A comparison of two forms of treatment for children with Attention Deficit Hyperactivity Disorder: Effects on executive functioning and behaviour.

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A research thesis submitted in partial fulfilment of the requirements for the degree, Doctor of Psychology (Clinical Neuropsychology)

August 2007

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Declaration

I, Monique Roper, declare that the Doctor of Psychology (Clinical Neuropsychology) thesis entitled, "A comparison of two forms of treatment for children with Attention Deficit Hyperactivity Disorder: Effects on executive functioning and behaviour" is no more than 40,000 words in length, exclusive of tables, figures, appendices and references. This thesis contains no material that has been submitted previously, in whole or in part, for the award of any other academic degree or diploma. Except where otherwise indicated, this thesis is my own work.

Signature: Date:

Acknowledgements

The following people have contributed so much in the completion of this thesis and I am extremely grateful to them all. Thank you.

My academic supervisor, Dr Alan Tucker, has been a great source of inspiration. His knowledge, passion for the field and unwavering ability to always look on the bright side has been motivating and invaluable. Also thank you to the paediatricians who were so generous in their support of this thesis. Their input and assistance with the recruitment process was much appreciated.

A very special thank you is extended to my unbelievably supportive parents and family, wonderful friends and sympathetic work colleagues who have endured the long and arduous thesis rollercoaster ride with me. They have been a constant source of moral support and have kept my sanity intact along the way!

I am especially grateful to my partner, Andrew, for his belief, patience, good humour and endless support throughout the duration of my thesis journey. I'm sure the process of writing a thesis was just as difficult for him as it was for me! But finally I can provide a satisfactory answer to his long-standing question, "Are you finished yet?"

Finally, to all the children and families who generously volunteered their valuable time to participate in the study, I thank you. Without you this study would not have been possible.

Abstract

Attention Deficit Hyperactivity Disorder (ADHD) is a common childhood disorder presenting with a variety of behavioural and cognitive manifestations. Recent conceptual theories of ADHD have implicated a key impairment in executive functioning, namely inhibition, however inconsistencies in the pattern of findings warrant further investigation. As a consequence of increasing awareness of the underlying deficits in ADHD, studies have emerged dedicated to investigating the efficacy of interventions, including psychostimulants and multimodal treatments (medication and behavioural therapy). Whilst the short term efficacy of these treatments is supported, there is a relative absence of convincing empirical evidence to support long term treatment in improving behaviour and cognition. Furthermore, the additive benefit of a combined treatment approach remains contradictory. The aim of the present study was to explore the executive function profile of ADHD and determine the impact of two treatments: medication alone and low intensity, family centred combined therapy treatment on cognition and behaviour. Cognitive test performance and parent ratings relative to published norms were assessed among 27 school age children with ADHD. These children were either assigned medication alone or combined therapy and were followed up over six months to compare treatment efficacy. Changes in performance based executive functioning and parent reported behaviours were evaluated at baseline, three months and six months for the two treatment groups. Results showed that the ADHD children performed poorly relative to test norms across most cognitive and behavioural measures sensitive to executive functions at baseline, however not all children demonstrated significant impairments in inhibition. Both treatment groups evidenced significant improvements with treatment over the six month follow-up period, however this is one of the first studies showing that the combined group was associated with greater and wider ranging improvements than medication alone treatment. Correlational analyses revealed mostly non-significant or low to moderate relationships between objective and subjective executive function measures. These results are discussed in the context of Barkley's theory of ADHD (1997a) and the value of low intensity, family centred combined treatment for ADHD.

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Chapter 1: Attention Deficit Hyperactivity Disorder: An Introduction

1.1 Definition and Current Classification

Attention Deficit Hyperactivity Disorder (ADHD) is a common childhood disorder that presents with a variety of behavioural and cognitive manifestations typically associated with significant detrimental effects upon a child's academic performance, social development, peer functioning and self-esteem. Specifically, the disorder's broad range of difficulties can often lead to poor academic achievement, learning difficulties, suspension or expulsion from school, conduct problems, delinquency, and poor peer and family relations. Its persistence into adolescence and adulthood is also associated with a range of serious long term negative consequences, including chronic peer and social difficulties, elevated rates of depression and anxiety, substance use, criminality and limited vocational outcomes (Barkley, 1997a; Berwid et al., 2005). Furthermore, its impact upon families and society is enormous in terms of financial cost, stress to families and disruption to schools.

According to the Diagnostic and Statistical Manual - Fourth Edition - Text Revision (DSM-IV-TR), current prevalence rates of ADHD are estimated in the range of 3% to 7% in school age children, however recent Australian statistics have revealed figures that at least double those estimates, with the National Survey of Mental Health and Wellbeing (1998) reporting that approximately 11% of Australian children aged between six and 17 have a diagnosis of ADHD (Sawyer et al., 2000). It is also well documented in the literature, that the disorder is reported with increased frequency in males than among females, by ratios ranging from 4:1 to 9:1 depending on the setting (Schachar, Mota, Logan, Tannock & Klim, 2000).

The term ADHD is currently used to describe a set of broad behavioural characteristics consisting of developmentally inappropriate degrees of inattention, hyperactivity and impulsivity. These overt core symptoms manifest in children as a myriad of behavioural and cognitive impairments with diagnoses being made based

upon qualitative behavioural observations of the presence of these set of symptoms. Inattention is revealed in behaviours such as difficulties in sustaining attention, poor task persistence or avoidance of tasks requiring mental effort, shifting from one uncompleted task to another, failure to listen to instructions, forgetfulness, episodes of staring and by distraction of irrelevant stimuli or a need for frequent stimulation (Barkley, 1999; Wagner, 2000). Hyperactivity may manifest in such behaviours as fidgetiness, excessive and inappropriate running or climbing, difficulty engaging in and sustaining activities and excessive talking. Whilst impulsivity is typically manifested as low frustration tolerance, impatience, blurting out of answers inappropriately, unpredictable behaviour, frequent interrupting and difficulty waiting one's turn (American Psychiatric Association [APA], 2000).

The DSM-IV-TR describes the essential feature of ADHD as a "persistent pattern of inattention and/or hyperactivity-impulsivity that is more frequent and severe than is typically observed in individuals at a comparable level of development" (APA, 2000, p85) In order to fulfil diagnostic criteria for ADHD, the symptoms must have developed prior to age seven and be relatively pervasive across multiple contexts, typically in the home and school environments. ADHD is also considered to be relatively chronic during childhood development therefore a diagnosis can only be made after the symptoms have been present for a period of at least six months. In accordance with diagnostic criteria, there must also be interference of developmentally appropriate social, academic or occupational functioning resulting in significant impairment in these major life activities. This must not be better accounted for by severe intellectual disability, pervasive developmental disorder, psychosis or any other mental disorder (APA, 2000). Although most children display symptoms of both inattention and hyperactivity/impulsivity, many exhibit one predominant pattern of behavioural symptoms. The DSM-IV-TR therefore presents ADHD as subdivided into three categories, reflecting the possible combination of the two dimensions: ADHD, predominantly hyperactive-impulsive type; ADHD, predominantly inattentive type and ADHD, combined type (APA, 2000). To meet the

criteria for diagnosis of an ADHD subtype, a child must present with six or more symptoms from each behavioural category.

1.2 Aetiology of ADHD

Despite there being a substantial body of research investigating the underlying causes of ADHD, various aspects of the aetiology of the disorder remain largely unclear and poorly characterised. However, recent reviews of studies examining the underlying pathogenesis of ADHD generally support a complex, multi-factorial aetiology (Berlin, Bohlin, Nyberg & Janols, 2004; Biederman, 2005; Bradley & Golden, 2001). Based on current literature, it appears that a complex interplay of genetic, biological, and psychosocial factors all contribute to the manifestation of ADHD symptoms. Furthermore, it is likely that there are probable adverse cumulative effects among all of these variables.

Confirmatory evidence for a genetic component in the aetiology of ADHD has emerged from various family, twin and adoption studies, with these consistently yielding support for a strong pattern of heritability in the disorder (Aron & Poldrack, 2005; Castellanos & Tannock, 2002). In particular, Biederman (2005) documented markedly elevated rates (two to eight fold) of ADHD amongst families with a parent or sibling with the disorder. Furthermore, Faraone, Biederman and Friedman (2000) reported that 30-35% of children with a full sibling diagnosed with ADHD, also fulfilled the diagnostic criteria for the disorder, a percentage that is substantially more than that found in the normal population. Twin studies have also proved useful in demonstrating the heritability of ADHD, whilst evidence from adoption studies has also provided convincing support for the notion of an inherited component in the aetiology of ADHD (Aron & Poldrack, 2005; Bradley & Golden, 2001; Castellanos & Tannock, 2002; Denney, 2001; Sprich, Biederman, Crawford, Mundy & Faraone, 2000).

A number of studies have also established a biological basis for the development of ADHD. Neuroanatomical, neuroimaging and neurochemical research findings have repeatedly demonstrated the significance of specific brain regions and certain neurotransmitters as underlying causes for the disorder. Abnormalities have been consistently identified in specific neuroanatomical systems, namely the frontal-sub-cortical pathways, whilst neurochemically, imbalances in the dopaminergic and noradrenergic systems have been postulated to contribute to the pathophysiology of ADHD (Biederman, 2005; Bradley & Golden, 2001; Bush, Valera & Seidman, 2005; Casey et al., 1997; Faraone et al., 2005; Hale, Hariri & McCracken, 2000; Raz, 2004; Solanto, 2002; Wagner, 2000).

Various other deleterious biological and environmental factors are also thought to possibly play a role in the pathogenesis of ADHD. These include exposure to maternal smoking or alcohol use during pregnancy, low birth weight, pre-natal and post-natal complications, head injuries, lead contamination, food additives and diet, parental mental health and adverse social factors (Biederman, 2005; Bradley & Golden, 2001; Purdie, Hattie & Carroll, 2002).

From a brief review of current literature, it is evident that the aetiology of ADHD is vastly complex and very likely to be multifactorial in nature. Bradley and Golden (2001) propose that genetic defects and neurobiological dysfunction represent the primary pathophysiology of ADHD with environmental factors increasing the risk to an underlying predisposition. It is also likely that there is a strong additive effect between these variables, with studies showing significantly increased prevalence of ADHD in the presence of more than one of these factors (Levy, Hay & Bennett, 2006).

1.3 History of ADHD Conceptualisation

The view of ADHD has been reconceptualised numerous times since it first appeared as a diagnostic category in the Diagnostic and Statistical Manual 2nd Edition, with these various revisions closely reflecting the ongoing debate regarding the underlying aetiology of the disorder. Historically, ADHD was first classified as being a hyperkinetic syndrome of childhood, with the core dysfunction proposed as one of excess motor activity (Chess, 1960). However, as a result of the development of new models of understanding of the disorder, ADHD has witnessed a conceptual re-shaping over the past few decades. Traditionally characterised as a syndrome of "hyperactive" behaviour, ADHD has become increasingly viewed as a disorder of behavioural symptomatology in the context of deficient cognitive functioning.

To review briefly, the publication of the DSM-III in 1980 introduced a new clinical label, with the motor-based hyperkinetic disorder being given the new diagnostic name of Attention Deficit Disorder (ADD). Along with the name change, came a refinement of the diagnostic criteria, with the introduction of two subtypes of ADD: Attention Deficit Disorder with Hyperactivity and Attention Deficit Disorder without Hyperactivity (APA, 1980). No longer viewed as a disorder of excessive motor activity, the primary dysfunction underpinning the overt behavioural manifestations was proposed as a core impairment in key aspects of cognitive attentional processing (Douglas, 1972; Sergeant, Geurts, Huijbregts, Scheres & Oosterlaan, 2003; Wilding, 2003). However even though this model of deficient attention received early widespread interest and despite its centrality to the name of the disorder, the "attention deficit" theory postulated by Douglas in the early 1970's, has, in recent years, waned in its popularity and viability (Das & Papadopoulos, 2003; Nigg, 2001; Roodenrys, Koloski & Grainger, 2001; Schachar, 1991).

Further developments in the conceptualisation of ADHD have been made in the last decade, which are reflected in the new diagnostic criteria in the DSM-IV-TR. Two broad categories of symptoms now exist, Inattention and

Hyperactivity/Impulsivity, with the addition of impulsive symptoms into the clinical picture reflecting the more contemporary approaches to the underlying mechanisms of the disorder. Moving away from an explanation in terms of basic attentional processes, some of the more recent models have implicated higher order cognitive processes of executive functioning or impairments in motivational processes as the core deficits in ADHD.

A number of motivation based dysfunction models have recently been proposed to account for the observed behavioural and cognitive difficulties in ADHD. Sonuga-Barke (2005) argues that children with ADHD can be described as delay aversive. Specifically he claims that disturbances in motivation contribute to the manifestation of ADHD symptoms. This theory suggests that children are not incapable of controlling their impulsive behaviour rather they are unwilling to delay their need for immediate gratification. According to Sonuga-Barke (2005) this makes children appear impulsive as a result of their attempt to minimise any delay in receiving rewards or reinforcement. An alternative motivational explanation has been provided by Quay (1997). He postulated that ADHD is likely attributed to diminished activity in the brain's behavioural inhibition system (BIS) which fails to adequately inhibit behaviour in response to punishment or non-reward (Crone, Jennings and van der Molen, 2003; Quay, 1997). Both of these motivational models, the delay aversion hypothesis and the BIS theory, continue to receive attention and support in the current literature (Kuntsi, Oosterlaan & Stevenson, 2001; Quay, 1997; Slusarek, Velling, Bunk & Eggers, 2001; Sonuga-Barke, 2005).

1.4 Current ADHD Conceptualisation

1.4.1 Neuropsychological Conceptualisation of ADHD – The Executive Dysfunction Hypothesis

The most recent and widely accepted conceptual theories of ADHD have implicated a central impairment in executive functioning as the primary underlying deficit of the disorder. Current models are presently utilising a neuropsychological framework, emphasizing impaired "higher order" cognitive functioning as the key deficient process in the manifestation of ADHD symptomatology. Recent theories have attempted to unify the myriad of symptoms of ADHD by examining them within the context of a general "executive function impairment". The broad concept of executive functioning, however, does not represent a unitary cognitive process, rather the term encompasses inter-related processes responsible for higher order regulatory and purposeful goal directed behaviour (Willcutt, Doyle, Nigg, Faraone & Pennington, 2005). Specifically defined, executive functions are 'the ability to maintain an appropriate problem-solving set for attainment of a future goal" (Welsh & Pennington, 1988, p.201). Disparities tend to exist with regard to the specific cognitive and behavioural processes associated with executive functions, however common elements are discernable. The principal executive functioning processes include goal selection, strategic planning, initiation, self-regulation, mental flexibility, attentional allocation, working memory and inhibitory control (Anderson, 2002). According to neuropsychological theory, executive dysfunction in ADHD is thought to interfere with the normal capacity to direct and maintain behaviour, resulting in problems with impulsivity and inattention (Brocki & Bohlin, 2006).

The current working hypothesis of ADHD therefore incorporates executive dysfunction, as characterised by these higher order cognitive processes, as the central deficit in ADHD. This notion has been widely supported by a number of recent empirical studies and reviews, with an extensive literature confirming poor performances of children with ADHD on neuropsychological tasks purportedly sensitive to executive dysfunction. A decade ago, Pennington and Ozonoff (1996) published a meta-analytic

review systematically examining 18 studies investigating the profile of executive functioning in children with ADHD. In their comprehensive review of the available evidence for an executive function deficit, they revealed that 15 out of the 18 evaluated studies reported significantly impaired performances in children with ADHD on a range of executive function measures when compared with normal children. From their investigation, the authors concluded that consistent weaknesses can be identified across many executive domains in ADHD (Pennington & Ozonoff, 1996).

More recently, Willcutt and colleagues (2005) replicated and extended the findings of Pennington and Ozonoff's meta-analysis in an updated review of 83 studies. Analogous to the previous review, Willcutt et al., (2005) sought to validate the neuropsychological theory suggesting that the behavioural and cognitive manifestations of ADHD arise from a primary impairment in executive functions. In line with the original review, the results of their analysis also revealed significantly poorer performances of children with ADHD as compared to a normal control sample on all measures of executive functioning with the overall effect size falling in the moderate range (.54). Findings of executive dysfunction were the most significant and consistent in the domains of response inhibition, planning, vigilance, and working memory. Based on these results, the authors concluded that executive dysfunction is associated with the observed deficits in ADHD.

In further support of a core impairment in executive functioning, Seidman and colleagues (1997b) compared the neuropsychological functioning of 140 children with ADHD to 120 age-matched paediatric controls. Performances on a range of executive tasks revealed pervasive higher order cognitive impairments, including weaknesses in interference control, cognitive flexibility, self-monitoring, impulsivity and planning (Seidman, Biederman, Faraone, Weber & Ouellette, 1997b). Similarly, Muir-Broaddus, Rosenstein, Medina and Soderberg (2002) compared the neuropsychological test performance of 78 children with ADHD to published normative data on numerous cognitive tasks of executive functioning. In support of the executive dysfunction hypothesis, ADHD children exhibited weaknesses relative to norms on most of the

measures sensitive to executive functions, including a Continuous Performance Test, the Wisconsin Card Sorting Test, and Digit Span.

Furthermore, Shallice et al., (2002) administered a battery of executive function measures to 31 children with ADHD aged between seven and 12 years in an attempt to further characterise the cognitive profile of the disorder. Based on performances on a variety of executive measures, including the Stroop Color and Word Test, a Letter Fluency Task and Working Memory Test, it was concluded that there were confirmed executive function deficits in their clinical sample. Finally, differential patterns of executive functions were examined in 94 children with ADHD in a study by Houghton and colleagues (1999). Children with ADHD were found to be significantly impaired on neuropsychological functioning compared to a sample of age-matched controls. These impairments were evident on the Wisconsin Card Sorting Test, the Stroop Color andWord Test, the Trail Making Test and the Tower of London.

In reviewing the literature employing neuropsychological methodology to evaluate the cognitive profile of children with ADHD, the existence of well established and reliable deficits on a range of executive function measures provides substantial confirmatory evidence to support the executive dysfunction hypothesis (Brocki & Bohlin, 2006; Fischer, Barkley, Smallish & Fletcher, 2005; Fuggetta, 2006; Lazar & Yitzchak, 1998; Reeve & Schandler, 2001; Rhodes, Coghill & Matthews, 2005; Seidman et al., 1997b; Sergeant, Geurts & Oosterlaan, 2002; Stins et al., 2005). However despite this seemingly overwhelming empirical support for the notion of a general deficit in executive processing in ADHD, such findings are not always consistently replicated, with many studies revealing inconsistent or inconclusive results. For example, there are studies reporting executive function test scores within the normal range, with a number of recent studies revealing only a proportion of their ADHD sample to demonstrate significant executive function impairments (Brown, 2006; Doyle, Biederman, Seidman, Weber and Faraone, 2000; Nigg, 2005). In addition, impairments are not always demonstrated across the range of executive function measures, with numerous studies citing deficits on particular tasks, such as those assessing interference control, working memory, verbal

fluency, attention set shifting and planning ability (Grizenko, Bhat, Schwartz, Ter-Stepanian & Joober, 2006; Mahone, Koth, Cutting, Singer & Denckla, 2001; Pocklington & Mayberry, 2006; Savitz & Jansen, 2003; Seidman et al., 1997a) whilst other studies fail to replicate these findings (Grodinsky & Diamond, 1992; Kempton et al., 1999; Scheres et al., 2004; van Goozen et al., 2004). Furthermore, variations within executive function performances have also been documented, with children exhibiting deficits in one executive function domain, whilst demonstrating no impairments in others. For example, in their study of 30 children with ADHD, Kempton and colleagues (1999) found that executive function deficits were not pervasive across all measures or all performance indices, with impairments only being exhibited on specific aspects of task performance.

This pattern of contradictory results may be in part attributable to persistent methodological limitations in the current empirical literature. A lack of consistent findings may represent differences across studies in the selection criteria utilised in defining and categorising ADHD, limited sample size, lack of control for co-morbidity and effects of gender and IQ, limited use of sensitive and valid neuropsychological measures, and failure to account for current medication status (Barkley, Grodinsky & DuPaul, 1992). For example, Scheres et al., (2004) found that after controlling for age and intelligence, previously identified deficits in response inhibition, letter fluency and planning were no longer significant. A consistent pattern of results may also be elusive due to the inherently broad nature of the domain of executive functions and the many skills that it encompasses. Consequently, the most recent empirical studies investigating the underlying nature of ADHD have focussed on determining the precise nature of deficits in a well defined aspect of executive functioning.

Chapter 2: Barkley's Unifying Theory of ADHD

Barkley (1997a) used the converging lines of evidence from neuropsychological research and existing theoretical frameworks to develop a model of ADHD based on a primary deficit in one key aspect of executive functions. In an attempt to account for the well documented executive function deficits and observed behavioural manifestations associated with ADHD, Barkley proposed a comprehensive unifying theory of ADHD that both complimented and built upon existing models of ADHD conceptualisation. In his theory, Barkley conceptualised the core deficit in ADHD (predominantly hyperactive-impulsive and combined ADHD subtypes) as being a specific developmental impairment in one domain of executive functioning, namely inhibition (Barkley, 1997a). To elaborate, Barkley defines inhibition as three interrelated processes: (1) inhibition of a prepotent response – a response for which immediate positive or negative reinforcement is available, or has previously been associated with that response, (2) inhibition of an ongoing response – this provides for a delay in the decision to respond or continue responding in the context of feedback and (3) interference control – which results in the protection of this period of delay from disruption by competing events and responses when engaged in self-regulatory tasks and goal-directed actions (Barkley, 1999). Therefore inhibition comes into play in situations requiring withholding or sudden interruption of an ongoing action or thought or in the suppression of information that one wishes to ignore.

Barkley's model also comprises a hierarchical component, with inhibition being viewed as primary to other executive functions. Specifically, inhibition must be the first process to occur in order to create a time delay during which subsequent executive functions may then be executed (Barkley, 1999). According to the theory, a failure to inhibit or delay a behavioural response leads to secondary impairments in four other executive functions that are dependent on inhibition for their effective execution and maintenance (Barkley, 1999). These four executive functions – working memory, internalisation of self-directed speech, self-regulation and reconstitution – all serve to bring behaviour under the control of internally represented information and self-directed

actions. By doing so, the four functions permit greater goal-directed action and task persistence (Barkley, 1997a). Despite regarding inhibition as primary to other executive functions, this model does not propose a direct causal influence of inhibition on these functions, rather it "sets the occasion" for their occurrence by providing an interference free delay necessary for them to occur (Barkley, 1997a, p.68).

According to Barkley (1999), deficits in inhibitory control create the characteristic cognitive and behavioural symptoms of ADHD, such as acting impulsively without thinking, because children with ADHD miss out on the benefits of these important control strategies. Barkley (1999) therefore proposes that ADHD symptoms previously associated with inattention can now be conceptualised as a lack of goal oriented persistence directly resulting from deficient inhibition.

2.1 Barkley's Unifying Theory: The Empirical Evidence

Various studies in the recent literature have utilized Barkley's theoretical framework as a basis for research, with many lending support for his unifying model of ADHD. Consistent with his theory, a relatively large number of empirical studies have documented findings showing that children with ADHD tend to under-perform on a range of neuropsychological measures of response inhibition in comparison to their peers.

An executive function test that has been extensively utilised in the ADHD literature as a classic measure of response inhibition is the Stroop Color and Word Test (SCWT) (Golden, 1978). The sensitivity of the SCWT to the proposed core inhibitory deficits of children with ADHD has been repeatedly demonstrated, with several studies citing significant impairments on the important interference trial of the task. A poor performance on this component of the SCWT is thought to be indicative of a decreased ability to inhibit competing over-learned responses, and is therefore considered to be a reliable measure of inhibition (Berlin et al., 2004; Cohen, Weiss & Minde, 1972; van Goozen et al., 2004). Two meta-analytic reviews by Barkley and colleagues (1992,

1997a) comparing ADHD children to matched controls on the SCWT, revealed significant group differences across this task, with the children with ADHD performing significantly worse than normal controls (Barkley, 1997a; Barkley et al., 1992). Similarly in a large sample of ADHD children (n = 118), Seidman et al., (1997b) provided confirmatory evidence for deficient inhibitory control, demonstrating impairments across three SCWT components in their ADHD children comparative to a sample of normal controls. Likewise, Reeve and Schandler (2001) reported findings to support Barkley's hypothesis, with ADHD adolescents exhibiting significantly poorer results on the color, color-word and interference components of the SCWT relative to matched controls. Finally, in a review of 287 children with ADHD across three research centre sites, Nigg, Willcutt, Doyle and Sonuga-Barke (2005) reported medium to large effect sizes (.5 to .84) for performances on the color word condition of the SCWT. Based on these and other similar findings, the SCWT has been argued to be one of the most sensitive tests to the proposed inhibition impairments in ADHD (Barkley, 1997a; Pennington, Groisser & Welsh, 1993; Pennington & Ozonoff, 1996; Sergeant et al., 2002).

In contrast, however, a number of researchers have been more cautious in their support of a response inhibition deficit in ADHD based on performances on the SCWT. For instance, Nigg (2001) argued that many studies have failed to reliably control for speed of response on the SCWT (based on word and colour naming ability), claiming that observed impairments on the SCWT may be equally attributed to poor speeded response as to interference control deficits. In support of this, Pocklington and Mayberry (2006) provided substantial support for speed of processing deficits in ADHD children rather than deficient inhibitory processes in their meta-analytic review of 13 SCWT studies. Similarily, van Mourik, Oosterlaan and Sergeant's meta-analysis (2005) of 17 studies also provided only minimal support for an interference deficit in ADHD, as measured by the SCWT. Their findings revealed poorer performances of ADHD children on the color naming and word reading components, with no differences being found on the inhibition sensitive interference component. Finally, in their review of 33 published studies, Homack and Riccio (2004) found ADHD children to perform poorly relative to controls

across *all* SCWT components. Contrary to expectations they did not find exclusive deficits on the color-word and interference trials, as would be expected if deficient inhibitory control, and not slowed information processing, was the core impairment.

In contradiction to these findings Seidman, Biederman, Mounteaux, Weber and Faraone (2000) reported significant impairments on the interference control condition even after controlling for processing speed, suggesting that there is indeed evidence for an inhibition deficit on the SCWT in ADHD children. Other studies examining the SCWT interference score, when corrected for reading and naming speed, have also clearly reported significant group differences (Lufi, Cohen & Parish-Plass, 1990; MacLeod, 1991). Currently however, there seems to be inconclusive evidence to confirm the presence of an ADHD inhibitory control deficit on the SCWT or to exclude the contribution of a processing speed problem.

Response inhibition is also operationalised by the processes involved in Continuous Performance Tests (CPTs), popular clinic based measures for the evaluation of selective attention, vigilance and impulsivity (Riccio, Waldrop, Reynolds & Lowe, 2001). The ability to sustain attention and focus on the CPT is evaluated by the number of omission errors (failure to respond to target stimuli), while the ability to inhibit responses to non-target stimuli (commission errors) is reported to be a measure of impulsivity (Bradley & Golden, 2001). A variety of CPTs have been routinely employed in an array of empirical studies investigating Barkley's model, with numerous studies yielding reliable support for an inhibitory deficit. Muir-Braoddus et al., (2002) compared the neuropsychological test performance on a variety of cognitive measures in a group of 78 children with ADHD relative to published test norms. Despite reporting pervasive impairments across the board on a range of executive function measures, the authors concluded that the CPT (along with performance on a memory task) was the most sensitive measure in detecting deficits in their ADHD sample. Consistent with this finding, Stins and colleagues (2005) utilised the CPT in a study investigating the cognitive profile of ADHD in a well defined sample of 34 boys with the disorder. Compared to controls, their findings revealed evidence for greater impulsivity in the ADHD children, with the authors associating this

with a reduced ability to accurately inhibit responses. Furthermore, Fischer et al., (2005) examined the quantitative and qualitative performances on a CPT in a large group of young adults with ADHD. They demonstrated that in comparison to controls, the ADHD group produced significantly higher rates of inhibition errors, while also displaying elevated levels of ADHD symptoms during completion of the task.

