No
Have you asked all the questions you want?	Yes/No
Have you had your questions answered in a way you understand?	Yes/No
Do you understand it's OK to stop taking part at any time?	Yes/No
Are you happy to take part?	Yes/No

If you don't want to take part, don't sign your name.

If you do want to take part, you can write your name below

Your name _____

Date _____

The researcher who explained this project to you needs to sign too:

Print Name _____

Sign _____

Date _____

Thank you for your help.

When completed: 1 for participant, 1 for researchers site file.

Appendix H: ADHD sample assent form

Learning Assessment & Neurocare Centre Ltd

CITY UNIVERSITY LONDON

A feasibility study to investigate the effect of neurofeedback and stimulant medication on children with Attention Deficit Hyperactivity Disorder (ADHD)
Research Study

Assent Form

	✓ Yes	X No
 Has somebody explained this project to you?		
 Do you understand what this project is about?		
 Have you asked all the questions you want to?		
 Have you understood the answers to your questions?		
 Do you understand that it is okay to stop taking part at any time?		
 Are you happy to be part of this study?		

Your Name: _____

Today's Date: _____

Researcher's Name: _____

Appendix I: Typically developed sample consent form

PSYETH(UPTD) 12/13 56

CITY UNIVERSITY LONDON

Participant Identification Number

"Effect of Neurofeedback Home Training on Concentration Abilities in Typical Children"

Research Study

Consent Form For Parents/Guardian

Information regarding the research will be verbally discussed with the patient and the parents. This will take approximately 10 minutes, with an opportunity for the patient and parent to ask questions. This will be done conjunction with providing written information in this consent form for parents and a written consent/assent form for children.

1. I confirm that I have read and understand the information sheet for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily. ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without my medical care or legal rights being affected. ☐
3. I agree for my child's teacher to be involved in this study. ☐
4. I agree to take part in the above study. ☐

Name of Participant's Parent/Guardian	Signature	Date
Name of Researcher	Signature	Date

When completed: 1 for participant, 1 for researchers site file.

Participant Details:

Child's Full Name:	
Child's Date of Birth:	
Child's Age:	
Child Left or Right Handed:	
Child have any clinical diagnosis? (e.g. ADHD, Autism, Aspergers, Tourettes)	
Parent's Full Name:	
Email Address:	
Child's School:	
Child's School Teacher:	
Child's School Teacher Email:	

Contact details (if child/parent wish to discuss options later)
Email: [redacted]

Appendix J: ADHD sample consent form

Learning Assessment & Neurocare Centre Ltd

CITY UNIVERSITY LONDON

A feasibility study to investigate the effect of neurofeedback and stimulant medication on children with Attention Deficit Hyperactivity Disorder (ADHD)
Research Study

Consent Form Parents/Guardian

	Please tick box
I agree to take part in the above City University London research project. I have had the project explained to me, and I have read the participant information sheet, which I may keep for my records. I understand this will involve: • QEEG • Conner's Continuous Performance Test (CPT) • Conner's questionnaire for parents and teachers • Personality questionnaire for the child • If appropriate, complete neurofeedback at home, twice a week for 15 weeks • If appropriate, take medication everyday for 15 weeks	
This information will be held and processed for research purposes only. I understand that any information I provide is confidential, and that no information that could lead to the identification of any individual will be disclosed in any reports on the project, or to any other party. No identifiable personal data will be published. The identifiable data will not be shared with any other organisation.	
I understand that my participation is voluntary, that I can choose not to participate in part or all of the project, and that I can withdraw at any stage of the project without being penalized or disadvantaged in any way.	
I agree to City University London recording and processing this information about me. I understand that this information will be used only for the purpose(s) set out in this statement and my consent is conditional on the University complying with its duties and obligations under the Data Protection Act 1998.	
I agree to my child's GP as well as school teacher to be contacted and informed that my child is taking part in this research study.	
I agree to take part in the above study.	

Name of Parent	Signature	Date
Name of Researcher	Signature	Date

Participant's Details:

Child's Full Name:	
Child's Date of Birth:	
Child's Age:	
Child Left or Right Handed:	
Clinical Diagnosis:	
Medication:	
Parent's Full Name:	
Email Address:	
Child's School:	
Child's School Teacher:	
Child's School Teacher Email:	
Child's GP:	
Child's GP address:	

Contact details (if child/parent wish to discuss options later)
Email: [redacted]

Appendix K: ADHD sample letter informing GP

Learning Assessment & Neurocare Centre Ltd

CITY UNIVERSITY LONDON

Ref: HW

Date:
Dr

Dear Dr

Re:

This patient has decided to take part in a research study taking place at the Learning Assessment and Neurocare Centre which is being conducted by research student, Hannah Wachnianin, with City University London, who is investigating the effect of neurofeedback and stimulant medication on children with a main clinical diagnosis of ADHD. This is a pragmatic study whereby the researcher will be observing the effects of usual treatment variants; therefore, the patient will receive the necessary specialist care from their consultant at the Learning Assessment and Neurocare Centre throughout the duration of the study and once the study has diminished. In addition to receiving the usual treatment at the Learning Assessment and Neurocare Centre, the patient will undergo a further QEEG brain wave reading, Conners' Continuous Performance Test, Conners' rating scales and a personality questionnaire, after receiving treatment for 15 weeks.

Yours sincerely

Hannah Wachnianin
Assistant Psychologist at the Learning Assessment and Neurocare Centre
PhD Student at City University London

Appendix L: BIS/BAS questionnaire

Please indicate if you agree or disagree with the following statements. For each question, indicate your answer by using the 4-point scale below the question. There are no right or wrong answers to these questions, simple provide your level of agreement or disagreement.

