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Short and Long Term Effect of Neurofeedback and Metacognitive Training on Children's Attention Deficit/Hyperactivity Disorder Symptoms

by

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Abstract

This study consists of two parts: evaluating both the short- (part one) and long-term (part two) impact of a 40-session combined neurofeedback and metacognitive training program on the severity of attention deficit hyperactivity disorder (ADHD) symptoms in children. The first study analyzed the existing data of 318 participants, who were 6-17 years old and diagnosed as having ADHD, to investigate the short-term impact of the training. Significant improvements in both hyperactive and inattentive symptoms were reported from pre to post training. A subset of 22 participants were recruited from the first part of the study to examine whether the gain from the 40-sessions of training were maintained at the follow up point. Continuous improvements in both hyperactive and inattentive symptoms were found for an average of 4.86 years after completion of training. Therefore, the results provide evidence for both short- and long-term effectiveness of neurofeedback combined with metacognitive training.

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Chapter 1: Introduction

Context of the Problem

It is typical for preschoolers to be active, move around all the time, have difficulties sustaining attention, and be impatient for their turns because they are curious to explore the world around them. However, if these symptoms persist after the age of 7, they are considered to be developmentally inappropriate, and comprise the core symptoms of Attention Deficit Hyperactivity Disorder (ADHD). According to the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV-TR*), 3%–7% of school-age children suffer from ADHD (American Psychiatric Association, 2000). Specifically in Canada, Romano, Baillargeon, & Tremblay (2002) found that 5% -17% of 2 to11-year-old girls and 9% - 23% of 2 to11-year-old boys often display hyperactive-impulsive behaviours. The presence of the ADHD symptoms frequently impairs student's daily life activities across multiple settings and often persists into adulthood (Polanczyk & Rohde, 2007). Therefore, it is crucial to help these children to improve their behaviours in order for them to reach their full potential.

Typically, medication is the most common and convenient treatment to address the symptoms of ADHD (Banaschewski *et al.*, 2010). Despite the effectiveness of the psychostimulant medications, which is the most commonly prescribed medication to individuals with ADHD (Barkley, 2006), they are criticized for being short-term solutions and for having negative side effects (Connor, 2006). The three primary types of side effects with psychostimulant medications are (1) cardiovascular effects including change of heart rate and blood pressure; (2) physical effects including appetite suppression, weight reduction, insomnia, and headaches; (3) physical and behavioral complaints including daydreaming, irritability, anxiety, staring, and nail biting (Van der Oord *et al.*, 2008).

In addition to these criticisms, 25% of school-age children with ADHD do not respond positively to psychostimulant medication (Rapport & Denney, 2000; Weyandt, 2007). Consequently, in an effort to address these issues interventions geared towards environmental support such as behavioural interventions have been employed to address the symptoms of ADHD (Banaschewski *et al.*, 2010). The goal of behavioural interventions is to alter a student's environment in order for them to replace undesired behaviours with more appropriate behaviours (Fabiano *et al.*, 2009; Banaschewski *et al.*, 2010). Unfortunately, reported drawbacks of this approach are similar to those reported for medications:

effectiveness is not consistent, no evidence supports long-term effectiveness, and not all students respond to treatment (Miller *et al.*, 1998; Waschbusch & Hill, 2003).

For these reasons, many researches have been exploring alternative treatments for individuals with ADHD, including neurofeedback training. Unlike the medication and behavioural approaches, which attempt to suppress or manage the symptoms, this strategy is geared towards changing the way in which the brain is functioning to reduce ADHD symptoms. Responding to research evidence of premature brain wave development in individuals with ADHD (Lubar & Lubar, 1984), the goal of neurofeedback training for the ADHD population is to shift the brain activation towards an age appropriate pattern. In the past 30 years, a growing group of researchers has been investigating how the neurofeedback training affects the symptoms of ADHD in children. Most of them have shown positive results including improvement in attention, academic performance, intelligence quotient (IQ), and social behaviours (e.g. Lubar & Lubar, 1984; Thompson & Thompson, 1998; Vernon, Frick, & Gruzelier, 2004; Fox, Tharp, & Fox, 2005). However, due to identified limitations to this research, including weak research designs, small sample sizes, and limited generalizability, neurofeedback has not been universally adopted as a viable intervention option with this population (Loo & Barkley, 2005). By incorporating a component such as metacognitive training with the neurofeedback program, it allows student to learn and practice skills in their daily life, which can address the generalization criticism. Furthermore, researchers are only just beginning to focus on the long-term effects of neurofeedback training, with some early promising results. For instance, Gani and colleagues (2008) found that half of the students in their study, initially diagnosed with ADHD, no longer met diagnostic criteria at a two year follow up point after a biofeedback intervention. To support this growing body of research, this study will investigate the short-term effects of neurofeedback, using a large sample size and incorporating metacognitive training to improve on the criticisms from pervious studies and will also explore the long-term effects with preliminary inquiry.

Statement of Purpose

This current study will evaluate the short term and long term impact of a 40-session neurofeedback training program combined with metacognitive strategy training. The goals of this study are: (1) to determine whether the number of ADHD symptoms rated by caregivers and the performance in the computerized performance tests changed from pre-training to post-training; and (2) to determine whether the perceived gain made by the children with ADHD after 40 sessions of NFB training is sustained after at least one year completed the training.

Chapter 2: Literature Review

The following section will include (i) an overview of ADHD, including diagnostic criteria, etiology, and treatment of ADHD; (ii) an overview of neurofeedback training, including definition and application to individuals with ADHD; (iii) an overview of the metacognitive training, including definition and application to individuals with ADHD.

Overview of ADHD

ADHD is one of the most commonly diagnosed psychiatric disorders among school-age students (Barkley, 2006). This disorder is characterized by the three core symptoms: inattention, hyperactivity, and impulsivity (Kropotov, 2009). The presence of these symptoms often interferes with the daily functioning of individuals with ADHD. For example, children with ADHD may be more inattentive, be more impulsive, and have poorer concentration than children without ADHD. As a result of these symptoms, children with ADHD may have more difficulty organizing and expressing their ideas in a coherent way in their school work (Weyandt & DuPaul, 2008). Consequently, they often underachieve in school, which in turn may affect their self-esteem (Selikowitz, 2009). In addition, being unable to inhibit their behaviours may disturb other student in class, which results in a lack of friendships (Selikowitz, 2009). Therefore, the ADHD symptoms can cause some serious impacts in these children's lives.

Diagnostic Criteria for ADHD. The first clinical report of ADHD symptoms was made by a German physician Dr. Hoffman in the 1800s (Weyandt, 2007). Since that time the terminology and diagnostic criteria of ADHD has evolved. From minimal brain dysfunction (MBD), to hyperactive child syndrome, to hyperkinetic reaction of childhood, and then to Attention Deficit Disorder (ADD) (Weyandt, 2007) the name applied has reflected changes in our understanding of this disorder. In 1994, revisions were made to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV), which is commonly used in North America to diagnose ADHD, and the diagnostic category was regrouped into three subtypes: ADHD, predominately inattentive type (ADHD-I); ADHD, predominately hyperactive-impulsive type (ADHD-HI); and ADHD, combined type (ADHD-C) (American Psychiatric Association, 1994). The latest version of DSM-IV-TR has maintained these same subtypes (American Psychiatric Association, 2000).

According to DSM-IV-TR diagnostic criteria, the symptoms have to be present for at least 6 months to a degree that causes impairment to school performance, intellectual functioning, social skills, and occupational functioning and must be present before age of 7 years old in order to make a diagnosis of ADHD (American Psychiatric Association, 2000). Specific symptoms are categorized into the previously identified subtypes, and a minimum number are required for a diagnosis to be made. For a diagnosis of ADHD-I, the individual has to fulfill at least six out of nine items on the inattention symptoms list in the DSM-IV-TR, including fail to give close attention to details; have difficulties focusing on the task; miss details in conversation; fail to follow instructions and finish task; have trouble organizing activities; avoid task that required mental effort for a long period of time; lose things needed for tasks; are easily distracted; and have difficulties remembering things (American Psychiatric Association, 2000).

For a diagnosis of ADHD-HI, an individual has to show at least six out of nine items from the hyperactivity & impulsivity list, including fidgets with hands or feet or squirm in seat; have difficulties to remain in their seats; run around in inappropriate situations; talk excessively; have trouble playing or enjoying leisure activities quietly, are always "on the go"; blurt out answers before questions have been finished; have trouble wait in line or take turns during group activities; and interrupt others during activities (American Psychiatric Association, 2000). Since these symptoms are more noticeable, parents and teachers often complain that these motor overflow movements often impair the student's school, occupational, and social life.

Lastly, if both ADHD-I and ADHD-HI conditions are fulfilled, it is classified as ADHD-C (American Psychiatric Association, 2000). In general, these criteria have framed our current understanding of individuals with ADHD: individuals with ADHD are a heterogeneous group with a high variability in the frequency and pervasiveness of the inattention, hyperactivity, and impulsivity symptoms (Weyandt, 2007).

ADHD at Different Developmental Stage. During development, the three core symptoms of ADHD may emerge at different ages or developmental stage due to the interaction of maturation and environmental expectation. At the prenatal, infancy, toddler and preschool years, Marsh, Gerber, and Peterson (2008) found that the typical brain development begins in the sensorimotor areas, so children at this age tend to be more active and explore the world with different movements. Compared with their peers at this age, children with ADHD are even more accident-prone, aggressive, noncompliant, defiant, and have negative temperaments (Weyandt, 2007). The symptoms of hyperactivity and impulsivity are almost always present before age of 7; therefore, the most predominate type of ADHD among these youngsters is the hyperactivity and impulsivity subtypes (Wicks-Nelson & Israel, 2003). During middle childhood, the environmental expectations of school start to increase starting from the elementary school years, and the students need to sustain their attention required for school work. Therefore, more inattentive symptoms or attention problems are often recognized by the parents and teachers at this stage (Weyandt, 2007). As these children with ADHD enter adolescence and adulthood, there is increasing activity in the frontal cortices, which allows for the maturation of higher-order cognitive functions. At this stage, the hyperactive-impulsive symptoms often become less obvious and are internalized as inner feeling of jitteriness, which may still cause impairment in daily life (Barry *et al*., 2003; Hart *et al*., 1995). As a result, these internal hyperactive-impulsive symptoms are often perceived as inattention symptoms in these older age groups. Barkley (2006) stated that 80% of children with ADHD-I or ADHD-C often carried their inattentive symptoms into the adolescence, and 70% of these children continue to retain these symptoms into their adulthood.

Etiology of ADHD. Due to the complexity of this disorder, no single cause has been identified to explain its etiology. Instead, researchers have identified a complex interaction between neurological, biological, and environmental factors to create the symptom cluster of ADHD (DuPaul & Stoner, 2003).

Neurological Factors. Evidence has shown that the maladaptive behaviours in ADHD are associated with brain dysfunction, structural brain differences, hypocortical arousal deficits, executive function deficits, and abnormal levels of neurotransmitters.

Individuals with ADHD have been identified as having a smaller brain volume, smaller right frontal area, and smaller caudate nucleus areas than the control group (Castellanos *et al.*, 2002). In particular, Barkley (2006) notes that although in typically developing children the size of the caudate nucleus increases with age, this is not the case for individuals with ADHD. Furthermore, using magnetic resonance imaging (MRI) researchers have found that children without ADHD have a left greater than right caudate asymmetry whereas children with ADHD have a right greater than left caudate asymmetry (Schrimsher, Billingsley, Jackson, & Moore, 2002). The caudate nucleus is associated with voluntary motor control, learning, and memory. Schrimsher *et al.* (2002) and Konrad & Eickhoff (2010) suggested that the anatomical difference of the caudate in individuals with ADHD is correlated with the inattention, hyperactivity, and impulsivity symptoms in individuals with ADHD.

Besides the structural brain difference, another plausible cause of hyperactivity in individuals with ADHD is hypocortical arousal deficit. Several studies have indicated that children with ADHD have decreased cerebral blood flow and glucose utilization in the prefrontal regions and pathways connecting it to the limbic system (caudate nucleus and globus pallidus) through the striatum (Banaschewski, *et al.*, 2010). As a result, the metabolic rate of the frontal area in children with ADHD is slower than in children without ADHD (McEwan, 1998). The under-aroused frontal lobe is believed to cause the hyperactivity symptoms in the individuals with ADHD as the hyperactive movements function to help their under-aroused brain maintain a normal level of arousal (Barkley, 2006).

As a result of cortical hypoarousal, different brain wave development has been identified in individuals with ADHD. Oades (2005) and Barry *et al.* (2003) found that individuals with ADHD have an increase in theta waves and a decrease in beta waves in the frontal region of the brain, which is similar to a younger individual's brain pattern. This immature brainwave development in the frontal lobe is associated with difficulty staying focused and remaining calm.

Barkley (2006) proposed that the abnormality in the frontal lobe reported in the previous section may be associated with executive functions. Barkley's proposed model of executive function deficits in individuals with ADHD involves four kinds of abilities: (1) nonverbal working memory, (2) verbal working memory, (3) self-regulation, and (4) reconstitution. First, with poor nonverbal working memory, individuals with ADHD are unable to sense past and future, have limited self-awareness, and have problems storing and recalling facts from long-term memory (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Second, delayed verbal working memory inhibits individuals with ADHD from internalizing speech and self-questioning to control their behaviours and direct future actions (Spencer, Biederman, Wilens, & Faraone, 2002). Third, immature self-regulation limits individuals with ADHD in the self-regulation of their emotion, motivation, and arousal (Barkley. 2006). Lastly, impaired reconstitution hinders individuals with ADHD in their abilities to take a complex problem apart, analyze it, and reorganize the pieces into new ideas (Barkley. 2006). Most individuals with ADHD have these executive function deficits; however, due to the heterogeneity in ADHD, not all individuals with ADHD would experience all of these problems with executive function (Lambek, Tannock, Dalsgaard, Trillingsgaard, Damm & Thomsen, 2010).