However, while many studies demonstrate a trend for inhibition deficits on the CPT, a number of published studies have been less convincing in their results. Whilst Seidman et al., (1997b) found impairments on an auditory CPT in their sample of 118 children with ADHD, significant group differences were only found in the number of omission errors produced, not the number of commission errors made, suggesting problems in the ADHD group with focussed attention rather than impulsivity. This was exemplified by Douglas (1999) who found ADHD children's performances to be characterised by errors of omission, leading to his conclusion that poor attention is the core deficit in ADHD. Likewise, in a study employing the CPT in a sample of ADHD and control pre-school children, Mariani and Barkley (1997) reported that whilst the overall accuracy of the ADHD children was impaired compared to the non-ADHD group, no difference was evident in the number of commission errors made across the two groups. Finally, in a study evaluating the clinical utility of the CPT, Preston, Fennell and Bussing (2005) failed to find any significant differences between their "ADHD" group and "subclinical control" group on a visual CPT. However, confounding variables in their research design (some of which were controlled for in secondary analyses) may have potentially limited the findings of their study, including lack of formal diagnostic assessment of ADHD, significant co-morbidity (over half met the criteria for conduct disorder or oppositional defiant disorder) whilst almost 50% of children in the "ADHD" group were medicated with psychostimulants.

The Stop-Signal paradigm is an alternative measure of inhibition that has been extensively utilised in the ADHD literature (Schachar & Logan, 1990). The concept of the Stop Task is theoretically derived from an explicit model of inhibitory processes (the race model; Logan & Cowan, 1984) and is a well established direct measure of inhibitory

control that allows a distinction between two independent processes: execution of an action (a "go" response) and the cessation of this action (a "stop" process), identified as inhibition. A slowed "stop" response on this task has typically been characterised as indicating deficient inhibitory control (Schachar & Logan, 1990). The stop signal paradigm has yielded promising findings in the ADHD literature. Schachar and Logan (1990) were the first to reveal deficient inhibitory control in ADHD on the stop signal task. In comparing a small sample of children with ADHD with three other clinical groups (conduct disorder, learning difficulties and emotional disorder), the authors found evidence to support the idea of impaired inhibition in ADHD, revealing slowed and more variable inhibitory processes in this group. Schachar, Tannock, Marriott and Logan (1995) replicated these findings using the stop signal paradigm in a larger sample of children with ADHD. In their investigation, they demonstrated slower and increased variability in the response times of children with ADHD relative to age matched normal controls, which they interpreted as a deficit in inhibitory control. More recently, in their meta-analysis of eight published studies of behavioural inhibition using the stop signal paradigm, Oosterlaan, Logan and Sergeant (1998) concluded that despite contrasting methodological approaches between studies, there remained consistent evidence for less efficient inhibitory processes in ADHD. Numerous other studies have also supported the notion of impaired inhibitory control in ADHD, as characterised by slowing and higher variability in the execution of a response, with overall medium effect sizes (.64) for the stop signal paradigm (Barkley, 1999; Fugetta, 2006; Konrad, Gauggel, Manz & Scholl, 2000; Nigg, Blaskey, Huang-Pollack & Rappley, 2002; Rubia, Oosterlaan, Sergeant, Brandeis & van Leeuwen, 1998; Schachar et al., 2000).

Although thought to generally be indicative of a deficit in inhibition, findings of slow and variable responses have also been interpreted as a generalised deficit in speed of processing, impaired arousal, poor self-regulation and deficient motivation rather than a problem in inhibitory control (Daugherty, Quay & Ramos, 1993; Jennings, van der Molen, Pelham, Brock & Hoza, 1997; Kuntsi et al., 2001; Nigg, 1999). For example, Nigg (2001) interpreted his findings of slowed stop signal responses and high response variability in 25 children with ADHD as evidence for impaired arousal. Furthermore,

Kunsti and colleagues (2001) also explained their findings of slow, variable and inaccurate responses on the stop signal task as providing support for a reduced level of arousal and effort, rather than poor inhibitory control. Although there is emerging evidence supporting the idea of a generalised slowing of responses, to date the stop signal paradigm primarily remains a measure of response inhibition.

Numerous other studies also support the concept of central inhibition deficits based on the performance of ADHD children on a variety of executive function measures of inhibitory control. For example, children with ADHD have consistently been shown to make more inhibition errors with slowed performances on the Matching Familiar Figures Test, Trail Making Test Part B and Go/No-Go tasks (Berwid et al., 2005; Borger & van der Meere, 2000; Brown, Jaffe, Silverstein & Magee, 1991; Iaboni, Douglas & Baker, 1995; Seidman et al., 1997a; Shallice et al., 2002; Shue & Douglas, 1992). Other executive function measures of inhibition have also been utilised in the ADHD literature, although their use is more limited and they have produced less consistent results. Despite being recognised as reliable and sensitive indicators of executive dysfunction, measures such as the Controlled Oral Word Association Test (COWAT) and those assessing working memory (such as the Digit Span Backwards Test from the Wechsler Intelligence Scales), have been inconsistent in their ability to discriminate between children with ADHD and normal controls (Barkley & Grodinsky, 1994; Snow, Blondis & Brady, 1988; Spreen & Strauss, 1998). For example, many empirical studies have yielded results which have failed to find any significant difference between children with ADHD and normal controls with regards to performance on measures of phonemic and/or semantic verbal fluency (Barkley, Grodinsky & DuPaul, 1992; Fischer, Barkley, Edelbrock & Smallish, 1990; Loge, Staton & Beatty, 1990; Scheres et al., 2004; Shallice et al., 2002). In contrast, other studies have demonstrated the presence of impairments in verbal fluency in children with ADHD (Felton, Wood, Brown, Campbell & Harter, 1987; Grodzinsky & Diamond, 1992; Pineda, Ardila & Rosselli, 1999).

Despite its fundamental role in Barkley's model of ADHD, relatively few studies to date have employed measures of working memory with results thus far remaining

inconclusive. In a study of 51 children described as "pervasively hyperactive", Kuntsi and colleagues (2001) demonstrated working memory deficits, however these did not remain after controlling for the effects of IQ. Scheres et al., (2004) also failed to find working memory deficits in a group of 23 boys with ADHD, whilst Shue and Douglas (1992) were unable to demonstrate significant differences between ADHD children and controls on their working memory task. All of these aforementioned studies, however, employed measures of non-verbal working memory. In contrast, Shallice and colleagues (2002) revealed the presence of verbal working memory deficits in a well defined sample of 31 children with ADHD whilst Roodenrys et al., (2001) also found children with ADHD (with co-morbid reading disorder) to perform worse than controls on measures of verbal working memory. Finally, Berlin et al., (2004) and Roberts and Pennington (1996) both argued that working memory deficits contribute independently to the impairments observed in ADHD children.

Overall, the current trend in the ADHD literature appears to be one of support for Barkley's theory of inhibition with a substantial proportion of the empirical research providing confirmatory evidence for deficient inhibitory control in children with ADHD. However it is also clear that Barkley's inhibition hypothesis has not been universally supported. In reviewing the recent literature, it is evident that studies citing evidence against Barkley's theoretical formulations do exist, with several different lines of research producing results that are in contradiction to the inhibition model. In fact, Nigg and colleagues (2005) estimate that less than half of all ADHD children can be convincingly characterised as being impaired on any measure of inhibition. These overall inconsistencies in the pattern of findings warrant further investigation, with Barkley himself endorsing the need for more extensive research into the executive functions in children with ADHD (Barkley, 1997a).

2.2 Barkley's Unifying Theory of ADHD: Neuroimaging Evidence

The paradigm shift evidenced in the neuropsychological literature has also been supported by the emergence of electrophysical, biochemical and neuroanatomical evidence for a frontal lobe connection in the proposed executive dysfunction in ADHD. Firstly, the frontal lobes, particularly the prefrontal cortex, have been implicated to play a fundamental role in the executive functioning processes thought to be involved in ADHD, with lesions in these anterior brain regions producing the behavioural features characteristic of ADHD, including distractibility, impulsivity and hyperactivity (Solanto, 2002). Secondly, a parallel has been identified between the cognitive deficits observed in children with ADHD and those resulting from frontal lobe injuries, with damage to frontal circuitry presumed to be responsible for poor performances on neuropsychological tasks of executive functioning in ADHD (Bradley & Golden, 2001). Finally, neuroimaging research has demonstrated structural, electrical and metabolic abnormalities in the anterior regions of the brain, including asymmetric-dysmorphic conditions, abnormal electrical activity and hypoperfusion in the frontal (particularly prefrontal) lobes, in children with ADHD. In particular, a recent review of the empirical literature concluded that structural and functional deficiencies of the complex circuitry linking the frontal lobes to the subcortical structures specifically contribute to the executive dysfunction underlying the deficits observed in ADHD (Himelstein, Shultz, Newcorn & Halperin, 2000).

Recent neuroanatomical research, employing computerised tomography (CT) and magnetic resonance imaging (MRI), has consistently demonstrated structural brain differences in children with ADHD comparative to their age and gender matched normal peers. Based on structural imaging studies, consistent evidence has emerged implicating neuroanatomical abnormalities of the prefrontal cortex and subcortical structures in ADHD. Specifically, volumetric measures have detected smaller right prefrontal regions (including the dorsolateral prefrontal cortex) with reductions also observed in the anterior cingulate in children with ADHD (Castellanos et al., 1996; Hale et al., 2000) results which were not accounted for by age, weight, height, IQ or stimulant medication use

(Raz, 2004; Wagner, 2000). These findings have been significantly correlated with poorer neuropsychological performance on inhibition, verbal fluency and working memory measures (Casey et al., 1997; Seidman, Valera & Makris, 2005). Abnormalities in the right prefrontal cortex and reduced volumes in basal ganglia structures, including the caudate nucleus and globus pallidus, also appear to be correlated with higher levels of impulsivity (Castellanos et al., 1996). Consistent with earlier research, Seidman and colleagues (2005) replicated previous findings of abnormalities in the fronto-striatal networks in their recent meta-analysis. Furthermore, they found evidence for abnormalities in the corpus callosum, cerebellum, temporal, parietal and occipital lobes, proposing that the locus of brain dysfunction in ADHD may be more extensive than previously hypothesized.

In line with the structural neuroimaging research, functional imaging studies have produced findings consistent with the notion that the anterior and subcortical regions of the brain subserve executive functions. Studies utilising functional neuroimaging techniques, such as positron emission tomography (PET), single photon emission computed tomography (SPECT) and functional magnetic resonance imaging (*f*MRI), have revealed that specific executive functions can be localised and lateralised to particular brain regions. For example, the processes involved in response inhibition and interference control have been found to activate the right prefrontal cortex and anterior cingulate (Aron, Fletcher, Bullmore, Sahakian & Robbins, 2003; Bench et al., 1993; Carter, Mintun & Cohen, 1995); working memory ability is thought to be under the control of the dorsolateral prefrontal cortex (Owen, Evans & Petrides, 1996; Smith, Jonides & Koeppe, 1996), whilst verbal fluency tends to activate the left dorsofrontal cortex (Royall et al., 2002).

Dysfunction in inhibitory control characteristic of ADHD has subsequently been associated with abnormal activation of these regions and their connecting networks, with recent studies revealing significant hypoperfusion and hypometabolism in the frontal regions and subcortical structures in the brain in ADHD. For example, in a series of pioneering studies using SPECT technology in children with ADHD, Lou, Henricksen,

Bruhn, Borner & Nielsen (1984,1990) discovered reduced metabolic rates in the prefrontal cortices and decreased perfusion in the striatum in ADHD.

More recently, functional MRI (*f*MRI) studies have reported parallel findings in ADHD children who have undergone brain imaging techniques whilst performing cognitive tasks of inhibition. Rubia and colleagues (1999) investigated the frontal-striatal circuits of 7 unmedicated adolescents with ADHD using *f*MRI during completion of the Stop Task paradigm. Results supported anterior hypofunctionality, with performance throughout the Stop Task producing a reduced activation of the right prefrontal cortex and left caudate nucleus. Tamm, Menon, Ringel and Reiss (2004) also reported prefrontal hypofunction in the dorsal anterior cingulate cortex in ADHD adolescents during performance on a Go-No/Go task, whilst Bush et al., (1999) provided further support for the hypothesized anterior cingulate dysfunction in ADHD revealing decreased activity in this region on a Stroop task. Finally, Vaijdya et al., (1998) reported significantly reduced frontal and striatal activation in ADHD children compared to normal controls during performance on Go-No/Go tasks, with stimulant medication improving performance and levels of activity in these areas.

At a neurochemical level, dysfunction in the neurotransmitters predominantly mediating the anterior brain circuits has been implicated in the pathogenesis of ADHD. Specifically, it has been proposed that an under-activity of dopamine and norepinephrine results in a dys-regulation in the systems modulated by these catecholamines; the frontal and subcortical regions (Lou et al., 1984).

Specifically, Lou et al., (1984) proposed a theory of the pathophysiology of ADHD which posited that dopamine deficiencies in the mesencephalon-prefrontal cortex pathways play a role in the primary symptomatology of ADHD, namely deficits in hyperactivity and inattention. Further, Lou and colleagues theorised that methylphenidate re-activates these dopaminergic pathways leading to a reduction in the symptoms of inattention and hyperactivity. The dopamine theory has received support by animal

models and stimulant medication studies, confirming an inhibitory dopaminergic effect at the prefrontal level in the brain (Levy & Swanson, 2001).

Chapter 3: ADHD Intervention

3.1 Pharmacological Intervention

3.1.1 Mechanisms of Action

As a consequence of the increasing knowledge of the underlying mechanisms of ADHD, numerous studies have emerged dedicated to investigating the efficacy of various forms of intervention on the behavioural symptomatology of ADHD. Pharmacological intervention, primarily psychostimulant medications such as methylphenidate (MPH) and dexamphetamine, are the most frequently used and probably best supported treatment option for children with ADHD (Biederman, Spencer & Wilens, 2004; Forness, Kavale & Crenshaw, 1999; Greydanus, Sloane & Rappley, 2002). This is reflected in, and in part supported, by the considerable number of children prescribed stimulant medications for the treatment of ADHD. Recent figures estimate upwards of 50,000 Australian children with ADHD to be using psycho-stimulants to treat their symptoms, with recent trends indicating that the rate of psychostimulant use is continuing to rise (Howard Florey Institute, n.d.). In fact research estimates that stimulant medication prescriptions double every four to seven years (Riccio et al., 2001).

Although there is wide individual variation in metabolism and effect of MPH and dexamphetamine, both stimulants generally produce clinical effects within about 30 minutes of ingestion, with diminished benefits becoming apparent after about three hours (though dexamphetamine has a half life of approximately 11 hours compared with the methylphenidate half-life of around three hours) (Markowitz & Patrick, 2001). Recent research has also highlighted a divergence in stimulant medications with regards to independent effects on behavioural and cognitive mediated symptoms, with Pelham (1999) and Solanto (2002) both revealing longer lasting stimulant induced reductions in behaviour (ie, a 7-8 hour improvement in hyperactivity/increased motor activity) as compared to cognition (ie, 2-3 hours improvement in inattention).

Stimulant medications have been hypothesized to improve the clinical signs of ADHD symptomatology by acting on dopamine in the frontal and sub-cortical brain regions, areas implicated in hyperactivity, impulsivity and inhibition (Volkow, Wang, Fowler & Ding, 2005). Therefore, evidence suggests that the likely locus of therapeutic benefit for stimulant medications is in the facilitation of dopaminergic activity in these areas of the brain (Raz, 2004). Though similar in their clinical effects, stimulants are thought to differ in their specific neurochemical mechanisms. MPH appears to increase extra-cellular dopamine by facilitating the release of stored dopamine in the brain, while dexamphetamine is believed to also inhibit the re-uptake of dopamine post-synaptically, in addition to releasing newly synthesized dopamine (Shenker, 1992).

Converging evidence from neuroimaging and psychopharmacological studies have also lent support to dopamine involvement in the action of psychostimulants. Results of functional imaging studies generally corroborate the findings regarding the mechanism of stimulant medications in ADHD symptomatology. For instance, Lou, Henricksen, Bruhn, Borner and Nielsen (1989) have demonstrated that hypoperfusion in the frontal lobes and caudate-striatal regions in children with ADHD can be effectively ameliorated by the administration of psychostimulants. Similarly, Kim, Lee and Cho (2001) reported an increase in regional cerebral blood flow in the DLPFC and caudate nucleus after MPH administration in a group of previously stimulant naïve children with ADHD. Finally, using *f*MRI technology, Vaijdya et al., (1998) demonstrated increased activation in the frontal and striatal regions of the brain, with corresponding improved performances on a go/no-go task of inhibition in a group of 10 boys with ADHD following the administration of MPH. Therefore, overall findings from neurochemical and neuroimaging studies provide evidence to support that the beneficial therapeutic effects of stimulant medications lie in ameliorating the dysfunction in dopamine regulation with consequent increases in fronto-striatal brain activity.

3.1.2 Pharmacological Intervention: Behavioural Outcomes.

Extensive clinical research published over the last twenty years attests to the efficacy of stimulant medications in reducing the core behavioural symptoms in children with ADHD. Reports on the positive therapeutic behavioural effects of psychostimulants are well documented in the literature, with the majority of studies demonstrating rapid and significant improvements in terms of reducing the core behaviours of ADHD, including parent and teacher reports of hyperactivity, impulsivity and inattention, as well as associated overt behaviours, such as social interactions and oppositional and defiant behaviour (Barkley, 1998) Recent figures estimate therapeutic benefits of stimulant medications in approximately 70% to 80% of children diagnosed with ADHD, with associated large effect sizes (in the range of .5 to .9) on behavioural outcomes (Barkley, 1990; Pelham, Wheeler & Chronis, 1998; Schachter, Pham, King, Langford and Moher, 2001). The benefits of pharmacological intervention also tend to represent dose dependent effects, with larger doses typically producing greater improvements on overt behaviours (Pearson et al., 2004; Rapport & Kelly, 1991; Schachar & Tannock, 1993).

Several well controlled clinical studies and reviews have provided consistent evidence for the *short term* efficacy of stimulant medications in alleviating the core behavioural symptoms of ADHD. An early meta-analysis on the effects of medication on hyperactivity by Thurber and Walker (1983) demonstrated that the strongest drug-related improvements were found on measures of attention and distractibility as measured by parental and teacher rating scales. Further, a meta-analysis by Ottenbacher and Cooper (1983) focusing on the behavioural outcomes of children with ADHD following administration of MPH, similarly revealed that stimulant medication was effective in reducing hyperactive behaviour. More recently, in their thorough review of 115 clinical studies, Crenshaw, Kavale, Forness and Reeve (1999) replicated these findings, demonstrating overall moderate to large effect sizes for the short term efficacy of stimulant medications on a range of parent and teacher reported assessments of behaviour, a finding also consistent with Purdie et al's (2002) meta-analysis of 74 studies of children with ADHD.

Schachter and colleagues (2001) published an updated synthesis of the treatment literature in a comprehensive review of 62 randomized placebo controlled studies completed between 1981 and 1999. Their meta-analysis compared the effectiveness of MPH and placebo in almost 3000 children with a primary diagnosis of ADHD on a range of behaviourally defined outcome measures, assessed via parent and teacher reports. Improvements in ADHD symptoms with MPH were observed with medium to large effect sizes found on all primary outcome measures.

In view of the findings from these and numerous other studies in the literature, there appears to exist an overwhelming favorable consensus in regards to the short term efficacy of stimulant medication on behavioural symptoms of ADHD. Yet despite yielding such conclusive results, these ostensible positive outcomes are often tempered by pervasive publications biases and poor methodological quality which limit the robustness, generalisability and external validity of the findings. In particular, Schachter and colleagues (2001) highlighted a range of weaknesses common among the many clinical studies utilized in their meta-analysis. These included poor reporting or identification of co-morbid diagnoses, unclear inclusion or exclusion criteria, biased publishing of significant results (including not reporting those findings in which children on stimulant medication performed less well than those receiving placebo), small sample sizes and inadequate description of methodology, including poor detailing of interventions and randomization processes, with the authors concluding that serious doubts may be cast on the strength of their findings (Schachar et al., 2002; Schachter et al., 2001). Furthermore, despite the well established benefits of stimulant medications on behaviour, Pelham and colleagues (1998) challenge the commonly held view that reported improvements on behavioural outcomes represent clinically significant changes. Rather, their findings indicate that even though behaviour is improved in 70%-80% of children, complete normalization is not achieved, with a substantial proportion of these children still remaining below age expected levels. Moreover, in his review of 14 studies, Barkley (1990) reported that although two thirds of children with ADHD improved with medication, almost one third remained unchanged, with some even demonstrating a deterioration in their behavioural functioning. These findings constitute a relatively small

part of the literature, with the vast majority of studies supporting the widely held notion that stimulant medications produce pervasive clinically significant improvements in behaviour in children with ADHD.

Empirical research into the short term benefits of stimulant medications constitute a considerable portion of the ADHD intervention literature, in contrast however there exists a clear lack of well controlled extended outcome treatment studies in children with ADHD. A review of the published meta-analyses revealed that many 'long term' studies have been rated as being unsatisfactory in terms of methodological quality and on average, duration of follow-up of psychostimulant treatment lasted only three weeks, with the vast majority of studies reporting treatment follow-up of no more than ten days (Schachar et al., 2002; Schachter et al., 2001; Vitiello 2001). Of the available studies that have extended follow-up beyond a month, few of them have demonstrated any uniform treatment effects, with many failing to achieve statistical significance in treatment outcomes (Schachter et al., 2001). For example, Lewin and Fletcher (1993) were unable to demonstrate continued beneficial effects of stimulant medications on behaviour following 12 months of pharmacological intervention, whilst Kupietz, Winsberg, Richardson and Maitinsky (1988) found that stimulant medications failed to produce ongoing benefits to behaviour in a group of children with ADHD over a period of six months of psychostimulant alone intervention. In addition, Schachter et al., (2001) failed to find treatment effects of stimulants beyond four weeks of intervention, with Swanson and colleagues (1993) cautioning that stimulant medications are only responsible for temporary reductions in the behavioural symptoms of ADHD.

Despite a somewhat limited literature, there are a few studies documenting continued behavioural improvements with extended stimulant medication use. In their randomized placebo controlled study of 91 children with ADHD, Schachar, Tannock, Cunningham and Corkum (1997) provided supportive evidence for ongoing beneficial effects of stimulant medications over four months of continued treatment. Results of their study found a significant reduction in the core behavioural symptoms of ADHD in the classroom as evaluated by teachers on the Iowa Conners Rating Scale. However, in

contrast to their initial hypothesis supporting general situational improvements, medication benefits were only limited to the school environment, with parents failing to report significant improvements in problem behaviours at home. Conversely, Gillberg et al's., (1997) randomized, double blind placebo controlled study of 62 children with ADHD between the ages of six and eleven, produced similarly positive although somewhat divergent findings. Significant improvements were observed across an extended treatment follow-up period, with 15 months of continued stimulant medication use resulting in superior improvements in attention, hyperactivity and other associated disruptive behaviour problems compared to placebo as measured by the Conners Teacher and Parent Rating Scales. However, unlike Schachar and colleagues (1997) findings, Gillberg et al's., (1997) study revealed that the most marked behavioural improvements were reported by the parents in the home environment. Particular constraints to this study, however, warrant caution in the interpretation of results. For instance, although all children met diagnostic criteria for a primary diagnosis of ADHD, almost half of the sample had co-morbid diagnoses, ranging from autism and intellectual difficulties to conduct disorder and oppositional defiant disorder. The presence of a co-morbid disorder has been reported to influence response to medication, which would argue for the use of a more homogenous sample of ADHD children in determining medication effects (Grizenko et al., 2006). In addition, publication biases were indicated, with ten of the original cohort being excluded due to being refractory to treatment and experiencing side effects and a very high attrition rate in the placebo group (over 70%). These limtations question the suitability of this group as a reliable comparison.

In a well controlled randomization study that extended treatment follow-up to five years, Charach, Ickowicz and Schachar (2004) investigated the long term effectiveness of stimulant medication use in three groups of children with ADHD: those adhering to medication, non-adherents prescribed medication and non-adherents not taking medication (N=79), all of whom had received systematic annual evaluations of their behaviour as rated by their parents and teachers on the Ontario Child Health Survey Diagnostic Instrument – Revised. The results of the study showed that children who consistently used stimulant medications exhibited consistently greater reductions in

behaviour at consecutive yearly evaluations over a five year follow-up period, than those children not adhering to a medication regime. Charach and colleagues (2004) findings are among the first to provide support for the benefits of ongoing medication use over a period of time more in line with that typically observed in clinical practice.

Aside from the noticeable observation that there are a limited number of well controlled extended treatment outcome studies, a review of the literature also reveals a pervasive trend for the routine utilization of parent and teacher reported behavioural measures, such as questionnaires and checklists, as primary indicators of outcome in measuring the efficacy of stimulant medications in ADHD. Evaluating the overt behavioural manifestations of ADHD is a popular method of assessment, because, as opposed to the frequently heterogeneous cognitive impairments, the behavioural profile of ADHD tends to be more homogenous, with behaviour rating scales and questionnaires often demonstrating much larger effect sizes in response to treatment than measures of cognition (Hale et al., 1998; Solanto, 2002). However, one shortcoming of these subjective behavioural techniques is their generally limited ability to provide objective information about the core cognitive processes in ADHD, such as the proposed response inhibition deficits. Furthermore, behavioural measures based on parent and teacher's subjective assessment of behaviour may be influenced by halo effects, biases in recall, and other potentially confounding factors (Seidman et al.,1997b).

A useful adjunct to subjective behavioural measures may be the use of standardized cognitive tests. This form of evaluation directly and objectively measures cognitive functioning, which is important in determining whether stimulant medication treatment demonstrated to improve overt behavioural symptoms, can also be beneficial to the underlying cognitive control processes implicated in ADHD. Cognitive performance measures also have sound psychometric properties having attained their validity and reliability from systematic and comprehensive empirical studies with normal controls and clinical populations (Nigg, 2001). However, despite these inherent advantages of standardized neuropsychological tests, the majority of empirical research examining the effects of stimulants on ADHD symptoms solely measure outcomes in terms of change in

behaviour based on subjective reports. This leaves relatively few that empirically evaluate the efficacy of stimulant medications in children with ADHD based on cognitive outcomes from neuropsychological measures.

3.1.3 Pharmacological Intervention: Cognitive Outcomes

Whilst there exists extensive available evidence providing support for the short term use of stimulant medications in reducing the overt behavioural symptoms of ADHD, there is a more limited body of literature investigating the effects of psychostimulant medication on the core cognitive processes in children with ADHD.

To date there have only been a handful of well controlled published studies that have investigated the cognitive domain of executive functioning outcome following therapy with stimulant medication. Of the available studies, the majority of them, which largely consist of short term studies, (ie., single dose or brief randomised placebo controlled trials) are in support of at least an acute amelioration of a variety of core cognitive deficits, including improvements on cognitive measures of attentional set shifting, visual search, focused attention, cognitive flexibility, working memory, planning ability and perseveration (Aman, Roberts & Pennington, 1998; Douglas, Barr, Desilets & Sherman, 1995; Everett, Thomas, Cole, Levesque & Michaud, 1991; Fugetta, 2006; Hazel-Fernandez, 2004; Hood, Baird, Rankin & Issacs, 2005; Kempton et al., 1999; Mehta, Goodyer & Sahakian, 2004). However, preliminary findings from Mehta and colleagues (2004) study of the performance of 14 boys with ADHD on the Cambridge Neuropsychological Test Automated Battery (CANTAB), revealed that whilst psychostimulants generally have the effect of enhancing executive functioning, the administration of stimulant medications may also be detrimental to performance, with results of their study showing that a medium dose of methylphenidate actually induced planning deficits in their sample of ADHD children.