1. A person's family is the most important thing in life.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

2. Even if something bad is about to happen to me, I rarely experience fear or nervousness.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

3. I go out of my way to get things I want.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

4. When I'm doing well at something I love to keep at it.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

5. I'm always willing to try somethinG new if I think it will be fun.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

6. How I dress is important to me.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

7. When I get something I want, I feel excited and energised.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

8. Criticism or scolding hurts me quite a bit.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

9. When I want something I usually go all-out to get it.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

10. I will often do things for no other reason than that they might be fun.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

11. It's hard for me to find the time to do things such as a get a haircut.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

12. If I see a chance to get something I want I move on it right away.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

13. I feel pretty worried or upset when I think or know somebody is angry at me.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

14. When I see an opportunity for something I like I get excited right away.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

15. I often act on the spur of the moment.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

16. If I think something unpleasant is going to happen I usually get pretty "worked up."

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

17. I often wonder why people act the way they do.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

18. When good things happen to me, it affects me strongly.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

19. I feel worried when I think I have done poorly at something important.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

20. I crave excitement and new sensations.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

21. When I go after something I use a "no holds barred" approach.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

22. I have very few fears compared to my friends.

1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

23. It would excite me to win a contest.

1	2	3	4

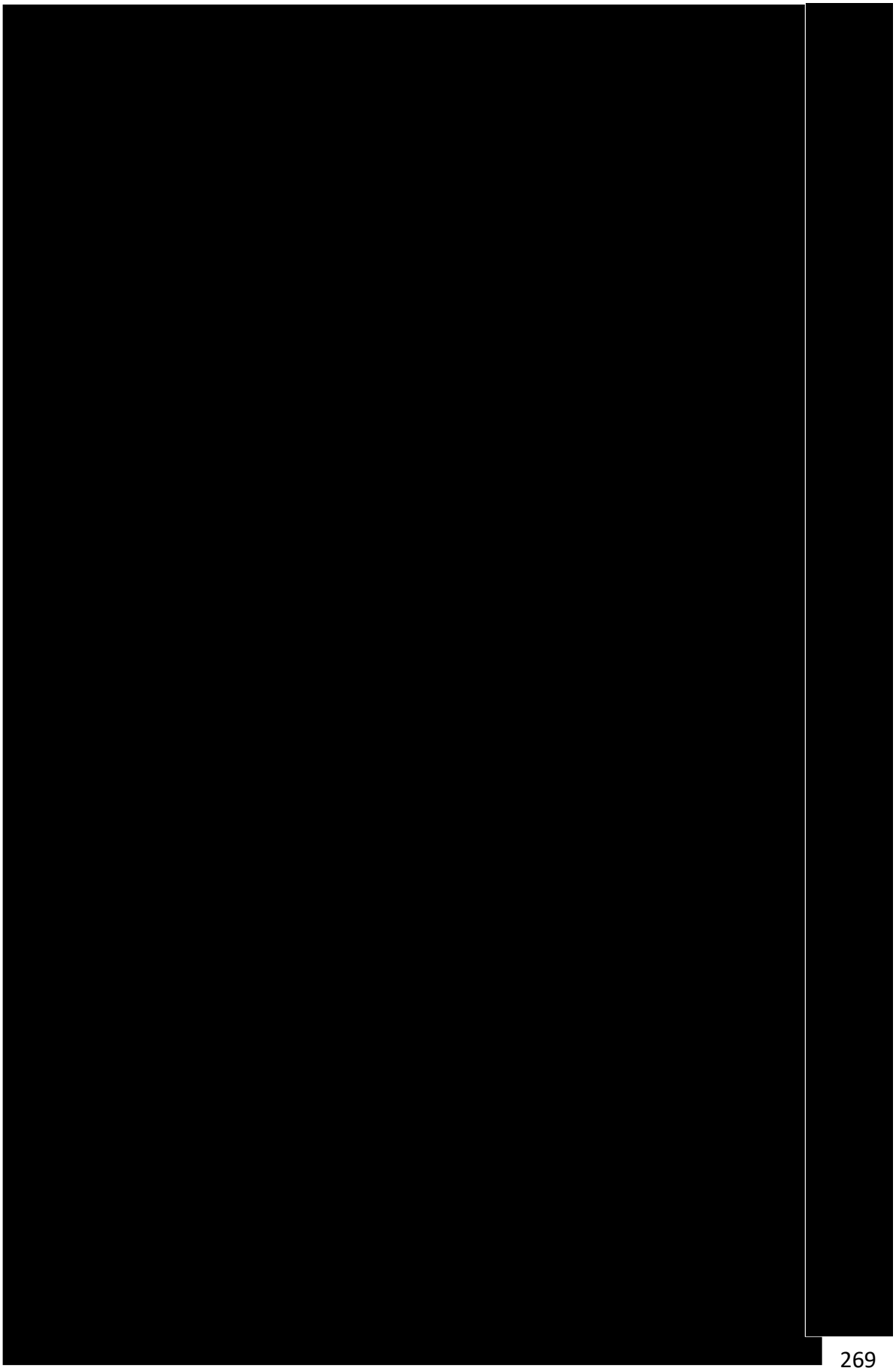
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