Another plausible cause of ADHD is the imbalance of neurotransmitters. Four neurotransmitters have been linked to ADHD symptoms: serotonin, dopamine, epinephrine, and norepinephrine (Weyandt, 2007). Due to the complexity of the brain, no single neurotransmitter has been identified as the leading cause. High levels of serotonin have been found in individuals with ADHD, which is associated with the impulsivity symptoms in ADHD (Novkovic, Rudan, Pivac, Nedic, & Muck-Seler, 2009); however, this is not unique to ADHD since this neurotransmitter is also implicated in other disorders such as impulsive personality disorders, violent behaviour, and depression (Strauss, 2010). Another set of neurotransmitters, known as catecholamines (dopamine, epinephrine, and norepinephrine), have been linked to alertness, attention, and motor control (Weyandt, 2007). Irregular levels of catecholamines, such as dopamine, are caused by the structural differences in the limbic area (caudate nucleus and globus pallidus) as mentioned above. As a result of the malformed dopamine network, individuals with ADHD have more difficulties in sustaining attention. In general, disturbances in serotonergic and catecholaminergic systems are commonly found in individuals with ADHD (Strauss, 2010).

Biological Factors. In addition to the neurological factors, genetic factors are another strong predictor of ADHD. Twins studies showed that the concordance rate in monozygotic twins is 79% and in same-gender dizygotic twins is 32% (Millichap, 2008). Similarly, other identical and fraternal twins, siblings, and family studies have provided further evidence that ADHD symptoms are highly heritable (Banaschewski, et al., 2010). Consequently, many researchers have taken this inquiry to a molecular genetic level in an attempt to identify the "ADHD gene" (Barkley, 2000). The researchers investigated the relationship between the genes associated with the production, regulation, and functioning of the specific neurotransmitters and the phenotypic expression of ADHD symptoms (Weyandt, 2007). Although no single specific gene was identified, the most studied genes are dopamine-related genes: the dopamine receptor gene (DRD-4) and the dopamine transporter gene (DAT-1) (DuPaul & Stoner, 2003). DRD-4 is a gene on chromosome 11 that allows the dopamine receptor to inhibit complex dominant transmission pathway (DuPaul & Stoner, 2003). Spencer et al. (2002)

showed that 50-60% of children with ADHD share this defective DRD-4 gene with their families whereas only 30% of the non-ADHD families share this gene. This evidence supported that the biological factors interplay with the neurological factors to define the fundamental cause to ADHD.

Environmental Factors. In addition to biological and neurological influences, the environment has been identified as playing a significant role in affecting the expression of ADHD symptoms. Zappitelli, Pinto, & Grizenko (2001) suggested environmental toxins, including maternal smoking, prenatal alcohol use and increased exposure to lead, may increase the risk of developing ADHD. The nicotine in tobacco could cause damage to the dopamine system in the fetus' brain, which leads to attention deficit (Zappitelli et al., 2001). Similarly, any fetal alcohol exposure could initiate attention and memory problems. Exposure to lead may come from lead-based paint, ceramic dishes, and old copper pipes, which may result in serious biological, cognitive, and behavioural damage; however, only less than 4% of individuals with ADHD are caused by constant exposure to high levels of lead (Barkley, 2006). Moreover, high sugar and low vitamin diet may increase the neurotransmitters imbalance and exhibit ADHD-related symptoms, such as irritable and inattentive (Sears & Thompson, 1998). With the exposure in the hazardous environment, it will increase the chance of triggering the ADHD symptoms.

Since ADHD is a complex disorder, the etiology is a combination of neurological, biological, and environmental factors. The genetic deficit is probably the primary cause that triggers the neurological imbalance in the individuals with ADHD, and the environmental factors may contribute as a secondary cause (Millichap, 2008).

Treatments for ADHD

With such a wide range of known contributors to the development of ADHD, researchers have similarly addressed neurological, biological, and environmental influences in developing the treatment for individuals with ADHD.

Medication and ADHD. For many years, medication has been the most popular treatment for ADHD. Aside from the side effects, ADHD medications often provide short-term benefit in improving students' academic, social, and behavioural functioning. Many studies including randomized, double-blind, placebo-controlled clinical trials confirmed the effectiveness of the ADHD medication (Connor, 2006; Monastra et al., 2002; Greenhill, 2001). Among all ADHD medications, psychostimulant medication is the most common medication prescribed to individuals with ADHD. Psychostimulant medication function by inhibiting the dopamine reuptake to increase the arousal in the under-aroused central nervous system (CNS) which in turn improves attention, concentration, motion planning, motor control, and self-regulation (Barkley, 2006). Out of all psychostimulant medications, methylphenidate is the most popular: 80% of children with ADHD have been treated with methylphenidates (DuPual & Stoner, 2003). Yet, 30% of the children with ADHD did not respond to psychostimulant medications, so other medication such as antidepressant and antihypertensives may be recommended (Bender, 1997). In addition, psychostimulant medication also carries some adverse side effects including decreased appetite, weight loss, irritability, sleep disturbances, headaches, stomachaches, mood changes, abdominal pain, and nervous tics (Weyandt, 2007). In addition, children who have motor tics and/or anxiety disorders might not be good candidates for this treatment, and other medication might be a more appropriate option for this population (Bender, 1997).

Although medication is the most common form of treatment for ADHD, it is only effective in reducing the symptoms in the short term and has no long term benefical effects. Jensen *et al.*'s 3 year follow-up study (2007) showed that the behavioural improvements in individuals with ADHD while using medication were no longer present at 3-year follow-up point after medication had been discontinued. With no long-term improvement evident, aversive side effects, and large number of non-response rate to medication, researchers started to seek for alternative form of treatment including neurofeedback training.

Behavioural Intervention and ADHD. Behavioural intervention takes a different approach to treatment of ADHD, and it is grounded in learning theory (Fabiano *et al.*, 2009). Most behavioural interventions are geared towards modification of a student's environment in order to help students learn to reduce problematic behaviours and replace them with more desirable behaviours. For example, caregivers or teachers can increase positive behaviour through praise, positive attention, and tangible rewards and reduce unwanted behaviour through planned ignoring, time-out, and other non-physical discipline techniques (Knight, Rooney & Chronis-Tuscano, 2008). Although behavioural interventions are widely used, their effectiveness is not consistent, there is no evidence that supports long-term effectiveness, and not all students respond to this approach

(Miller *et al.*, 1998; Waschbusch & Hill, 2003).

Neurofeedback. Neurofeedback is a type of behavioural training which allows users to develop skills to self-regulate their own brain activity. By attaching electrodes to the human scalp, the brain's activity patterns, measured through electroencephalograms (EEG), can be recorded and displayed as images on a computer screen virtually instantly (Heinrich, Gevensleben, &Strehl, 2007; Thompson & Thompson, 1998). The process of neurofeedback is to learn how to alter and normalize undesired brainwave pattern by manipulating the image into a desired brainwave pattern (Thompson & Thompson, 1998). Based on the operant conditioning theory, the behaviour of producing a particular desired brainwave patterns will strengthen if users are rewarded for producing ideal brainwave patterns during their neurofeedback training; for example, the award could be achieving certain audio and visual cues on the computer screen (Thompson & Thompson, 1998). The goal is to be able to recognize the different cognitive states, such as being inattentive and focused, and learn to regulate the desired brainwave pattern through neurofeedback.

Neurofeedback Training for ADHD. Based on research evidence of premature brain wave development for individuals with ADHD (Oades, 2005), the goal of neurofeedback training for individuals with ADHD is to shift the EEG activation towards an age appropriate pattern by decreasing the theta waves and increasing the beta waves. The pioneer ADHD neurofeedback researchers Lubar & Lubar (1984) found that the theta-beta work on individuals with ADHD yielded positive results with improvements towards desired brainwave patterns and subsequent reductions in observed ADHD behaviours. After the 1990s, more research was focused on evaluating the short-term effectiveness of neurofeedback training in individuals with ADHD. Most researchers such as Fuchs et al. (2003), Thompson & Thompson (1998), found positive results including both cognitive and behavioural improvement. Furthermore, Monastra et al. (2002) evaluated the effects of Ritalin, neurofeedback, and parenting style on the primary symptoms of ADHD, and they reported the effectiveness of neurofeedback training is equivalent to medication. Recently, some researchers found that there is a potential for long term maintenance even after completing neurofeedback training sessions (Gani et al., 2008; Monastra et al., 2002).

Metacognition Training and ADHD. Metacognition was first introduced by Flavell (1979), who defined it as the experience and knowledge that people have about their own cognitive processes (Perfect & Schwartz, 2002). Flavell (1979) reported that metacognition goes beyond learning or behavioural strategies, as it includes the monitoring and regulating of one's own cognitive progress and to ensure that a cognitive goal has been met. Therefore, students with good metacognitive skills can monitor their learning process, plan what strategies to use and when to use each strategy in a given situation, and increase awareness of their cognitive outcome. Researchers, such as Fernandez-Duque, Baird, & Posner (2000) and Bryce (2007) have investigated the relationship between executive functioning and metacognition. Although the precise relationship between executive functions and metacognition is not yet established, it is clear that executive function is a necessary component of metacognition (Whitebread *et al.*, 2010). Given that students with ADHD have deficits in their executive function (Barkley, 2006), use of metacognitive strategies has been considered as another way to help students think about thinking and reflect on what they know about knowing (Thompson & Thompson, 1998). Moreover, Solanto, Marks, Wasserstein, Mitchell, Abikoff, Alvir,. & Kofman (2010) found that metacognitive therapy alone produced significant improvements in severity of

ADHD symptoms. Therefore, students were able to learn the necessary skills to increase awareness of their learning processes and organize and synthesize materials to finish tasks.

Rationale of Combined Training. By combining metacognitive training with neurofeedback training two goals are accomplished: (1) addressing the academic underachievement for the students with ADHD by teaching them learning strategies and (2) increasing the generalization of the neurofeedback training effect by practicing academic tasks that are similar to the ones students encounter in other non-treatment settings while learning to produce the desired mental state (neurofeedback training) at the same time. Thompson & Thompson (1998) have provided early positive results for intervention approaches that combined these two approaches. Specifically, they found that students with ADHD showed significant improvements in EEG theta/beta ratio, ADHD symptoms, Intelligence Quotient (IQ) score, and academic performance after 40-sessions of training.

Conclusion from the Literature

The development of ADHD is the result of the ongoing interactions between the neurological, biological, and environment factors. Many researches supported that ADHD is a neurologically-based disorder due to the evidence of structural brain difference, hypo-cortical arousal deficit, executive function deficits, and abnormal levels of neurotransmitters. Typically, medication is used to target these deficits in the brain; however, the effect of medication is short-lived and a number of students with ADHD do not respond to the medication. Similarly, the effectiveness of behavioural intervention is not consistent. Therefore, alternative treatments such as neurofeedback training are started to blossom. Neurofeedback training is focused on restoring the abnormal brainwave pattern in individuals with ADHD, and consequently reducing the ADHD symptoms. In the past decades, many studies have investigated the effectiveness of neurofeedback training for individuals with ADHD and found positive results in alleviating ADHD behaviours. Yet, both short-term effectiveness studies with a larger sample size and long-term effectiveness studies are still lacking at the present moment.

Based on the neurological etiology and criticisms of the commonly used treatments, the combination of neurofeedback training and metacognitive training may be able to address the ADHD symptoms. This research consisted of two related studies in which both the short- and long-term effectiveness of the combined training was investigated. The research questions in this study were as follows:

(1) Whether the number of ADHD symptoms rated by caregivers and the performance in the computerized performance tests changed from pre-training to post-training?

(2) Is there any factors including gender, age, intelligence level, subtype of ADHD, and medication intake that will moderate the training effects?

(3) Whether the perceived gain made by the children with ADHD after 40 sessions of NFB training is sustained.

The first study is a secondary data analysis, which will examine the short-term effects of the neurofeedback training combined with the metacognitive training by determining whether there is a gain after having 40-sessions of neurofeedback training based on both parent rating questionnaires and computerized performance tests. This will provide an up-to-date evaluation of the combined intervention with a larger sample size, which hopefully will converge with the previous evaluation of this combined program, which was done in 1998. Aside from this primary objective, since the ADHD symptom expression may influence by gender (Gaub& Carlson, 1997), age (Marsh, Gerber, and Peterson, 2008), subtype of ADHD (Loo & Barkley, 2005), intelligence level (Cooter & Cooter, 2004), and medication intake (Loo & Barkley, 2005), these potential moderators are going to be examined to determine possible factors that influence the training effect.

Gender. The meta-analysis from Gaub& Carlson (1997) concluded that there is no gender difference in impulsivity symptoms, academic performance, social functioning, and fine motor skills, but girls with ADHD showed more intellectual impairment, less hyperactivity symptoms, less inattention symptoms, less peer aggression, and less externalizing and internalizing behaviours. Although the research study showed there is a slight gender difference, the findings from this study does not support that there is a gender difference in the treatment effect.