Specific studies examining medication effects on the proposed core inhibitory processes of ADHD are significantly more limited, with only a small number of well controlled clinical trials currently available in the published literature. Due to the restricted range of investigations and consequent lack of replication of findings, uniform treatment effects have not been routinely observed across studies. Some of the research, however, has documented positive therapeutic effects of medication on inhibitory control in children with ADHD. One study demonstrating the efficacy of pharmacological intervention in adolescents with ADHD was an early placebo controlled, cross over study by Klorman, Brumaghim, Fitzpatrick and Borgstedt (1991), who demonstrated improved inhibitory performance on a modified version of the CPT following three weeks of treatment with stimulant medication. Similarly, in their meta-analytic reviews of CPT outcome studies, Losier, McGrath and Klein (1996) and Riccio and Reynolds (2001) concluded that for the most part, ADHD children's performance benefited from the introduction of pharmacological treatment, as evidenced by significant short term improvements in attention and impulsivity as measured by this task. However, they also found that this was not a pervasive finding across all studies included in their reviews, with some clinical trials reporting the absence of positive medication effects on the CPT outcome variables.

Another study investigating the acute effects of stimulant medication on the cognitive profile of ADHD was conducted by Kempton and colleagues (1999) who examined the cognitive performance of 30 children with ADHD (15 treated with stimulant medication, with the other 15 being medication naïve) on a range of executive function measures. In their between group design, the two groups performances on the CANTAB were compared, with findings showing that the stimulant medication group exhibited enhanced executive functioning in comparison to the children receiving no medication. Similarly, Tamm (2001) examined the short term effects of stimulant medications on the task performance of 19 children diagnosed with ADHD on the Stop Signal task, with the author concluding that stimulant therapy significantly improved performance, including increasing efficacy and accuracy, on the task of behavioural inhibition.

In Tucha et al's., (2006) double blind, placebo controlled study examining the acute effects of stimulant medication on executive functioning, 58 children with ADHD completed a computerized battery assessing inhibitory control and attention. They initially completed these tasks whilst stimulant medication free, and were then re-tested seven days later following the administration of medication. Comparisons of performance on and off medication revealed that treatment with stimulant medication was associated with small to moderate improvements in performance on a range of measures sensitive to executive functions, including response inhibition, attention and cognitive flexibility. However, despite the reported improvements in functioning following stimulant medication use, normalisation of cognition, or a return to age expected levels, was not achieved, with considerable impairments remaining in response inhibition. Notably, this is not the first study to report such findings. A study by Bedard, Ickowicz and Tannock (2002) also provided supportive evidence for non-optimal inhibitory functioning following treatment with stimulant medication. Using the Stroop Color and Word Test to measure response interference, three fixed doses of methylphenidate were administered to 31 children aged six to twelve with ADHD in a five day randomised, double blind, placebo controlled, cross-over trial. Contrary to their initial hypotheses, and despite finding positive therapeutic effects on the word and colour naming conditions, they found that response interference, as measured by the Stroop Color and Word Test, was not significantly improved by methylphenidate.

Further to the conflicting findings regarding the overall acute effects of stimulant medications on the central inhibitory processes in ADHD, disagreement also exists in the literature concerning the nature of the relationship between dosage of medication and treatment response. Specifically, optimal cognitive performance has been found to be obtained at varying levels of medication dosage across separate studies. In parallel with behavioural findings, which tend to suggest that higher doses of stimulant medications are associated with greater improvements in parent and teacher ratings of behaviour, higher stimulant doses have often also been found to produce optimal inhibitory functioning. For instance, Pearson et al., (2004) investigated this dose response hypothesis in a group of 24 children with a diagnosis of ADHD and intellectual

difficulties. Utilising the CPT and Gordon Diagnostic System to measure inhibitory control, results from their study indicated that methylphenidate was associated with gains in response inhibition, with optimal performance observed at the highest medication dose (0.6mg/kg) compared to a lower dose (.15mg/kg). A clinical trial by Douglas et al., (1995) also demonstrated empirical support for this linear dose response relationship, providing evidence of greater cognitive improvements in children with ADHD with successively higher doses of stimulants (up to 0.9mg/kg). Whilst Tannock, Schachar, Carr, Chajczyk and Logan (1989) demonstrated continued beneficial effects of stimulant medications on inhibitory processes up to the high dose of 1.0mg/kg. Furthermore, Berman, Douglas and Barr (1999) and Rapport and Kelly (1991) found that not only were higher doses more effective in enhancing core inhibitory processes than lower doses, they also revealed that the highest stimulant doses tended to produce the most significant improvements on the most complicated and demanding tasks of inhibition.

Although findings from these studies support linear improvements in response inhibition at successively higher doses, Tannock, Schachar and Logan (1995) demonstrated a nonlinear dose response relationship across three doses of stimulants (0.3, 0.6 and 0.9mg/kg) and placebo on a complex version of the stop-signal task. In their acute double blind, placebo controlled trial of 28 children with ADHD they found a curvilinear dose effect on inhibition, observing a decrement in inhibitory functioning at the highest dose, with the lower doses being the most optimal relative to placebo. Moreover, Scheres et al., (2003) failed to find the presence of any dose effect in their study comparing the efficacy of three doses of methylphenidate on inhibition in 23 boys with ADHD. As hypothesized, methylphenidate was associated with improvements across most measures of inhibitory control, including inhibition of a prepotent and ongoing response. However, in contrast to initial hypotheses, the effect of stimulant medication dose was not significant. In reviewing these studies, it is clear that a consistent dose response relationship of stimulant medication on response inhibition in ADHD has not yet been achieved.

Further to the limited research and associated ambiguity of short term stimulant medication efficacy on executive functions, namely inhibition, clinical studies examining the longer term effects of psychostimulant use in ADHD are not well established, with extended treatment studies being almost absent from the literature. In fact, there exists only a handful of well controlled extended follow-up treatment studies investigating the long term effects of medication use on response inhibition, and unfortunately a range of clinical and methodological limitations inherent in these studies make it difficult to reliably determine a consistent pattern of treatment effects. For instance, Vance, Maruff and Barnett (2003) investigated the long term effects of psychostimulant medication on non-verbal executive functions in children with ADHD. The study sample comprised 40 primary school aged children who were stimulant medication naïve; 26 children who had received long term psychostimulant treatment and 26 control children, with performances on the CANTAB being compared across groups. Despite concluding that long term stimulant medication use was associated with better executive function performance, extent of the follow-up period was not known, as they did not report the duration of medication use in the "long term psychostimulant" group. In addition, the design of this between groups study was such that there was no longitudinal follow-up, whilst they also failed to incorporate baseline testing to help determine the pre-treatment cognitive profile of the groups necessary to identify the presence of any potential confounding characteristics that could influence treatment effects.

Aggarwal and Lillystone (2000) completed a twelve month prospective follow up study of 18 children using a form of the CPT, the Test of Variables of Attention (TOVA). Following an initial medication free baseline evaluation, a 12 month follow-up re-assessment was conducted, whilst the children were again free from medication. Results from this within group study revealed a significant reduction in the number of commission errors made following extended stimulant therapy, however improvements were not observed on omission errors, response time and response variability. However these findings of somewhat limited cognitive improvement after a period of prolonged stimulant treatment perhaps represents a consequence of the study sample being un-medicated at the time of the 12 month follow-up evaluation rather than ineffectiveness of

the medication itself. This hypothesis would be consistent with Zeiner's (1999) findings, who reported that the beneficial effects of medication dissipate rapidly once treatment is ceased and therefore improved performances are dependent on continued stimulant treatment.

In a double blind cross over study designed to compare the effectiveness of methylphenidate, clonidine and placebo on response inhibition, van der Meere, Gunning and Stemerdink (1999) evaluated 53 ADHD children's performances on a go-no/go task. Following a medication free baseline evaluation, children were randomly assigned to one of the three treatment conditions for seven weeks, after which time their performance was re-assessed. Not dissimilar to the findings of Aggarwal and Lillystone (2000), the results of this study revealed that there was no significant difference in task performance between the three treatment groups after seven weeks of continued treatment, indicating that there was no significant drug effect on response inhibition.

In contrast, Epstein and colleagues (2006) study examining the long term effects of stimulant medications on cognitive performance reported more positive outcomes following extended stimulant medication treatment. In their study, 316 children were assessed using the Conner's CPT at a 24 month follow-up assessment, with performances of 190 children taking stimulants on the day of testing being compared to 126 children free from medication. Results of the study demonstrated beneficial medication effects, revealing significantly better cognitive performances in the medication group, with associated moderate to large effect sizes, as compared to those children not using stimulant treatment. Furthermore, in one of the only studies to analyse the relationship between neuropsychological and behavioural outcome measures, positive correlations were reported between performances on the CPT and parent and teacher reported behavioural ratings. Despite yielding such promising results, methodological design issues inherent in this study somewhat limited the validity of their conclusions. For instance, despite the importance of medication status in this study, details of medication dose, length of time from ingestion to testing, adherence to medication regimes and duration of actual treatment were surprisingly not reported. Furthermore, although

medication free on the day of testing, almost half of the children in the "non-medicated" group had been using stimulants over the preceding months as part of their treatment program. In addition, their pattern of results were based on performances on only one, single cognitive outcome measure, which limits the ability to generalise their findings. Finally, a lack of baseline testing meant that there was no indication of whether any potential confounds were present prior to testing which may, in part, account for the observed differences in performances.

In reviewing the available literature, it is clearly apparent that there is an absence of convincing empirical evidence to support long term medication use in children with ADHD. This paucity of well controlled prospective long term clinical research and ambiguity of current results is of great concern given that children routinely receive stimulant medications in clinical contexts for lengthy periods, most of which almost definitely extend well beyond the length of follow-up observed in these empirical studies. In addition, the frequency and duration of treatment of children diagnosed with ADHD appears to be increasing as a result of the persistence of the disorder well into adolescence and adulthood. It is integral, therefore, that the current lack of evidence pertaining to the long term efficacy of stimulant medications finally receives the attention in the ADHD literature that it deserves.

3.2 ADHD Intervention: Behavioural Intervention

In comparison to the relative myriad of clinical research studies dedicated to investigating the short term effectiveness of stimulant medications on behaviour, and to a lesser extent cognition, in children with ADHD, non-pharmacological therapies, such as cognitive and behaviourally focussed interventions, generally receive far less attention in the ADHD literature. As a result of this absence of comparative research, conclusions regarding the efficacy of non-pharmacological therapies in ameliorating behavioural and cognitive impairments in ADHD children remain equivocal. For instance, in their comprehensive meta-analytic review of available extended treatment outcome research,

Schachar and colleagues (2002) reported the need for further replication of the efficacy of behavioural and cognitive interventions, finding limited outcomes on behavioural interventions with only two studies providing evidence for the benefits of cognitive behaviour therapy (CBT) and EEG biofeedback. Schachar et al's., (2002) systematic review of the research closely reflects the broader ADHD literature in which the vast majority of intervention studies exclusively focus on the effects of stimulant medication, with many tending to ignore the contribution of non-pharmacological interventions. In saying that, many of those that have used a non-medical management approach in their treatment studies have often failed to provide convincing evidence pertaining to their effectiveness when used in isolation. Although interventions such as CBT have inherent appeal in their use of self-monitoring and self reinforcement techniques to change thought patterns, there exists little empirical support for CBT as a lone treatment modality in the treatment of children with ADHD (Frazier & Merrell, 1997). For instance, in his meta-analysis of cognitive treatment studies, Abikoff (1991) found little documented efficacy in the clinical utility of CBT in children with ADHD. In addition, many other studies have also failed to confirm the effectiveness of cognitive therapies for this disorder (Abikoff & Gittelman, 1985; Brown, Borden, Wynne, Schlesher & Clingerman, 1986; Hinshaw et al., 2000; Swanson et al.,1993).

Of the non-pharmacological interventions employed in the treatment of children with ADHD, behavioural therapy is probably the most well established and widely supported alternative therapy to stimulant medication in current clinical practice (Pelham et al., 2005). Behavioural interventions represent a broad set of specific strategies that share a common goal of modifying a child's environment in order to change behaviour (Thorpe & Olson, 1997). Specifically, behavioural therapy uses principles of positive and negative reinforcement to increase adaptive and appropriate behaviour with positive rewards, and to reduce or eliminate problematic or inappropriate behaviours by removing reinforcement (Damico and Armstrong, 1996; Martin and Pear, 1999). In the context of ADHD, behaviour modification programs are typically carried out by professionals in comprehensive and intensive treatment programs (for example, summer camps) or implemented by parents and teachers who receive training in behaviour therapy

principles, specifically in the application of different techniques and strategies to reward positive behaviour and apply consequences for undesirable behaviours in order to manage and shape the child's behaviour.

A number of empirical studies over the past few decades have attested to the efficacy of behaviour modification techniques, yielding considerable benefits across multiple domains of outcome both in the home and school environments and on parent and teacher rating scales. Recent studies implementing intensive behaviour modification treatment programs with ADHD children have reported moderate to large effect size improvements for behavioural interventions, with these treatment effects being similar in magnitude, across some outcome domains, to low to moderate doses of stimulant medications (Northup et al., 1999; Pelham, 1999; Pelham & Waschbush, 1999; Pelham et al., 2005). For example, in their investigation of the effectiveness of behaviour modification in a summer treatment program (STP), Pelham and colleagues (2005) revealed a pattern of significant improvements across multiple domains of behavioural functioning in a group of 27 children with ADHD. Children's involvement in an intensive eight week STP (nine hours a day, five days a week) was found to produce behavioural improvements reported to be comparable to a moderate dose of stimulant medication, with improvements attributed to behavioural intervention being equivalent to the benefits observed with pharmacological intervention, depending on the outcome measure (Pelham et al., 2005).

Further promising findings have also been documented in treatment outcome studies employing less intensive behavioural modification programs. In a study evaluating the efficacy of teacher implemented behaviour therapy strategies and techniques in the classrooms of 29 primary school children with ADHD, Miranda, Presentacion and Soriano (2002) found that there were substantial reductions in problematic ADHD behaviours following the implementation of a behaviour modification program. Results indicated that their teacher training program (consisting of eight three hour education sessions), which focused on behaviour therapy principles, was effective in improving a range of parent and teacher reported behavioural difficulties. However despite these

documented improvements in behaviour, parents reported that very few of the children had actually returned to age appropriate levels of functioning (only 17%) following behaviour therapy. Furthermore, little benefit was achieved on neuropsychological test performance. Similarly, Michel, Kerns and Mateer (2005) compared the effects of three reinforcement conditions (no reinforcement, immediate reinforcement and delayed reinforcement) on response inhibition in 20 children with ADHD and matched controls. Results of the study revealed significantly improved inhibitory performances in the ADHD children when provided with immediate and delayed reinforcement whilst performing a stop signal task, however consistent with Miranda et al's., (2002) findings, their ability to appropriately inhibit their responding was not normalised, with their performance remaining below that of the control group.

Behavioural interventions have several inherent advantages which make them appealing treatments for children with ADHD. For instance, unlike stimulant medication, behavioural therapy is not associated with any known physical side effects and doesn't have the potential for any long term harm (Frazier & Merrell, 1997). Behavioural treatments may also yield a more complete and broader effect upon ADHD symptoms, not having the temporal effects that are often associated with stimulant medication. For example, behavioural techniques and strategies are effectively employed in the early morning or late evening when the effects of medication dissipate. Finally, parents tend to prefer behavioural methods over the prescription of stimulant medications, often reporting higher levels of satisfaction for behavioural treatment programs compared to medication management, which may be associated with increased compliance rates (MTA Cooperative Group, 1999).

However, despite evidence for some favourable outcomes and the advantages of behavioural treatment programs, a considerable amount of debate still remains regarding its effectiveness as a uni-modal treatment, with a convincing body of literature existing to challenge the effectiveness of behavioural interventions in the treatment of children with ADHD (Jadad, Boyle, Cunningham, Kim & Schachar, 1999; Klein & Abikoff, 1997; Miller et al., 1998; Wigal et al., 1999). For instance, despite the research by Miranda et

al., (2002) and Pelham et al., (2005) it is a widely supported notion that behavioural treatments, when used in isolation, rarely produce improvements on important domains of behavioural and cognitive functioning to the extent observed with stimulant medications. In fact, in their study, Pelham et al., (1993) revealed that stimulant medications had double the effect size of a behaviour modification program. Furthermore, behaviour therapy often fails to produce a sufficient level of improvement to completely normalise functioning, whilst an absence of any significant treatment effects has also been repeatedly documented with behaviour therapy programs. In addition there is a lack of empirical evidence confirming the long term effects of behavioural interventions, with limited support for maintenance effects once therapy has faded. This has resulted in some authors concluding that uni-modal behaviour therapy has not been substantiated as an effective intervention for the treatment of ADHD symptoms (Barkley, 2000; Ercan, Varan & Deniz, 2005; Frazier & Merrell, 1997; Jensen, 1999; Klassen, Miller, Raina, Lee & Olsen, 1999; Pelham et al., 2000).

The lack of successful outcomes of behaviour therapy alone can be in part attributed to the nature and design of the behaviour modification programs. Behaviour therapy treatment programs implemented in empirical research typically tend to be highly complex, time-consuming, intensive and expensive with little ecological validity, all of which often present as a barrier to parental compliance and adherence. Additionally, in contrast to pharmacological intervention, behavioural programs usually require a great degree of time and energy to be expended by parents and caregivers, often with little improvement or benefit to the child, which can prove a challenge to maintain over extended periods. Finally, behaviour therapy programs are customarily conducted in the context of group therapy and as such strategies and techniques are not always customised to the individual child and family's needs (Barkley, 2000; Boyle, 1999; Frazier & Merrell, 1997).

3.3 ADHD Intervention: Multimodal Intervention

As a result of the limitations of behavioural therapy when used alone, a number of studies have emerged advocating for the now popular clinical approach of combining therapies. For several reasons, a multimodal treatment approach, consisting of a combination of pharmacological and behavioural therapy, has intrinsic appeal as an alternative to using either treatment approach alone. For instance, multiple treatments assist in maximising therapeutic impact by providing a more comprehensive and multi-faceted approach to the profusion of behavioural and cognitive problems characteristic of children with ADHD. Furthermore, limitations of using pharmacological therapy in isolation also support the implementation of a multimodal treatment approach over either treatment alone. For example, despite the well documented beneficial therapeutic effects of stimulant medications, it is estimated that at least 30% of children with ADHD are non-responsive to a purely pharmacological regime, with many more failing to reach normal levels of functioning in terms of their ADHD symptoms (Pelham et al., 2000; Whalen & Henker, 1991). In addition, there is no evidence to support any generalisation or carry-over effects of psychostimulants and the beneficial effects of stimulant medications are proposed to be short lived, dissipating once the treatment is discontinued (Zeiner, 1999). Therefore the positive therapeutic impact of medication is reserved only for a limited number of hours after ingestion. These limited temporal effects of a purely medication management regime may however be enhanced when combined with behaviour therapy, which can continue to be effective even after the effects of stimulant medication have worn off.

Although there is an assumption in the literature that multimodal treatment approaches optimise therapeutic outcome, the value of a combined approach, in terms of being superior to either treatment alone, remains contradictory (Purdie et al., 2002). Unfortunately, despite an emerging outcome literature on combined treatment approaches, multimodal therapy, at present, still remains relatively understudied, especially in comparison to pharmacological intervention. However, similar to the results of behaviour therapy alone, many studies have failed to demonstrate a superiority of

multimodal treatment effects over pharmacological interventions when used alone. In a recent study examining the effects of combined medication management and parent training on the behavioural symptoms of 83 children with ADHD, Ercan et al., (2005) demonstrated their multimodal treatment approach to be successful in improving core ADHD symptoms and associated behaviours, as rated by parents and teachers, over a five month treatment period. However, further analyses revealed that the most significant gains were achieved prior to the introduction of the parent training component, suggesting that the medication alone was largely responsible for the reduction in ADHD symptoms. A similar pattern of findings have also been replicated in other studies, demonstrating significant improvements on parent and teacher rating scales with combined therapy, with further analyses revealing no additive effects of behaviour therapy with stimulant medications over medication management alone (Abikoff & Gittleman, 1985; Brown et al., 1986; Greenhill, Halperin & Abikoff, 1999; Ialongo et al., 1993; Klassen et al., 1999; Klein & Abikoff, 1997).

One of the most influential multimodal therapy studies in the ADHD treatment literature has been the comprehensive, large scale study completed by the Multimodal Treatment Study of Children with ADHD Co-operative Group (1999). Un-paralleled in the field of ADHD to date, the MTA Co-operative Group evaluated the long term efficacy of four treatment approaches in a prospective follow-up investigation of a large clinically referred sample of children with ADHD. Specifically, in their extensive randomised clinical trial, 579 children aged seven to nine years with ADHD were followed-up over an extended 14 month treatment period of either medical management, intensive behavioural intervention, a combination of both these treatments or standard community care. Outcomes were assessed across multiple domains of behavioural functioning, including core ADHD symptoms, social skills, oppositional/aggressive behaviours, internalising symptoms, and parent-child relationships, prior to and following the completion of the treatment programs. Study findings revealed significant reductions across all four treatment groups on the multiple behavioural efficacy outcomes as rated by parents and teachers over the 14 months, however the best results were achieved with the medical management and combined treatment programs, yielding greater

improvements in core ADHD symptoms than the behavioural treatment alone and standard community care. Notably, few differences were noted between the treatment groups on the other associated domains of functioning, such as social skills and oppositional/aggressive behaviours. Despite finding a superiority of the medication alone and combined treatment approaches over the two other interventions in reducing core ADHD symptoms, and contrary to initial hypotheses, the multimodal therapy failed to produce significantly greater benefits than the medical management approach. This failure to achieve additive effects with a multimodal approach parallels findings from the more recent research by Ercan and colleagues (2005).

Much has been written about the pivotal and important research undertaken by the MTA Cooperative Group, with due attention given to the valuable contribution it has made to the ADHD treatment literature. However, whilst there is little doubt that this is one of the most influential and comprehensive clinical studies of interventions for ADHD in light of the extensive time, effort and resources employed in the organisation and execution of the research, the study's methodological design has also been the focus of several critiques and reviews. One of the pervasive criticisms of the MTA study is the lack of ecological validity afforded by the extremely intense, complex and expensive behavioural and medication interventions. Although certainly not alone in the field of intervention research, the comprehensive nature of the intervention has consistently been criticized to lack real world applicability with both Barkley (2000) and Boyle (1999) questioning the relevance of the components of the interventions to clinical practice. And when one considers the demanding and costly nature of the therapies (28 day double titration trials for stimulant medications; 27 group and eight individual family sessions for parent training; 10-16 school based intervention sessions with the teacher and an eight week all day summer treatment program for the children) it is indeed difficult to imagine that this impressive level of intensity could be widely available or readily adopted by clinicians, parents, teachers and children in normal clinical practice (Pelham, 1999). Furthermore, Greene and Ablon (2001) argue that in contrast to the individually titrated medication regime, the intensive behavioural intervention in the MTA study was not appropriately individualised to meet the specific needs of families. Like Barkley (2000) and Boyle (1999), they query whether these standardised interventions could be

successfully incorporated into clinical contexts, advocating for a more adaptive and individually tailored approach. In addition to the intensive behaviour therapy implemented in the MTA study, a similar "intensive" level of medical management was also employed, with high doses of stimulant medication (average of approximately 38mg daily) being used in the medication groups. Therefore failure to find a superiority of combined treatment in the MTA study may be possibly explained in the context of findings from cross over trials that show that high doses of medication rarely add incremental benefits to behaviour therapy, with greatest benefits being achieved with low doses of medication (Carlson, Pelham, Millich & Dixon, 1992; Chacko et al., 2005; Pelham, 1999; Pelham et al., 2005).

Another critical issue of the study design is the use of 19 dependent measures, all of which assess outcome in terms of behavioural characteristics. In fact, consistent with the vast majority of intervention studies, the MTA study provides no advancement of knowledge regarding the various treatment approaches impact upon the proposed core cognitive deficits, such as inhibition, as outlined in Barkley's theoretical model of ADHD. Finally, whilst a pervasive fault of many outcome studies, interpretation of findings in the MTA study were analysed at the group level, with no attention paid to the more informative and clinically relevant results of the percentage of individuals showing change in functioning. Barkley (2000) contends that group results are not particularly important clinically, and can often be misleading, arguing that this level of analysis may mask any children who show no change in functioning, or perhaps even worsen as a result of therapy.

Despite many studies failing to demonstrate a statistically significant advantage for a multimodal treatment approach over uni-modal therapies, more recent research has attempted to address some of the limitations of the MTA study, with the results seeing the emergence of more promising results. In a secondary analysis of the MTA study, Conners et al., (2001) adopted an overall single composite dependent measure in place of the original multiple behavioural outcome scores in an attempt to further explore the non-superiority of the combined treatment over stimulant medications. With this increased statistical power to detect the presence of significant differences, these post hoc analyses

revealed that the combined treatment program was significantly better than all other treatment approaches, including medical management, with the magnitude of effect ranging from small (.28) to moderately large (.70). Based on these new interpretations of the MTA findings, the authors concluded that multimodal therapy does indeed have a statistically and clinically significant superiority over all other therapies. Additionally, in further analyses of the original MTA study, Swanson et al., (2001) also found that the combined treatment was more successful than medical management, behavioural therapy and community care.

Dopfner and colleagues (2004) adopted a more clinically driven approach in their innovative research design intended to more closely reflect treatment strategies typically implemented in clinical practice. In contrast to the fixed, standardised treatment rationale adopted in most research designs, including the MTA study, a more ecologically valid approach was utilised, in which their treatment programs mimicked decision making in clinical practice. They used adaptive, individualised uni-modal and multimodal treatments of varying intensity in 75 children with ADHD, which were either continued, augmented or stopped depending on the treatment effects. After two months of treatment, teacher and parent ratings of ADHD behaviours showed significant improvements with the combined therapy approach, however there were no reports of superiority of either treatment. However despite these observed benefits, children were not completely normalised, with parents continuing to report the presence of ADHD behaviours.

In a more recent study by van der Ord, Pins, Oosterlaan and Emmelkamp (2007), the merits of a combined multimodal behaviour therapy/stimulant medication program were evaluated and compared to a standard psychostimulant therapy approach in the treatment of ADHD behaviours. Analogous to the Dopfner et al., (2004) article, the design of this study was uniquely clinically driven, devised to be suitable for, and readily adaptable to normal clinical practice. Specifically, they implemented an integrated behavioural program consisting of ten weeks of group therapy involving parents, ten weeks of group CBT for children and a one two-hour workshop for teachers. They hypothesized that the

additive effects of this brief, but comprehensive, multimodal therapy would be superior to the medication alone treatment in improving parent and teacher rated ADHD behaviours. However, despite finding significant improvements overall on most of the behavioural outcome measures, there was no evidence to support their initial expectations that the multimodal behaviour therapy would enhance the effects of methylphenidate. Although the practical design of this study's multimodal therapy program is one which appears to be able to be readily adopted into standard clinical practice, the authors themselves conclude that yet more of an individualised treatment approach tailored to the specific needs of each child and family (as opposed to most group therapy programs), needs to be advocated for in both research and clinical practice in order to achieve optimum outcomes.