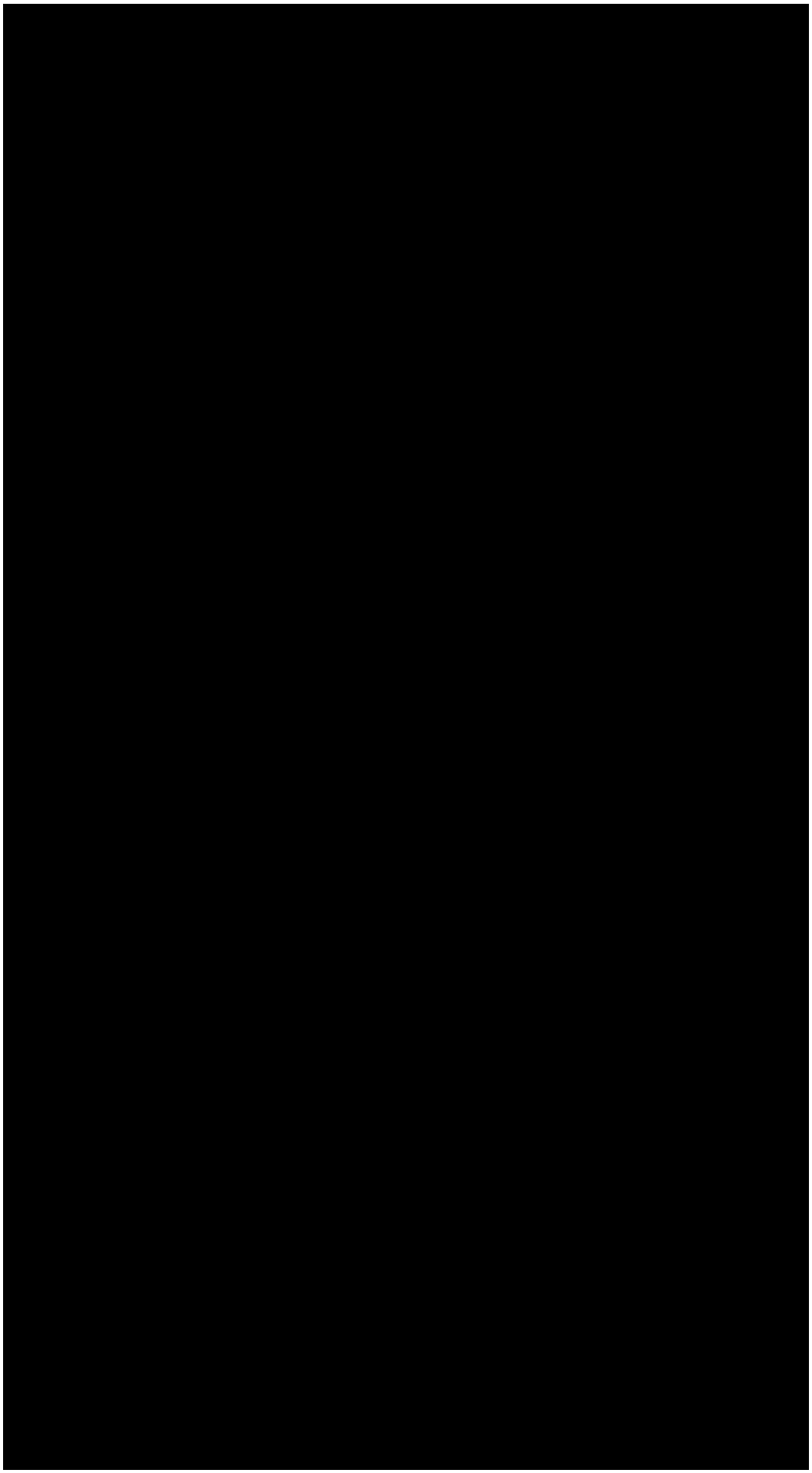
24. I worry about making mistakes.

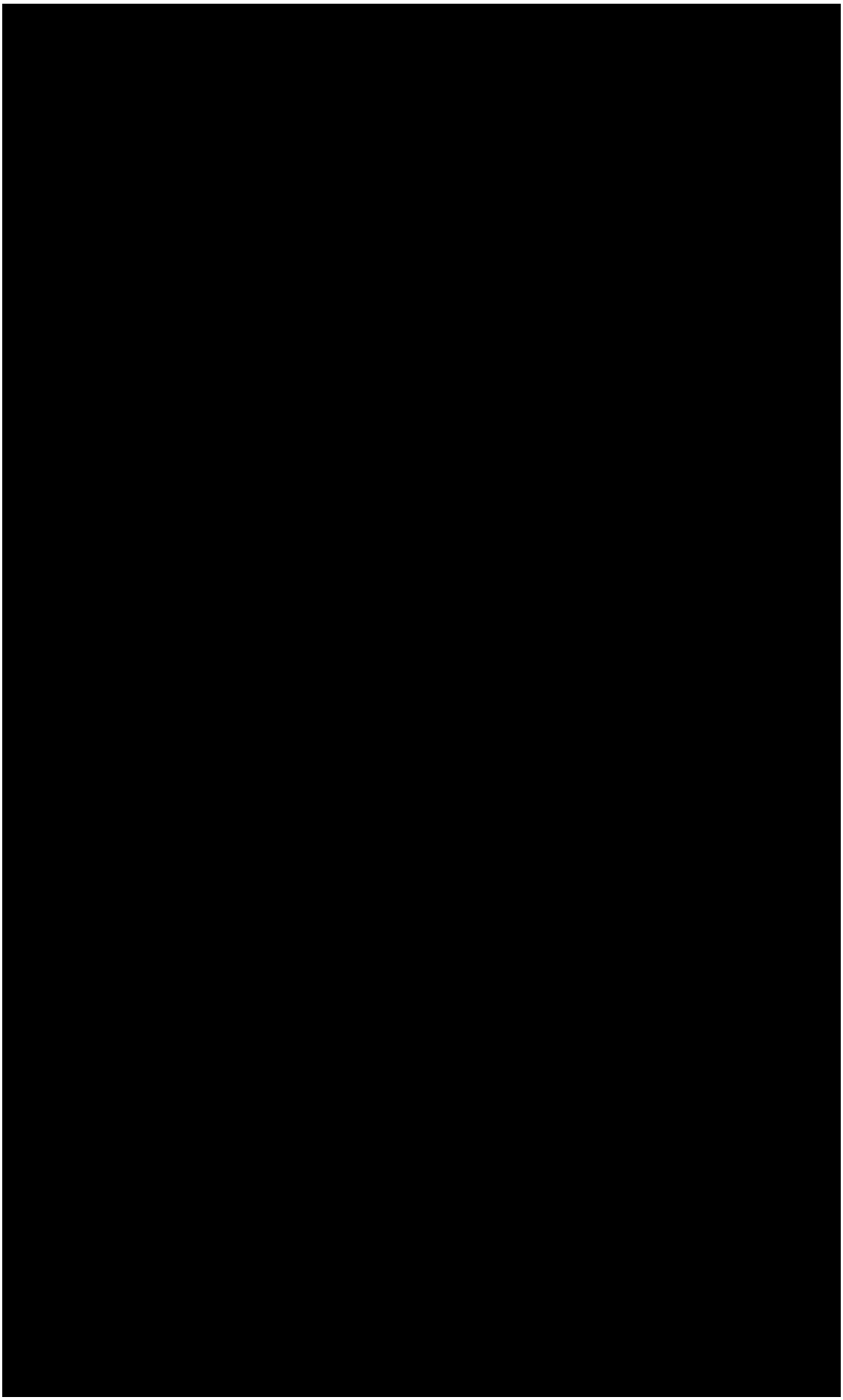
1	2	3	4
Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree

Thank you very much for completing this questionnaire.

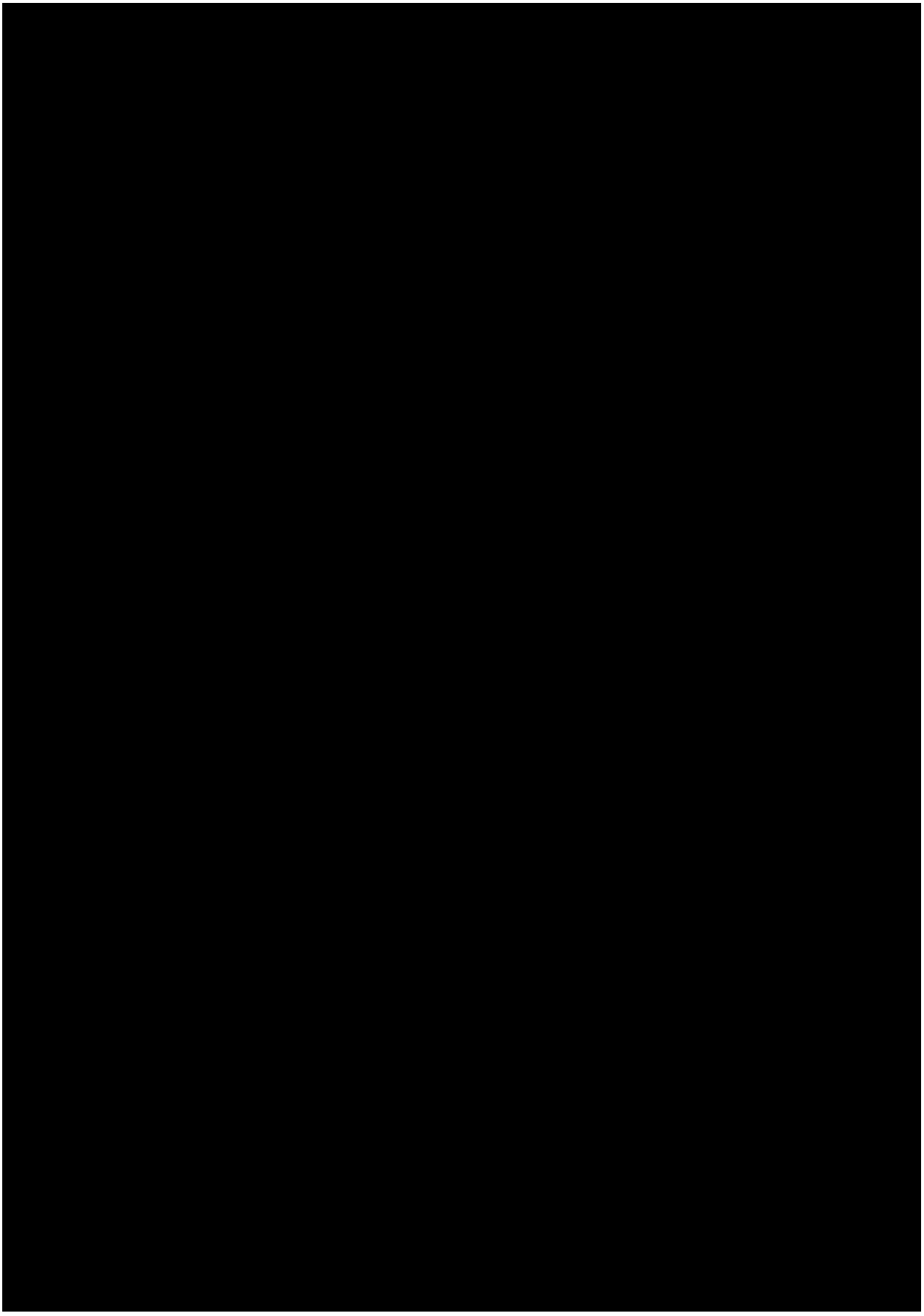
Appendix M: Conners' 3 Parent Rating Scale