Age. Given that developmental evidence mentioned in the previous section suggesting that the brain development is different during childhood and adolescence, the present study will examine the training effects between children (6-12 of age) and adolescents (13-17 of age).

Subtype of ADHD. Evidence suggests that different ADHD subtypes have their unique EEG patterns. Loo & Barkley (2005) also mentioned that individuals with ADHD- Combined Type displayed greater underarousal and more maturational delay than those with ADHD- Inattention Type; however, Thompson & Thompson (1998) suggested that both subtypes showed significant improvement after receiving the 40-sessions of neurofeedback training. Therefore, it is expected that different subtypes of ADHD will not affect the training effect.

Intelligence Level. As Lubar & Lubar (1984) mentioned that neurofeedback training based on operant learning theory to increase self-awareness of their own brain activity. Therefore, students with below average Full Scale Intelligence Scale (FSIQ) (between 70 and 85), as known as the slow learners (Cooter & Cooter, 2004), may need more time to achieve the same treatment effect as students who have average and above FSIQ.

Medication Intake. Loo & Barkley (2005) suggested that neurofeedback training is a form of behavior therapy. If this is the case, the students having medication intake throughout the training will show more improvements in both ADHD symptoms than the students that did not have medication.

The second study is a pilot study that extends the work from Thompson & Thompson (1998) to determine whether the perceived gain made by the children with ADHD after 40 sessions of NFB training is sustained for a subsample from the first study.

References

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders DSM-IV-TR Fourth Edition (Text Revision)*. Washington, DC: American Psychiatric Publishing, Inc.

Banaschewski, T., Coghill, D., Santosh, P., Zaddas, A., Asherson, P., Buitelaar, J., Dankaerts, M., Dopfner, M., Faraone, S.V., Rothenberger, A., Sergeant, J., Steinhausen, H.C., Sonuga-Barke, E.J.S., & Taylor, E. (2006). Long-acting medications for the hyperkinetic disorders: A systemic review and European treatment guideline. *European Child and Adolescent Psychiatry*, 15, 476-495.

Barkley, R. A. (2006). *Attention Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment (3rd ed.)*. New York, NY: Guildford Press.

Bender, W.N. (1997). *Understanding ADHD: A Practical Guide for Teachers and Parents*. Columbus, Ohio: Prentice-Hall.

Barry R.J., Clarke A.R. & Johnston, S.J. (2003). A review of electrophysiology in Attention-Deficit/Hyperactivity Disorder: I. Qualitative and quantitative electroencephalography. *Clinical Neurophysiology*, 114:171–183.

Bryce, D. (2007). Investigating the relationship between metacognitive skill and executive functions: A pilot study to assess developmental changes in four to eight-year-olds. Unpublished MPhil thesis, University of Cambridge, UK.

Castellanos, F.X., Lee, P.P., Sharp, W., Jeffries, N.O., Greenstein, D.K., Clasen, LS., *et al.* (2002). *Developmental trajectories of brain volume abnormalities in children and adolescents with attention-deficit/hyperactivity disorder.* Journal of the American Medical Association, 288, 1740-1748.

Connor, D.F. (2006). Stimulants. In R.A. Barkley (ed.), *Attention Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment (3rd ed.,* pp. 608-648). New York, NY: Guilford Press.

DuPaul, G.J. & Stoner, G. (2003). *ADHD in the Schools* (2nd ed.). New York, NY: Guildford Press.

Fernandez-Duque, D., Baird, J. A., & Posner, M. I. (2000). Executive attention and metacognitive regulation. *Consciousness and Cognition*, 9, 288–307.

Flavell, J. H. (1979). Metacognitive and cognitive monitoring: a new area of cognitive developmental inquiry. *American Psychologist,* 34, 906– 911.

Fox, D. J., Tharp, D. F., & Fox, L.C. (2005). Neurofeedback: An Alternative and

Efficacious Treatment for Attention Deficit Hyperactivity Disorder. *Applied Psychophysiology and Biofeedback,* 30(4), 365-373.

Heinrich, H., Gevensleben, H., & Strehl, U. (2007). Annotation: Neurofeedback – train your brain to train behaviour. *Journal of Child Psychology and Psychiatry*, 48(1), 3-16.

Jensen, P.S., Arnold, L.E., Swanson, J.M., Vitiello, B., Abikoff, H.B., Greenhill, L.L., Hechtman, L., Hinshaw, S.P., Pelham, W.E., Wells, K.C., Conners, K., Elliott, G.F., Epstein, J.N., Hoza, B., March, J.S., Molina, B.S.G., Newcorn, J.Y.H., Severe, J.B., Wigal, T., Gibbons, R.D., & Hur, K. (2007). 3-year follow-up of the NIMH MTA study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46(8), 989-1002.

Konrad, K. & Eickhoff, S.B. (2010). Is the ADHD Brain Wired Differently? A Review on Structural and Functional Connectivity in Attention Deficit Hyperactivity Disorder. Human Brain Mapping, 31, 904-916.

Kropotov, J.D. (2009). *Quantitative EEG, Event-Related Potentials and Neurotherapy*. Boston, MA: Academic Press.

Lambek, R., Tannock, R., Dalsgaard, S., Trillingsgaard, A., Damm, D., Thomsen, P. H. (2010). Validating neuropsychological subtypes of ADHD: how do children *with* and *without* an executive function deficit differ? *Journal of Child Psychology & Psychiatry*, 51(8), 895-904.

Lubar, J. O., & Lubar, J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. *Biofeedback and Self-Regulation*, 9 (1), 1 - 23.

Lubar, J.F. (1991). Discourses on the development of diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback and Self Regulation*, 16(3), 201-224.

McEwan, E.K. (1998). *The principal's guide to attention deficit hyperactivity disorder*. Thousand Oaks, California: Corwin Press.

Millichap, J.G. (2008). Etiologic Classification of Attention-Deficit/Hyperactivity Disorder. 121(2), 358-365**.**

Monastra,V. J., Monastra, D.M.,& George, S. (2002). The effects of stimulant therapy, EEG biofeedback, and parenting style on the primary symptoms of attention-deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback*, *27,* 231–249.

Novkovic, V. H., Rudan, V., Pivac, N., Nedic, G., & Muck-Seler, D. (2009). Platelet Serotonin Concentration in Children with Attention-Deficit/

Hyperactivity Disorder. *Neuropsychobiology*, 59 (1), 17-22.

Oades, R.D. (2005). *Brain Maturation - it covers three decades: Considerations of the development of ADHD.* Essen, Germany: University Clinic for Child and Adolescent PsychiatryPerfect, T.J. & Schwartz, B.L. (2002). *Applied Metacognition*. New York, NY : Cambridge University Press.

Polanczyk, G. & Rohde, L.A. (2007). Epidemiology of attention-deficit/hyperactivity disorder across the lifespan. Current Opinion in Psychiatry, 20(4): 386-392.

Rapport, M.D., & Denney, C.B. (2000). Attention deficit hyperactivity disorder and methylphenidate: Assessment and prediction of clinical response. In L.L. Greenhill & B.B. Osman (Eds.), *Ritalin: Theory and practice* (2nd ed., pp. 45-70). Larchmont, NY: Mary Ann Liebert.

Romano, E., Baillargeon, R. H., & Tremblay, R.E. (2002). *Prevalence of Hyperactivity-Impulsivity and Inattention Among Canadian Children: Findings from the First Data Collection Cycle (1994-1995) of the National Longitudinal Survey of Children and Youth: Final Report*. Ottawa, Ontario: Human Resources Development Canada.

Schrimsher, G. W., Billingsley, R. L., Jackson, E.F., Moore, III, B. D.. (2002).

Caudate Nucleus Volume Asymmetry PredictsAttention-Deficit Hyperactivity Disorder Symptomatology inChildren. *Journal of Child Neurology*, 17 (12), 877-885.

Sears, W. & Thompson, L. (1998). The A.D.D. Book: New Understandings, New Approaches to Parenting Your Child. New York: Little, Brown & Co.

Solanto, M.V., Marks, D.J., Wasserstein, J., Mitchell, K., Abikoff, H., Alvir, J.M.J. & Kofman, M.D. (2010). Efficacy of Meta-Cognitive Therapy for Adult ADHD. *The American Journal of Psychiatry*, 167, 958-968.

Spencer, T.J., Biederman, J., Wilens, T.E., & Faraone, S.V. (2002). Overview and neurobiology of attention-deficit/hyperactivity disorder. *Journal of Clinical Psychiatry*, 63(Suppl12), 3-9.

Strauss, L. (2010). Attention-Deficit/Hyperactivity Disorder. *Journal of Biomedical Therapy*, 4 (1), 18-21.

Thompson, L, & Thompson, M. (1998). Neurofeedback combined with training in metacognitive strategies: Effectiveness in students with ADD. *Applied Psychophysiological Biofeedback, 23*, 243-263.

Van der Oord S., Prins P.J., Oosterlaan J., & Emmelkamp P.M. (2008). Efficacy of methylphenidate, psychosocial treatments and their combination in school-aged children with ADHD: a meta-analysis. *Clinical Psychology Review*, 28,783–800.

Vernon, D., Frick, A, & Gruzelier, John. (2004). Neurofeedback as a treatment for ADHD: A methodological review with implications for future research. Journal of Neurotherapy. Vol.8(2), 53-82.

Weyandt, L.L. (2007). *An ADHD primer (2nd ed.).* Mahwah, N.J.: Lawrence Erlbaum Associates.

Whitebread, D., Almeqdad, Q., Bryce, D., Demetriou, D., Grau, V. & Sangster, C. (2010). Chapter 11: Metacognition in Young Children: Current Methodological and Theoretical Development. In Efklides, A. & Misailidi, P. (Eds.), *Trends and prospects in metacognition research* (pp. 233-258). New York, NY : Springer Science and Business Media.

Wicks-Nelson, R. & Israel, A.C. (2003). *Behavior Disorder of Childhood* (5th ed.). New Jersey: Pearson Education, Inc.

Willcutt, E.G., Doyle, A.E., Nigg, J.T., Faraone, S.V., & Pennington, B.F. (2005). Validity of the Executive Function Theory of Attention-Deficit/Hyperactivity Disorder: A Meta-Analytic Review. Biological Psychiatry, 57 (11), 1336-1346.

Zappitelli, M., Pinto, T., & Grizenko, N. (2001). Pre-, Peri-, and Postnatal Trauma in Subjects With Attention-Deficit Hyperactivity Disorder. *Canadian Journal of Psychiatry,* 46(6), 542-548.

Chapter 3: Effectiveness of Neurofeedback and Metacognitive Training on Children's Attention Deficit/Hyperactivity Disorder Symptoms: A Secondary Data Analysis

Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder, which is clinically characterized by attention deficits, hyperactivity, and impulsivity (American Psychiatric Association, 2000). This is one of the most commonly diagnosed psychiatric disorders among school-age students (Barkley, 2006). Meta-regression analysis by Polanczyk *et al*. (2007) found that the worldwide prevalence rate was 5.29%; specifically, 6.5% of school-age children and 2.7% of adolescents were diagnosed with ADHD. The presence of these ADHD symptoms may result in significant impairments in students' social, academic, or occupational functioning. For example, students with ADHD often have poor concentration and organization skills, which affect their ability to organize and express their ideas in a coherent way in writing assignments (Weyandt & DuPaul, 2008). As a result of the academic underachievement, students may have a low sense of self-esteem (Selikowitz, 2009). In addition, a student's inability to inhibit their behaviours, such as, difficulty waiting for their turns or touching other students in class, may lead to a lack of friendships (Selikowitz, 2009). Since these ADHD symptoms have serious impacts in student's lives, it is important to search for effective intervention to reduce these ADHD symptoms.

The most common mainstream treatments for individuals with ADHD are medication and behavioural intervention (Banaschewski *et al.*, 2010). Typically, medication is considered as the first-line treatment for ADHD (Centers for Disease Control and Prevention, 2005). Barbaresi *et al.* (2002) found that 86.5% of children with ADHD were being treated by psychostimulant medication. Despite its effectiveness, medication is only able to provide short-term reduction of ADHD symptoms (Wigal, 2009), it has negative side-effects (Wigal, 2009; Van der Oord *et al.*, 2008), and it does not work for about 25% of individuals diagnosed with ADHD (Connor, 2006). Therefore, medication may not be the best solution to treat ADHD symptoms.

Behavioural intervention takes a different approach in comparison to the medication, and it is grounded in learning theory (Fabiano *et al.*, 2009). Most behavioural interventions involve modification of a student's environment in order for students to learn to reduce problematic behaviours and replace them with more desirable behaviours. For example, caregivers or teachers can increase positive behaviour through praise, positive attention, and tangible rewards and reduce unwanted behaviour through planned ignoring, time-out, and other non-physical discipline techniques (Knight, Rooney & Chronis-Tuscano, 2008). Although behavioural intervention is widely used, it does not have a consistent effect, there is no evidence of long-term effectiveness, and high number of individuals who do not respond to the intervention (Miller *et al.*, 1998; Waschbusch & Hill, 2003). Due to the limitations of both medication and behavioural interventions other approaches have been explored to help individuals with ADHD.