There are a small number of other studies in the current literature that have provided supportive evidence for the efficacy of combined treatment programs over uni-modal therapies. These studies have revealed multimodal approaches to be beneficial in improving functioning across several domains of outcome, including increasing on task behaviour, rule following, social skills and peer relationships (Chacko et al., 2005; Jensen, 1999; Mathes & Bender, 1997; Pelham et al., 2000; Pelham et al., 2005) However despite multimodal therapy being such a popular notion, conclusive empirical evidence is currently lacking to support the idea that combined treatments are superior in improving behaviour to all other treatment options, whilst there is an absence of studies to date in the literature investigating the amelioration of cognitive deficits with combined treatment. This is in part due to the limitations inherent in current studies and also due to the fact that this is a developing treatment area that is presently understudied. These constraints only highlight the need for further research into this exciting new area.

Chapter 4: Study Rationale

In reviewing the literature, it is evident that a consistent behavioural profile of ADHD has been well established, which undoubtedly reflects the vast amount of research that has been conducted dedicated to investigating the core behavioural characteristics of the disorder. However recently there has been a shift in the focus of ADHD research, with the most prominent theories now emphasizing deficient cognitive processes, rather than underlying behavioural problems, as the central impairment in the manifestation of ADHD characteristics. Although an extensive research literature generally supports an impaired cognitive profile characterised by deficits in executive functioning, the pervasiveness of this dysfunction is not conclusive, with a number of studies failing to demonstrate impaired performances on a range of measures sensitive to executive functions. However, the domain of executive functioning is a broad one, encompassing numerous different cognitive processes, hence when considered in this context, inconsistencies in findings across the range of executive functions is hardly surprising. In his pivotal theory of ADHD, Barkley (1997a) narrowed the focus of ADHD conceptualisation by proposing a specific theory encompassing a key aspect of executive functioning as the core deficit in ADHD, namely response inhibition. Despite receiving a great deal of attention for his work, with much empirical support for his theory, the existing evidence base would suggest that there is not conclusive support for his hypotheses, with numerous studies exposing inconclusive or contradictory evidence. These inconsistencies, however, may be attributed, in part, to the methodological flaws and publication biases evident in the literature which unfortunately may limit the reliability and validity of findings. Inconsistent selection processes and criteria for categorising ADHD, inclusion of co-morbid disorders in ADHD samples, evaluation of cognitive functioning while medicated with psychostimulants and utilisation of a restricted range of outcome measures are characteristic of some of the design limitations, whilst a frequent publication weakness is the pervasive reporting of group findings only, with very little attention given to the more meaningful and clinically relevant individual level of analysis. The current study, therefore, plans to contribute to the existing literature by assisting in the conceptualisation of the executive functioning profile of

ADHD, with a specific focus on evaluating the nature of response inhibition in a relatively homogenous sample (ie, no co-morbid disorders) of medication free children who have received a diagnosis of ADHD by their paediatrician. Furthermore, clinically meaningful individual level of analysis will be provided rather than just group results, with the proportion of children who are impaired compared to tests published normative data being reported.

As a result of the well documented behavioural profile of ADHD, interventions aimed at reducing the symptoms of the disorder have typically focussed on remediating the maladaptive behaviours manifested as part of the disorder. Consequently, there exists an extensive literature attesting to the short term efficacy of stimulant medications on improving behaviour, with many studies revealing substantial improvements with associated moderate to large effect sizes on a range of subjective behavioural questionnaires. However although these are undeniably important findings, the obvious limitation of these intervention studies is the restriction of findings to primarily behavioural symptoms, preventing these findings from being generalised to the core underlying cognitive deficits proposed to be implicated in ADHD. Since a focus on behavioural outcomes has dominated the intervention literature, comparatively little research has been undertaken to investigate the important effects of stimulant medications on cognitive inhibition. As such the acute effects of psychostimulants on inhibition are not very well established, making it quite difficult to conclude with any degree of confidence or certainty about the efficacy of intervention on this key aspect of cognition. Given that current empirical evidence supports the presence of underlying cognitive deficits in ADHD, the focus of this study will be to investigate the successfulness of psychostimulants in improving the core cognitive processes of ADHD, whilst also examining the effects on overt behavioural symptoms. The study will achieve this aim through the implementation of standardised, objective cognitive measures of inhibition, and outcome measures evaluating the behavioural correlates of executive functioning, with a specific focus on behavioural inhibition. This present study will be one of the first to examine the impact of stimulant medications on both cognitive and behavioural measures of inhibition as defined in Barkley's model of ADHD (1997a). Furthermore, it

will be among the first in the literature to explore the relationship between objective cognitive measures of inhibition and parent rated behavioural questionnaires.

Whilst extending knowledge about ADHD conceptualisation and short term psychostimulant treatment outcomes, the current study is also making a valuable and timely contribution to the literature regarding the long term efficacy of psychostimulant intervention on cognitive and behavioural ADHD symptoms. At present, there is a concerning lack of extended psychostimulant treatment outcome studies, with only a mere handful of long term clinical studies currently available in the clinical research literature. With this paucity of research comes a lack of uniform treatment findings for long term stimulant medication use. Specifically, some studies reveal only temporary medication effects without maintenance of benefits over time, a few report improvements without complete normalisation of behaviour or cognition, whilst others show no beneficial drugs effects at all over weeks to months of continued stimulant medication treatment. This leaves very few that demonstrate a consistent pattern of positive long term effects of psychostimulant treatment in children with ADHD. Further to the ambiguous treatment findings, this limited body of empirical research is unfortunately also characterised by a range of clinical and methodological limitations which make it difficult to clearly determine treatment effects. These include no documentation of important medication details (for example, no reports of length, dose and duration of medication), using a between group design with assessments at one time point rather than incorporating a more informative and valuable prospective longitudinal design across multiple time points, absence of baseline testing to determine pre-medication functioning, follow-up assessment whilst not medicated, the use of only one outcome measure, and using "stimulant naïve" groups who have in fact been previously medicated.

These inconsistent and ambiguous findings of limited extended clinical outcome studies highlights the absence of convincing supportive research for the extended use of stimulant medications in the treatment of children with ADHD. Given that extended stimulant medication use is often a central component to treatment programs for ADHD, with many children typically continuing psychostimulant treatment for several years into

adolescence and even into adulthood, the discrepancy between the lack of empirical evidence to support long term medication use and the considerable number of children prescribed stimulant medications for ADHD is both astounding and concerning. Moreover, longitudinal studies investigating the course of ADHD throughout development have consistently revealed that ADHD symptoms continue to have an impact on social and occupational functioning leading to negative outcomes, whilst cross sectional and retrospective studies have revealed children with ADHD are at increased risk for developing psychiatric disorders and have a serious potential for leading to substance abuse and criminality.

In light of there being relatively few well designed and methodological sound longitudinal follow-up studies investigating the longer term efficacy of treatments in ADHD children, this current study will make an important contribution to the literature by prospectively following up a relatively homogenous sample of children with ADHD across a series of assessments from baseline to six months follow-up on a range of behavioural and cognitive measures. In doing this, we may be able to help identify those interventions that are effectively able to reduce or even eliminate the cognitive and behavioural manifestations of ADHD in children, in turn providing greater success for the improvement of their prognosis into adolescence and adulthood.

Another significant aspect of the current study's design is the comparison of the effectiveness of stimulant medications with the now common multimodal approach of combining psychostimulants with behavioural therapy. Although there is an emerging body of literature dedicated to investigating multimodal treatment approaches, at the present time this area still remains understudied with an associated pattern of inconclusive findings and study limitations. To date, the pervasive weakness of most of the research evaluating the behavioural intervention component is the nature of the behaviour therapy implemented. Typically complex, demanding and intensive, the behavioural interventions are characteristically neither ecologically valid nor relevant in standard clinical care. Specifically, they generally fail to parallel the needs of normal clinical practice, which necessitates an individualised, flexible approach tailored to the

needs of the child and family, one which is carried out and monitored on an ongoing basis to suit changing requirements. As it stands, the current literature has not provided suitable empirical evidence for the impact of a more ecologically valid, lower intensity, individualised behavioural intervention program, one which is conducted by parents in a naturalistic environment over an extended period of time. This study hopes to make a substantial contribution to this area of research by conducting a study adopting a family centred cognitive behavioural therapy approach to ADHD treatment. This involves brief individually tailored education sessions addressing the key principles of behaviour modification relevant to the family dynamics, with key members of the child's family implementing the behavioural therapy techniques in the home environment. This process is neither time-consuming nor too labour intensive for the family, is designed to have a direct impact on the maladaptive ADHD behaviours of the child, and is practical and achievable for parents to consistently and successfully implement. Inherent in this process is a real world applicability that has been noticeably absent from much of the previous research.

Chapter 5: Aims and Hypotheses

This current study aims:

1. To investigate the cognitive test performances of a clinical sample of children with ADHD relative to the published norms that accompany the tests. More specifically, this study will examine whether children with ADHD exhibit impaired executive performance on cognitive and behavioural measures relative to age related published norms, as proposed by Barkley's model of ADHD.

2. To examine whether two forms of intervention, medication alone and combined medication and family centred cognitive behaviour therapy, have any effect in reducing the core cognitive and behavioural executive function symptoms associated with ADHD and whether there is a superiority of either treatment over the other.

3. To examine the relationship between neuropsychological performance on cognitive measures of executive functioning and of parents rating of behaviour relating to executive functioning.

It was hypothesized that:

1. Children with ADHD will perform significantly worse on cognitive measures relative to age-related published norms for each test. Specifically, it is expected that children with ADHD will exhibit cognitive and behavioural impairments in inhibition, interference control, high level attention and working memory relative to the published norms.

2. It was predicted that cognition and behaviour relating to executive functioning, such as inhibition, would show improvements after six months of either stimulant medication therapy or combined family centered cognitive behaviour therapy and stimulant medication therapy. Specifically, it was hypothesized that performances on standardized cognitive measures of inhibition would be significantly improved from pre-treatment baseline to six month follow-up after either stimulant medication treatment alone or stimulant medication in combination with behavioural therapy. In addition, the same pattern was predicted on parent rated measures of behaviour relating to inhibition, with a reduction in problem behaviours expected following six months of continued pharmacological treatment alone or in combination with behavioural therapy.

3. There will be an association between cognitive and behavioural measures of executive functioning. More specifically, it is expected that there will be a strong correlation between the performance based executive function measures and parent reported behaviour on the Behavior Rating Inventory of Executive Functioning (BRIEF).

Chapter 6: Methodology

6.1 Participants

Participants in the study comprised a total sample of 27 children between the ages of seven and twelve (mean age 8.8 years) all of whom had received a recent diagnosis of ADHD by their consulting paediatrician. Culturally, the majority of participants were from an Anglo-Saxon background (90%) and most of the sample were male (85%). The entire sample was recruited through four paediatricians in private outpatient consulting rooms located in the Northern and Western suburbs of Melbourne.

All participants met the diagnostic criteria for ADHD (either predominantly hyperactive/impulsive or ADHD combined type) based on thorough diagnostic evaluations performed by their consulting paediatrician, which consisted of clinical interviews with the parents and the administration of standardised questionnaires (the Child Behaviour Checklist and the Rowe Behaviour Rating Inventory). In accordance with current DSM-IV-TR criteria, a diagnosis of ADHD was made based on parental report of at least six inattention and/or six hyperactive/impulsive symptoms deemed present for at least six months in at least two settings, and with an onset dating back prior to age seven. These symptoms were also considered to be maladaptive and inconsistent with the child's developmental level (APA, 2000).

As a result of their current diagnosis of ADHD, all children had been prescribed stimulant medication by their paediatrician. Most of the children had recently commenced their medication regime (prior to participation in the study) however there were some children who were beginning their medication following completion of their initial baseline assessment. Children were prescribed either methylphenidate or dexamphetamine and doses were deemed by the paediatrician to be at an appropriate therapeutic level for each child. The sample of children with ADHD were receiving no other formal therapy at the time of the study's initial evaluation.

6.1.1. Exclusion Criteria

All children were screened via a phone call prior to inclusion in the study. The researcher, who was not involved in the clinical care of participants, conducted the phone interview and subsequently determined eligibility. Parents were required to complete a brief semi-structured interview over the phone regarding their child's background history, which requested demographic information and a medical and developmental history in order to assist in confirming eligibility for the study. Children were only deemed eligible to participate once the following exclusion criterion was avoided. They were precluded from participating if there was any evidence of intellectual difficulties, (measured by a full scale intelligence quotient of 80 or lower on the Wechsler Intelligence Scale for Children – Fourth Edition [WISC-IV]) or in the presence of any known or undiagnosed learning disability, as these difficulties have been associated with executive impairments independent of a diagnosis of ADHD. . Identification of undiagnosed learning difficulties in the sample was based upon comparisons made between each child's WISC-IV full scale intelligence quotient (FSIQ) and their performance on a measure of academic achievement (reading and mathematics on the Wide Range Achievement Test – Third Edition [WRAT-3]). Children with a significant discrepancy between these indices, characterised by academic functioning of at least one standard deviation (or $\geq$ 15 points) below their overall general intellectual functioning, were identified as having a possible learning disorder and were excluded from participation. The intelligence and academic measures were obtained at an initial baseline assessment prior to the introduction of treatment. Based on paediatrician and/or parental report, children with ADHD were also excluded from the sample if they presented with a history of acquired or developmental insult to the central nervous system (including traumatic brain injury or seizure disorder), an acute or chronic medical illness or co-morbid Axis I psychological or emotional disturbance, including diagnosed conduct disorder, oppositional defiant disorder, depression, anxiety, Asperger's syndrome or Tourette's syndrome.

Thirty six children were referred by the paediatricians for inclusion in the study. Of these, four children with ADHD were excluded from participating due to the presence of intellectual disabilities, three parents declined their child's participation due to limited time/availability, and two parents refused to cease their child's medication for the preliminary baseline assessment. Twenty seven children in total completed the initial baseline evaluation.

Once participation in the study was confirmed, the sample was divided into one of two treatment groups. Thirteen of the participants were selected for inclusion into the "medication alone group", in which children received pharmacological intervention only. The remaining 14 participants were assigned to the "combined group", in which both a combination of pharmacological intervention and family centred cognitive behaviour therapy was implemented. Data was collected concurrently for all children.

6.2 Measures

The dependent measures used fell into two broad categories: (a) standardised cognitive tests assessing general intelligence, academic achievement and a number of domains of executive functioning, with a particular focus on inhibition and working memory; and (b) parent rating measures assessing the behavioural correlates of executive functions.

6.2.1 Executive Function Tasks

6.2.1.1 The Stroop Color and Word Test – Children's Version

The Stroop Color and Word Test – Children's Version (Golden, Freshwater & Golden, 2003), was selected as one of the primary measures of cognitive inhibition. Specifically, this task measures resistance to interference and the ability to suppress a prepotent response in favour of a novel and unusual one. The Stroop Color and Word

Test (SCWT) is a highly reliable and valid neurocognitive test of inhibition, producing very good reliability co-efficients of .90, .83 and .91, for the three primary task components, when examining test-retest reliability using a one month interval (Spreen & Strauss, 1998).

The SCWT consists of three cards with 100 stimuli ordered in five columns of twenty items. In the first section (the word reading condition) the children were presented with a word page with randomised colour names (i.e., red, green and blue) printed in black ink. In accordance with the standard procedures described in the test manual (Golden et al., 2003), children were instructed to read the words down the column as quickly as they could, continuing in this manner until the end of the 45 second time limit, with the number of completed items being recorded. On the second page (the color naming condition), coloured Xs, printed in either red, green or blue, were presented to the children in random order. Children were instructed to name, as quickly as they could, the colour of the ink in which the Xs were printed within the 45 second time limit. In the final condition of the SCWT, children were presented with coloured words printed in colours incongruent with the written word. For example, the word blue was printed in red ink. In this color/naming condition, children were required to name the colour ink in which words were printed, whilst inhibiting the dominant tendency to read the actual word. In other words, they were instructed to name the colour of the ink, but *not* read the actual word.

Performance was compared across the three task components: word reading, color naming and colour-word naming, with the number of items completed in each condition being recorded to yield three basic scores. Errors were not counted across the three conditions, however an incorrect response required repetition of that item, resulting in a lower score overall. A secondary score measuring interference was also calculated (color naming subtracted from color word naming). According to Golden et al., (2003) this results in a pure measure of interference, independent of the child's reading or colour naming speed. Both raw scores and T scores were calculated and included in the analyses. Low scores (T score of less than 30) on the first three conditions of the

SCWT indicate an impaired level of functioning, however the reverse was true for the interference index, with a high score (T score of more than 70) reflecting a higher level of interference, thus indicating impairment.

6.2.1.2 Controlled Oral Word Associated Test (COWAT)

The COWAT (Spreen and Strauss, 1998) evaluates the ability to spontaneously produce words under restricted search conditions (phonemic prompts) within a limited time period. This verbal fluency task is sensitive to impaired frontal lobe functioning and has been documented to be a valid executive function measure of problem solving ability, the capacity to resist distraction and perseveration (Crocket, 1996; Spreen & Strauss, 1998).

In the COWAT, the child was required to generate as many words as possible beginning with specific letters of the alphabet (F, A and S) over three separate trials. Specifically, using the phonemic prompts, children were instructed to rapidly and spontaneously produce words within the allocated 60 second time limit for each letter. Children were told to adhere to two rules during the task: they must not produce words beginning with capital letters (for example, names like “Florence” or “Fiona”) and they must generate different words each time, with any repeated words being disallowed. Therefore inadmissible words produced during the three trials, including repetitions, proper nouns, and intrusions (of other letters), were marked as incorrect. The number of words produced for each letter, and in total for the three letters, was recorded as the measure of verbal fluency, with a higher number of words produced representing better verbal fluency.

6.2.1.3 Controlled Animal Fluency Test (CAFT)

The CAFT, originally developed by Monti (1984) is a measure of verbal fluency evaluating children’s ability to access semantic lexicon under various regulation conditions. This task was chosen for its utility with children, due to its un-reliance on

knowledge of grapheme-phoneme rules. Furthermore, it requires children to access a category with which they are familiar (animals).

The CAFT consists of three conditions, all of which require children to name as many animals as they can (under different conditions) in a 60 second time period (see Appendix A). The first condition (animals automatic) involves recalling animals without any restriction, whereby children were instructed to name as many different animals as rapidly as they could. In the second condition (animals by size), children were required to name animals in order of size, starting with the smallest animal and slowly progressing to the largest animal. Animals recalled out of order were disallowed, however they were counted if the recalled animal was larger by virtue of height or weight. For instance, zebra followed by horse or horse followed by zebra was counted as an acceptable ordering of animals. The final condition commenced with the child reciting the alphabet. This was carried out to ensure competency with the alphabet in order for the child to proceed with the task without cognitive difficulty. This condition (animals by alphabet) required children to recall as many animals as they could in alphabetical order, beginning with an animal starting with the letter A, then B and so on until the completion of the 60 time limit. If the child omitted a letter of the alphabet no point was awarded for that letter and if an incorrect letter was used, for example “pheasant” for the letter F, no credit was given.

The number of animals produced under each condition was recorded, with more animals generated indicative of better verbal fluency. An additional measure, the relative difficulty size score, was also calculated. This index was used to compare the difference in raw scores between the automatic and animals by size conditions in order to assess the difference in difficulty level experienced during these two conditions. A higher relative difficulty score represented a significant discrepancy between the performances on these two conditions, thereby indicating an overall greater level of difficulty on the more complex (animals by size) component of the task (Tucker, Ewing & Oguzkaya, 2007; Tucker, Ewing & Ross, 1996).

6.2.1.4 Continuous Processing Task (CPT)

To assess the children's ability to inhibit a prepotent response, the present study included the CPT as another primary measure of inhibition. The CPT is purported to test sustained attention and impulsivity and was first developed by Rosvold and colleagues in the 1950's (Rosvold, Mirsky, Sarason, Bransome & Beck, 1956).

In the CPT, a series of 360 visual stimuli, were presented sequentially to children on the screen of a laptop computer. Letters of the alphabet appeared at the rate of one per second with target stimuli intermixed amongst a series of distracters. Children were instructed to respond, by only pressing the 'yes' button (on a button box) when a specific target letter combination appeared: the letter X preceded by the letter A (see Appendix B). In addition, children had to refrain from responding to non-target stimuli (any other combination of letters), by pressing the 'no' button. This version of the CPT contained 70 target letter combinations and 290 distracter letters. Children first completed a brief practice trial (6 target letters and 25 distracter letters), where correct and incorrect responses were accompanied by different audible tones, thereby providing feedback for their responses. Children then proceeded to the actual task which was void of any tones to indicate right or wrong responses. The duration of the CPT was 12 minutes and upon completion participants received feedback about their performance with a total score of the number of correct responses.

Performance was measured by the number of correct and erroneous responses produced by the child which provided two primary measures: omission errors and commission errors. Omission errors reflected missed targets, and thus were indicative of deficits in sustained attention or vigilance with more errors indicative of poorer functioning. Commission errors reflected rapid responses to non-target stimuli and a higher rate of errors was indicative of impulsivity. These two measures of errors were employed in the statistical analyses. Both measures have been documented to have adequate test-retest reliability and stability over time (Spreen & Strauss, 1998).

6.2.1.5 Trail Making Test – Part B (TMT)

The Trail Making Test (Parts A and B) has been reported to be a valid and reliable measure assessing rapid complex visual sequencing and attention (Reitan & Wolfson, 1995). Part B, in particular, is thought to reflect prefrontal or executive functioning, as it requires cognitive flexibility, working memory and the ability to execute and modify a plan of action. The focus of the current study was to specifically examine the nature of executive functioning in children with ADHD, thus for this reason, only Part B of the TMT was administered as part of the test protocol.

The TMT - Part B Children's (Intermediate) version is a timed pencil and paper task requiring connections to be made between 15 encircled numbers and letters in alternating order. In accordance with instructions provided in the Neuropsychological Assessment of the School Aged Child manual (Anderson, Lajoie & Bell, 1995), children were asked to begin at number one and draw a line from this number to the letter A and then to number two and so on as quickly as they could in alternating numerical and alphabetical order until they reached the end. To ensure adequate competency with task instructions, children were required to successfully complete a practice trial prior to proceeding to Part B. Once achieved, children then progressed to Part B and were instructed to complete this task as quickly as they could. Errors made during completion of the task were immediately drawn attention to the participant with the instruction that they were to proceed from the point at which the mistake occurred. Total completion time of the TMT - Part B was recorded as the primary measure, however total number of errors committed were also counted and contributed to a slower completion time overall. Longer completion times indicated greater executive difficulty.

6.2.1.6 Digit Span Test

The Digit Span test is a verbal subtest of the WISC-IV and is one of two core subtests comprising the WISC-IV Working Memory Index (Wechsler, 2003). The

Digit Span task comprises two components: digit span forwards and digit span backwards, with the digits backwards component being one of the most well documented measures of working memory, with high reliability and validity (Spreen and Strauss, 1998). Digit span forwards involved a series of trials in which the child was required to immediately recall a string of increasing numbers, where digits were said at a rate of one per second. Initially, two digits were required to be recalled, followed by three digits, then four digits and so on. Successful completion of two trials at each level was necessary to avoid the discontinuation criterion. The second component of the digit span test, required the child to recall the digits in reverse order (for example 5 – 2 would be recalled as 2 – 5), where again an increasing number of digits was presented. As in the digits forward condition, the test was discontinued once both trials of a particular number of digits backwards was failed.

For each trial of the Digit Span test (both forwards and backwards) one point was scored for each correct response. Scaled scores were obtained by comparing performances with age matched norms. These scaled scores, along with those derived from the Letter Number Sequencing task (see below) were combined and transformed into the Working Memory Index (mean = 100, standard deviation = 15). This composite score was utilised in the statistical analysis.

6.2.1.7 Letter Number Sequencing

The Letter Number Sequencing Test is the second core subtest of the WISC-IV Working Memory Index (Wechsler, 2003). In this task, increasing sequences of numbers (from one to ten) and letters of the alphabet were orally presented in a random order, with the children being required to subsequently recall the numbers first in ascending order, with the letters following in alphabetical order. Initially, only two numbers and letters (i.e., 3 – H) were recalled, followed by increasingly complex sequences of more digits and letters. The task was discontinued after consecutive failures on all three trials of a particular item.

For each trial, one point was scored if all the numbers and letters were recalled in their correct sequence with the total sum of scores being compared to aged matched norms to obtain an overall scaled score. As mentioned previously, this score contributed to the Working Memory Index.

6.2.2 General Intelligence and Academic Achievement Tasks

6.2.2.1 Wechsler Intelligence Scale for Children – Fourth Edition (WISC-IV) Wide Range Achievement Test – Third Edition (WRAT-3)

The WISC-IV is the most commonly used measure of general intellectual ability in children (Wechsler, 2003) The core subtests of the WISC-IV were administered to all participants as part of the baseline cognitive assessment only. The WISC-IV was administered in accordance with the standard procedures outlined in the test manual and was conducted for the purpose of screening for intellectual (Wechsler, 2003). Full Scale IQ scores (mean = 100, standard deviation = 15) were calculated and used in the analyses.

6.2.2.2 Wide Range Achievement Test – Third Edition (WRAT-3)

The WRAT-3 is one of the most frequently employed tests of academic achievement (Wilkinson, 1993). The Reading and Arithmetic sections of the WRAT-3 were administered to all participants at the baseline assessment and were conducted for the purpose of screening for leaning difficulties (Wilkinson, 1993). WRAT-3 Reading and Arithmetic standard scores (mean = 100, standard deviation = 15) were calculated and used in the analyses.

6.2.3 Parent Rating Measures

6.2.3.1 Behavior Rating Inventory of Executive Functioning (BRIEF) – Parent Form

The BRIEF is a questionnaire comprising 86 statements identifying eight sub-domains of behaviour relating to executive functioning, which takes approximately 10-15 minutes to complete (Gioia, Isquith, Guy & Kentworthy, 2000). Parents were required to rate aspects of their child's behaviours by completing the questions related to domains of executive functioning, including inhibition, cognitive flexibility, emotional control, initiation, working memory, organisation, planning and self-monitoring. Parents responded according to three categories: Never, Sometimes or Often, based on the occurrence of their child's behaviours over the past month. Although raw scores and T scores for all BRIEF domains were documented and calculated, for the purpose of the current study the primary executive functioning domains examined were inhibition and working memory, therefore these were the only two indices included in the statistical analyses. On these scales, a higher numerical score signified poorer behavioural functioning relating to the particular sub-domain.

The BRIEF was chosen for its utility with children, having been standardised and validated for use with boys and girls ages five through to 18 years from wide ranging demographic and social backgrounds (Gioia et al., 2000). Furthermore, it has been documented to have good internal consistency and good inter-rater reliability, whilst the sub-domains of working memory and inhibition have correlated well with various ADHD scales (for example, the Conner's Rating Scale) (Gioia & Isquith, 2001) and have low correlations with behavioural and emotional difficulties unrelated to inattention and hyperactivity (Gioia et al., 2000).

6.2.3.2 Child Behaviour Checklist (CBCL) and Rowe Behavioural Rating Inventory (RBRI)

Parents also completed two standardised behaviour rating scales, the CBCL and the RBRI, at the time of the consultation with their paediatrician. These were administered prior to the baseline cognitive assessment by the consulting paediatrician, in combination with the clinical interview, in order to provide further supportive evidence for a diagnosis of ADHD.

The CBCL for ages 4-18 is a psychopathology checklist completed by parents, which provides a broad survey of emotional and behavioural problems in children (Achenbach & Edelbrock, 1991). The questionnaire obtains parent's ratings of 113 problem items, in addition to descriptions of behavioural, cognitive, social and academic strengths and weaknesses of the child. It typically takes approximately 15 minutes to complete. The CBCL consists of two broad subscales rating disturbance with emotional (internalising) problems and behavioural (externalising) problems, with other associated domains such as attention and social problems, anxiety and aggression.