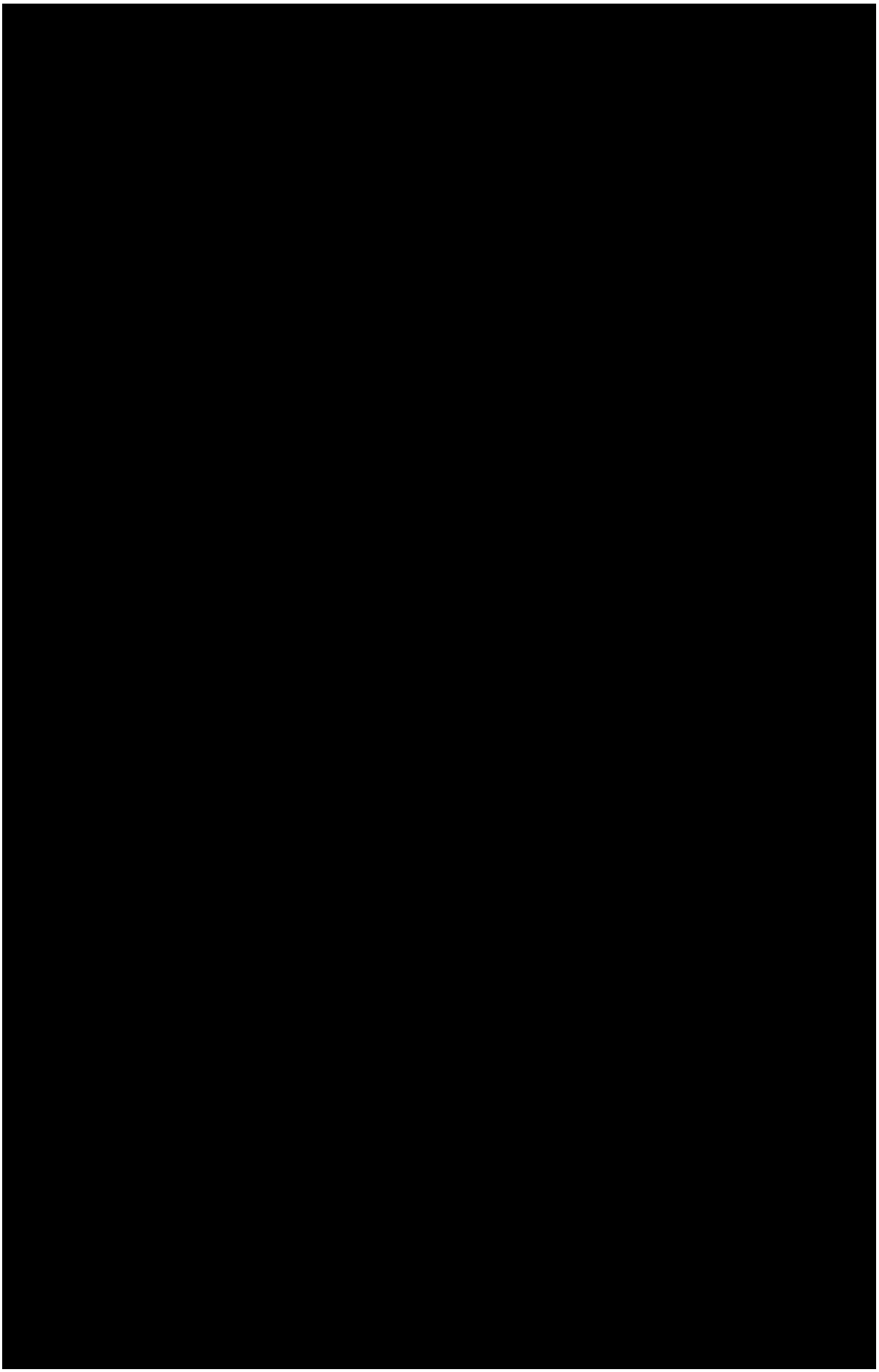


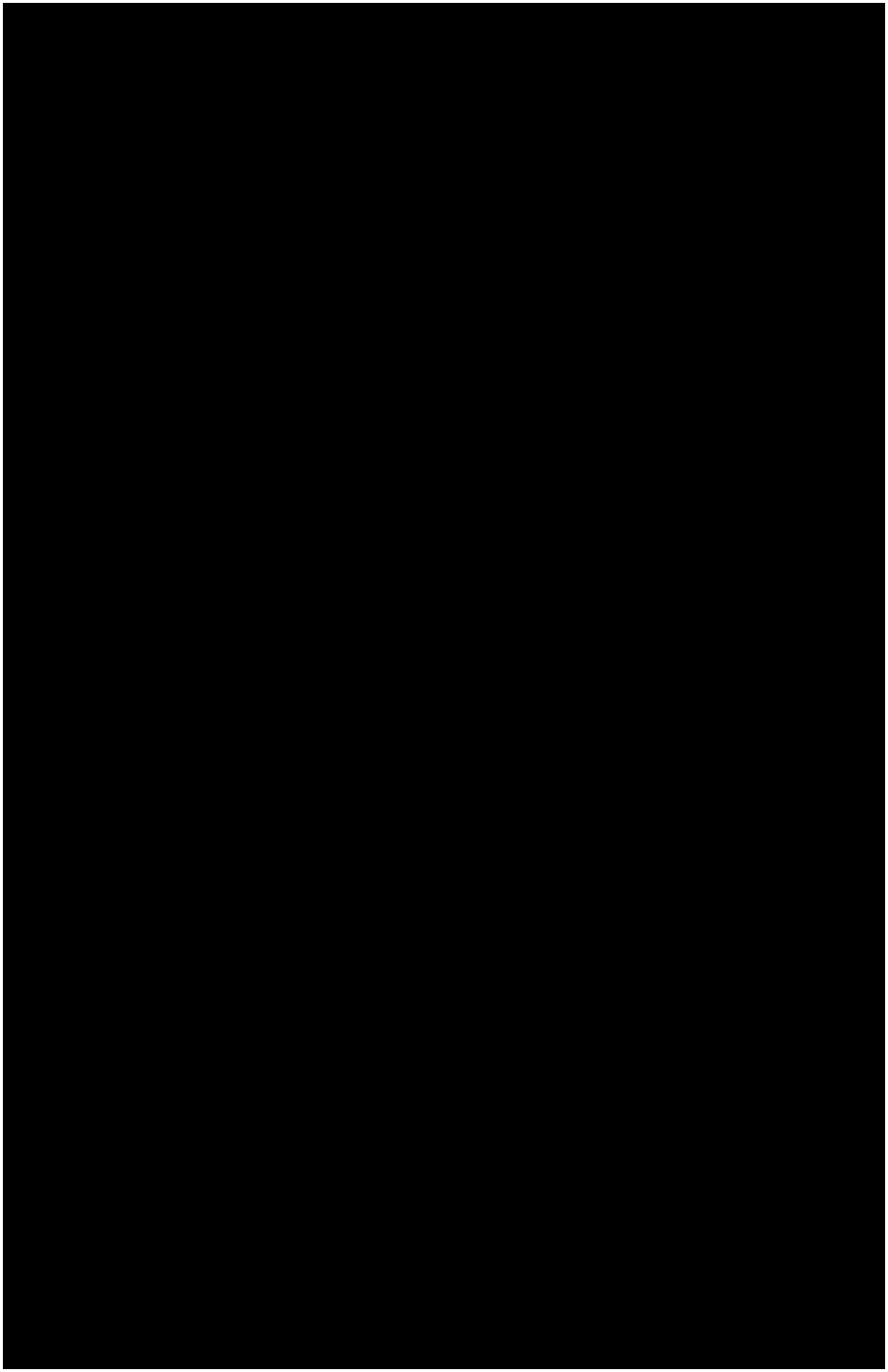




Appendix N: Conners' 3 Teacher Rating Scale







Appendix O: Typically developed sample neurofeedback home training guidelines

This would be personalised for the ADHD sample.

Neurofeedback Home Training Guidelines

I have agreed that .....(participants name) will complete neurofeedback sessions on .....(day) at .....(time) and .....(day) at .....(time) at home with the supervision of .....(parent/guardian).

After every 2 sessions, the researcher will provide the participant will some written feedback via email about their completed sessions.

Pre-Training Software Guidelines

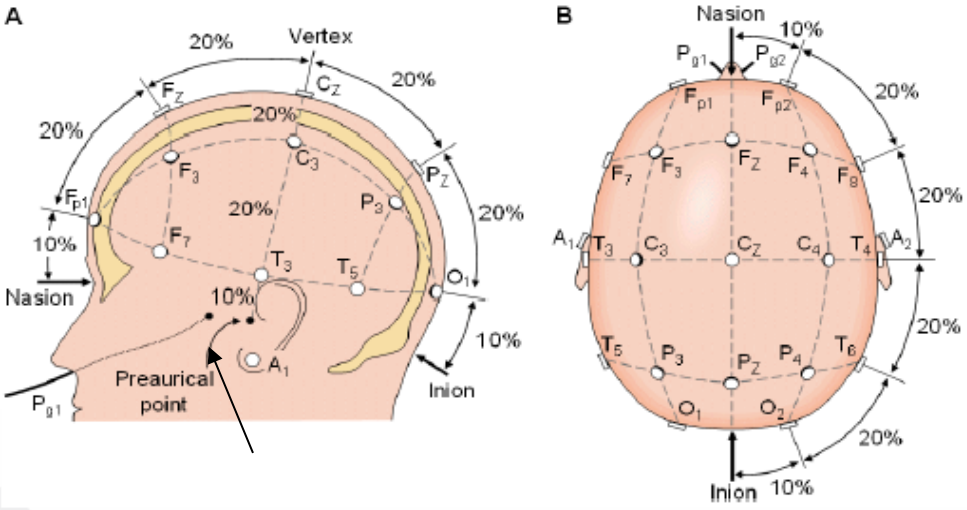
1. Insert the purple dongle.
2. Double click on the appropriate design icon on the desktop for the training session
   - C3 Pacman
   - Cz Boxes
   - C4 Video

Pre-Training Hardware Guidelines

1. Switch PET on. Check light is on.
2. Plug PET USB silver dongle into the computer. You MUST always use the same USB port as identified at identified by your neurofeedback therapist.

Attaching Electrode at C3

1. Clip a disposable electrode onto the black and yellow leads.
2. Strap the PET around the right upper-arm with the electrode leads at the top. Ensure it is Velcro tightly.