Neurofeedback and ADHD

Neurofeedback is a complementary treatment option for individuals with ADHD that has existed for almost 30 years (Gevensleben *et al.*, 2009). The goal of neurofeedback training is to modify the immature brainwave development in individuals with ADHD, who typically have excess theta and decreased beta activity in the frontal lobe in people with ADHD (Oades, 2005). Unlike medication and behavioural intervention, neurofeedback combined both neurological and operant learning components to equip users to develop the skills to self-regulate their own brain activity, which in turn is believed to reduce their ADHD symptoms. Lubar& Lubar (1984) are pioneers when it comes to examining behavioural changes in individuals with ADHD after neurofeedback training. They found that training children with ADHD to increase Sensorimotor EEG rhythm (SMR: 12-15Hz) or beta activity (16-20 Hz) and inhibit theta activity (4-8 Hz) helps these children to reduce their ADHD symptoms. After Lubar's first publication of successful treatment of a child with ADD using neurofeedback in 1976, clinicians started to employ this intervention with individuals with ADHD and researchers started to investigate the effectiveness of this intervention.

Monastra *et al.* (2002) conducted a randomized, controlled study to evaluate the effects of Ritalin and neurofeedback on the primary symptoms of ADHD by comparing the medication group against the combined group (medication plus neurofeedback). In addition, both groups also received school consultation and parenting counselling. They found that both groups showed significant improvement. Monastra & Monastra (2004) followed up with forty-three participants from the initial study two years after the treatment ended. They found that 70% of the participants in the combined group had reduced their medication dosage, whereas 85% of the participants in the medication group increased their dosage. Similarly, Fuchs *et al.* (2003) also evaluated the effectiveness of both stimulant medication and neurofeedback treatment. The finding from their study showed significant improvements on parent and teacher behavioural ratings as well as computerized tests of attention in both groups. Gevensleben *et al.* (2009) also found that the neurofeedback group training resulted in greater decline in ADHD symptoms than the control groups (computerized attention skills training) based on parent and teacher ratings.

Despite the efforts in evaluating the effectiveness of neurofeedback training, Loo and Barkley (2005) commented that methodological problems such as no random assignment, no control for additional therapist time, and small sample size in the studies conducted shed doubt on the positive findings, and that results may simply reflect time spent with therapists rather than from the training itself. Another criticism Loo and Barkley (2005) suggested is generalization to non-clinicial settings. They proposed that this problem may be addressed by setting up a natural environment in the training session such as building in tasks that students encountered in school or at home. Last criticism made by Loo and Barkley (2005) was that neurofeedback training is possibly another form of cognitive-behavioural therapy because there is no evidence showing the treatment effects. Sherlin *et al.* (2010) argued that the study by Gevensleben *et al.* had control groups and randomization, which suggested there is a training effect; however, more research with better methodology is needed to continue evaluating the effectiveness of the neurofeedback training program and to determine the mechanism by which it contributes to the success.

Metacognition and ADHD

Nearly 80% of students with ADHD struggle with their academic performance due to their poor concentration, disorganization, and poor time management (Weyandt, 2007). Metacognitive training is based on a cognitive-behavioural principle that it is important to allow students to develop executive self-management skills (Solanto *et al.*, 2010). Barkley (2006) proposed that individuals with ADHD have frontal lobe abnormality, which is associated with executive functions including self-regulation, therefore, employing metacognitive training with students with ADHD could be an effective approach (Thompson & Thompson, 1998). Some metacognitive strategies used with students diagnosed with ADHD are time management, organization, and actively applying strategies to reading, listening, essay writing, and math (Thompson & Thompson, 1998).

Combined Training and ADHD

In Thompson & Thompson's study (1998), they found significant improvements in EEG theta/beta ratio, ADHD symptoms, Intelligence Quotient (IQ) score, and academic performance after 40-sessions of metacognitive training combined with neurofeedback training. Moreover, Solanto *et al.* (2010) found that metacognitive therapy reduced the severity of ADHD symptoms. This combination of training approaches also addresses the problem of generalization: students can practice similar academic tasks to those they encounter in other settings while learning to produce the desired mental state using neurofeedback training.

Potential Moderators

Researchers have shown that gender (Clarke *et al.*, 2001a), age (Marsh, Gerber, and Peterson, 2008), and subtype of ADHD (Loo & Barkley, 2005), may potentially affect the treatment outcome of students with ADHD. For intelligence level, Cooter & Cooter (2004) suggested that students with students with below average FSIQ (between 70 and 85) are slow learners, who require more time and more repetition in order to understand new concepts. Therefore, having low intelligence level may hinder the training effect.

Moreover, medications often reduce ADHD symptoms. MTA Cooperative Group (1999) found that both medication and the combination of medication and behavioural training were equally effective in reducing ADHD symptoms; however, the combined training was more superior because it was the only group that showed improvements in other internalizing and externalizing behaviours, social skills, and reading achievement. If neurofeedback training is a form of behavior therapy as Loo & Barkley (2005) suggested, the students having medication intake throughout the training will show more improvements in both ADHD symptoms than the students that did not have medication. By understanding these moderator effects, it will be helpful to determine who or in what situation would there be a greater benefit most from this treatment.

Summary of Hypotheses

This study improves on one of the methodological issues previously identified for investigation of neurofeedback as a treatment for ADHD by having a larger sample size and increasing the generalizability of the intervention by incorporating metacognitive strategies. The goal of this study is to evaluate the effect of the combined neurofeedback and metacognitive strategies training and provides some Canadian data outcomes. In this study, the effect of 40-sessions of neurofeedback training on the severity of ADHD symptoms will be investigated based on both subjective measurement (parent's ratings) and objective measurement (computerized test of attention). The potential moderating roles of gender, age, intelligence level, subtype of ADHD, and medication intake will also be considered. The research hypotheses for this study are as follows:

1. Students will reduce their inattentive and/or hyperactive-impulsive symptoms after the completion of 40-sessions of neurofeedback training according to three parent ratings questionnaires and two computerized tests of attention.
2. The effect of neurofeedback training on the severity of ADHD symptoms will be different for different gender, age, subtypes of ADHD, intelligence level, and whether the students had medication from the beginning of the training. Specially, students with average and above FSIQ will perform better than students with below average FSIQ and students with both medication and neurofeedback training will perform better than students with just neurofeedback only.

Method

Research Design

This study is a secondary data analysis research study. The data from this study comes from files review of clients who attended training session at the ADD Centre in Mississauga, Ontario, which is a private clinic offering assessments and neurofeedback training to their clients. The primary goal of this study is to determine whether the completion of 40-sessions of neurofeedback training could reduce the ADHD symptoms in school-age students. A descriptive, correlational design was used to analyze secondary data collected from 1995 to 2010.

A sample of 318 participants who met the following inclusion criteria was extracted from the existing database for this study: (1) were 6-17 years old at the time of training, (2) were diagnosed as having ADHD without any comorbidity, and (3) completed 40-sessions of one-hour neurofeedback training combined with metacognitive strategies training. These cases are further divided into groups to investigate the moderator effect: (1) gender (male, n = 252, and female, n = 66); (2) age at the time of training (age 6-12, n=212, and age 13-17, n=67 with an average age of 10.48 ranging from 6 to 17); (3) IQ at the time of training (Below

Average: FSIQ below 85, n= 20; Average: FSIQ 85 to 115, n=97; Above Average: FSIQ above 115, n=42 with an average FSIQ of 103.9 ranging from 68.0 to 139.0). Since not all participants had received an intelligence test prior to training, only the participants who completed the Wechsler Intelligence Scale for Children (WISC) would be included in this section of the analysis; (4) medication intake at the time of training (have medication, n=69, and no medication, n=209); and (5) ADHD subtypes (ADHD Combined Type, n= 95; ADHD Inattentive Type, n=96; ADD, n=19; ADHD without a labeled subtype, n=69).

The study was approved by the University of Alberta, and all caregivers provided informed written consent to the ADD Centre at the time of their child's training.

Training Program

After the caregivers decided to enroll their child into the paid program at the ADD Centre, the student received 40-sessions of one-hour neurofeedback combined with metacognitive strategies training. The participating children were being measured on the severity of their ADHD traits prior to training using parent behaviourial rating scales (Conner's Global Index – Parent Version, DSM symptom list, and ADD-Q) and computerized test of attention [Test of Variables of Attention (T.O.V.A.) and Integrated Visual and Auditory Continuous Performance task (IVA+)]. The participant's background information included gender (male and female), age (children: aged 6-12 and adolescent: aged 13-17), and ADHD-related medication intake prior to the training (has medication and no medication). The information was collected by the clinician during the clinical interview with the caregivers prior to the training.

After the series of pre-tests and interview, the clinician in the ADD Centre diagnosed the participants with the current version of the Diagnostic and Statistical Manual of Mental Disorders (DSM) at the time of assessment (DSM-III, DSM-III-R, DSM-IV and DSM-IV-TR) to determine whether the participants fit with the diagnosis of ADHD. There are four diagnoses of ADHD used in the ADD Centre: ADHD, Combined Type; ADHD, Inattentive Type; ADHD without a labeled subtype; and ADD. For the purpose of this study, only the students diagnosed with either ADHD Combined Type, or ADHD Inattentive Type will be included to examine the role of the diagnosis moderator.

After the diagnosis, participants received 40 sessions of 50-minute, typically twice a week over a 20-week period. For children who participated in the study, the information on the severity of ADHD symptoms was collected before and after the training program.

Before each training session, the trainer explained the instructions and placed the electrodes accurately using measuring tape. For most participants, the electrode was placed at Cz and referential placement was to the left ear lobe. For some participants, the electrode was placed at C3 instead of Cz when there was a need to strengthen the functions related to left hemisphere such as language. The protocol for the training screen included slow wave (theta waves: 4-8 Hz or 3-7 Hz), fast wave (beta wave: 15-18 Hz or sensorimotor rhythm: 13-15 Hz targeting hyperactivity and impulsivity symptoms), and electromyography (EMG) (52-58 Hz). During each training screen, participants practiced to reducing the average theta activity and EMG and increasing the average beta activity.

During training, the trainers set the thresholds for the computer programs, coached the participants by giving positive verbal feedback (i.e. “good job” or “stay focused”), and collected brainwave data. Metacognitive training was delivered for 5-10 minutes during each session to teach strategies related to academic tasks while receiving auditory feedback, so the participants would notice how well they are paying attention while working on tasks. For example, if they were paying attention while working on the tasks, the auditory feedback would continue whereas if they started to daydream, the auditory feedback would stop. Strategies taught during the sessions included: weekly and daily scheduling, planning for events/homework/project, organizing and synthesizing material to help recall, active reading skills, active listening skills, essay writing strategies (i.e. hamburger method), tricks for times table, solving math word problems, employing mnemonic strategies, and preparing studying notes.

Throughout the session, participants earned tokens based on their performance as appreciation and motivation. They were encouraged to save their points in their bank account and exchange the tokens to get rewards from the "toy store" in the clinic. Prizes ranged from small dollar-store type toys/pencils/stickers to board games and bookstore/ music gift certificates.

Measures

Severity of ADHD symptoms. The two dimensions of ADHD symptoms, inattention and hyperactivity-impulsivity, were measured by using three parent's behavioural rating questionnaires and two computerized tests of attention.

Parent Rating. Three questionnaires were used in this study: the Conners 3rd Edition Global Index–Parent (Conners 3GI–P), the SNAP-IV, and the ADD-Q. The Conners 3GI–P is a parent's observation rating scale to assess his/her child or adolescent’s (6-18 years) current behavioral, academic, and emotional functioning. The Conners 3GI–P consists of 10 items on a 4-point scale ranging from Never (0) to Almost Always (3). There are three scores available for tracking behavioural and emotional functioning of the child: Total Score, Restless-Impulsive subscale, and Emotional Lability subscale. The total raw score can be transferred into T-scores based on age and sex. The cutoff T-score for a diagnosis of ADHD is 65. For the purpose of this study, the total raw score will be used to compare the treatment effect. Since the training only took place within 20 weeks time frame, using raw score can be more precisely to define a difference between pre- and post-training.

The SNAP -IV is a parent’s observation rating scale to assess the presence and severity of ADHD symptoms: inattention, impulsivity, hyperactivity, and peer interaction. This is a 23-items checklist, which is a 4-point scale ranging from Not at All (0) to Very Much (3), to assess children and adolescents based on the DSM-IV diagnostic criteria. Since this scale is not normed, only the raw scores were available for comparison of improvement.

The ADD-Q was developed by Sears and Thompson (1998) to use at the ADD Centre. This is a parent’s observation rating scale to assess the presence and severity of ADHD symptoms: inattention (attention span and organization), hyperactivity-impulsivity (spontaneity and activity level), and emotion. The ADD-Q consists of 30-items on a 4-point scale ranging from Never/Very Rarely (0) to Almost Always (3). This questionnaire is not normed so the raw scores were used for comparison. Thompson, Thompson, & Reid (2010) suggested that scores above 35 nearly always are associated with a diagnosis of ADHD.

Computerized Test of Attention. Two computerized tests of attention were used in this study: Test of Variables of Attention (T.O.V.A.) and Integrated Visual and Auditory Continuous Performance task (IVA). The Test of Variables of Attention (T.O.V.A.) is a visual continuous performance computerized test that measures variability of response time (consistency), response time, commission (impulsivity), and errors of omission (inattention). This test is for individuals aged 6 through adult, and takes approximately 21 minutes. After the assessment, an ADHD score was generated to compare to an age/gender specific ADHD group. This score is a total z-score of the three subscales mentioned above, which ranges from -9.0 to +9.0. The validity and reliability of T.O.V.A. are well-established by Leark, Greenberg, Kindschi, Dupuy & Hughes (2007).