The RBRI is an empirically derived rating scale used to obtain valid and reliable measures of children's levels of disruptive and difficult behaviour (Rowe & Rowe, 1997). It was completed by paediatricians, incorporating parental ratings of behaviour and primarily provides a measure of three dimensions of externalising behaviour: antisocial, restless and inattentive behaviour. These measures were not included in the statistical analyses.

6.3 Procedure

Approval to conduct the study was granted by the Victoria University Human Research Ethics Committee (see Appendix C).

Parents of children who had received a recent diagnosis of ADHD were offered the chance to participate in the study by way of consulting paediatricians at the time of consultation for ADHD diagnosis or treatment. Parents were given a detailed invitation to participate form by the paediatrician at the time of the consultation, which provided a thorough written explanation of the study, including what was required of both parents and children if participation was agreed upon (see Appendix D). At this time, parents indicated their interest in the study and verbal consent was obtained for the paediatrician to pass on their contact details to the researcher. Parents were subsequently contacted by phone in order to provide further details and confirm their willingness to participate. In addition to the written information provided, it was re-iterated to parents that their participation in the study was entirely voluntary and that they had the option to withdraw at any time. Furthermore, they were verbally informed that declined participation or withdrawal in no way influenced the medical treatment they were receiving from their paediatrician.

Once parents agreed, and prior to the initial baseline assessment, children were assigned to one of two treatment groups: those receiving medication alone or those receiving a combination of pharmacological intervention with cognitive behaviour therapy within a family dynamics framework (see Figure 6.1). Assignment of children to treatment group was determined by a combination of factors. In the first instance, simple alternation was applied, with the first child being assigned to the combined treatment group, the next to the medication alone group. Attempts were also made to match participants from both treatment groups on age and gender, subsequently this matching process had some influence on which treatment group children were assigned to. Furthermore, parents were allowed some choice over which treatment group their child was assigned. Specifically, parents were provided with verbal information about

both treatment groups, following which time they were informed which group their child was assigned to. If parents expressed unhappiness with their child's group assignment, they were allowed to request their child to be placed in the other group. This situation arose on only two occasions, with both families requesting their child to be moved from the medication alone group into the combined group. Therefore the majority of children were assigned to their treatment group by the foregoing principals of alternation and matching.

Once group membership was confirmed, a baseline cognitive assessment was organised. For the purpose of obtaining baseline results, and with the permission of the consulting paediatrician and the child's parents, all children already commenced on medication were instructed to withhold their regular dose of stimulants on the day of testing. This was clarified with each parent by way of a phone call in the 24 hours preceding the assessment and verbally confirmed with the parent at the commencement of the testing session. Due to the short half life of stimulant medication, this practice ensured that there were only minimal amounts of drugs, if any, circulating at the time of testing, consequently, eliminating any medication effect on the child's performance on baseline cognitive testing. Regular doses were resumed for those children already on medication, or commenced for those who had previously been un-medicated, following the completion of the initial cognitive evaluation.

Informed parent-signed consent was obtained from the parents at the time of testing (see Appendix D). Children also gave verbal assent to participate. All children in the study participated in an individual baseline cognitive assessment session lasting approximately 120 minutes, in which all cognitive measures were administered with a short break incorporated if required. All testing was conducted in the consulting rooms at the Victoria University Psychology Clinic at the St.Albans campus. Children either participated in a morning or afternoon assessment session and were assessed with a battery of standardised neuropsychological measures of general intellectual ability, academic achievement and executive functioning. In the initial baseline assessment all children were administered the core subtests of the WISC-IV to provide a measure of general intellectual functioning and to obtain a working memory index. In addition, an

achievement test was administered, namely the reading and arithmetic sections of the WRAT-3, to provide a measure of academic ability to compare to IQ in order to identify the presence of undiagnosed learning difficulties. Executive function measures, including the CPT, the TMT - Part B, the SCWT, the CAFT and the COWAT, were also administered during the initial evaluation. The order of task administration was counterbalanced in two different test orders to control for the possible effects of fatigue, any test learning factor and for effects of declining medication efficacy. Order A consisted of the WISC-IV, followed by the WRAT-3, the SCWT, the CAFT, the TMT – Part B, the COWAT and the CPT. Order B included the COWAT followed by the CAFT, the Trail Making Test, the Stroop, the CPT, the WISC-IV and the WRAT. All children were provided with verbal information at the start of the session about the reasons for assessment and the nature of the testing. Instructions for all tests were administered in accordance to the standardised procedures. Once the testing session was complete, children were provided with stickers as a thank you for their involvement.

Whilst the children were being assessed, parents completed a questionnaire (the BRIEF) which focused on their child's behaviour during the preceding month of being un-medicated. Parents were instructed to retrospectively rate behaviour for those children who had already commenced on medication prior to the current testing. A postage-paid self-addressed envelope was provided to parents if insufficient time resulted in an uncompleted questionnaire. Upon completion of the initial assessment, a brief one page neuropsychological report documenting the child's performance on the baseline assessment was completed and forwarded to the consulting paediatrician and parents at no charge.

6.3.1 Intervention

6.3.1.1 Medication Alone Treatment

The 13 children assigned to the medication alone group received a dose of stimulant medication only. Each dose was deemed to be at a clinically therapeutic level for each individual child as determined by their consulting paediatrician, with these prescribed doses remaining relatively stable over the duration of the six month treatment follow-up period. Children assigned to this group received no other formal therapy over the six months of follow-up however they continued to be monitored by their paediatrician as per standard clinical care. In addition they received follow-up phone calls by the researcher every six weeks to ensure they were adhering to their medication regime.

6.3.1.2 Combined Treatment

For those 14 children in the combined treatment group an additional family centred behavioural therapy component was added to their paediatrician prescribed stimulant medication treatment program. This involved participation in one, two to three hour psycho-education session involving family education and child focussed behaviour therapy for each individual child. This was typically carried out in the few days following completion of the initial baseline assessment. In order to achieve a consistent approach to treatment in each family, it was important that all those involved in the primary care of these children attended these sessions. All primary caregivers were encouraged to attend, and as a result, these sessions were typically attended not only by parents, but also by grandparents, aunties and uncles, and in some instances close friends of the family.

There were two main components of the intervention program: the psycho-educational component and the family centred cognitive behavioural therapy component. The first stage, the psycho-educational component, commenced with a read through and discussion of a hand-out explaining ADHD, including such

information as prevalence, causes, features, treatment and prognosis. This was followed by a presentation of the basic theory and general principles of behaviour therapy to the parents and primary caregivers, which included providing them with a written hand-out re-iterating all points discussed, including positive reinforcement and time-out techniques (see Appendix E). The practical family-centred cognitive behavioural therapy component consisted firstly of the completion of a problem behaviour questionnaire by the family and caregivers in order to identify the child's main problem behaviours and to ascertain the circumstances surrounding the occurrence and maintenance of these behaviours (see Appendix E). Once a limited number of target behaviours were identified for that particular child, an interactive dialogue followed of how next to proceed in order to successfully target the behaviour. This involved the researcher presenting simple, individualised strategies, typically involving the utilisation of positive reinforcement techniques, to target specific behaviours. In other words, parents and close others identified target ADHD problem behaviours and were encouraged to reward the child for not engaging in the problematic targeted behaviour with the goal of reducing or eliminating unwanted behaviours, subsequently increasing the frequency of wanted behaviours.

Specifically, real life examples of the child's target behaviour, including antecedents and consequences of the behaviour, were reviewed with a resulting 'workshop' discussion considering appropriate strategies to implement and practical ways to consistently implement them. For example, if the parents identified that their child had great difficulty sitting for any length of period and completing a task without getting distracted, the primary caregivers were instructed to encourage and reward the child with something appealing to them (i.e., 15 minutes on the play-station) if the child was able to sit and complete a task for five minutes without distraction. To extend the length of the desired behaviour, the caregivers were then instructed to introduce increasing intervals (i.e., ten minutes without distraction) before rewards were given. All primary care-givers were provided with written information on the specific behaviours to be targeted and the strategies suggested for each behaviour. They were encouraged to consistently and reliably apply these principles to the children involved.

To monitor this and to ensure families were consistently implementing the set program, parents or other caregivers of children in the combined treatment group completed a weekly progress diary detailing the frequency and perceived successfulness of the behaviour therapy techniques (see Appendix E). Follow-up phone calls every six weeks were also made to the families to provide encouragement and further consultation if required.

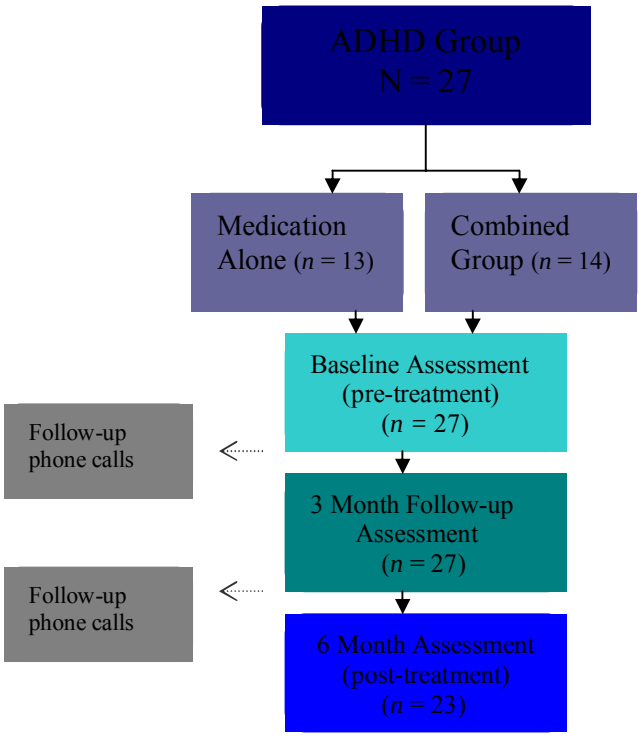
6.3.2 Three Month Follow-up

As mentioned previously, all children in the study were followed-up at approximately six weeks post initial assessment by way of a phone call to monitor medication regimes and implementation of family centred behaviour therapy, and to address any issues that may have arisen regarding intervention. After approximately 12 weeks of treatment (either medication alone or combined therapy), 27 children (100% of the original sample) returned to the Victoria University Psychology Clinic for a three month follow-up treatment assessment (see Figure 6.1). The protocol of this evaluation included the re-administration of all previously administered cognitive measures in a counterbalanced order, including the SCWT, the CPT, the TMT – Part B, the COWAT, the CAFT and the Digit Span and Letter Number Sequencing subtests from the WISC-IV in order to obtain a Working Memory Index. The remaining core subtests of the WISC-IV and the WRAT-3 were not re-administered as these measures were only used to establish general intellectual functioning and academic skills in order to ascertain eligibility. Parents also completed another BRIEF – Parent Form which detailed their child's behaviour in the preceding three months, whilst on medication and/or whilst engaged in the cognitive behavioural therapy program. All children at the three month follow-up were assessed within two hours of taking their regular dose of stimulant medication in order to establish medication effects. Paediatricians and parents once again received a brief neuropsychological report documenting the results of the post treatment assessment.

6.3.3 Six Month Follow-up.

A sub-group of the sample of children with ADHD were also followed up at approximately six months post initial baseline cognitive assessment ($n = 23$) (see Figure 6.1). There was a small attrition rate, with four children not being assessed at six months. The reason these children were not followed-up was due to a lack of current contact details. This assessment was conducted in order to establish the stability of treatment effects over an extended time period. Therefore, all children re-tested at the six month time assessment session, had continued taking either medication alone or in combination with family centred behavioural therapy (according to their initial group assignment) through out the duration of the research study. The test protocol at this third assessment time point remained identical to the three month follow-up assessment, with tests being given in a counterbalanced order. In addition, the BRIEF – Parent Form was also completed again by the parents. Analogous to the procedure followed at the second assessment session, all children had been medicated with psychostimulants (as per their usual medication schedule) in the two hours prior to being assessed. Parents and consulting paediatricians again received a one page report documenting the child's performance in the context of their treatment program.

Figure 6.1 Flow chart of assignment to treatment groups and of assessment time points.



Chapter 7: Results

7.1 Data Analysis

Data were analysed using the Statistical Package for the Social Sciences – Windows Version 14.0. Preliminary data analysis was carried out to assess normality of the sample's scores on the dependent variables in order to assist in the appropriate selection of statistical techniques. Descriptive data regarding skewness and kurtosis of the dependent variables were obtained, with an acceptable level of skew and kurtosis, in the range of +2 to -2 being achieved on all dependent variables for the sample. Tests of normality, using the Shapiro-Wilk statistic for small sample size, were ascertained to determine the presence of any significant deviation from normality, with the normality of the distribution of scores not being violated on any outcome measure. As these assumptions were met, parametric statistics were selected as the most appropriate techniques to analyse the primary results.

Demographic and subject characteristics were analysed for the whole ADHD sample and compared across the two treatment groups using independent samples *t*-tests for the continuous variables (for example, age) and nonparametric chi square analyses for the categorical variables (for example, gender).

Single sample *t*-tests were carried out to compare the performances of the ADHD group against the age matched normative data accompanying each dependent variable. Each test was considered separately, with multiple single sample *t*-tests being computed in order to determine whether the group means for the present sample differed significantly from the normative test means. Interpretations of significance were based on standard definitions of statistical significance ($p<.05$). For the purposes of performing comparative examinations of the normative data against the ADHD group, standardized scores were used in the analyses. For those outcome measures in which standard scores could not be derived (Trail Making Test – Part B, the COWAT and the CAFT), raw scores were transformed into z-scores and one sample *z*-tests were performed in order to

allow for a comparison of the ADHD group's scores against one normative standardized score. Individual percentages of those children whose performances on the dependent variables fell in the impaired range at baseline and at the six month treatment follow-up assessment were also calculated.

To determine the magnitude of potential change in scores in the whole ADHD group across the range of dependent variables over the three treatment time points, a series of repeated measures analysis of variance (ANOVA) were conducted, with time (three levels: baseline, three month follow-up and six months follow-up) as the within subjects factor and group as the between subjects factor. Multiple repeated measures ANOVAs were similarly performed for each dependent variable for the two conditions of the independent variable, the medication alone group and the combined treatment group. Despite having a considerable number of outcome measures, univariate procedures, rather than multivariate tests, were considered the most appropriate statistical analysis to compare each dependent variable. The fundamental rationale underlying the selection of univariate analyses relates largely to the modest sample size in the current study. Whilst multivariate approaches provide protection against inflated Type I error rates, a univariate approach has comparatively more power than a multivariate design when participant numbers are relatively small. Specifically, Maxwell and Delaney (1990) advise against the use of a multivariate approach when sample sizes are limited due to the associated loss of power. Alternatively, they advocate running univariate analyses with appropriate statistical adjustments, to minimize the possible risk of inflating familywise errors. Accordingly, the current study adopted an alpha level of .01, but also presents findings at an alpha level of .05 for heuristic purposes.

Another issue in the decision to adopt a univariate approach concerns sphericity, or the conclusion that there are equal variances in the differences between treatment levels. The effect of violating the assumption of sphericity can potentially produce an invalid F ratio with an associated loss of power. Multivariate analysis of variance (MANOVA) is often considered an advantageous statistical approach in order to avoid the assumption of sphericity inherent in univariate repeated measures designs. However Field (2005)

recommends that when participant numbers are modest and the assumption of sphericity is maintained (as was the case in the greater part of the data in the current study), then the more powerful and hence preferred approach are univariate analyses, rather than multivariate tests. Of the few dependent variables that did not satisfactorily uphold this sphericity assumption in the current study, appropriate adjustments to correct for these violations were made. Specifically, the Greenhouse-Geisser correction was used when estimates of sphericity were lower than .75, with Huynh-Feldt epsilon being utilized when they were greater than .75 (Field, 2005).

Significant effects were further analysed with post hoc tests to determine the source of differences in mean scores over the three evaluation points for the ADHD group as a whole and also separately for the two treatment groups. Bonferroni corrections were applied to the multiple pairwise comparisons due to the robustness of this univariate technique to control Type I error rates in small samples (Field, 2005). This method of correcting for multiple comparisons also acceptably increases test power if there are unsatisfactory departures from sphericity (Maxwell, 1980).

In addition to the reporting of statistical significance through the estimates of probability (ie., *p* values), estimates of effect size were also included. By providing details of the magnitude of effects, significant results in the current study were able to be interpreted in a more practical and meaningful way by not only illustrating the strength of effects, but also having the important benefit of allowing for a more reliable and uniform comparison with comparable previous research in the field (Schuele & Justice, 2006). Furthermore, effect sizes have an additional advantage, in that, unlike statistical significance, effect sizes are interpreted independent of sample size. In other words, there is no association between sample size and the likelihood of finding significant effects, as is the case with achieving statistical significance (Zakzanis, 2001). Cohen's *d* was used to assess the magnitude of effect size, following the convention of .20 equating to a small effect, .50 a medium effect and .80 representing a large effect (Cohen, 1988).

Mixed between-within groups ANOVAs with time as the within groups variable and group as the between groups variable were utilized to assess for the presence of any significant main or interaction effects between the treatment group on changes in the dependent variables over time. Again, there were very few violations to the assumption of sphericity, however these were appropriately corrected when required. In addition, in light of the multiple within subject analyses conducted, a conservative alpha level of .01 was selected to achieve significance to assist in minimizing the possible risk of Type I error. Effect sizes were also calculated utilizing the partial Eta squared based on Cohen's (1988) criteria. Polynomial contrasts were also performed to examine the presence of significant trends in the data. An investigation of the linear and quadratic components of trend were made as an additional analysis to assess the pattern of change in performance of either treatment group over the three assessment time points.

Finally, relationships among the parent reported BRIEF inhibit index and working memory index and performances on the performance based cognitive measures of inhibition were investigated by calculating Pearson product-moment correlation co-efficients. Partial correlations were subsequently calculated between parent ratings of behaviour on the BRIEF and on the correlated cognitive measures controlling for intelligence (FSIQ) and academic ability (WRAT-3 Reading and Arithmetic). Preliminary analyses were performed to ensure no violation of the assumptions of normality, linearity and homoscedasticity. Raw score variables were used in the correlational analyses.

7.2 Sample Characteristics

The demographics of the whole sample are summarized in Table 7.1. The 27 ADHD children had a mean age of eight years and eight months at the time of pre-treatment baseline assessment. In line with reported gender prevalence rates of ADHD, the majority of the participants were male (85%). In terms of diagnosis of ADHD subtype, the sample was predominantly classified by their treating paediatricians as ADHD

Combined type (63%), with the other third receiving a diagnosis of ADHD predominantly Hyperactive type (37%). No children with a diagnosis of ADHD predominantly Inattentive type were included in the sample, as their specific cognitive profile has been suggested in the literature to be different from the two aforementioned diagnostic categories upon which Barkley's inhibition theory is based.

Full Scale Intelligence Quotients (FSIQ) from the WISC-IV (Wechsler, 2003) were measured using the Australian normative data for each child in the sample at the baseline evaluation as a screening measure to identify the presence of any undiagnosed intellectual difficulties. Overall for the sample, the mean IQ was within the average range (Mean = 91.2, Standard Deviation (S.D.) = 7.0) and all participants had a FSIQ of 80 or above (range 80 – 105). Using a two tailed single sample t-test, in which the WISC-IV Australian published normative data was used to represent the population mean (i.e., M = 100, S.D. = 15), the sample mean's FSIQ, although in the average range, was significantly below the population mean of the test [$t(25) = -6.1$, $p < .05$].

Academic achievement scores (reading and mathematics) as measured by the WRAT - 3 (Wilkinson, 1993) were also assessed initially at the pre-treatment evaluation in order to detect the presence of any potential learning difficulties in the sample of ADHD children. The mean standard score of the ADHD sample for reading achievement (M = 94.4, S.D. = 11.3) and arithmetic skills (M = 91.5, S.D. = 9.6) both fell within the average range, with participants scoring above 80 on both academic measures (range 80 – 121 for reading, and 80 to 119 for arithmetic). Single sample t-tests, however, revealed both reading [$t(25) = -2.6$, $p < .05$] and arithmetic [$t(25) = -4.7$, $p < .05$] to be significantly below the test's published normative means.

Table 7.1. Demographic Characteristics of the ADHD Group at Baseline.

Demographic Variable		ADHD Group (N=27)
Age (Years)	Mean (SD)	8.8 (1.6)
	Range (Years)	7 - 12
Gender	Male (%)	23 (85%)
	Female (%)	4 (15%)
ADHD Subtype	Hyperactive (%)	10 (37%)
	Combined (%)	17 (63%)
FSIQ*	Mean (SD)	91.2 (7.0)
WRAT^		
Reading	Mean (SD)	94.4 (11.3)
Arithmetic	Mean (SD)	91.5 (9.6)

* Composite Score
^ Standard Score

7.2.1 Group Comparisons on Sample Characteristics

Among the 27 children with ADHD in both treatment groups (medication alone and combined therapy), no significant differences were identified when comparing the two treatment groups on any demographic characteristic at the pre-treatment baseline evaluation. Statistical analyses, including independent samples t-tests for the continuous variables (i.e., age) and chi-square analyses for the categorical variables (i.e., gender), revealed that the medication group (n = 13) and combined group (n = 14) were evenly matched on all demographic variables at the baseline assessment, prior to the introduction of the interventions (Table 7.2). Both groups were approximately eight years of age, predominantly male and with a mean average FSIQ and mean low average to average academic ability. There was a relatively even diagnostic rate of ADHD subtypes across the two treatment groups.

Table 7.2. Comparison of Treatment Groups on Demographic Characteristics at Baseline

Demographic Variable		Medication Alone Group (n=13)	Combined Group (n=14)	*p* value
Age	Mean (SD)	8.7 (1.7)	8.8 (1.6)	.70
Gender	Male (%)	11 (85%)	12 (86%)	
	Female (%)	2 (15%)	2 (14%)	.86
ADHD Subtype	Hyperactive (%)	4 (31%)	6 (43%)	
	Combined (%)	9 (69%)	8 (57%)	.76
FSIQ*	Mean (SD)	92.6 (6.8)	90.3 (7.3)	.42
WRAT^				
Reading	Mean (SD)	94.0 (11.2)	92.7 (9.8)	.75
Arithmetic	Mean (SD)	92.9 (10.3)	88.8 (7.4)	.24

* Composite Score
^ Standard Score

A comparison of the demographic variables between the treatment groups over the two subsequent treatment assessment points are presented in Table 7.3. Similar to the baseline evaluation, the two treatment groups were evenly matched at both the three and six month post baseline testing on demographic characteristics, including age, gender and ADHD subtype, with no significant differences noted between the two treatment groups on any of these variables. Furthermore, the mean number of days the second (three month) and third assessment (six month) sessions were conducted from the baseline evaluation were closely comparable for the two treatment groups, with no statistically significant differences between the groups.

Table 7.3. Comparison of Treatment Groups on Demographic Characteristics over the three and six month treatment evaluations.

		ADHD Group (N=27)					
		2nd Assessment (3 months)			3rd Assessment (6 months)		
Demographic Variable		Medication Alone (n = 13)	Combined Group (n = 14)	p	Medication Alone (n = 11)	Combined Group (n = 12)	p
Age	Mean (SD)	8.9 (1.7)	9.1 (1.7)	.71	9.2 (1.7)	8.9 (1.7)	.69
Gender	Male (%)	11 (85%)	12 (86%)		10 (83%)	10 (83%)	
	Female (%)	2 (15%)	2 (14%)	.85	2 (17%)	2 (17%)	1.00
ADHD	Hyperactive (%)	4 (31%)	6 (43%)		2 (17%)	3 (25%)	
	Combined (%)	9 (69%)	8 (57%)	.76	10 (83%)	9 (75%)	.52
No. of Days since Baseline Assessment							
	Mean (SD)	98 (14.3)	100 (21.1)	.70	208 (34.6)	214 (40)	.71

7.2.2 Group Comparisons on Medication Status

At the time of the initial baseline evaluation, the majority of children had recently commenced on stimulant medication as prescribed by their consulting pediatrician. All children, however, were free of medication at the time of baseline testing, with those who had already commenced their psychostimulant treatment ceasing medication for an average of 32 hours (range 20 hours to 76 hours) prior to participation in the first cognitive assessment. During the active intervention phase, which commenced immediately following the initial baseline assessment, children began taking a dose of either methylphenidate (82%) or dexamphetamine (18%) clinically determined by their pediatrician to be at a therapeutic level for the individual. These dosages ranged from 10mg/kg to 20mg/kg a day, which is considered comparable with average daily doses administered in clinical settings (Department of Health, 2005). Assessments were carried out over two daily session times, with the majority of children being tested in the

morning session (81%). There was no significant difference between the medication alone group and the combined treatment group in the time of day their assessment sessions were conducted [$\chi^2(25) = .724, p < .05$].

Table 7.4. Whole ADHD Group Medication Status at Baseline.

Medication Status		ADHD (N=27)
Daily dose (mg)	Mean (SD)	18 (4)
	Range (mg)	10 - 20
Type of Medication		
	Methylphenidate *n* (%)	22 (81%)
	Dexamphetamine *n* (%)	5 (19%)
Hours since Medication		
	Mean (SD)	32.3 (15)
Duration (days)		
	Mean (SD)	147 (136)
	Range (days)	0 – 265
Time of Assessment		
	Morning (%)	22 (81%)
	Afternoon (%)	5 (19%)

Children in both the medication and combined treatment groups continued to take medication throughout the six month intervention period, with daily medication doses for children in both treatment groups remaining stable across the six month treatment phase, with no significant differences between the two treatment groups in the daily dose of medication taken at baseline [$t(25) = -.83, p > .05$], three month follow-up [$t(25) = -.669, p > .05$], or the six month evaluation [$t(25) = .635, p > .05$]. At the three month and six month follow-up assessment, all children were evaluated whilst actively taking medication, with all participants being assessed within one and a half hours of ingesting their regular dose of either methylphenidate or dexamphetamine in order to document medication effects. There was no statistically significant difference between the medication alone group and combined group in the time between taking their medication

and being assessed both at the three month assessment time period [$t(25) = -1.7$, $p > .05$] and at the six month evaluation [$t(25) = 1.5$, $p > .05$].

7.3 Group Results

7.3.1 Hypothesis One:

It was predicted that children with ADHD would perform worse compared to published age norms for each of the dependent variables. More specifically, it was expected that children with ADHD would exhibit cognitive and behavioural impairments in inhibitory control as measured by a range of measures of cognition and parental reports of behaviour.

In order to investigate this initial hypothesis, cognitive test performances and parent ratings of behavioural functioning of the ADHD group (N=27) were compared to the age matched published test normative data accompanying each cognitive and behavioural outcome measure using a series of single sample *t*-tests. Results of these analyses revealed that the ADHD group as a whole performed well below the tests normative means across a range of the cognitive and behavioural dependent measures of executive functioning. In terms of behaviour, mean parent ratings of the ADHD group's behaviour on the two key BRIEF indices measured, were significantly worse than the tests normative means, with both the inhibition index [$t(25) = 13.6$, $p < .001$] and working memory index [$t(25) = 13.5$, $p < .001$] falling well below the 5th percentile, indicating that the ADHD sample's scores were more than two standard deviations below the normative mean. Specifically, children were rated by their parents at baseline to be exhibiting significantly more problem behaviours than age matched norms, with parents endorsing high levels of disinhibition (for example, gets out of seat at the wrong times) and working memory problems (for example, when sent to get something, forgets what he/she is supposed to get). Associated effect sizes for the behavioural measures were considered

to be in the large range, with both BRIEF indices having effect sizes of .80 (Cohen , 1998).