3. Clean the skin at the position of the mastoids (see diagram) by rubbing some Nuprep on the skin.
4. Wipe the residual Nuprep off with a tissue.
5. Remove the plastic film and stick the black and yellow electrodes at the position of the mastoids (see diagram)
6. Clip a disposable electrode onto the blue lead.
7. Remove the plastic film and put some Ten20 paste onto this electrode. Spread a thin film of the paste all over the electrode and leave a pea sized amount in the centre of the electrode.
8. Using the diagram below locate the desired electrode location.
9. Clean the desired electrode location by rubbing some Nuprep on the scalp.
10. Position the blue electrode at the identified location. If necessary put a little 3 X 3 cm piece of kitchen towel paper on top and around the electrode to ensure that it remains in its position.



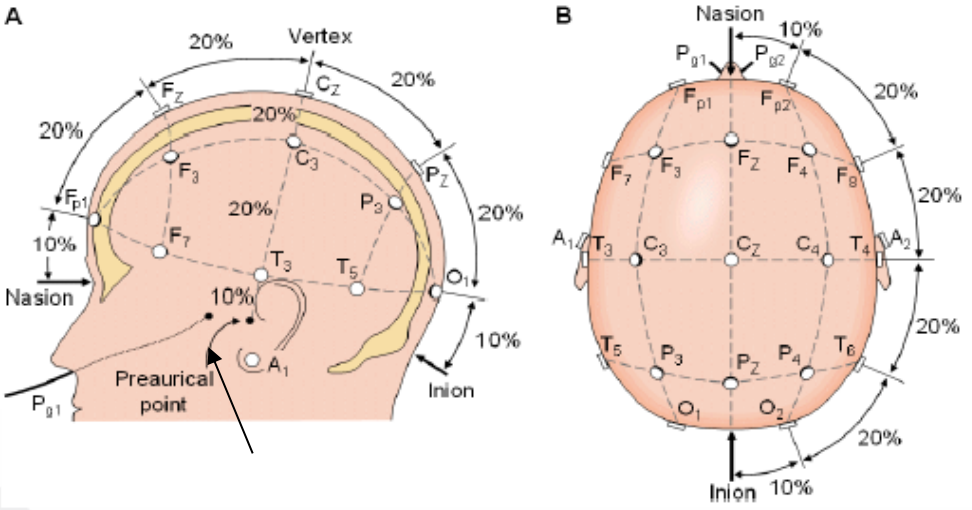
Software Guidelines

1. You need to have the 'Instruments 1' window open, so if necessary click on 'Window', and click on 'Instruments 1'.
2. Click on 'Play' and check for good signal.
3. Click on 'Record'.
4. Save the file including the date and electrode location e.g.....C3Pacman
5. Click Save.
6. Click OK in the 'Session Info' box.
7. Click on 'Window', and select 'Instruments 2'.
8. The client can now do the session.