The Integrated Visual and Auditory Continuous Performance task (IVA) is a combined visual and auditory continuous performance computerized test to assess ADHD symptoms for individuals aged 6 through adult. The ADHD symptoms are measured as the Full Scale Response Control Quotient (impulsivity) and the Full Scale Attention Quotient (inattention). Each full scale quotient is then divided into the Auditory and Visual Response Control Quotient scores. This assessment takes approximately 15 minutes. After the assessment, the four full-scale scores were generated to compare to an age/gender specific ADHD group in standard scale with a mean of 100 and standard deviation of 15.

Intelligence Testing. The participants were assessed with the current version of Wechsler Intelligence Scale at the time of assessment (WISC-R, WISC-III, WISC-IV, for ages 6–16; WAIS-R and WAIS-III for those 17 years) to measure intelligence level. Canadian norms were used in the scoring when available. All intelligence testing was done by the clinical psychologist at the ADD Centre, and the intelligence level was determined by the Full Scale Intelligence Quotient (FSIQ). There are three levels for this variable: Below Average (FSIQ < 85), Average ($85 \leq$ FSIQ ≤ 115), and Above Average (FSIQ ≥ 116).

Statistical Analysis

For this secondary data analysis, repeated-measures MANOVA tests were computed for both groups of ADHD symptoms: inattention and hyperactive-impulsivity symptoms. This analysis met the following four underlying assumptions of MANOVA tests: multivariate normality, homogeneity of variance, homogeneity of variances and covariances, and multivariate outliers (Quinn & Keough, 2002).

This procedure addressed the first research question: determining whether the completion of 40-sessions of neurofeedback training will reduce student's ADHD symptoms. To answer the second research question, 15 repeated-measures ANOVA were computed for each moderator: gender, age, intelligence level, ADHD subtype, and medication intake. Tukey's post hoc comparison was used to determine significant differences of multiple pair-wise comparisons if needed. Lastly, Pearson's correlation was calculated for each type of ADHD symptom: inattention and hyperactive-impulsivity. It was used to determine whether the subjective and objective measures were measuring the same construct. Significance level was set at .05 for all analyses.

Results

Reliability and Validity of the Parent's Rating Questionnaires

Before investigating the research questions, the subjective measuring tools (three parent's rating questionnaires) used in this study were be evaluated. Since the three questionnaires are subjective measure, it is important to ensure the reliability and validity of the ratings. Both reliability and validity were calculated by using the pre-training data. The internal consistency reliability is used to examine whether the items in the questionnaires measure the same construct as expected. The Cronbach's alpha for Conners 3rd Edition Global Index–Parent (Conners 3GI–P) is at an acceptable level (10 items; $\alpha = .695$) whereas the Cronbach's alpha for the SNAP-IV and the ADD-Q are much higher, (23 items; $\alpha = .852$ and 30 items; $\alpha = .866$ respectively). Therefore, the internal consistency reliabilities for all three questionnaires were between the acceptable to good levels.

The construct validity was examined to determine whether the items in the questionnaires measure the construct that they intended to measure: hyperactivity-impulsivity and inattention symptoms. The construct validity for each questionnaire is measured by factor analysis procedure. After the analysis, all 10 items in the Conner's 3GI–P loaded into two factors, which match the subscales in this questionnaire (Restless-Impulsive and Emotional Lability). The 23 items in SNAP –IV loaded into 4 factors as expected in the measures (hyperactivity, impulsivity, inattention, and peer interaction). The 30 items in ADD-Q loaded into three factors, which also match the number of categories in the measures inattention (attention span and organization), impulsivity-hyperactivity (spontaneity and activity level), and emotion. Overall, all three questionnaires were found to have good internal consistency and construct validity.

Alignment of Objective and Subjective Measures

In some analysis, only the subjective parent's rating questionnaires will be used, which may not be the best practice because it is difficult to detect a potential rater bias by only using one single type instrument, so it is important to ensure the subjective measuring tools are consistent with the objective tools. Pearson's correlations were used to determine the correlation between the two objective and three subjective measures for both inattention and hyperactive-impulsivity symptoms. The correlations were calculated based on the mean difference between the pre- and post- measures (mean difference = Mean measure at Time

2 – Mean measure at Time 1). The expected correlations between subjective and subjective or objective and objective measures will be significantly positively correlated. The expected correlations between subjective and objective measures will be significantly negatively correlated.

For inattention symptoms, there were strong positive significant correlations between the two subjective measures (SNAP-IV and ADD-Q) [r = 0.506, n = 240, $p<0.0001$] and between the two objective measures (T.O.V.A. and IVA) was found [r = 0.259, n = 92, $p=0.013$]. There was a negative significant correlation between the SNAP-IV and T.O.V.A. [r = -0.230, n = 95, $p=0.025$]. Overall, the trend of the measuring tools was found to be consistent with each other.

For hyperactivity-implusivity symptoms, there were strong positive significant correlations among the three subjective measures [Conner's 3GI–P and ADD-Q: r = 0.783, n = 257, $p<0.0001$; SNAP-IV and ADD-Q: r = 0.811, n = 202, $p<0.0001$; Conner's 3GI–P and SNAP-IV: r = 0.780, n = 229, $p<0.0001$]. A positive significant correlation was found between the two objective measures (T.O.V.A. and IVA) [r = 0.273, n = 92, $p=0.008$]. There are negative correlations found in the pair of Conner's 3GI–P and T.O.V.A. and the pair of ADD-Q and T.O.V.A.; however, these negative correlations were not statistically significant.

In conclusion, the overall trend of all measuring tools also displayed consistent results with each other for the hyperactivity-implusivity symptoms.

The Effectiveness of the Neurofeedback Training

Three parent rating questionnaires and two computerized tests of attention are used to measure the presence and severity of the ADHD symptoms before and after the 40-sessions of the neurofeedback training. Two repeated-measures MANOVA tests were calculated to reveal the overall difference in hyperactivity-impulsivity and inattention symptoms at the two time points. Significant behavioural improvement in hyperactive-impulsivity symptoms was found on the three questionnaires and two computerized assessments ($F(4,132)=969.200$, $p<0.0001$) as listed in Table 3-1. Similarly, there are significant behavioural improvements in inattentive symptoms as well based on two questionnaires (SNAP-IV and ADDQ) and two computerized assessments (T.O.V.A. and IVA) ($F(3,123)= 389.440$, $p<0.0001$) as listed in Table 3-1.

The Potential Moderators

There are five potential moderators to be considered in this study: gender, age at the time of training, IQ at the time of training, medication intake at the time of training, and ADHD subtypes. For the purpose of this study, the subjective parent rating questionnaires were used to measure the moderators' effect on the overall training outcome. 15 repeated-measures ANOVA were calculated to determine whether the moderators alter the effect of the neurofeedback intervention. Due to multiple comparisons, the significance level was set at .01 for all analyses. No significant effect was detected for any of five moderators on the overall training outcome. Mean scores for each group as listed in Table 3-2 (Gender), 3-3 (Age), 3-4 (Intelligence Level), 3-5 (Medication), 3-6 (Subtypes of ADHD. Effect size tests were used to follow up with each group; however, the effect size was small and the means of pre-training measures were close between group.

Discussion

In this study, the effects of a neurofeedback training program with metacognitive training on ADHD symptoms were evaluated with both subjective and objective measuring tools. Responding to gaps in the literature, this study has improved on the sample size limitation from the previous studies and provides supporting evidence for the combination of neurofeedback and metacognitive training in reducing the symptoms of ADHD.

The Effectiveness of the Neurofeedback Training

Both hyperactivity-impulsivity and inattention symptoms showed significant reduction after the completion of 40-session of neurofeedback training, which is consistent with the findings in the literature (Lubar & Lubar, 1984; Thompson & Thompson, 1998; Fox, Tharp, & Fox, 2005). In addition, students showed more improvement in hyperactivity/impulsivity symptoms than inattention symptoms. According to developmental evidence, the hyperactivity-impulsivity symptoms often appear to decease as age increases, (Barry *et al.*, 2003) suggesting this decline in hyperactivity-impulsivity symptoms may due to students' maturation or a combination of maturation and treatment effect. Future studies can address this limitation by incorporating a control group. Typically, inattention symptoms tend to maintain throughout lifespan (Barry *et al.*, 2003). Yet after treatment the inattention symptoms showed significant decline, so this suggests potential treatment effect after the completion of 40-sessions of neurofeedback training.

The Potential Moderators

After a series of analysis, all five potential moderators did not show any significant difference between groups, consistent with the previous research. This result suggests that the combined neurofeedback and metacognitive training is equally effective to both genders, both children and adolescence, and different type of ADHD diagnoses. Similarly, unlike previous research, intelligence level and medication were not found to have an impact on treatment response. One plausible explanation for having no difference in intelligence level is because of an uneven distribution among the groups: there is only a few participants with below average FSIQ compared to a large number of participants who had average and above FSIQ; therefore, future study should investigate the role of intelligence level on neurofeedback training with a larger sample size of participants with below average FSIQ.

Lastly, no difference was found between whether the students were on medication at the time of training or not. There are two plausible explanations to this finding. MTA Cooperative Group (1999) found that both medication and the combination of medication and behavioural training were equally effective in reducing ADHD symptoms; however, the combined training was more superior because it was the only group that showed improvements in other internalizing and externalizing behaviours, social skills, and reading achievement. If neurofeedback training is a form of behavior therapy as Loo & Barkley (2005) suggested, the students having medication intake throughout the training (combined training) should show more improvements in both ADHD symptoms than the students that did not have medication. Yet, the result from this study showed that having both the neurofeedback training and medication was not superior to the neurofeedback training alone. Therefore, it suggests that neurofeedback training may not just be a form of behaviour therapy as Loo & Barkley (2005) proposed. The second plausible explanation is that the students who had medication may have more severe symptoms prior to the training compared to the students without medication. Although students with medication may have more improvement, the moderation effect may be disguised by the characters of these groups. Further studies are needed to compare the different treatment effects between the neurofeedback training and medication.

Limitations and Future Directions

This study has three key limitations. First, similar to previous research, this is not an experimental study as there is no control group involved. Thus, the positive findings may result from factors besides the treatment effect such as maturation, pre-testing, and statistical regression. Specifically the participants may change or mature throughout the training process, may get used to the format of the T.O.V.A. and IVA during pre-test, or may all be the participants who were referred to the clinic for training have severe symptoms compared to the rest of the population (extreme group) (Johnston & Christensen, 2007). Future studies should involve blind control groups and random assignment to control for these threats and support the robustness of this treatment.

The second limitation is that this study treated both hyperactivity and impulsivity as a single dimension because there are not many distinctions at the behavioural level (Achenbach, 2001). Yet, Barkley & Loo (2005) have suggested that there is a distinction at the EEG level: impulsivity symptoms are associated with the frontal–central theta activity whereas hyperactivity symptoms are related the central beta activity. Future studies should separate this dependent variable into two different dependent variables to determine whether these symptoms change differently after the completion of the training.

The last limitation is that the intervention in this study is a combined training, so it is difficulty to define whether the training result is solely due to neurofeedback or metacognitive or the combination of the trainings. Therefore, future study can further investigate the effectiveness of each component in the combination training.

Implications

The results suggest that the combined neurofeedback and metacogntive training is an effective intervention for individuals with ADHD, hence it provides another intervention option for individuals with ADHD that is safer than medication and yields more consistent training effect than behavioural intervention. By implementing neurofeedback in school it could help the students with ADHD to improve their behavioural problems. In turn, it improves their social and academic functioning that is caused by the ADHD symptoms because the students could focus better on their academic work and have a better relationship with their peers and family when the ADHD symptoms have been reduced. Due to the serious impact of ADHD it is important to employ early intervention to target these ADHD symptoms. Although the finding from this research suggests that age does not moderate the training effect, early intervention can be a more proactive strategy to prevent future behavioural, academic, and social problems that encountered by these students with ADHD. However, if the students do not receive training later in life, improvements can still be seen. In general, neurofeedback is usually an expensive intervention, but the parents and the school could probably reduce the financial cost in a long run with this safer, consistent, and positive intervention options.

Conclusion

With the limitations of the ADHD medication and behavioural interventions, alternative treatments such as neurofeedback training were examined to improve ADHD symptoms in students. Although neurofeedback is a non-invasive alternative treatment of ADHD, only a small number of research studies have evaluated the effectiveness of this treatment option. This present study examined the effectiveness of the treatment by evaluating the student's behavioural changes with three parent rating questionnaires and two computerized tests of attention. The results of this study show that students with ADHD reduced their hyperactivity, impulsivity, and inattention symptoms significantly after the completion of 40-sessions of neurofeedback combined with metacognitive training. This suggests potential effectiveness of neurofeedback training combining with metacognitive training. Future studies that address the limitations of this study can look at providing control groups and randomization to strengthen the casual relationship between neurofeedback training and ADHD symptoms.

References

Achenbach, T. M. (2001). *Manual for the Revised Child Behavior Profile and Child Behavior Checklist*. Burlington, VT: University of Vermont.

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders DSM-IV-TR Fourth Edition (Text Revision)*. Washington, DC: American Psychiatric Publishing, Inc.

Arcia, E. & Conners, C. (1998). Gender differences in ADHD? *Journal of Developmental & Behavioral Pediatrics,* 19, 77-83.

Banaschewski, T., Coghill, D., Danckaerts, M., Dopfner, M., Rohde, L., Sergeant, J.A., *et al.* (2010). *ADHD and Hyperkinetic Disorder*. New York, NY : Oxford University Press.