Executive function performance based measures of the ADHD sample as a whole were also found to be significantly worse at baseline across the range of cognitive tests compared with the normative data provided in the test manuals. Performance on the Working Memory Index (WMI) from the WISC-IV approached one standard deviation below the normative mean, with group performances placing the ADHD sample in the low end of the average range. Analysis with a single sample *t*-test found the ADHD children to be performing significantly worse than the standardized normative data [t (25) = -6.01, p <.001] with an associated low to moderate effect size (.40). At baseline, the sample of ADHD children also performed well below the tests normative means on the CPT. Specifically, they exhibited significantly higher numbers of commission errors [t (25) = 9.74, p <.001] and omission errors [t(25) = 5.64, p<.001] compared to the normative data, with the mean number of impulsive and inattentive errors both being more than two standard deviations above normative mean values. Effect sizes associated with both measures were considered to be in the medium to large range (.79 and .56 respectively). . Mean performances on the important inhibition related color/word [t(25) = -5.00, p<.001] and interference conditions [t(25) = -4,230, p<.001] on the SCWT were similarly significantly below age expected levels when compared to the published norms. The strength of the differences between the ADHD group and the normative means was in the low range (.30 and .25 respectively). As a group, the ADHD children's performances were at least one, to one and a half standard deviations below the normative mean for the test. In contrast, the ADHD group performed in the normal range on the word [t(25) = -0.33, p>.54] and color [t(25) = -0.96, p>.77] components of the task, with the group performances not differing significantly from the published norms on these two less demanding SCWT conditions.

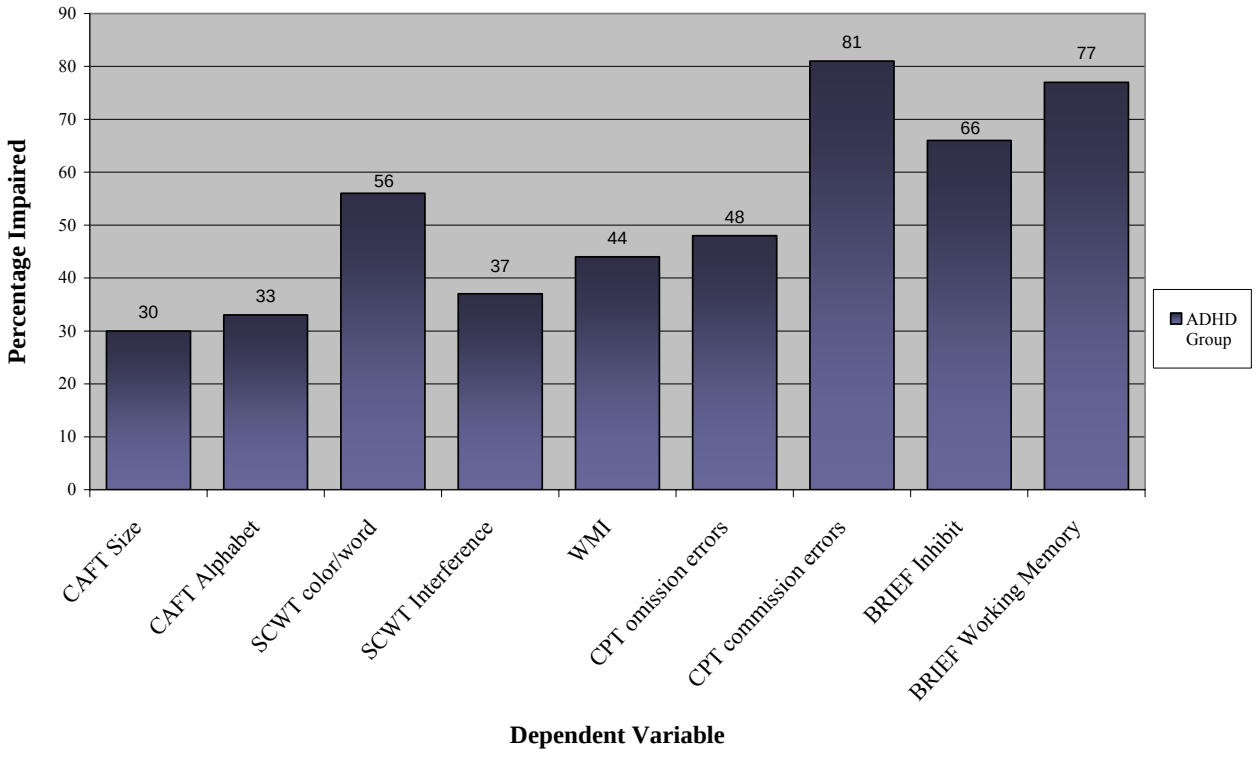
Single sample *z*-tests were conducted for the remaining dependent variables. Results of these analyses found the ADHD children to be impaired at baseline compared to age matched norms on the more cognitively demanding conditions of the verbal fluency

based CAFT. Specifically, performances placed their functioning in the low average range on the size condition [$z = -2.6$, $p < .02$] and on the alphabet condition [$z = -6.6$, $p < .000$], with the number of words generated on these conditions falling significantly below those reported in the normative data. In contrast, the ADHD sample performed well within the average range for the total number of words produced on the less complex automatic condition on the CAFT [$z = -1.14$. $p > .28$] and on the measure of relative difficulty on the CAFT [$z = -.90$, $p > .93$] (see Appendix F for normative data). Furthermore, the ADHD group performed within the normal range on the COWAT, with no statistically significant differences being found on the total number of words produced on this phonemic fluency task when compared to the age matched normative data [$z = -.1.10$, $p > -.59$]. Finally, response times recorded on the Trail Making Test – Part B revealed significantly faster performances than those of the normative means, with the ADHD group's functioning being placed in the average to above average range overall [$z = 5.05$, $p < .001$].

Despite the presence of statistically significant differences between the ADHD sample's overall group performance and the test's published normative means across many of the cognitive and behavioural measures of inhibition, it is well documented in the literature that children with ADHD manifest a significant degree of individual difference in both their behavioural and cognitive profile in the patterns and magnitude of impairments they exhibit. Although the series of single sample *t*-tests and *z*-tests comparing the ADHD group means with the published normative means demonstrated the presence of significant differences at a whole group level on a range of outcome measures, it failed to provide us with the more important and clinically relevant individual level of analysis, showing specifically how many individuals in the group actually exhibited clinically significant impairments in inhibition. Therefore an additional method of analyzing the cognitive and behavioural data are presented here. The percentage of ADHD children in the sample whose cognitive performance and parent ratings of behaviour were at least 1.5 standard deviations below the mean were computed (ie, approximately functioning at or below the 7th percentile). This individual level of analysis provides a more clinically meaningful picture of the percentage of individuals

who were actually functioning in the impaired range across the various outcome domains, rather than presenting just whole group deficits which has the potential to mask any individual differences or unique patterns in the data. Figure 7.1 summarizes the percentage of children whose cognitive performance or behaviour ratings fell within the impaired range across those dependent variables where a significant difference was found between the performances of the ADHD children compared with the aged matched normative data. Individual data presented here reveal that despite finding significantly different group means between the ADHD sample and the normative data, there were considerable variations in the percentage of children that were actually impaired on these tests, ranging from only 30% of the total group on two conditions of the CAFT to a considerable 81% being impaired on the CPT (commission errors).

Figure 7.1 Percentage of ADHD Children Impaired Overall at Baseline



7.3.2 Hypothesis Two:

It was predicted that cognition and behaviour relating to executive functioning, such as inhibition, would show improvements after six months of either stimulant medication therapy or combined family centered cognitive behaviour therapy and stimulant medication therapy. Specifically, it was hypothesized that performances on standardized cognitive measures of inhibition would be significantly improved from pre-treatment baseline to six month follow-up after either stimulant medication treatment alone or stimulant medication in combination with behavioural therapy. In addition, the same pattern was predicted on parent rated measures of behaviour relating to inhibition, with a reduction in problem behaviours expected following six months of continued pharmacological treatment alone or in combination with behavioural therapy.

7.3.2.1 Effects of Treatment on the ADHD Group over Six Months

To determine the magnitude of change in the ADHD group as a whole across the range of outcome measures over time (regardless of treatment group), a one way repeated measures analysis of variance (ANOVA) was conducted. The means, standard deviations, significance levels and pairwise comparisons are displayed for each dependent measure across the three follow-up times in table 7.5 (significance levels were rounded to three decimal points). Over all dependent variables, a significant within groups treatment effect for time was found, with all but one measure (CAFT automatic condition, significant at .05) achieving significance at the .01 alpha level. Specifically, these pre and post-test analyses revealed a substantial reduction in parent reported behavioural problems, and significant improvements in executive cognitive processes on all outcome measures over the six months of continued treatment with either medication alone or combined therapy. Furthermore, partial Eta squared calculations suggest that treatment effects over the six months were sizeable across the majority of the outcome measures, with an average moderate effect size overall (average effect size was .45; ranging from .15 to .93) (Cohen, 1988).

To further break down these significant effects of time, post hoc test comparisons were performed comparing each of the three levels of time across the dependent variables for the whole ADHD group. Pairwise comparisons with Bonferroni corrections revealed that baseline scores at time one were significantly different from scores on the second assessment after three months of treatment, with substantial improvements noted across the three months on all dependent variables, with the exception of the CAFT Relative Difficulty Score and the SCWT Interference. Despite both of these measures exhibiting improvements over the first three months, they failed to achieve significance at the .01 alpha level. Planned comparisons revealed that performances at the third assessment session at six months post treatment were also significantly better than baseline scores across all of the dependent variables. Finally, comparison of time two and three performances revealed significant differences between dependent variable scores on seven of the outcome measures, including the Trail Making Test - Part B, the CAFT (size and alphabet conditions), the SCWT (word, color and color/word trials) and the CPT (commission errors), with six month scores found to be superior to three month performances on these measures (see Table 7.5).

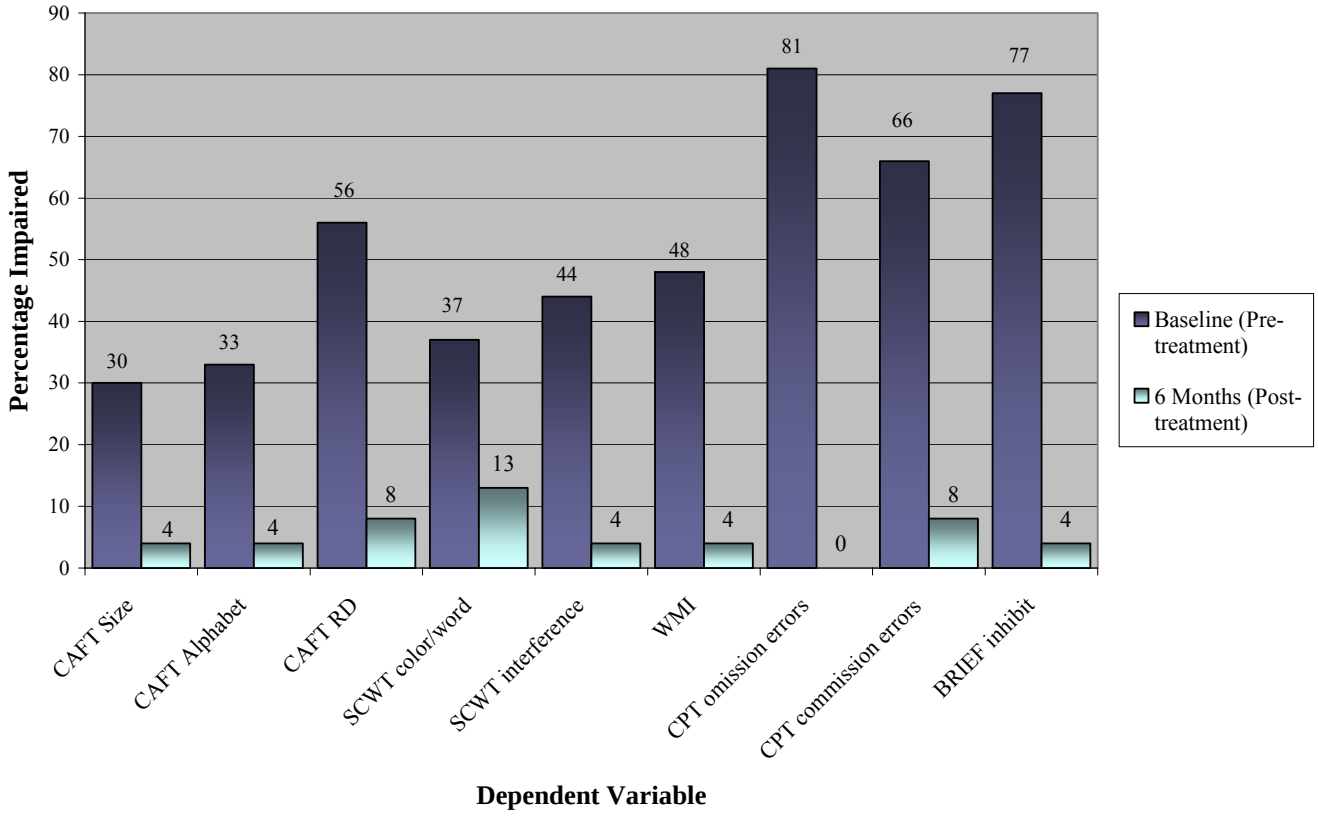
An additional individual level of analysis was also performed at the six month evaluation to determine the percentage of children who had remained in the impaired range on behavioural and cognitive dependent measures following six months of either medication alone or combined treatment. Figure 7.2 displays a comparison of these data with the percentage of children with ADHD impaired at initial pre-treatment baseline. As is clearly illustrated in Figure 7.2, a comparison of individual percentages at baseline and six months demonstrate a significant reduction in problem behaviours and improvements in cognition in the ADHD group following treatment. Only a very small percentage of children remained in the impaired range after six months of treatment, suggesting that treatment (either medication alone or a combination of medication and behaviour therapy) was successful in normalizing behaviour and cognition in the vast majority of children with ADHD.

Table 7.5: One way within group ANOVA: Comparison of the performance of the whole ADHD group over the three assessment time points.

Measure	Baseline	3 months	6 months			
	Mean (SD)	Mean (SD)	Mean (SD)	*p*	Partial Eta^2	Pairwise Comparison
WMI	89.5 (9.5)	101.3 (7.8)	101.0 (8.9)	.000**	.58	Time 2 & 3>Time 1
Trails B Time	81.4 (32.3)	60.5 (25.0)	47.8 (12.6)	.000**	.46	Time 2 &3 > Time 1; Time 3> Time 2
COWAT (Total)	17.6 (7.4)	20.7 (8.1)	22.0 (7.3)	.003**	.29	Time 2 & 3>Time 1
CAFT (Automatic)	13.5 (4.7)	15.5 (4.1)	15.3 (3.6)	.016*	.21	Time 2 & 3>Time 1
CAFT (Size)	6.3 (2.7)	7.2 (2.0)	8.5 (2.4)	.000**	.37	Time 2 &3 > Time 1; Time 3> Time 2
CAFT (Alphabet)	5.7 (2.3)	7.3 (1.8)	8.0 (2.3)	.000**	.42	Time 2 &3 > Time 1; Time 3> Time 2
CAFT (RD)	55.3 (15.4)	52.5 (11.9)	44.2 (11.2)	.004**	.21	Time 3>Time 1 & 2
SCWT (Word)	52.4 (10.7)	57.0 (11.6)	60.2 (11.4)	.000**	.40	Time 2 &3 > Time 1; Time 3> Time 2
SCWT (Color)	41.5 (7.4)	45.6 (7.2)	47.8 (6.7)	.000**	.66	Time 2 & 3>Time 1; Time 3> Time 2
SCWT (C/W)	17.6 (10.8)	24.5 (4.8)	28.2 (5.1)	.000**	.40	Time 2 &3 > Time 1; Time 3> Time 2
SCWT (Interference)	-22.2 (5.1)	-21.1 (6.6)	-19.6 (5.8)	.006**	.15	Time 3>Time 1 & 2
CPT (Comm. Errors)	19.1 (6.8)	12.4 (3.5)	6.0 (3.3)	.000**	.93	Time 2 &3 > Time 1; Time 3> Time 2
CPT (Omiss. Errors)	12.9 (5.0)	7.3 (3.5)	6.6 (4.7)	.000**	.39	Time 2 & 3 > Time 1
BRIEF (Inhibit)	25.0 (4.3)	17.4 (4.0)	17.4 (4.1)	.000**	.67	Time 2 & 3 > Time 1
BRIEF (WM)	25.0 (3.0)	18.0 (4.1)	18.0 (4.8)	.000**	.63	Time 2 & 3 > Time 1

*$p < .05$; **$p < .01$

Figure 7.2.Comparison of the percentage of ADHD children impaired overall at pre-treatment baseline with six month treatment follow-up.



7.3.2.2 Comparison of Pre-Treatment Dependent Measures

In the evaluation of the pre-treatment phase thus far, only group results and individual percentages have been presented for the ADHD sample as a whole. Independent sample *t*-tests however were also performed to compare the performances of the medication alone group with the combined treatment groups on the objective cognitive measures and parental behavioural ratings of inhibition at the baseline evaluation to determine the presence of any differences in pre-treatment functioning (see Table 7.6). Results of the *t*-tests revealed no significant differences between the medication alone group and the combined treatment group on any pre-treatment dependent variable at the .01 significance level. It is worth noting that significant differences were found on pre-treatment scores between the two treatment groups on the number of CPT commission errors made, $t(25)$

= -2.15, $p<.043$, and the BRIEF Inhibition Index, $t(25) = -2.23$, $p<.035$, at the .05 significance level, but these were not significant at the critical .01 level. Specifically, the combined treatment group committed more errors of commission on the CPT at baseline, whilst they were also reported as having more inhibition problems as rated by parents, than the medication alone group on initial pre-treatment testing. Despite only achieving significance at the .05 level, baseline scores for these two variables were entered into the statistical analyses comparing the two group's performances to control for differences. Overall, however, these findings confirm that the two treatment groups were relatively homogenous in terms of their cognitive and behavioural functioning at baseline.

Table 7.6: Pre-Treatment Baseline Scores on Cognitive and Behavioural Dependent Measures for the Medication Alone and Combined Treatment Groups.

	Medication Alone	Combined Treatment		
Dependent Measure	Mean (SD)	Mean (SD)	*t*	*p*
WMI	91.69 (10.2)	86.64 (7.9)	1.440	.162
Trails B (Time)	75.69 (18.1)	84.2 (43.9)	-.669	.513
Trails B (Errors)	1.15 (.99)	1.42 (1.6)	-.520	.608
COWAT (Total)	19.54 (6.8)	15.50 (7.2)	1.481	.151
CAFT (Automatic)	15.23 (3.9)	12.07 (4.9)	1.828	.079
CAFT (Size)	7.23 (1.6)	5.86 (3.1)	1.460	.160
CAFT (Alphabet)	6.38 (1.9)	4.93 (2.6)	1.658	.110
CAFT (RD Score)	50.85 (11.5)	53.86 (21.3)	-.450	.657
SCWT (Word)	54.77 (11.2)	49.76 (9.5)	1.248	.224
SCWT (Color)	41.38 (6.2)	41.35 (8.3)	.010	.992
SCWT (C/W)	18.53 (5.4)	16.57 (14.1)	.472	.641
SCWT (Interference)	-22.85 (5.4)	-21.29 (5.4)	-.746	.462
CPT (Comm. Errors)	16.54 (4.9)	21.93 (7.9)	-2.15	.043*
CPT (Omiss. Errors)	13.61 (6.3)	11.57 (3.5)	1.058	.300
BRIEF (Inhibit)	24.16 (4.4)	27.21 (2.6)	-2.23	.035*
BRIEF (WM)	24.77 (2.6)	26.71 (3.5)	-1.65	.111

* $p < .05$

7.3.2.3 Effects of Treatment on Cognitive and Behavioural Measures over Six Months

Table 7.7 and Table 7.8 presents baseline, three month and six month means and standard deviations, and the statistical results of the repeated measures ANOVA main effects for time for the behavioural and cognitive dependent measures for the medication alone and combined treatment groups respectively.

7.3.2.4 Changes in the Medication Alone Group over the Six Month Treatment Follow-up.

Findings from the one-way repeated measures ANOVA revealed significant effects of time on ten of the fifteen cognitive and behavioural dependent variables at a significance level of .01, indicative of improvements on these measures across the six months of continued medication alone treatment. Planned post hoc pairwise comparisons found significant differences between the initial pre-treatment baseline and the first follow-up treatment evaluation at three months on all of these ten measures (see Table 7.7). Specifically, significant improvements from baseline to three months were noted on a range of cognitive measures and behavioural measures, with the exception of three of the CAFT conditions, the COWAT total number of words produced and the interference trial on the SCWT, which failed to achieve significance.

A further investigation of the significant statistical comparisons revealed the presence of significant differences between pre-treatment baseline performances and scores produced at the six month post-treatment assessment time point. Specifically, pairwise comparisons, with Bonferroni corrections, and examination of mean values revealed the presence of significant improvements from time one to time three on all of the same dependent variables identified as showing improvements over the first three months of treatment. These improvements were associated with moderate to large effect sizes across both the cognitive and behavioural measures, with an average medium effect size

of .42 overall (ranging from .38 to .70). More specifically, the two behavioural measures averaged larger effect sizes (.56) than the cognitive variables (.39).

Notably, when specifically examining the significant contrasts from the three month evaluation to the six month follow-up assessment, only three of the nine cognitive variables that showed initial improvements (TMT – Part B, SCWT color naming, and SCWT color and word condition) continued to show statistically significant improvements from three to six months, with neither of the behavioural indices showing any further changes from time two to time three. These data collectively suggest that after significant improvements over the first three months, there was an absence of continued significant improvements across most domains of performances from three months to six months of medication alone treatment.

7.3.2.5 Changes in the Combined Group over the Six Month Treatment Follow-up.

A series of one way repeated measures ANOVA's revealed a significant effect for time on all of the dependent variables, with the exception of one cognitive outcome measure (the word reading of the SCWT). An investigation of effect sizes for these changes over time were found to be in the range of small to large effects, with an average moderate effect size of .55 overall (ranging from .29 to .80) (see Table 7.8). More specifically, larger average effect sizes were found on the two behavioural variables (.65) in comparison to the cognitive outcomes (.42).

Post-hoc comparisons, with Bonferroni corrections, found significant differences between performances at the initial baseline at time one compared to the three month assessment scores, with superior performances observed after three months of combined medication and behaviour therapy on nine of the dependent variables (see Table 7.8). Furthermore, pairwise analyses comparing time one and time three scores revealed significantly superior scores at the six month assessment on fourteen of the fifteen

outcome measures compared to initial baseline functioning, with all measurements at the end of six months of continued combined treatment being significantly better than those produced at time one.

More importantly, comparisons of time two and three scores revealed that more than half of the dependent variables continued to show significant improvements from the second assessment at three months to the final assessment at six months. Specifically, pairwise comparisons revealed performances on nine dependent variables to be significantly superior at the third evaluation point in comparison to the second assessment time. This suggests that improvements were not only observed after three months of continued combined therapy, but that this group continued to demonstrate significant improvements in cognition and behaviour over the six months of treatment.

Table 7.7: One way within group ANOVA: Comparison of the performance of the Medication Alone group over the three assessment time points.

Measure	Baseline	3 months	6 months			
	Mean (SD)	Mean (SD)	Mean (SD)	*p*	Partial Eta^2	Pairwise Comparison
WMI	91.7 (10.6)	102.5 (7.5)	101.6 (11.2)	.000**	.56	Time 2 & 3>Time 1
Trails B Time	73.3 (16.7)	54.8 (14.3)	42.9 (12.7)	.000**	.70	Time 2 &3 > Time 1; Time 3> Time 2
COWAT (Total)	19.66 (7.1)	21.00 (6.5)	20.83 (6.6)	.607	.04	
CAFT (Automatic)	15.17 (4.1)	14.91 (3.1)	14.42 (3.2)	.615	.04	
CAFT (Size)	7.17 (1.6)	7.50 (1.4)	7.75 (1.9)	.586	.05	
CAFT (Alphabet)	6.58 (1.8)	7.67 (1.6)	8.08 (2.1)	.006**	.38	Time 2 & 3 > Time 1
CAFT (RD)	50.92 (12.0)	48.01 (12.9)	45.57 (13.7)	.391	.08	
SCWT (Word)	54.75 (11.7)	58.67 (9.4)	62.83 (8.6)	.000**	.63	Time 2 &3 > Time 1; Time 3> Time 2
SCWT (Color)	41.83 (6.3)	46.42 (6.4)	48.42 (6.9)	.000**	.71	Time 2 & 3 > Time 1
SCWT (C/W)	18.75 (5.6)	24.17 (3.6)	27.75 (4.2)	.000**	.71	Time 2 &3 > Time 1; Time 3> Time 2
SCWT (Interference)	-23.08 (5.6)	-22.25 (5.7)	-20.75 (5.6)	.181	.15	
CPT (Comm. Errors)	13.92 (6.5)	7.17 (3.8)	8.00 (4.8)	.010*	.42	Time 2 & 3 > Time 1
CPT (Omiss. Errors)	17.00 (4.8)	8.08 (3.8)	7.33 (3.2)	.000**	.68	Time 2 & 3 > Time 1
BRIEF (Inhibit)	23.83 (4.4)	17.08 (3.6)	19.06 (3.3)	.000**	.60	Time 2 & 3 > Time 1
BRIEF (WM)	25.00 (2.5)	18.67 (3.1)	20.58 (4.7)	.001**	.52	Time 2 & 3 > Time 1

*$p < .05$; **$p < .01$

Table 7.8: One way within group ANOVA: Comparison of the performance of the Combined group over the three assessment time points.

Measure	Baseline	3 months	6 months			
	Mean (SD)	Mean (SD)	Mean (SD)	*p*	Partial Eta^2	Pairwise Comparison
WMI	87.00 (8.5)	100.7 (8.7)	102.33 (6.6)	.000**	.68	Time 2 & 3 >Time 1
Trails B Time	86.33 (45.8)	62.25 (27.6)	53.58 (11.4)	.000**	.33	Time 3 > Time 1
COWAT (Total)	15.67 (7.9)	21.58 (9.6)	23.58 (8.6)	.000**	.76	Time 2 & 3 > Time 1
CAFT (Automatic)	12.33 (5.1)	16.25 (4.9)	16.08 (4.0)	.000**	.62	Time 2 & 3 > Time 1
CAFT (Size)	5.66 (3.3)	7.17 (2.6)	9.42 (2.7)	.000**	.67	Time 3 > Time 1 & 2
CAFT (Alphabet)	5.00 (2.7)	6.92 (2.2)	8.08 (2.8)	.002**	.47	Time 2 & 3 > Time 1
CAFT (RD)	58.07 (19.1)	54.97 (11.4)	41.09 (9.7)	.025*	.31	Time 3 > Time 1 & 2
SCWT (Word)	50.25 (10.1)	55.33 (14.6)	57.33 (14.2)	.072	.25	
SCWT (Color)	41.58 (8.6)	45.58 (8.3)	47.17 (7.3)	.000**	.64	Time 2 & 3 > Time 1
SCWT (C/W)	16.00 (15.2)	25.92 (6.0)	29.58 (6.2)	.024*	.37	Time 3 > Time 1 & 2
SCWT (Interference)	-21.50 (5.2)	-19.66 (8.0)	-17.58 (6.0)	.037*	.29	Time 3 > Time 1 & 2
CPT (Comm. Errors)	12.08 (3.5)	7.58 (3.5)	4.50 (2.1)	.001**	.60	Time 2 &3 > Time 1; Time 3> Time 2
CPT (Omiss. Errors)	21.92 (7.9)	11.33 (5.4)	5.67 (3.4)	.000**	.72	Time 2 &3 > Time 1; Time 3> Time 2
BRIEF (Inhibit)	27.25 (2.7)	19.00 (4.0)	17.17 (3.4)	.000**	.80	Time 2 &3 > Time 1; Time 3> Time 2
BRIEF (WM)	25.92 (3.0)	18.25 (4.3)	16.83 (3.7)	.000**	.80	Time 2 &3 > Time 1; Time 3> Time 2

*$p < .05$; **$p < .01$

7.3.2.6 Comparison of the Medication Alone Group and Combined Group over the Six Month Treatment Follow-up.

The effects of treatment group were investigated separately for each of the dependent variables by utilizing a mixed between-within design ANOVA with time (three levels) as the within groups factor and the two treatment conditions as the between groups variable. All effects were considered significant at the alpha .01 level, however those with significance levels below .05 are also reported.