9. At the end of the session, close down Bioexplorer and select the next appropriate design icon on the desktop for the next session.

Attaching Electrode at Cz

1. Replenish Ten20 paste onto the blue electrode if necessary.
2. Using the diagram below locate the desired electrode location.
3. Clean the desired electrode location by rubbing some Nuprep on the scalp.
4. Position the blue electrode at the identified location. If necessary put a little 3 X 3 cm piece of kitchen towel paper on top and around the electrode to ensure that it remains in its position.

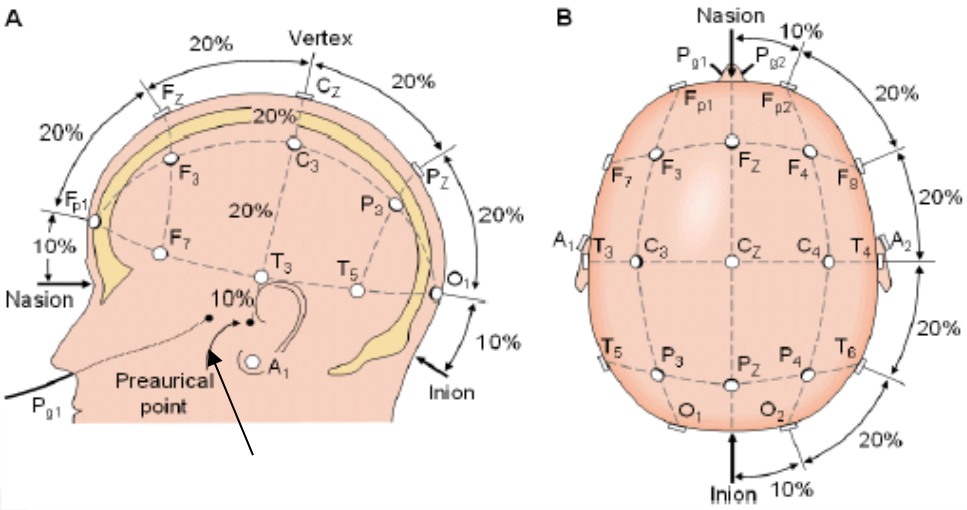


Software Guidelines

1. You need to have the 'Instruments 1' window open, so if necessary click on 'Window', and click on 'Instruments 1'.
2. Click on 'Play' and check for good signal.
3. Click on 'Record'.
4. Save the file including the date and electrode location e.g.....CzBoxes
5. Click Save.
6. Click OK in the 'Session Info' box.
7. Click on 'Window', and select 'Instruments 2'.
8. The client can now do the session.
9. At the end of the session, close down Bioexplorer and select the next appropriate design icon on the desktop for the next session.

Attaching Electrode at C4

1. Replenish Ten20 paste onto the blue electrode if necessary.
2. Using the diagram below locate the desired electrode location.
3. Clean the desired electrode location by rubbing some Nuprep on the scalp.
4. Position the blue electrode at the identified location. If necessary put a little 3 X 3 cm piece of kitchen towel paper on top and around the electrode to ensure that it remains in its position.



Software Guidelines

1. As you are intending to do a session with video feedback you may want to change the videos. To do this click on 'Window', and click on 'Signal Diagram'.
2. You may need to scroll to the right. Right-click on the 'Video-Player 1' box.
3. Click 'Properties'. Click 'Remove' to remove any videos you do not want, and click 'Add' to select those you do. The 'Video' folder is on the desktop, from which the video files can be selected.
4. You need to have the 'Instruments 1' window open, so if necessary click on 'Window', and click on 'Instruments 1'.
5. Click on 'Play' and check for good signal.
6. Click on 'Record'.
7. Save the file including the date and electrode location e.g.....C4Video
8. Click Save.
9. Click OK in the 'Session Info' box.
10. Click on 'Window', and select 'Instruments 2'.
11. The client can now do the session.
12. At the end of the session, close down Bioexplorer and select the next appropriate design icon on the desktop for the next session.

Post-Training Software Guidelines

1. If the session is the final one for the day, close Bioexplorer.
2. Send all saved sessions to hannah.wachnianin@lanc.uk.com

Post-Training Hardware Guidelines

1. At end of session take electrodes off scalp and mastoids.
2. Dispose of electrodes.
3. Unplug the PET USB dongle from the computer.
4. Turn off PET.
5. Unplug the purple dongle.
6. Place all hardware in packaging.

Battery Charging (This can be done at anytime)

1. Remove battery from PET.
2. Insert the battery into the battery charger.
3. Plug the battery charger in to a wall socket.
4. Once charged insert the battery into the PET.
5. Place the battery charger back in the packaging.

Appendix P: Typically developed sample active control group guidelines

Computer Game Training Guidelines

I have agreed that .....(participants name) will complete computer game training sessions on .....(day) at .....(time) and .....(day) at .....(time) at home with the supervision of .....(parent/guardian).

Before starting the games, set a timer for 30 minutes. You can use the following online timer. Simply write the website into your browser http://timer.onlineclock.net/, click on the little drop down arrow and select 30 minutes. Ensure the sound on your computer is on. The timer will start straight aware.