Barkley, R. A. (2006). *Attention Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment (3rd ed.)*. New York, NY: Guildford Press.

Barbaresi, W. J., Katusic, S. K., Colligan, R. C., Pankratz, S., Weaver, A. L., Weber, K.J., *et al.* (2002). How common is attentiondeficit/hyperactivity disorder? *Archives of Pediatrics and Adolescent Medicine*, *156*, 217–224.

Barry R.J., Clarke A.R. & Johnstone S.J. (2003). A review of electrophysiology in Attention-Deficit/Hyperactivity Disorder: I. Qualitative and quantitative electroencephalography. *Clinical Neurophysiology*, 114:171–183.

Biederman, J., Kwon, A., Aleardi, M., Chouinard, V.A., Marino, T., Cole, H., Mick, E. & Faraone, S.V. (2005). Absence of gender effects on attention deficit hyperactivity disorder: findings in non-referred subjects. *The American Journal of Psychiatry*, 162(6), 1083-1089.

BrainTrain. (2011). IVA+*Plus – Visual & Auditory Attention Testing*. Retrieved from http://www.braintrain.com/professionals/adhdtesting/ivaplus_pro.htm

Centers for Disease Control and Prevention (2005). Prevalence of diagnosis and medication treatment for attention-deficit/hyperactivity disorder—United States, 2003. *Morbidity and Mortality Weekly Report*, 54, 842-847.

Clarke, A. R., Barry, R. J., Dupuy, F.E., Heckel, L.D. & McCarthy, R., Selikowitz, M. & Johnstone, S.J. (2010). Behavioural differences between EEG-defined subgroups of children with Attention-Deficit/Hyperactivity Disorder. Clinical Neurophysiology, 122, 1333-1341.

Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2001a). Age and sex effects in the EEG: differences in two subtypes of attention-deficit/hyperactivity disorder. *Clinical Neurophysiology, 112,* 815–826.

Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2001b). EEG-defined subtypes of children with attention-deficit/hyperactivity disorder. *Clinical Neurophysiology, 112,* 2098–2105.

Connor, D.F. (2006). Stimulants. In R.A. Barkley (ed.), *Attention Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment (3rd ed.,* pp. 608-648). New York, NY: Guilford Press.

Cooter, K.S. & Cooter, Jr., R.B. (2004). One size doesn't fit all: Slow learners in the reading classroom. *Reading Teacher*, 57(7), 680-684.

Fabiano, G.A., Pelham, W.E., Coles, E.K., Gnagy, E.M., Chronis-Tuscano, A. & O'Connor, B.C. (2009). A meta-analysis of behavioral treatments for attention-deficit/hyperactivity disorder. *Clinical Psychology Review*, 29(2), 129-140.

Flanagan, D.P. & Harrison, P.L. (2005). *Contemporary Intellectual Assessment, Second Edition: Theories, Tests, and Issues.* New York, NY: Guilford Press.

Fuchs, T., Birbaumer, N., Lutzenberger, W., Gruzelier, J. H. and Kaiser, J. (2003).

Neurofeedback treatment for attention-deficit/hyperactivity disorder in children: a comparison with methylphenidate. *Applied Psychophysiology Biofeedback*, 28 (1), 1-12.

Gevensleben, H., Holl, B., Albrecht, B., Vogel, C., Schlamp, D., Kratz, O., et al. (2009). Is neurofeedback an efficacious treatment for ADHD? A randomised controlled clinical trial. *Journal of Child Psychology and Psychiatry*, 50, 780–789.

Gaub, M. & Carlson, C. (1997). Gender differences in ADHD: a meta-analysis and critical review. *Journal of the American Academy of Child & Adolescent Psychiatry,*36 (8):1036-1045.

Hart, E. L., Lahey, B. B., Loeber, R., Applegate, B. & Frick, P. J. (1995). Developmental change in attention-deficit hyperactivity disorder in boys: A four-year longitudinal study. *Journal of Abnormal Child Psychology, 23,* 729–749.

Johnston, N., & Christensen, L. (2007). *Educational Research: Quantitative, Qualitative, and Mixed Approaches* (3rd ed.). Thousand Oaks, CA: Sage.

Knight, L.A., Rooney, M. & Chronis-Tuscano, A. (2008). Psychosocial Treatments for Attention-Deficit/Hyperactivity Disorder. *Current*

Psychiatry Reports, 10(5), 412-418.

Leark, R.A., Greenberg, L.M., Kindschi, C.L., Dupuy, T.R., & Hughes, S.J. (2007). *T.O.V.A.® Professional Manual: Test Of Variables of Attention Continuous Performance Test.* Retrieved from http://www.tovatest.com/manuals/tova_7_3_Professional_Manual_2007_02_27.pdf

Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback & Self Regulation, 16*, 201–225.

Lubar, J., & Lubar, J. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorder in a clinical setting. *Biofeedback and Self Regulation, 9,* 1-23.

Marsh, R., Gerber, A.J., Peterson, B.S. (2008). Neuroimaging Studies of Normal Brain Development and Their Relevance for Understanding Childhood Neuropsychiatric Disorders. *Journal of the American Academy of Child & Adolescent Psychia*try, 47(11), 1233-1251.

Monastra,V. J. & Monastra, D.M (2004). *EEG biofeedback treatment for ADHD: an analysis of behavioral, neuropsychological, and electrophysiological* *response over a three-year follow-up period*. Colorado: Annual Conference of the Association for Applied Psychophysiology and Biofeedback

Monastra,V. J., Monastra, D.M.,& George, S. (2002). The effects of stimulant therapy, EEG biofeedback, and parenting style on the primary symptoms of attention-deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback*, *27,* 231–249.

Miller, A., Lee, S., Raina, P., Klassen, A., Zupancic, J., & Olsen, L. (1998). A Review of Therapies for Attention-Deficit/Hyperactivity Disorder. Ottawa: Canadian Coordinating Office for Health Technology Assessment (CCOHTA).

MTA Cooperative Group. (1999). A 14-Month Randomized Clinical Trial of Treatment Strategies for Attention-Deficit/Hyperactivity Disorder.*Archives of General Psychiatry*, 56, 1073-1086.

Oades, R.D. (2005). *Brain Maturation - it covers three decades: Considerations of the development of ADHD.* Essen, Germany: University Clinic for Child and Adolescent Psychiatry.

Polanczyk, G., Lima, M.S., Horta, B.L., Biederman, J., & Rohde, L.A. (2007). The worldwide prevalence of attention deficit/hyperactivity disorder: a systematic review and meta-regression analyses. *American Journal of Psychiatry*, 164: 942-948.

Quinn, G. P. & Keough, M. J. (2002). *Experimental design and data analysis for biologists*. Cambridge, UK: Cambridge University Press.

Sears, W., & Thompson, L. (1998). *The A.D.D. book: New understandings, new approaches to parenting your child*. New York, NY: Little, Brown and Co.

Selikowitz, M. (2009). *ADHD: The Fact* (2nd Edition). New York, NY : Oxford University Press.

Sherlin, L., Arn, M., Lubar, J. & Sokhadze, E. (2010). A Position Paper on Neurofeedback for the Treatment of ADHD. *Journal of Neurotherapy*, 14(2), 66-78.

Solanto, M.V., Marks, D.J., Wasserstein, J., Mitchell, K., Abikoff, H., Alvir, J.M.J. & Kofman, M.D. (2010). Efficacy of Meta-Cognitive Therapy for Adult ADHD. *The American Journal of Psychiatry*, 167, 958-968.

Thompson, M., Thompson, L.& Reid, A. (2010). Neurofeedback Outcomes in 150 Clients with Asperger's Syndrome and 9 Clients with Autism. *Journal of Applied Psychophysiology and Biofeedback,* 35(1), 63-81.

Van der Oord S., Prins P.J., Oosterlaan J., & Emmelkamp P.M. (2008). Efficacy of methylphenidate, psychosocial treatments and their combination in school-aged children with ADHD: a meta-analysis. *Clinical Psychology Review*, 28,783–800.

Waschbusch, D. A., & Hill, G. P. (2003). Empirically supported, promising, and unsupported treatments for children with Attention-Deficit/Hyperactivity Disorder. In S. O. Lilienfield, S. Jay Lynn, & J. M. Lohr (Eds.), *Science and pseudoscience in clinical psychology*. New York, NY: Guilford Press.

Weyandt, L.L. & DuPaul, G..J. (2008). ADHD in college students: Developmental findings. *Developmental Disabilities Research Reviews*, 14 (4), 311-319.

Wigal, S.B. (2009). Efficacy and Safety Limitations of Attention-Deficit Hyperactivity Disorder Pharmacotherapy in Children and Adults. *CNS Drugs*, Supplement 1 (23), 21-31.

Table 3-1

Results for MANOVA tests from pre- to post-training

	SS	DF	MS	F	Sig of F
Symptoms					
Inattentive Symptoms					
Within cells	37139.32	123	301.95		
DV	352768.65	3	117589.55	389.44	0.000
Hyperactive-Impulsivity Symptoms					
Within cells	18725.26	132	141.86		
DV	549955.79	4	137488.95	969.20	0.000

Table 3-2

Mean Scores pre- and post- 40 sessions of training by Gender

Gender	Male		Female	
Time	Pre-Training	Post-Training	Pre-Training	Post-Training
Measuring Tools				
Inattentive Symptoms				
SNAP-IV	10.84	7.79	10.48	7.23
ADD-Q	32.08	24.02	30.75	22.07
Hyperactive-Impulsivity Symptoms				
SNAP-IV	17.80	12.64	14.04	9.90
ADD-Q	13.81	9.75	11.39	7.70
Conners 3G-P	13.27	9.71	11.48	7.90

Table 3-3

Mean Scores pre- and post- 40 sessions of training by Age Group

Age Group	Children (6-12 yrs. old)		Adolescents (13-17 yrs. old)	
Time	Pre-Training	Post-Training	Pre-Training	Post-Training
Measuring Tools				
Inattentive Symptoms				
SNAP-IV	10.77	7.77	10.77	7.36
ADD-Q	31.56	23.65	32.45	23.47
Hyperactive-Impulsivity Symptoms				
SNAP-IV	17.94	12.76	14.16	9.65
ADD-Q	13.87	9.80	11.53	7.82
Conners 3G-P	13.45	9.78	11.30	7.89

Table 3-4

Mean Scores pre- and post- 40 sessions of training by Intelligence Level

Intelligence Level	Below Average		Average		Above Average	
Time	Pre-	Post-	Pre-	Post-	Pre-	Post-
Measuring Tools						
Inattentive Symptoms						
SNAP-IV	11.30	8.10	11.07	7.61	10.19	7.33
ADD-Q	31.00	23.71	32.05	24.08	31.92	22.41
Hyperactive-Impulsivity Symptoms						
SNAP-IV	15.00	11.71	17.26	11.70	17.19	10.58
ADD-Q	13.65	9.17	13.97	9.38	12.70	8.73
Conners 3G-P	12.63	8.00	12.98	9.23	12.50	9.17

Note. Intelligence level was determined by the Full Scale Intelligence Quotient (FSIQ). There are three levels for this variable: Below Average (FSIQ < 85), Average (85 ≤ FSIQ ≤ 115), and Above Average (FSIQ ≥116).

Table 3-5

Mean Scores pre- and post- 40 sessions of training by Medication Usage during Training

Medication Usage	Medication		Without Medication	
Time	Pre-Training	Post-Training	Pre-Training	Post-Training
Measuring Tools				
Inattentive Symptoms				
SNAP-IV	11.27	7.92	10.59	7.61
ADD-Q	32.11	23.67	31.70	23.62
Hyperactive-Impulsivity Symptoms				
SNAP-IV	19.89	13.74	16.00	11.45
ADD-Q	14.44	10.40	12.89	8.97
Conners 3G-P	13.75	10.51	12.62	8.93

Table 3-6

Mean Scores pre- and post- 40 sessions of training by Subtype of ADHD

Subtype of ADHD	Combined Type		Inattentive Type	
Time	Pre-Training	Post-Training	Pre-Training	Post-Training
Measuring Tools				
Inattentive Symptoms				
SNAP-IV	10.92	7.97	10.46	7.74
ADD-Q	32.04	24.26	32.79	23.28
Hyperactive-Impulsivity Symptoms				
SNAP-IV	21.65	15.48	12.01	8.96
ADD-Q	16.27	11.35	10.36	7.33
Conners 3G-P	14.82	11.05	10.28	7.53

Chapter 4: Long-Term Effectiveness of Neurofeedback and Metacognitive Training on Children's Attention Deficit/Hyperactivity Disorder Symptoms: A Pilot Study

Inattention, impulsivity, and hyperactivity are the three hallmark symptoms of Attention Deficit/Hyperactivity Disorder (ADHD). These symptoms may interfere with a student's ability to focus in class or at work, get along with peers, and manage frustration with family members (Barkley, 2006). Although medication and behavioural interventions have been found to be effective in reducing these ADHD symptoms, the disadvantages and limitations of these interventions, such as short-term and/or inconsistent improvement, negative side effects, and a considerable rate of non-responders have been reported (Wigal, 2009; Van der Oord, Prins, Oosterlaan, & Emmelkamp, 2008; MTA Cooperative Group, 1999; Connor, 2006; Miller *et al.*, 1998). In addition, no evidence supports long-term effectiveness of either type of intervention (Jensen *et al.*, 2007; Waschbusch & Hill, 2003). Therefore, researchers are searching for other intervention options that avoid these negative impacts and provide long-term benefits to the individuals with ADHD.