Significant interaction effects for time and group were found on six of the dependent variables. A significant interaction effect between group and index scores on the BRIEF were found, indicating that scores differed on the two behavioural indices of this parent reported measure between the two treatment groups. A significant effect existed for the inhibit index [$F(1.5, 34) = 9.49$, $p < .001$] at the critical .01 alpha level, which remained significant even after controlling for pre-treatment baseline scores [$F(1.5, 34) = 8.68$, $p < .007$]. The working memory index [$F(2, 44) = 4.02$, $p < .025$] achieved significance at the .05 alpha level. An inspection of the graphical representation of these data (see graphs 7.3 and 7.4) revealed that although both groups demonstrated improvements over time on these variables, the combined treatment group exhibited more pronounced improvements over the six months with a linear trend being apparent in this group, whilst a distinct quadratic trend was identified in the medication alone group (note that a lower score on the BRIEF represents better functioning). The strength of the treatment effect for the inhibit index of the BRIEF was moderate (.47), whilst a smaller magnitude of difference between the two groups was noted for the working memory index of the BRIEF (.15).

Further to the significant interaction effects found on the measure of behavioural outcomes, significant interaction effects between time and group were also identified on four of the cognitive variables. Specifically, scores significantly differed between the medication alone group and the combined treatment group on three measures of verbal fluency, with the CAFT automatic condition [$F(2, 44) = 10.19$, $p < .000$], the CAFT size

condition, $F(2, 44) = 8.41$, $p < .001$, and the COWAT total number of words produced, $F(2, 44) = 6.79$, $p < .003$, all achieving significance at the .01 alpha level. Examination of mean values between the two treatment groups in conjunction with an inspection of the data presented graphically revealed that analogous to the behavioural indices, these three cognitive measures showed significantly greater improvements over time in the combined treatment group in comparison to the medication alone group (see graphs 7.5, 7.6 and 7.7). Again, despite both groups evidencing significant improvements over time, the combined group exhibited more marked improvements over time, with clear significant linear trends being observed across all measures. Whilst the medication alone group also demonstrated linear trends in the data for the CAFT size condition and the COWAT, a significant quadratic trend was found for the CAFT automatic condition. The effect sizes for all three dependent variables was in the small range (CAFT automatic condition = .32, CAFT size condition = .28; COWAT .24)

The final measure demonstrating a significant interaction effect between time and group was CPT commission errors, $F(1.5, 33) = 3.23$, $p < .007$, which remained significant even after co-varying for differences in pre-treatment baseline scores, $F(1.5, 33) = 3.19$, $p < .010$. In parallel with the previously mentioned findings, a significant difference was found between the medication alone group and the combined treatment group in the number of commission errors committed on the CPT, with the combined treatment group again showing more pronounced improvements over the six month treatment follow-up. A significant linear trend was found for the combined treatment group, suggesting continued significant improvements over the six months of continued treatment with a combined therapy approach. The magnitude of this effect was in the small range (.38).

No significant between subjects main effect was observed across any of the other cognitive or behavioural dependent variables, suggesting overall that scores were not significantly different for the medication alone and combined therapy groups after six months of continued treatment.

Figure 7.3 A Comparison of the Performance of the Medication Alone Group and Combined Treatment Group over time on the BRIEF Inhibit Index.

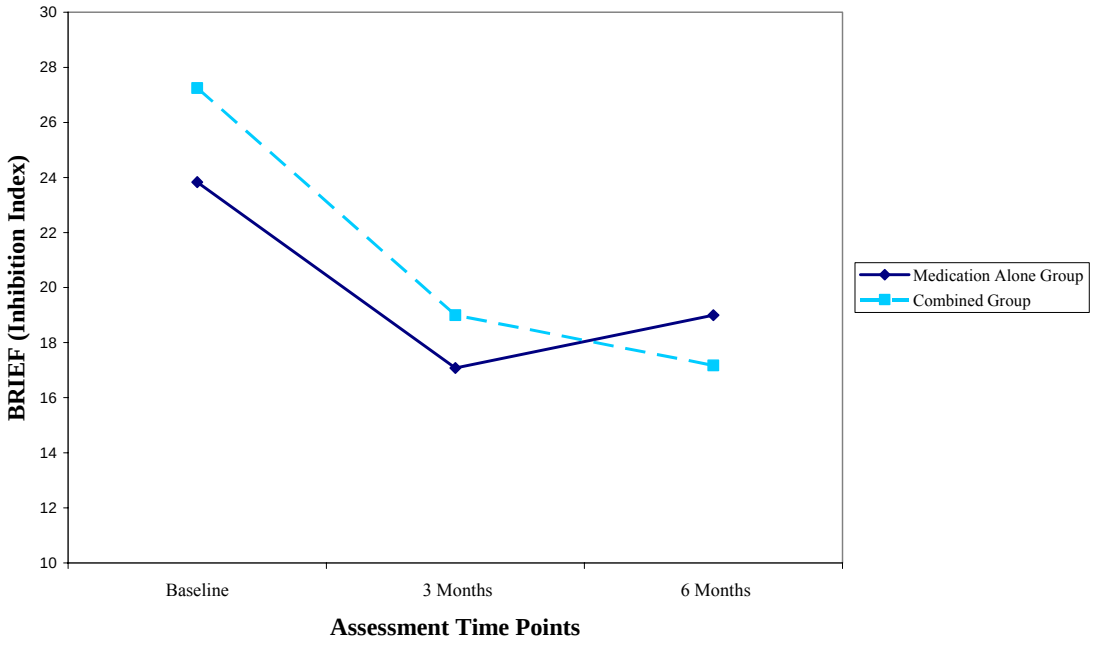


Figure 7.4. A Comparison of the Performance of the Medication Alone Group and Combined Treatment Group over time on the BRIEF Working Memory Index.

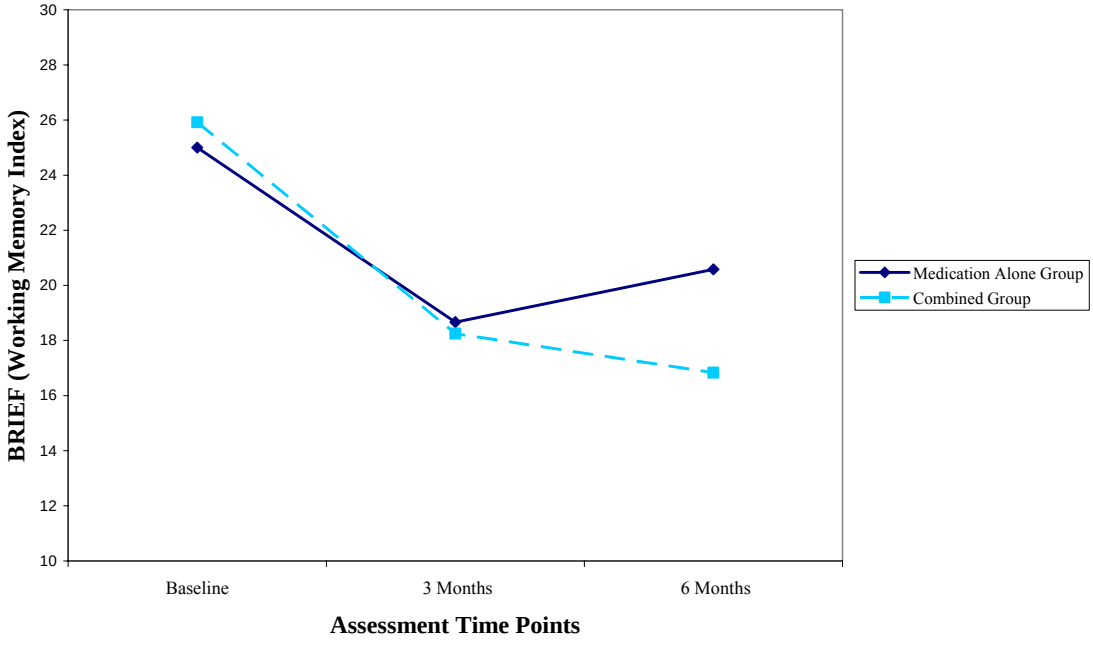


Figure 7.5. A Comparison of the Performance of the Medication Alone Group and Combined Treatment Group over time on the CAFT Automatic Condition.

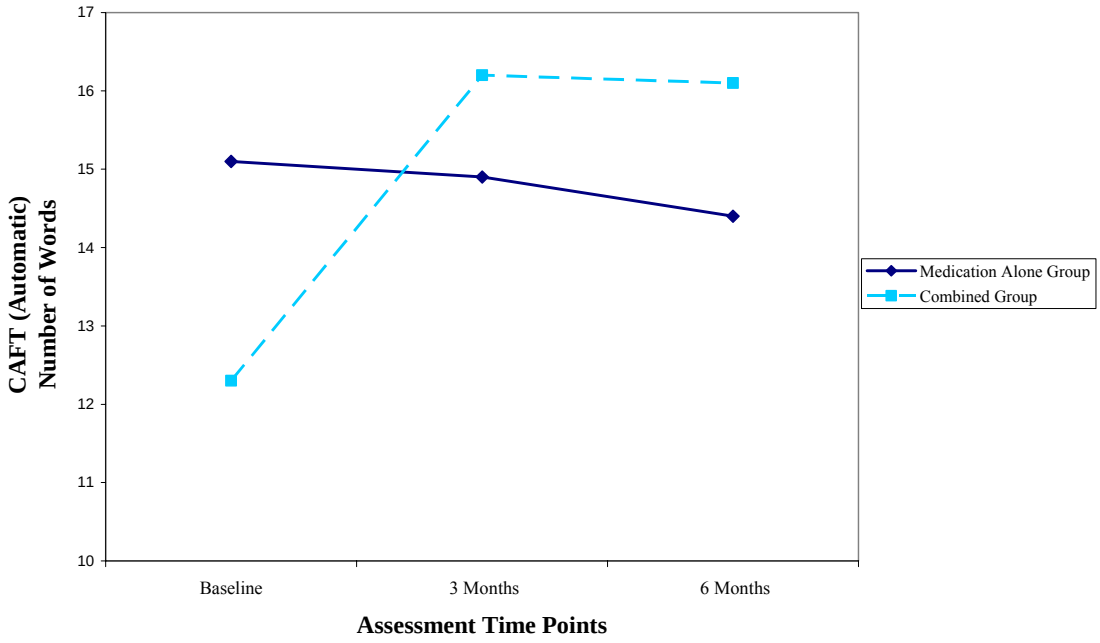


Figure 7.6. A Comparison of the Performance of the Medication Alone Group and Combined Treatment Group over time on the CAFT Size Condition.

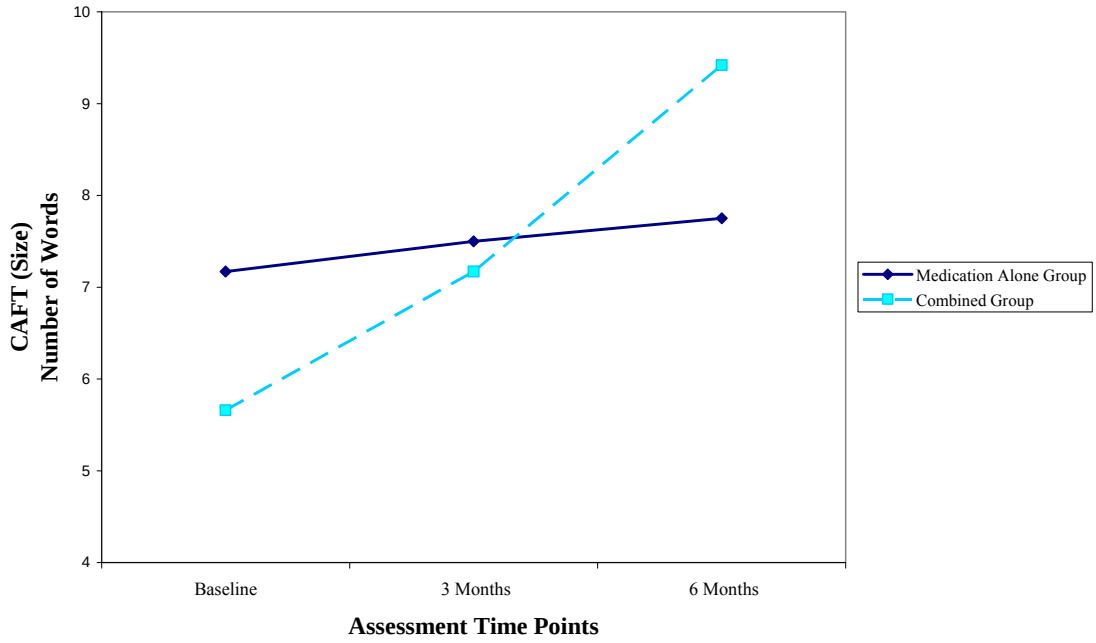


Figure 7.7. A Comparison of the Performance of the Medication Alone Group and Combined Treatment Group over time on the COWAT.

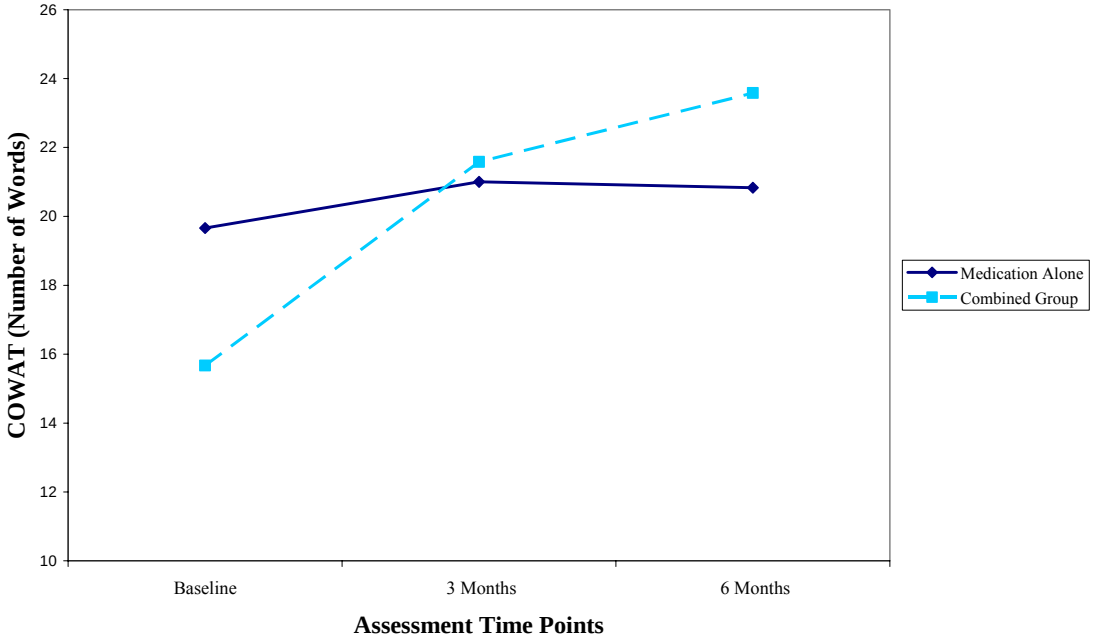
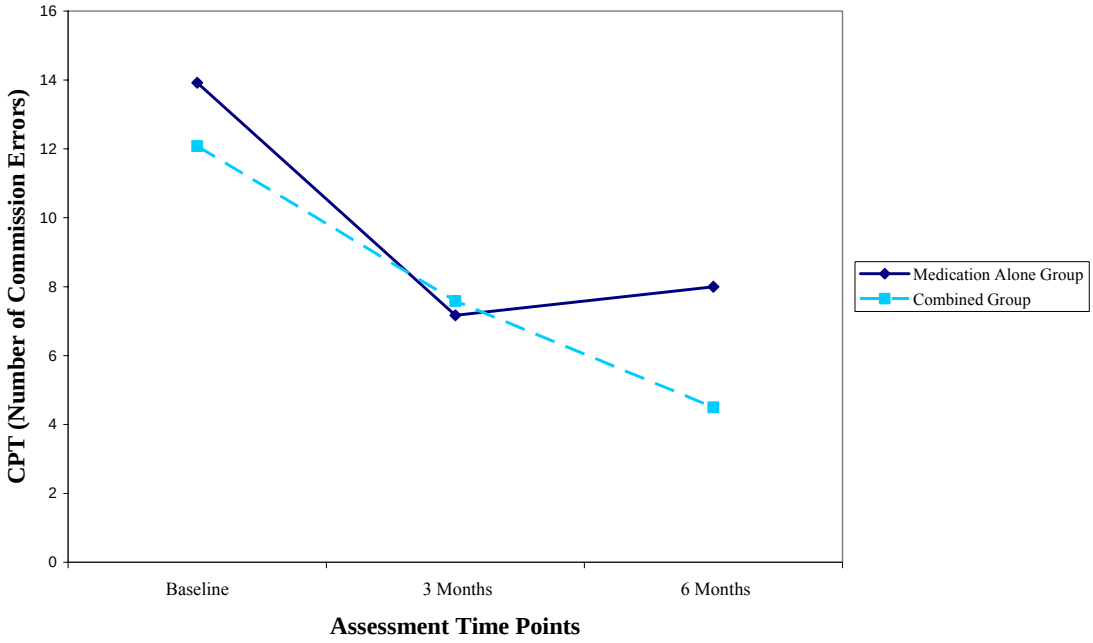


Figure 7.8. A Comparison of the Performance of the Medication Alone Group and Combined Treatment Group over time on the CPT (Commission Errors).



7.3.3 Hypothesis Three:

Finally, it was hypothesized that there would be a relationship between cognitive and behavioural measures of executive functioning. More specifically, it was expected that the results of the executive functioning cognitive measures would strongly correlate with the results of the BRIEF on the same domains.

Overall correlations were generally in the range of small to medium between the BRIEF subscales and the neuropsychological test measures, suggestive of weak relationships between the cognitive and behavioural variables overall. The exception to this was the relationship between the BRIEF Working Memory scale and the WISC-IV Working Memory index which produced a statistically significant correlation, suggesting quite a strong negative relationship between these two measures of working memory (r = -.501, n = 27, p < .01). Specifically, better performances of the WISC-IV working memory index were associated with lower parent reported working memory deficits. The CAFT Relative Difficulty Score, was also found to be moderately, but non-significantly, correlated with the BRIEF Working Memory scale, (r = -.361, n = 27, p <.10). Besides these two tests, the BRIEF Working Memory index did not produce strong correlations with any other standardized cognitive measure of inhibition.

The parent reported BRIEF inhibit scale produced moderate negative correlations with three of the neuropsychological test variables, only one of which was statistically significant at the .05 alpha level. The BRIEF inhibit index and the CAFT alphabet condition were moderately associated, with better performances on the demanding alphabet trial on the CAFT being correlated with parent's rating of lower behavioural symptoms of inhibition (r = -.386, n = 27, p <.05). The BRIEF inhibit index also produced marginal negative correlations with the number of CPT omission errors and on the automatic condition on the CAFT, however these failed to achieve significance at the .05 alpha level. Marginal and statistically significant correlations between the BRIEF indices and the neuropsychological test measures are presented in table 7.9. After using Bonferroni corrections for multiple comparisons by applying a more stringent alpha level of significance, no significant correlations remained, with the exception of the

relationship between the BRIEF working memory index and the WISC-IV Working Memory Index. None of the other cognitive variables were significantly correlated with the parent ratings of behaviour relating to inhibition or working memory.

Table 7.9: Correlations between parent ratings on the BRIEF Inhibit and Working Memory Scales and Cognitive Test Performance (only those achieving significance)

Measure	BRIEF Inhibit		BRIEF WM	
	r	sig.	r	sig.
CPT (Omission Errors)	-.338	.085*	-.157	.434
CAFT (Automatic)	-.337	.053*	-.189	.346
CAFT (Alphabet)	-.386	.047**	-.146	.416
CAFT (Relative Difficulty)	-.061	.763	-.361	.064*
WISC-IV WM Comp. Score	-.257	.196	-.501	.008**

* $p < .05$, ** $p < 01$

Partial correlations (controlling for FSIQ and WRAT-3 Reading and Arithmetic skills) were calculated between parent's ratings on the BRIEF and on the correlated cognitive measures. An inspection of the zero order correlation (r = -.528) indicated that controlling for intelligence (FSIQ) had very little effect on the strength of the relationship between parent's reports of working memory on the BRIEF and on the WISC-IV working memory scale. Controlling for intelligence also had little effect on the strength of the relationship between the BRIEF indices and the remaining correlated cognitive variables. Similarly, partialling out the effects of academic ability (reading and arithmetic) did not effect the strength of the significant correlations, with the exception of the relationship between the BRIEF inhibit scale and the CAFT alphabet, which became non-significant at the level of .05 when controlling for reading ability (r = -.385).

Chapter 8: Discussion

8.1 Neuropsychological Conceptualisation of ADHD

The first aim of the current study was to test the hypothesis of executive dysfunction in children with diagnosed ADHD. In line with initial expectations, and consistent with the growing research literature, the present findings confirm previously published reports of impairments in executive functions in children with ADHD compared to normative data. In line with predictions based on Barkley's unifying theory of ADHD, statistically significant group differences were found at baseline (prior to the introduction of treatment) between the 27 school age children with ADHD and age matched normative data across a range of developmentally appropriate cognitive and behavioural measures of executive functioning, namely inhibition. Specifically, baseline cognitive performances on executive tasks measuring inhibition, interference control and working memory were found to be significantly poorer in the ADHD group compared to each test's age matched normative data. In comparison to the norms, the sample of ADHD children committed significantly higher numbers of inhibitory (commission) errors and inattention (omission) errors on a measure of sustained attention. Furthermore, overall group findings suggested that the ADHD children were less efficient at inhibiting over learned responses on a measure of interference control, were more impaired in their working memory ability in comparison to the normative data, and were less competent at generating complex lists of semantically related words on a verbal fluency task.

In addition, parental reports indicated considerably higher levels of problem behaviours relating to executive functioning in the children with ADHD compared to the normative data, including impaired working memory ability, such as difficulty finishing tasks and forgetfulness, and poor inhibition skills, including interrupting others and engaging in distracting activities at inappropriate times. However, contrary to our initial hypothesis, there was limited evidence to suggest the presence of impairments in other domains of performance based executive functioning, namely phonemic fluency, aspects of semantic fluency and attentional set shifting.

8.1.1 Evidence for Executive Dysfunction

Findings of executive dysfunction in children with ADHD in the present study provides further evidence in support of the numerous existing studies that have previously reported deficits in these higher cognitive functions in ADHD children. In particular, the current data replicate findings from recent empirical studies and large scale meta-analyses demonstrating significant group differences between samples of ADHD children and normal controls (Muir-Broaddus et al., 2002; Pennington & Ozonoff, 1996; Seidman et al., 1997b; Shallice et al., 2002; Willcutt et al., 2005). Furthermore, informal comparisons of effect sizes between the present study and those from comparable recent investigations reveal similarly moderate effect sizes overall across a range of cognitive measures, demonstrating further support for executive function impairments in children with ADHD compared to normal controls (Willcutt et al., 2005).

The group data presented here also provide supportive evidence for Barkley's (1997a) conceptualization of ADHD as a disorder in a key aspect of executive functioning, namely inhibition. In line with Barkley's model and consistent with the growing body of recent literature, the present study established the presence of significant weaknesses on inhibition measures in the sample of children with ADHD (Barkley, 1997b; Berwid et al., 2005; Schachar & Logan, 1990; Seidman et al., 1997b; Shallice et al., 2002; Stins et al., 2005). Poorer performances by the ADHD children were found to be present across a range of executive function tests purported to be sensitive to inhibitory impairments and as such provides considerable support for a central inhibition deficit in ADHD.

In replication of previous studies, significant differences were found between the children with ADHD and the published normative data on the SCWT, with the ADHD children exhibiting significantly poorer performances on the two primary measures of inhibition on this test. Compared to the norms, the children with ADHD exhibited more difficulty on the color/word and interference conditions, impairments that have reliably been associated with deficient inhibitory processes and impaired interference control (Barkley et al., 1992; Berlin et al., 2004; Pennington & Ozonoff, 1996). Similar to a

report by Nigg and colleagues (2005), the present study revealed a small to moderate effect size between the ADHD children and norms on these two inhibition related SCWT conditions. Furthermore, these impairments were evident in the presence of age expected performances on the non-executive word reading and color naming trials, a finding which would appear to contradict the emerging line of evidence in the literature suggesting that generalised deficits in speeded processing, not inhibition, underlie the observed impairments in ADHD (Homack & Riccio, 2004; Nigg, 2001; Pocklington & Mayberry, 2006; van Mourik et al., 2005).

Consistent with previous findings, the current study also provided considerable evidence to support the CPT as a sensitive measure of inhibition in children with ADHD. The present study identified significantly elevated rates of commission errors and omission errors compared to published norms, reflecting the presence of significant impairments in aspects of both inhibition and attention in the sample of ADHD children. These significant group findings were associated with impressive effect sizes, which were particularly large for commission errors, thereby providing strong support for inhibition deficits in the sample of children with ADHD. This tends to reflect the similar significant group findings in the broader published literature, and provides further evidence against those few studies in which commission errors were reported as being absent (Douglas, 1999; Mariani & Barkley, 1997; Preston et al., 2005; Seidman et al., 1997a).

Impairment in working memory in the children with ADHD was also a robust finding in the present study, with the ADHD children performing significantly below the age matched normative data on the WISC-IV Working Memory Index. Effect sizes were considered to be in the moderate range, suggesting the presence of reliable differences between the ADHD sample and the norms. Despite the centrality of working memory to Barkley's theoretical model of ADHD, there currently exists limited research dedicated to elucidating the role of this executive function in ADHD. Unfortunately much of the limited published literature on working memory is compromised by a restricted range of working memory measures (usually non-verbal tasks) and design limitations (including samples with significant co-morbidity and children without confirmed diagnoses of

ADHD). Perhaps unsurprisingly, these studies have produced a range of inconsistent findings, including an absence of working memory deficits in ADHD in some instances (Kuntsi et al., 2001; Roodenrys et al., 2001; Scheres et al., 2004).

However, recent studies with more well defined samples and with those employing verbal measures of working memory have reported more convincing results, finding significant differences between their ADHD samples and normal controls (Berlin et al., 2004; Shallice et al., 2002). Along with these well controlled studies, findings from the present study also provide support for the presence of a working memory deficit in ADHD.

There was a failure in the present study to find significant group differences between the ADHD children and norms on the TMT – Part B. This is somewhat surprising given this test's well known demands on executive function processes, however this null finding is not entirely unprecedented. Although evidence does exist in the literature to support impaired performances on this measure, previous studies have suggested that executive function impairments such as set shifting ability and planning skills are often less robust than other executive function deficits, namely inhibition, in ADHD (Pennington & Ozonoff, 1996). Although the TMT- Part B is a recognised measure of inhibition, as with many tests of executive functions, normal performance depends not only on one cognitive process, rather they rely on an interplay of a variety of cognitive functions. One possible explanation for the non-significant finding on this test therefore is that the TMT- Part B might reasonably place more demands on other executive processes, such as set shifting and planning ability, than on inhibition. An alternative explanation might be related to the variable used to measure performance on the TMT- Part B. In the present study the conventional total time score was utilised as the primary measure, as this was thought to be an all inclusive score, taking into account number of errors made and processing speed. In hindsight however, perhaps the number of errors made might have been a more informative measure of inhibition, a finding which has been confirmed in at least one other study (Sergeant et al., 2002).