Depending on the child's age, please select the most appropriate website below:

Children aged between 7 and 10 - http://www.bbc.co.uk/bitesize/ks2/

Children aged between 11 and 14 - http://www.bbc.co.uk/schools/ks3bitesize/

Children aged between 15 and 17 - http://www.bbc.co.uk/schools/gcsebitesize/

The child can choose which activities they complete on that website. Please write down which activities are completed, and if a score is given, please write down the score gained. When one activity is completed, the child can choose another until they have completed 30 minutes worth of activities.

After 30 minutes, the online timer will start to beep. Once this has sounded, please close down the website with the timer and the website with the games – the session has ended.

Now email the researcher on [redacted] letting her know that the session has been completed and which activities your child completed.

Appendix Q: Typically developed sample child debrief

CITY UNIVERSITY LONDON

"Effect of Neurofeedback Home Training on Typical Children"

Research Study

Debrief Sheet

Thank you very much for taking part in this research study, conducted by Hannah Wachnianin with City University, London.

The aim of this study is to find if neurofeedback home training can help children to concentrate and if it can influence a child's personality. This is an area of little previous research.

It maybe that those children who receives neurofeedback will have the most improved concentration at school and home, and that those children who receives no neurofeedback will have no changes in academic performance and behaviour. It is hoped that neurofeedback in the home setting will be a success.

If you have any queries about this research or would like to ask any further questions, please contact the researcher or research supervisor using the contact details below.
If you would like to remove your information within the next four weeks, please tell your parents who will let the researcher know. You do not have to give a reason for your withdrawal.

Once again, thank you very much for helping me to complete this research. Your participation is greatly appreciated.
Yours sincerely,

Hannah Wachnianin

Researcher contact details:

Hannah Wachnianin
[redacted]

Appendix R: ADHD sample child debrief

Learning Assessment & Neurocare Centre Ltd

CITY UNIVERSITY LONDON

A feasibility study to investigate the effect of neurofeedback and stimulant medication on children with Attention Deficit Hyperactivity Disorder (ADHD)

Research Study

Debrief Sheet Children

Thank you taking part in this research study which is being conducted by research student, Hannah Wachnianin with City University, London. We hope to see if medication and neurofeedback together help children with ADHD to improve their concentration and being impulsive. We are also looking to see how helpful completing neurofeedback at home is. This is an area with little previous research.

It maybe that children who have medication and neurofeedback will have the most improved concentration compared to children who have no treatment. Also, it is hoped that neurofeedback in the home setting will be a success.

The findings let us know the best combination of treatments to help children with ADHD.

If you have any worries or concerns about this research or would like to ask any further questions, please speak to your parents or the researcher. You will not be in trouble.
Once again, thank you very much for taking part in this research. Your participation is greatly appreciated.
Yours sincerely,

Hannah Wachnianin

Researcher contact details:
Hannah Wachnianin
[redacted]

Appendix S: Typically developed sample parent debrief

CITY UNIVERSITY LONDON

"Effect of Neurofeedback Home Training on Concentration Abilities in Typical Children"

Research Study

Debrief Sheet

Thank you for allowing your son / daughter to take part in this research study, conducted by Hannah Wachnianin with City University, London. The aim of this study is to find if neurofeedback home training helps to improve concentration abilities in children. Additionally, we are looking to see if neurofeedback influences reward responsiveness and inhibition abilities. This is an area with little previous research.

It maybe that children who undergo neurofeedback will have the most improved academic performance and behaviour, and those individuals with no treatment strategies in place will have no changes in academic performance and behaviour. Additionally, it is hoped that neurofeedback in the home setting will be a success.

If you would like information about the results of this research, please contact the researcher who will email you the results as soon as these become available. Additionally, if you would like more information about neurofeedback or would like to receive neurofeedback sessions after your involvement in this study, please contact the Learning Assessment and Neurocare Centre [redacted] who provide a neurofeedback service in [redacted] Furthermore, if you have any concerns about your child's concentration and are interested in an assessment and/or long-term management, the Learning Assessment and Neurocare Centre will be able to help you further. Also, your family doctor will be able to advise you of services within your local area.

If you have any queries about this research or would like to ask any further questions, please contact the researcher or research supervisor using the contact details below. If you have any comments, concerns or observations about the conduct of the study or your experiences as a participant, please contact the Secretary to the Committee [redacted] quoting the project number PSYETH(UPTD) 12/13 56 on [redacted]

Once again, we would like to thank you for your valuable contribution to this research. Your participation is greatly appreciated.

Yours sincerely,

Hannah Wachnianin

[redacted]

Appendix T: ADHD sample parent debrief

Learning Assessment & Neurocare Centre Ltd

CITY UNIVERSITY LONDON

A feasibility study to investigate the effect of neurofeedback and stimulant medication on children with Attention Deficit Hyperactivity Disorder (ADHD)

Research Study - Debrief Sheet Parents

Thank you for allowing your son / daughter to take part in this research study, taking place at the Learning Assessment and Neurocare Centre, conducted by research student, Hannah Wachnianin with City University London. The aim of the study is to find if stimulant medication and neurofeedback together have a positive effect for children with ADHD. Additionally, the effectiveness of completing neurofeedback in the home setting is being examined. This is an area of little previous research.

The findings will inform us and other settings of the best combination of treatments to help children with ADHD to reduce their symptoms.

If you would like to withdraw your data within the next four weeks, please contact the researcher Hannah Wachnianin. You do not have to give a reason for your withdrawal.

If you have any queries about this research or would like to ask any further questions, please contact the researcher or research supervisor using the contact details below. Furthermore, if you would like to know the results from this study, please email the researcher who will provide you with these once they become available. If you have any comments, concerns or observations about the conduct of the study or your experiences as a participant, please contact the Secretary to the Committee [redacted] quoting the project number SREC 13-14 03 E 11 2 2014 on [redacted]

Once again, we would like to thank you for your valuable contribution to this research. Your participation is greatly appreciated.

Yours sincerely,
Hannah Wachnianin

[redacted]