Neurofeedback has become one of the alternative intervention options for individuals with ADHD for almost 30 years; however, it is still not considered as the mainstream treatment for ADHD. Although several studies have shown that neurofeedback training is effective in improving attention, academic performance, IQ, and social behaviours (Fuchs, Birbaumer, Lutzenberger, Gruzelier & Kaiser, 2003; Lubar & Lubar, 1984; Monastra, Monastra & George, 2002; Thompson & Thompson, 1998), few have focused on the evaluation of neurofeedback training's long term impact. Therefore, whether the improvement seen with neurofeedback training can be maintained over time is still under investigation.

Lubar (2003) performed the first study that examined the potential for long term maintenance, by utilizing telephone interviews to follow up with students who had completed neurofeedback treatment from a period ranging from less than 1 year up until 10 years prior. Most parents and teachers of the participants who received the neurofeedback training rated the students as showing a significant improvement on their ADHD symptoms with the Conner's rating scale. Although this was not a randomized controlled study, it suggested a potential long-term benefit from the neurofeedback training. In 2004, Monastra & Monastra followed up their initial randomized controlled study, which evaluated the effectiveness of neurofeedback. Two years after the treatment, they found that 70% of the participants who had received neurofeedback training reduced their medication dosage at the two year follow up point whereas 85% of the participants who did not undergo neurofeedback training had increased their dosage.

In a similar vein, another randomized controlled study from Leins *et al.* (2007) found that the neurofeedback training with either Theta/Beta Frequencies or Slow cortical potentials (SCPs) protocol displayed improvement in both attention and intelligence quotient (IQ) scores after the treatment and even at a 6-month follow-up point. In 2008, Gani and colleagues conducted a 2-year follow up study, and found similar results: half of the students initially diagnosed with ADHD no longer met diagnostic criteria at the 2-year follow up point. Recently, Gevensleben *et al.* (2010) found that children with ADHD reduced their ADHD behaviours significantly initially after receiving neurofeedback treatment and at the 6-month follow up point. These studies provide preliminary evidence suggesting that neurofeedback training may lead to long-term improvement of ADHD symptoms.

However, one of the criticisms of this training is that it does not generalize to non-treatment settings (Loo & Barkley, 2005). Consequently, to address this limitation, the ADD Centre decided to incorporate metacognitive training combined with the neurofeedback into the program for students with ADHD

(Thompson & Thompson, 1998). Metacognitive training allows students to develop executive self-management skills (Solanto *et al.*, 2010). The two goals of the metacognitive training are: (1) to address the academic underachievement for the students with ADHD by teaching them learning strategies and (2) to increase the generalization of the neurofeedback training effect by practicing academic tasks that are similar to the ones students encounter in other non-treatment settings while learning to produce the desired mental state (neurofeedback training) at the same time. The first to pair the two approaches, Thompson & Thompson (1998), found that students with ADHD showed significant improvements in EEG theta/beta ratio, ADHD symptoms, Intelligence Quotient (IQ) score, and academic performance after 40-sessions of combined neurofeedback and metacognitive strategies training. However, the long-term effects of this approach have not yet been determined, and therefore, this pilot study is going to extend the work from Thompson & Thompson (1998) to investigate the potential long-term benefit of the neurofeedback and metacognitive strategies training on ADHD symptoms and also provide some Canadian data outcomes.

Summary of Hypotheses

The goal of this paper is to evaluate whether the improvements in ADHD symptoms after 40 sessions of combined neurofeedback and metacognitive training are sustained after at least one year after the completion of the training (Thompson & Thompson, 1998).

Method

Participants

Twenty-two participants (average age was 14 years old ranging from 8 to 17) were recruited from the ADD Centre. The inclusion criteria were: (1) predominately diagnosed with ADHD without any comorbidity, (2) completed a minimum of 40-sessions of neurofeedback training at the ADD Centre at least a year prior to recruitment (with an average of 67 sessions ranging from 40 to 172 sessions), and (3) age between six to seventeen years at the time of the study. These twenty-two participants, who fit the inclusion criteria, were available to be contacted and interested in the study. They completed their 40-sessions neurofeedback ranging from one-year to ten-years as described in Figure 1 with an average 4.86 years. The ratio of males to females was approximately 16:8.

Training Program

The training program at the ADD Centre consisted of at least 40-sessions of neurofeedback combined with metacognitive strategies training. The participating children had the severity of their ADHD symptoms measured prior to training using parent behaviourial rating scales (Conner's Global Index – Parent Version, DSM symptom list, and ADD-Q) and computerized tests of attention [Test of Variables of Attention (T.O.V.A.) and Integrated Visual and Auditory Continuous Performance task (IVA+)]. Participants received a 50-minute training program, typically twice a week over a 20 week period. Neurofeedback sessions took place in the training rooms in the ADD Centre. Sessions were conducted at a time based on participants' preference (typically after school or during the weekend) two times a week on a regular schedule. Sessions missed due to assemblies, trips, holidays, weather, and illness absences were rescheduled.

Before each training session, the trainer explained the instructions and placed the electrodes accurately using measuring tape. For most participants, the electrode was placed at Cz and referential placement was to the left ear lobe. For some participants, the electrode was placed at C3 instead of Cz when there was a need to strengthen the functions related to left hemisphere such as language.

Each training session started with a 30-second assessment screen to assess the brainwave activity of the participants. Following the assessment screen, the participants worked on four to six 3-minutes training screens. The protocol for the training screen included slow waves (theta waves: 4-8 Hz or 3-7 Hz), fast waves (beta wave: 15-18 Hz or sensorimotor rhythm: 13-15 Hz targeting hyperactivity and impulsivity symptoms), and electromyography (EMG) (52-58 Hz). EMG measures electrical interference caused by muscle movements. This was tracked to insure a clean EEG signal. During each screen, participants practiced to reduce the average theta activity and EMG and increase the average beta activity. For children with both reading difficulties and impulsivity, both fast wave 15-18 Hz and 13-15 Hz are trained. Average Amplitude data (measured in mean uv) were then collected continuously for the three bands after each training screen. The trainers were responsible for setting thresholds for the computer programs, coaching the participants by giving positive verbal feedback (i.e. “good job” or “stay focused”), and collecting brainwave data.

Metacognitive strategies related to academic tasks were taught for 5-10 minutes during the sessions while receiving auditory feedback, so the participants would also notice how well they were paying attention while working on tasks. Strategies taught during the session were: weekly and daily scheduling, planning for events/homework/project, organizing and synthesizing material to help recall, active reading skills, active listening skills, essay writing strategies (i.e.

hamburger method), tricks for times table, solving math word problems, employing mnemonic strategies, and preparing studying notes.

Throughout the session, participants earned tokens based on their performance as appreciation and motivation. They were allowed to save the points in their bank account and exchange the tokens to get rewards from the "toy store" in the clinic. Prizes ranged from small dollar-store type toys/pencils/stickers to board games and bookstore/ music gift certificates. Lastly, the trainer debriefed with the participants and their caregivers. Experts from ADD centre reviewed the training progress periodically.

Procedure

Twenty-two participants were recruited through the ADD Centre (Mississauga) to participate in this follow up study. The ADD Centre identified those children who met the inclusion criteria (diagnosis of ADHD) and recruited the caregivers of these children by sending them recruitment materials along with a set of questionnaires that were the same forms that they filled out at the pre- and post-assessment point (Conner's Global Index – Parent Version, DSM symptom list, ADD-Q). The caregivers could then contact the researchers or administrative staff at the ADD Centre directly if they had further questions about participating in the study. Two to three weeks after the recruitment materials had been mailed out, the researchers or administrative staff performed a telephone follow-up with the potential participants to address any questions they might have about the study. The participating caregivers who were interested completed the set of questionnaires and returned them to the ADD Centre with the prepaid envelope. After the data was been collected, the scores on the questionnaires were compared to the participant's post-test scores, which were collected at the completion of the 40-sessions training session, to determine whether the gains have been sustained for at least 1-year post training.

Measures

Severity of ADHD symptoms. The two dimensions of ADHD symptoms, inattention and hyperactivity-impulsivity, were measured by using three parent's behavioural rating questionnaires: the Conners 3rd Edition Global Index–Parent (Conners 3GI–P), the SNAP-IV, and the ADD-Q.

Conners 3GI–P. A parent's observation rating scale that assesses the current behavioural, academic, and emotional functioning of his/her child or adolescent, who is 6 to 18 years old. The Conners 3GI–P consists of 10 items on a 4-point scale ranging from Never (0) to Almost Always (3). There are three scores available for tracking behavioural and emotional functioning of the child: Total Score, Restless-Impulsive subscale, and Emotional Lability subscale. The total raw score can be transferred into T-scores based on age and sex. The cutoff T-score for a diagnosis of ADHD is 65.

SNAP –IV. A parent's observation rating scale that assesses the presence and severity of ADHD symptoms: inattention, impulsivity, hyperactivity, and peer interaction. This is a 23-items checklist, which is 4-point scale ranging from Not at All (0) to Very Much (3), to assess child and adolescent based on the DSM-IV diagnostic criteria. Since this scale is not normed, only the raw scores were available for comparison of improvement.

ADD-Q. This questionnaire was developed by Sears and Thompson (1998) to use at the ADD Centre. This is a parent's observation rating scale to assess the presence and severity of ADHD symptoms: inattention (attention span and organization), hyperactivity-impulsivity (spontaneity and activity level), and emotion. The ADD-Q consists of 30-items on a 4-point scale ranging from Never/Very Rarely (0) to Almost Always (3). This questionnaire is not normed, so the raw scores were used for comparison. Thompson, Thompson, & Reid (2010) suggested that scores above 35 nearly always are associated with a diagnosis of ADHD.

Statistical Analysis

For this pilot study, data was analyzed using SPSS (SPSS for Windows, 2008). Missing data in some questionnaires, ranging from 0 to 6 participants, was resultant of one or two incomplete items. Two repeated-measures MANOVA tests were computed according to the inattention and hyperactive-impulsivity symptoms to measure the training effect from the pre-training time point to post-training and to follow-up point. This analysis met the following four underlying assumptions of MANOVA tests: multivariate normality, homogeneity of variance, homogeneity of variances and covariances, and multivariate outliers (Quinn & Keough, 2002).

Another four repeated-measures MANOVA test was used to further determine where the significant differences lay: (1) pre- and post-training time point and/or (2) post-training and follow-up time point. Due to multiple comparisons, the significance level was set at .01 for all analyses.

Results

Alignment of Objective and Subjective Measures

Since this study intended to measure the changes in day-to-day function of the students, the parent rating questionnaires were used as the primary measuring tools; however, there are limitations to only using subjective questionnaires. Therefore, it is important to ensure the subjective measuring tools are consistent with the objective tools in order to provide some evidence that any training effect reported may not just due to the parents' perceptions. Pearson's correlations were used to determine the correlation between the two objectives and three subjective measures for both inattention and hyperactive-impulsivity symptoms. The correlations were calculated based on the mean difference between the pre- and post- measures (mean difference = Mean measure at Time 2 – Mean measure at Time 1), and the mean scores for each measure were listed in Table 4-1 Overall, the trend of all subjective measuring tools showed that all subjective measures were consistent with each other (positive significant correlation) and also with one of the objective measures (negative significant correlation for both inattention and hyperactive-impulsivity symptoms.

The Follow-Up Measures

The scores from the three parent rating questionnaires were used to measure the presence and severity of the ADHD symptoms at the pre-training, the post-training assessment after the 40-sessions of the neurofeedback training and the follow up point. Two repeated MANOVA were calculated to determine the overall changes in hyperactivity-impulsivity and inattention symptoms between all three time points. Significant decrease in both inattention symptoms ($F(1,11)=101.36$, $p<0.0001$) and hyperactive-impulsivity symptoms ($F(2,22)=5.78$, $p<0.01$) were observed from pre-training to follow-up point.

Another four repeated-measures MANOVA were used to follow up the two significant results. From pre-training to post-training time point, both inattention symptoms ($F(3,9)=41.23$, $p<0.0001$) and hyperactive-impulsivity symptoms ($F(4,12)=150.49$, $p<0.0001$) revealed significant difference as shown in Table 4-2. From post-training to follow up time point, a significant decrease was detected in inattention symptoms ($F(1,13)=65$, $p<0.0001$) and hyperactive-impulsivity symptoms ($F(2,32)=779.56$, $p<0.0001$) as shown in Table 4-3. The result showed significant decrease in both the hyperactive-impulsivity symptoms and inattention symptoms even after completion of training.

Discussion

This pilot study is intended to provide preliminary evidence regarding the combined use of neurofeedback and metacogntive training, and specifically to determine whether students are able to continue practicing the more mature brainwave patterns that they learned in neurofeedback training even after at least

1-year of completing training sessions. Two key results emerged from this pilot study:

1) parents felt that both inattention symptoms and hyperactivity-impulsivity symptoms were further reduced from post-treatment levels, suggesting that not only were treatment effects maintained but that improvement continued to take place. Barry *et al*. (2003) and Hart *et al*. (1995) found that the hyperactivity-impulsivity symptoms seem to normalize as age increases, but the inattention and disorganization symptoms still continue as a problem throughout development and into adulthood. With this developmental evidence, the continued reduction of the inattention symptoms since treatment completion suggests that the maintenance and continuation of the symptom reduction may not be solely due to maturation.

Both neurofeedback and metacogntive training have often been used as a non-medication approach to treatment of ADHD. This study provides evidence that this approach may be more effective in the long-term than taking stimulant medications because the effects of medications do not last if the patient stops taking the medication. With the combined training, the result showed that a continuous reduction in both hyperactivity-impulsivity and inattention symptoms for an average of 4.86 years after completion of training.

Limitation and Future Direction

The goal of the present investigation was to contribute information to the field of neurofeedback about how the 40 sessions of neurofeedback combined with metacognitive training benefits students in the long run, and to provide some Canadian data outcomes. The findings of this study showed potential long-term effectiveness of the training; however, since this is not a randomized experimental study, the students' maturation may play a role in these positive findings. In order to rule out the maturation and support the treatment effect, future studies should involve blind control groups and random assignment.

Another limitation of this study is the usage of subjective measuring tools, although this study tried to provide evidence that both subjective and objective measuring tools are consistent when comparing pre- and post-training results. Ideally, future studies should incorporate both subjective and objective measuring tools to evaluate the severity of ADHD symptoms at the follow up point.

Another limitation of this study is the usage of subjective measuring tools, although this study tried to provide evidence that both subjective and objective measuring tools are consistent when comparing pre- and post-training results.

Ideally, future studies should incorporate both subjective and objective measuring tools to evaluate the severity of ADHD symptoms at the follow up point. Lastly, since this study is a pilot study, researchers should duplicate this study with a bigger sample size, a control group, and usage of objective measuring tools.

Traditionally, Lubar (1991) suggested that typically having 40 sessions of neurofeedback for ADHD individuals with theta and beta training should show clinical improvement. Clinical improvement was defined as an increase in the mean amplitude of beta brainwaves, a decrease in mean amplitude of theta brainwaves, and an improvement on certain psychometric tests (Lubar, 1991). In spite of this, some researchers found that slow cortical potential training showed improvement in ADHD symptoms (Gevensleben *et al.*, 2010; Heinrich *et al.*, 2004). More research is needed to determine which type of neurofeedback training is the most effective. Moreover, the training protocol and number of sessions are different among the limited number of the long-term evaluation research. In this study, half of the participants decided to continue the training after the 40-sessions; therefore, future studies should look into the relationship between the training effect and number of training sessions. Furthermore, researchers should focus on determining the most effective neurofeedback training protocol and the optimal number of training sessions for individuals with ADHD.

Conclusion

In conclusion, this study has shown that the long-term effectiveness of neurofeedback and metacognitive training provides a promising alternative treatment for individuals with ADHD. The findings of this study showed a decrease in both hyperactivity-impulsivity symptoms and inattention symptoms from post-training. The present investigation supports the past research on the long-term effectiveness of the neurofeedback training program. With the promising results, this combined training could be introduced to the school or community settings instead of medication to address the behavioural problems in the students with ADHD. Due to the serious impact of ADHD, the reduction of the ADHD symptoms could also improve students' social and academic functioning. Therefore, by implementing this combined training in school, students could improve on their behaviours and also on their academic work and social relationship with their peers and family that impaired by the ADHD symptoms.

References

Barkley, R. A. (2006). *Attention Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment (3rd ed.)*. New York, NY: Guildford Press.

Centers for Disease Control and Prevention (2005). Prevalence of diagnosis and medication treatment for attention-deficit/hyperactivity disorder—United States, 2003. *Morbidity and Mortality Weekly Report*, 54, 842-847.

Gani, C., Birbaumer, N., Strehl, U. (2008) Long term effects after feedback of slow coritcal potentials and of theta-beta-amplitudes in children with attention-deficit/hyperactivity disorder. *International Journal of Bioelectromagnetism*, 10, 4, 209 - 232.

Gevensleben, H., Holl, B., Albrecht, B., Schlamp, D., Kratz, O., Studer, P., Rothenberger, A., Moll, G.H., & Heinrich, H. (2010). Neurofeedback training in children with ADHD: 6-month follow-up of a randomised controlled trial. *European Child & Adolescent Psychiatry*, 19, 715–724. DOI: 10.1007/s00787-010-0109-5.

Heinrich, H., Gevensleben, H., & Strehl, U. (2007). Annotation: Neurofeedback – train your brain to train behaviour. *Journal of Child Psychology and Psychiatry*, 48 (1), 3-16.

Jensen, P.S., Arnold, L.E., Swanson, J.M., Vitiello, B., Abikoff, H.B., Greenhill, L.L., Hechtman, L., Hinshaw, S.P., Pelham, W.E., Wells, K.C., Conners, K., Elliott, G.F., Epstein, J.N., Hoza, B., March, J.S., Molina, B.S.G., Newcorn, J.Y.H., Severe, J.B., Wigal, T., Gibbons, R.D., & Hur, K. (2007). 3-year follow-up of the NIMH MTA study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46(8), 989-1002.

Leins, U., Goth, G., Hinterberger, T., Klinger, C., Rumpf, N., & Strehl, U. (2007). Neurofeedback for children with ADHD: A comparison of SCP and theta-beta protocols. *Applied Psychophysiology and Biofeedback*, 32, 73–88.

Loo, S.K. & Barkley, R.A. (2005). Clinical Utility of EEG in Attention Deficit Hyperactivity Disorder. *Applied Neuropsychology*, 12(2), 64-76.

Lubar, J.F. (2003). Neurofeedback for the management of attention deficit disorders. In M.S. Schwartz & F. Andrasik (Eds.), *Biofeedback: A practitioner's guide* (pp. 409-437). New York, NY: Guilford.

Lubar, J.F. (1991). Discourses on the development of diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback and Self Regulation*, 16(3), 201-224.

Lubar, J. O., & Lubar, J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. *Biofeedback and Self-Regulation*, 9 (1), 1 - 23.

Monastra,V. J., Monastra, D.M.,& George, S. (2002). The effects of stimulant therapy, EEG biofeedback, and parenting style on the primary symptoms of attention-deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback*, *27,* 231–249.

MTA Cooperative Group. (1999). A 14-Month Randomized Clinical Trial of Treatment Strategies for Attention-Deficit/Hyperactivity Disorder.*Archives of General Psychiatry*, 56, 1073-1086.

Oades, R.D. (2005). *Brain Maturation - it covers three decades: Considerations of the development of ADHD.* Essen, Germany: University Clinic for Child and Adolescent Psychiatry.

Quinn, G. P. & Keough, M. J. (2002). *Experimental design and data analysis for biologists*. Cambridge, UK: Cambridge University Press.

SPSS for Windows [2008, computer program]. Version 17.0.0. Chicago: SPSS Inc.

Van der Oord S., Prins P.J., Oosterlaan J., & Emmelkamp P.M. (2008). Efficacy of methylphenidate, psychosocial treatments and their combination in school-aged children with ADHD: a meta-analysis. *Clinical Psychology Review*, 28,783–800.

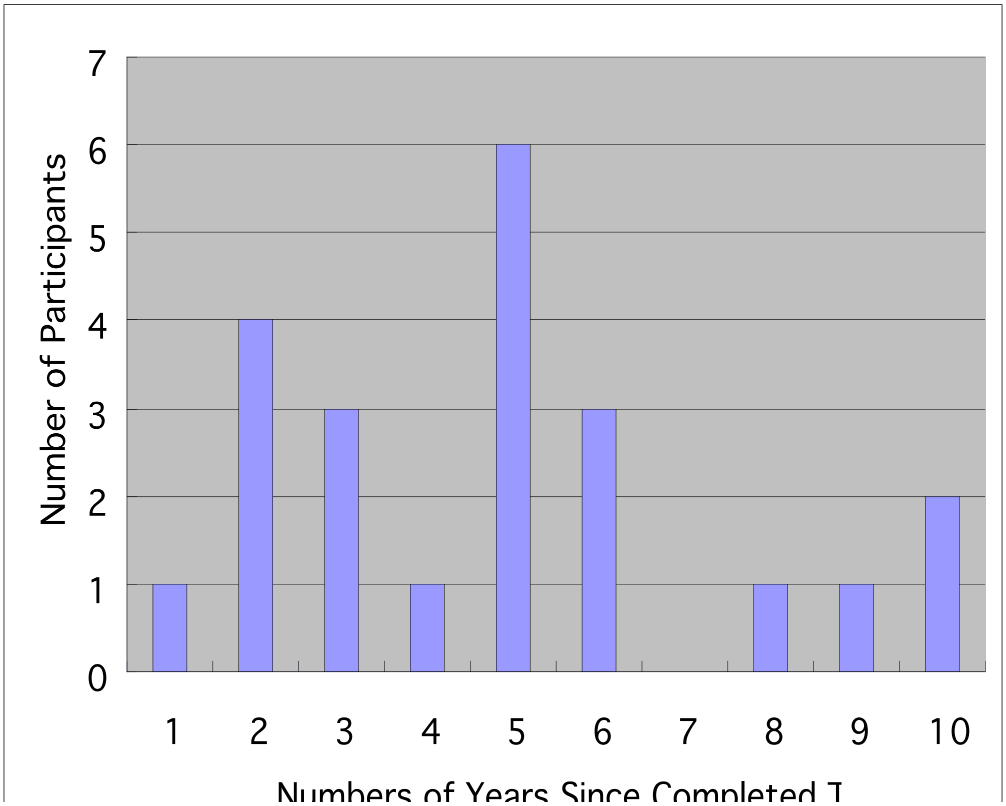

Figure 4-1. Number of years since the participants completed the 40-sessions training.

Table 4-1

Mean Scores at pre-training, post-training, and follow-up time point

Time Measuring Tools	Pre-Training	Post-Training	Follow-Up
Inattentive Symptoms			
TOVA	85.20	79.88	-----
IVA	66.29	77.43	-----
SNAP-IV	9.58	6.75	6.14
ADD-Q	16.58	11.50	8.41
Hyperactive-Impulsivity Symptoms			
TOVA	89.80	94.50	-----
IVA	89.29	89.43	-----
SNAP-IV	16.58	11.50	8.41
ADD-Q	11.65	9.25	6.20
Conners 3G-P	78.24	69.19	64.00

Note. T-scores for Conners 3G-P were used in this analysis.

Table 4-2

Results for MANOVA tests from pre- to post-training

	SS	DF	MS	F	Sig of F
Symptoms					
Inattentive Symptoms					
Within cells	2757.38	9	306.38		
DV	37892.13	3	12630.71	41.23	0.000
Hyperactive-Impulsivity Symptoms					
Within cells	1167.80	12	97.32		
DV	58582.00	4	14645.50	150.49	0.000

Table 4-3

Results for MANOVA tests from post-training to follow-up time point

	SS	DF	MS	F	Sig of F
Symptoms					
Inattentive Symptoms					
Within cells	391.31	12	32.61		
DV	2119.69	1	2119.69	65.00	0.000
Hyperactive-Impulsivity Symptoms					
Within cells	1617.59	32	50.55		
DV	78813.41	2	39406.71	779.56	0.000

Chapter 5: Conclusion

Neurofeedback has been an alternative treatment for individuals with ADHD for years; however, with limited research evaluating its short-term and long-term effectiveness, many people have undermined this treatment option. Noticing this gap in the field, I decided to investigate both the short-term and long-term impact of the neurofeedback training on the severity of ADHD symptoms. Results from this study showed that both hyperactive-impulsive and inattentive symptoms have decreased after 40-sessions of training and also at the follow-up point. This training effect does not appear to be affected by gender, age, subtype of ADHD, intelligence level, and medication intake. Therefore, neurofeedback has potential for its use as a short-term and long-term treatment for individuals with ADHD.

This thesis is just the first step of my research journey. Investigating the effectiveness of the neurofeedback and metacognitive training programs offered by the ADD Centre has number of challenges. One of the hurdles is that this program took place in a clinic. In clinical settings, it is very difficult to conduct controlled research studies because it is unethical to put the patients in a control group and not provide the best interventions for them. Without a control group or comparable group, it is important to determine whether the training effect established in this study could be found in a research setting in the future. It is also important to compare the neurofeedback and metacognitive training program to other attention or executive function training programs and medications to determine whether this training program is superior in the long run and to support the notion that this training is not simply another form of cognitive-behavioural therapy as Loo and Barkley suggested (2005).

If the neurofeedback and metacognitive training program is effective in decreasing ADHD symptoms in students, I think future research could explore generalization of the intervention to school settings. It would be ideal for schools to incorporate this training for students with ADHD to help reduce their disruptive ADHD behaviours and improve their ability to focus in class. Although neurofeedback is a costly intervention, it is also a non-invasive treatment of ADHD in comparison to medication. By implementing neurofeedback in school, the school can reduce 1:1 teacher assistance, save other extra costs in other intervention programs, and eventually, it may help the school reduce costs in a long run. When the ADHD symptoms have been reduced, other risk factors associated with the ADHD symptoms would also be improved such as social and academic functioning. Therefore, by employing the combined training in school, it is a more proactive strategy to address the any current and future behavioural, academic, and social difficulties that students with ADHD encountered.

Throughout this research journey, I realized that no one research study could address all the limitations. By recognizing the limitations of this study, it provides me with directions for future research. The most important lesson that I learned from this research project is learning to define an approachable size for your own research within the limited time frame. With the proper planning, it will make the rest of the project go much smoother.

The findings in this study suggest that the neurofeedback and metacognitive training could be apply as an alternative treatment with the promising short- and long-term training effect. Although this combined training may be costly and time consuming, having medication, which does not provide a long-term reduction of ADHD symptoms, can be more expensive over the long run. Therefore, with more researchers to support the results from this study in the future, hopefully this combined training could eventually become one of the mainstream treatments for individuals with ADHD.