The current study also found no impairment in phonemic fluency in the ADHD children when compared to the normative data, with modest evidence in support of semantic fluency deficits. Unfortunately, comparisons between the present study's results and those of similar published studies are somewhat limited due to the paucity of studies employing measures of verbal fluency in children with ADHD, however the non-significant findings of the present study are not unparalleled. Although some studies have reported differences between normal controls and ADHD children on measures of verbal fluency, these findings tend to be present in studies with significantly larger sample sizes than those achieved in the present study (Grodzinsky & Diamond, 1992), suggesting that limited sample sizes, the present study included, may lack power sufficient enough to detect any significant effects on verbal fluency measures. This might also imply that verbal fluency measures may not be robust or sensitive enough to detect deficits in ADHD. Specifically, similar to the TMT- Part B, verbal fluency tasks might place more demands on executive processes other than inhibition, or may even rely on non-executive processes, such as vocabulary. Moreover, standard measures of verbal fluency may not be sufficiently challenging to detect difficulties in ADHD children. These explanations could be interpreted as supporting the current pattern of findings. Although the present study failed to demonstrate significant group differences between the children with ADHD and norms on the simple word generation tasks (COWAT and automatic condition on the CAFT), as a group, the children with ADHD were significantly worse than the normative data on the more difficult conditions on the CAFT (size and alphabet conditions), both of which could reasonably be characterised as being more challenging and more demanding on inhibition than the two simpler conditions.

Overall, group data from the present study appear to confirm predictions from the initial hypothesis, by providing strong supportive evidence for impairments in aspects of executive functioning in children with ADHD compared to norms. In addition, consistent with Barkley's prevailing theory of ADHD, the ADHD children performed significantly poorer than the normative data on a range of executive function measures sensitive to inhibition. However, this was not a consistent finding, with unimpaired performances being documented across a few of the executive function tests. The interpretation of the

current pattern of findings suggested that only those executive function tests demanding enough on normal inhibitory control processes are likely to produce significant discrepancies in performances between children with ADHD and norms. Given that executive functioning is a multi-dimensional construct, with executive function tests generally requiring a combination of both executive and non-executive cognitive processes, on closer inspection of some of the measures used in the present study, it is perhaps not so surprising that we failed to find inhibitory deficits across all measures. Another difficulty in this area is that there is arguably not one "gold standard" measure of inhibition. As observed by the multiple dependent measures employed in the current study, it is difficult to find one measure that purely assesses inhibitory processes. Based on the current study, the CPT commission errors, SCWT color/word and interference conditions, the working memory index from the WISC-IV and the size and alphabet conditions on the CAFT appear to be sensitive measures in distinguishing between ADHD children and norms. Development and refinement of specific tests of inhibition are clearly needed, whilst replication of these results in larger samples would further help in the characterisation of the specific cognitive profile of children with ADHD.

The pattern of executive dysfunction displayed by the ADHD children in the current study provides support for the notion that there are key deficits in pre-frontally mediated aspects of inhibition. Accordingly, these findings provide indirect evidence to support the assertion that there are underlying dysfunctions in the prefrontal and sub-cortical regions of the brain previously implicated in inhibition processes (Bradley & Golden, 2001; Castellanos et al., 1996; Himelstein et al., 2000; Solanto, 2002).

The finding of significant group differences and the notable effect sizes in the present study are certainly impressive, especially in light of our rather modest sample size. Unfortunately a larger sample size was not possible given the overall limited time frame of the current study. Furthermore, there were pervasive difficulties with recruitment consistent across the four paediatricians. This apparently was directly related to an untimely significant decline in the number of children being referred for ADHD diagnosis during the study's recruitment phase. In addition, the small participant

numbers also came as a result of the desire to recruit a relatively homogenous sample of ADHD children not confounded by any co-morbid disorders that might exacerbate existing ADHD-related deficits. Although it could be argued that this relatively homogenous sample of ADHD children might not be representative of the wider ADHD population, due to the obvious common co-morbidity of ADHD with other psychopathologies, the purpose of the current study was to elucidate the cognitive profile of ADHD only, not attempt to disentangle deficits exacerbated by other disorders.

Another important issue is the decision to compare the ADHD children against age matched normative data rather than employ the traditional research comparison group of normal control children. As mentioned previously, it was considered important to the study to recruit a relatively homogenous sample of ADHD children, therefore all available time and effort was put in to achieving that recruitment aim. Henceforth, it was not possible to recruit a similar sized and comparable control sample of children in the limited time frame for completion of this intervention study. Therefore, the age matched normative data accompanying each test was utilised as a suitable comparison in which to compare the ADHD children. Comparing clinical samples to normative data is not unprecedented in the literature and as evidenced in the current study, produces significant results in parallel to published studies using control children (Muir-Broaddus et al., 2002). Furthermore, a comparison with normative data is typically utilised in standard clinical practice to determine cognitive and behavioural strengths and weaknesses of children.

Although not the focus of the present study, a brief exploration of the pattern of development in the data seems warranted. Developmentally appropriate measures were utilized in the present study, however despite finding significant differences between the ADHD children and age matched normative data across most variables, it is unclear what involvement age played in contributing to the significant group differences. Given that Barkley (1997a) argues that inhibition deficits in ADHD might be due to a delay in developing inhibitory control, it is reasonable to suggest that the degree of variation between ADHD and norms may vary at different developmental stages, with increasing

age being associated with the development of more efficient inhibitory processes. This is important to bear in mind when interpreting the present results. The current samples' performances were combined over a relatively large age range (seven to twelve year olds), with each age group containing small and unequal numbers, with a definite skewness towards the younger age groups (ie. there were more seven and eight year olds than any other age group). With the developmental hypothesis in mind, it is plausible that the younger children in the study may have contributed most to the findings of significant differences between the ADHD group and the normative data. For this reason, future research should use smaller age ranges and larger sample numbers in prospective longitudinal designs in order to elucidate the developmental trajectories of executive function deficits in ADHD.

Although not the primary focus of addressing the study's first hypothesis, it would be remiss to ignore the considerable behavioural deficits revealed in the study. Findings of significant impairments in behaviour very obviously replicate those of repeatedly reported findings of behavioural problems in this clinical population and parallel research noting impairments as measured by both parent and teachers (Willcutt et al., 2005). Furthermore, the current study replicated previous research by revealing impressive effect sizes between the ADHD children and the normative data on the behavioural measure. With the theoretical importance of executive functions in mind, the present study also extended and refined knowledge of the behavioural deficits in ADHD by specifically assessing behaviours relating to executive functioning.

8.1.2 A New Direction?

Whilst the group comparisons present a relatively consistent pattern of executive dysfunction in the children with ADHD, these overall group results fail to enlighten us about whether these significant differences are clinically, as well as statistically, meaningful. In light of the significant group findings, one might expect that these findings would logically imply a significantly impaired level of functioning in the ADHD children. Somewhat surprisingly, however, this assumption was not found to be strongly

supported. In an analysis of the percentage of ADHD children actually performing in the impaired range (defined as performances at least 1.5 standard deviations below the normal population mean), it was found that the proportion of children classified as functioning at a clinically significant impaired level ranged widely from 30% to 81%. These data indicate that although significant whole group differences were found between the ADHD children and normative data, not all children were actually impaired in their executive function performance. More importantly, approximately 20% to 70% of children were not exhibiting any significant impairment on the executive measures used in the present study. Although these findings still provide empirical support for the presence of inhibition deficits in children with ADHD, the individual data do not establish conclusive evidence to confirm executive dysfunction, namely inhibitory deficits, as the primary cause for impairments in all children with ADHD, as postulated in Barkley's conceptualization of ADHD.

The most feasible explanation for this finding is likely to be related to the clinical heterogeneity of the cognitive profile of children with ADHD. Although inhibitory deficits are undoubtedly a key feature among many ADHD children, impairment in this single executive function domain appears unlikely to be able to solely account for the pervasive impairments manifested in this disorder in all children (Willcutt et al., 2005). Rather, it appears more plausible that the aetiology of ADHD symptomatology is multi-factorial and complex, with a range of impairments encompassing a largely heterogeneous cognitive profile. Whilst not completely discounting Barkley's notion of inhibition as an important component of ADHD, the idea of clinical heterogeneity is more congruous with Nigg and colleagues (2005) and Sonuga-Barke's (2005) more recent theoretical models. Specifically, they propose multiple-causal models in the aetiology of ADHD. In particular, it is hypothesized that whilst one primary cognitive deficit, namely inhibition, remains a likely fundamental feature of ADHD in some children, this single cause is unlikely to produce the range of deficits observed in *all* children with ADHD. The implication being that there exists alternative aetiological pathways that are likely to contribute to the manifestation of ADHD. Therefore, whilst an executive dysfunction causal pathway is still implicated in some cases of ADHD,

alternative causal models (such as motivational impairments) may also be influential in the development of ADHD in some children (Sonuga-Barke, 2005).

Although addressing issues of clinical heterogeneity and multiple casual pathway models was not the primary intention of the present study, the individual data collated from the current study, in particular the finding that not all children with ADHD have executive dysfunction does provide some preliminary evidence in support of a multiple pathways model of ADHD. Taken together, these findings underscore the importance of routinely reporting both group and individual data in future research. Further research is also needed to elucidate and characterise the proposed alternative aetiologies in ADHD in order to assist with diagnosis and treatment.

8.2 Efficacy of Treatments

The second aim of the present study was to evaluate the long term efficacy of two forms of treatment on measures of executive functioning in children with ADHD. To our knowledge, this is currently the first extended duration, prospective follow-up study to examine the comparative effects of medication alone with a multimodal design consisting of paediatrician prescribed medication and low intensity, individualized family centred behavioural therapy on both behavioural and cognitive based measures of executive functioning. Specifically, in contrast to previous multi-component intervention studies which have adopted approaches encompassing high intensity, complex group therapy programs, the present study utilized a less intensive, individualized and family centred design more comparable to interventions incorporated into standard clinical practice. Additionally, in comparison to the published studies to date which have employed only behavioural outcome measures, the current study examined the effects of the two intervention programs not only on parental ratings of behaviour relating to executive functioning, but also on performance based measures of cognition. This appears to be unprecedented in the literature to date.

Findings from the present study revealed significant beneficial therapeutic effects for both forms of treatment on behaviour and cognition in children with ADHD. Specifically, substantial reductions were documented in parental reports of problem behaviours relating to executive functioning, namely inhibition and working memory, whilst treatment with either medication alone or combined therapy also yielded beneficial effects across all of the cognitive based measures of executive functioning over six months of continued treatment. The findings from the present study complement the extensive previous empirical literature demonstrating the effectiveness of treatment on behavioural outcomes, along with extending current knowledge on the positive effects of treatments on cognition.

Specifically, the present study documented statistically significant improvements on the two parent reported behavioural measures across the six months of continued treatment, a finding which was also strengthened by the impressive moderate to large effect sizes. This improvement in behaviour attributed to treatment, parallels findings from numerous other short term studies whilst it also extends findings from long term intervention studies. Most similar studies have reported large effect sizes for treatment changes on primary ADHD symptoms, such as the core behavioural features of hyperactivity, inattention and impulsivity, and for behavioural difficulties usually associated with the disorder, including social skills, self esteem, oppositional defiant behaviour and internalizing symptoms for treatment in general (Dopfner et al., 2004; Klein & Abikoff, 1997; Kavale et al., 1999; Pelham et al., 1998; Purdie et al., 2002; Schachter et al., 2001). To date however no comparable study exists measuring either short or long improvements on the specific construct of executive functioning related behaviour, a significant finding of improved behaviour which is unique to the current study.

Consistent with previous research, considerable improvements were also found with treatment across all of the performance based cognitive measures, including those sensitive to inhibition, interference control, working memory, verbal fluency and attention. Six months of treatment revealed remarkable post-test improvements, with an

overall average effect size in the moderate range for the cognitive measures of executive functioning when examining both forms of treatment collectively. Although positive effects of treatment have been repeatedly demonstrated in children with ADHD on behavioural outcomes, short and long term treatment effects on cognition are currently under studied in the literature and as a consequence efficacy of treatments are less well established (Bedard et al., 2002; Mehta et al., 2004; Riccio & Reynolds, 2001; Tucha et al., 2006). This may be in part due to the fact that only recently have theoretical models of ADHD illuminated the crucial role of cognition and more specifically, executive dysfunction, in ADHD. Prior to the last decade, the majority of research on ADHD had been dedicated to exploring and understanding the behavioural manifestations of the disorder, which is reflected in the vast amount of literature investigating this area of ADHD. Contemporary work, however, has shifted its focus with the emergence of theoretical models embedded in a cognitive framework. As such, there is an emerging body of literature devoted to studying the effects of intervention on cognition. Certainly, however, further research will need to be dedicated to investigating the long term effects of treatments on both the core cognitive and behavioural features of ADHD.

Analysis at the individual level revealed that these statistically significant group improvements with treatment importantly translated into clinically meaningful findings, in that the vast majority of children with ADHD achieved a normal level of behavioural and cognitive functioning following six months of treatment. In fact, the rates of normalization attained in the present study were extremely impressive. Pre-test levels of impairment (performances at least 1.5 *SD* below the mean) ranged between 30% and 81% across both cognitive and behavioural measures. On average, approximately half of the children were functioning at an impaired level on cognitive measures of executive functioning prior to the introduction of treatment, whilst almost three quarters of children were in the impaired range according to parental reports of behaviour. Incredibly, rates of impairment declined dramatically following six months of either medication alone or combined therapy, with almost complete normalization observed across the multiple cognitive measures of executive functioning and behaviour in children who were previously impaired. At least 87% of children were functioning in the normal range

across all of the outcome measures following treatment, with that figure rising to around 96% on the majority of the measures. Amazingly, none of the children remained in the impaired range on the number of commission errors made on the CPT, with 100% of the ADHD children functioning in the normal range. These results indicate that a very high percentage of children not only evidence significant improvements following treatment, but can also be normalized following six months of either medication alone treatment or multimodal therapy.

8.2.1 A Promising Outlook

Although it is probably premature to arrive at implications about the benefits of therapeutic intervention on the basis of the preliminary findings from the present study, especially given the modest sample size, these individual findings of high rates of normalisation of both behaviour and cognition in the present study are nonetheless extremely promising and very exciting. If these findings are replicated in larger samples in future research, these statistically and clinically significant improvements are likely to have significant implications for children with ADHD and have the potential to bring about dramatic differences in their day to day lives, in terms of improving their academic, psychosocial and emotional functioning. Further to these more immediate benefits, it is tempting to speculate that these significant improvements might also lead to a brighter long term prognosis. Given that there are well established serious long term negative consequences associated with the persistence of ADHD into adolescence and adulthood, including poor vocational outcomes, psychosocial difficulties and increased risk of mental health problems, it would be extremely important for future research to examine the relationship between the impact of treatment of ADHD in childhood and the effect on long term prognosis. To achieve this, a large scale long term prospective follow-up study is required, with treatment follow-up of years rather than weeks or months, more in line with treatment duration typically observed in clinical practice. Although the present study extends treatment duration compared to many other studies, it would certainly be interesting to see whether the significant improvements identified after six months of

treatment persist over subsequent years of continued intervention and also whether this is associated with improved long-term prognosis.

Without doubt, the interesting reporting of individual findings, along with effect sizes in this and similar studies, must be recognized as being more clinically relevant and informative than presenting just group differences and significant *p* values. Moreover, aside from providing more clinically meaningful results, the percentage of children impaired and effect sizes also allow for greater comparison of treatment effects between different studies. Accordingly, future intervention studies should adopt the practice of routinely reporting the proportion of children who remain in the impaired range of functioning and those who achieve normalization, rather than continuing to report group data only.

The finding of significant improvements in the sample of ADHD children in the present study should be replicated and validated against a control sample of normal children. A potential weakness of the current study is the absence of a matched control group of normal children with which to compare the performances of ADHD children over the course of the treatment. Ideally, a matched control sample of ADHD children receiving no treatment would be the most appropriate comparison group, however due to obvious ethical reasons this was not a viable option. Without having a control group, it is difficult to conclude with any degree of certainty that the significant improvements observed in the current ADHD group were directly attributable to treatment and not related to potential confounding effects such as practice or development. Although not ruling them out, it does however appear unlikely that such significant improvements could be attributed to practice or time effects. In particular, it would be unexpected that such high levels of normalisation of both behaviour and cognition would be the result of practice or maturational effects. The most likely explanation would appear to relate to the positive therapeutic effects of treatment.

8.2.2 Evidence for the efficacy of two forms of treatment for ADHD.

In addition to reporting treatment effects for the two treatment groups collectively, a separate analysis of the medication alone group and the combined treatment group was also performed. Medication alone treatment was found to be effective in reducing parent reported behavioural problems whilst it also significantly enhanced various aspects of cognition over the six month follow-up period, with these improvements being associated with moderate to large effect sizes. However, the greatest benefits of medication alone treatment were observed in the ADHD children over the first three months of this intervention. An examination of the mean differences in the data from three months to six months revealed a trend for a plateau in performances across many of the cognitive and behavioural measures with even a slight non-significant decline in some performances after the first three months of medication alone treatment.

The findings of improved behavioural and cognitive functioning after three months of medication alone treatment is consistent with the myriad of short term studies demonstrating the acute amelioration of problem behaviours and executive function deficits in children with ADHD. The short term benefits of psychostimulant medication are well established in the intervention literature, with a vast array of studies confirming improvements on both behaviour and cognition over days to weeks of medication treatment (Barkley, 1990; Kempton et al., 1999; Klorman et al., 1991; Losier et al., 1996; Mehta et al., 2004; Pelham et al., 1998; Schachter et al., 2001; Tamm, 2001; Tucha et al., 2006). Where uniform treatment effects have not been found, methodological design flaws, including un-medicated children during treatment testing and no comparative baseline evaluations, are likely explanations for the lack of significant findings (Aggarwal & Lillystone, 2000; Vance et al., 2003; van der Meere et al., 1999).

The finding from the present study of marked improvements in the first few months of treatment, followed by minimal change does however contradict the pattern of findings in some of the recent longer term intervention studies, which have documented continued improvement over many months, even up to five years of psychostimulant treatment in

some instances (Charach et al., 2004; Epstein et al., 2006; Gillberg et al., 1997; Schachar et al., 1997). A recent paper by Ercan and colleagues (2005) however reported similar findings to the current study. Analogous to present findings, they found the greatest gains in ADHD symptoms were achieved in the first month of medication treatment, with only minimal further improvements observed over the following two evaluations up to six months. Aside from the Ercan and colleagues (2005) paper, there is a noticeable disparity between many of the previous extended duration intervention studies and the present study. In most comparable research, performances are typically only evaluated at two time points, with no consecutive follow-ups mid-point in the treatment phase. This failure to incorporate additional evaluations into long term intervention studies may conceal any trends over the treatment period that may exist. For instance, the present study too would show steady, continued improvements with medication alone treatment if only performances at six months were compared to baseline.

Based on the current results, it would be tempting to interpret this finding as support for medication alone treatment being primarily beneficial in the short term. This interpretation would be consistent with that of Schachter and colleagues (2001) who cautioned that medications only provide temporary reductions of ADHD symptoms. However, despite the observed trends in the data, it is important to note that performances of the ADHD children in the medication group were still better at six months than initial baseline. Furthermore, even though there was evidence for some slight reductions in functioning after the initial gains made at three months, these changes were not statistically significant. An absence of statistical significance therefore casts some doubt on the clinical significance of these trends.

Analogous to the medication alone group a similar range of improvements were observed in the combined treatment group over the first three months of treatment. Comparisons of the initial baseline performances with those at six months also revealed that almost all cognitive and behavioural measures showed strong statistically significant improvements for the combined treatment group. This was a very positive and robust finding, with effect sizes being in the moderate to large range overall. Moreover, further

comparative analyses found that the combined group showed continued improvements on all of the outcome variables from three months to six months, with well over half of the measures reaching statistical significance. These findings indicate that the combined group not only improved significantly over the first three months of multimodal treatment, they also continued to show improvements over the entire six month follow-up period. Furthermore, the results indicate that the combined treatment may be associated with a greater range of improvement than the medication alone group, with evidence of slightly greater effect sizes in the combined group and more significant improvements on a wider range of outcome measures.

There is a relative paucity of empirical research studies evaluating multimodal treatment approaches, making it difficult to contextualize the current findings. Preliminary conclusions from a handful of recent studies have however in general confirmed the positive therapeutic effects of a combined treatment approach in children with ADHD (Conners et al., 2001; Dopfner et al., 2004; MTA Cooperative Group, 1999; van der Ord et al., 2007). Nevertheless, although these studies demonstrate significantly improved performances at end point evaluations following combined therapy, procedural design issues in these studies, predominantly an absence of multiple consecutive treatment evaluations, make it impossible to explicitly compare trends in the data over time with the present study. Moreover, the only study that did evaluate four consecutive performances over six months attributed their initial improvements to the medication component of the combined therapy, not the behavioural program (Ercan et al., 2005).

The improvements over the first three months in the combined group closely parallel those observed in the medication alone group, indicating that at least some of the initial improvements may be attributed to the acute effects of medication. Medication alone however is unlikely to account for all of the documented improvements in the combined group. A divergence in the degree of change in the two treatment groups after three months of treatment, with a trend for continued significant improvements in the combined group indicates at least some additional positive therapeutic effects of the behavioural therapy above and beyond medication alone. This interpretation is

compelling when one considers the theoretical principles of behavioural therapy. In contrast to medication, which is assumed to be acute acting with improvements in behaviour and cognition effective immediately, behaviour therapy, by its very nature, is not typically associated with rapid, instant effects. Rather, behavioural therapy programs generally take a period of time to produce any noticeable change. Therefore, although speculative, it is reasonable to suggest that at least some of the improvements noted in the combined treatment group in the present study, especially those observed after the initial three months, can be attributed to the efficacy of behavioural strategies. Studies and indeed clinical interventions in standard practice in the future need to take this effect into account and ensure that follow-up periods are lengthy enough to capture the full effects of a combination therapy. Furthermore, multiple evaluations over extended treatment durations are also advocated for to document trends in changes over time.

8.2.3. Evidence for a Combined Treatment Approach

Direct statistical comparisons of the two treatment groups revealed a significant interaction effect on a number of the outcome measures, suggesting a superiority of one treatment over the other on at least some of the dependent variables. Although the two treatment groups did not significantly differ on the majority of outcome measures, the combined treatment group was found to have made larger and more persisting changes on four of the cognitive measures and the two behavioural indices in comparison to the medication alone group. Specifically, significant interaction effects were found for the combined group on three measures of verbal fluency, inhibition as measured by commission errors on the CPT and both behavioural indices on the BRIEF, with the magnitude of effect being in the small to moderate range (ranging from .15 to .47). Even though this was not a pervasive effect across all domains of functioning, these findings do suggest that the combined treatment was statistically superior to medication alone on at least some of the outcome measures over the extended six month follow-up treatment period. Due to their being no significant differences in demographic characteristics, medication status or measurements of functioning at baseline between the two treatment

groups, these superior improvements were attributed to the success of the combined treatment program.

The findings from the current study provide modest support for the superiority of long term family centred multimodal therapy over medication alone treatment. Although preliminary evidence in the published literature attests to the efficacy of a multi-component intervention program in improving ADHD related behaviour, current empirical research as yet has failed to confirm any advantage of combined therapy over treatment with medication alone (Dopfner et al., 2004; Ercan et al., 2005; MTA Cooperative Group, 1999; van der Ord et al., 2007). The result from the present study, therefore, is amongst the first to provide evidence for the additive effects of a combined therapy program over medication alone treatment on aspects of both behaviour and cognition relating to executive functioning. The only other recent studies with comparable promising findings are those from Conners et al., (2001) and Swanson et al., (2001). In their post-hoc analyses of the originally non-significant MTA, they showed that the combined treatment was significantly more effective in reducing ADHD associated behaviours than medical management, behavioural management and community care in both studies. Conners et al., (2001) reported the magnitude of effect between the combined treatment and the medical management conditions to be in the small range (.28), which is remarkably similar to the average effect size for both behavioural and cognitive measures in the present study (.31). To date, these are the only studies providing evidence supporting a superiority of combined therapy over medication alone treatment in ADHD.

The large scale MTA study is the most renowned and influential combined intervention study in the ADHD literature to date, even though their initial analyses failed to reveal any significant superiority of the combined treatment over the medical management. This is not an isolated finding, with more recent studies also reproducing similar non-significant findings (Dopfner et al., 2004; van der Ord et al., 2007). One possible explanation for the absence of significant additive effects of the combined treatment over medication alone may relate to the high dosages of psychostimulants and

intense levels of behavioural therapy adopted in these previous studies, particularly the MTA study. Specifically, the average dose of medication in the MTA study's medication management group was 38mg a day and 31mg a day in the combined therapy group (compared with an average of 18mg a day in both groups in the present study). Previous research has tended to suggest that there is little additive benefit in combining high doses of medication with high intensity behaviour therapy, with studies specifically demonstrating little or no added benefit of behaviour therapy when coupled with high doses of medications (Carlson et al., 1992; Pelham et al., 1993). This might provide some clues as to why there were no superiority effects for the combined treatment group in the MTA study.

Conversely, the treatment groups in the current study were both prescribed significantly lower doses of medication and were similarly involved in lower intensity behavioural therapy programs in comparison to the MTA study. Yet despite this, the present study still achieved significant results, with moderate to large treatment effect sizes overall and high rates of normalisation of behaviour and cognition, results quite comparable with the much more intensive and comprehensive MTA study. Furthermore, modest evidence emerged in the present study to support the superiority of the combined treatment group in improving some aspects of behaviour and cognition. Although this is certainly a preliminary finding requiring further replication in much larger samples, the statistically and clinically significant findings of the present study would be amongst the first to support a low intensity, family centred combined therapy approach as a beneficial and viable long term treatment of both behaviour and cognition in children with ADHD. This interpretation obviously does not disregard the impressive results of the MTA study, which clearly produced significant improvements over the 14 month follow up in both the combined and medication management groups. The present results merely suggest that significant improvements in behaviour and cognition in ADHD may also be achieved with a lower intensity, family centred and individualised multimodal approach. Similar effect sizes across the two studies suggests that the magnitude of effects of the combined treatment group were quite comparable, however studies directly comparing low and high intensity multimodal approaches will be needed to confirm this preliminary finding.

8.2.4. A Combined Treatment Approach: The Benefits.

A finding of beneficial combined low dose multimodal therapy has several important clinical implications. Side effects of stimulant medications are proposed to be related to dose, with higher doses producing significantly higher rates of associated side effects. It was reported that an alarmingly high 64% of children experienced acute side effects in the MTA study. Although purely speculative, previous research would provide some support for the contention that the ADHD children in the current study may have lower rates of side effects than studies prescribing higher doses of psychostimulants, including the MTA study (Pelham et al., 2005). Based on these findings, it should be recommended that future intervention studies in children with ADHD routinely gather information on side effects of psychostimulants. Further to acute side effects, the long term risks of prolonged high dose psychostimulant use are also relatively unknown at present. However new evidence emerging from the MTA study suggests that improvements associated with high doses of medications came at the cost of adverse effects to long term height and weight (MTA Cooperative Group, 2004). From a public health perspective, it would therefore appear sensible to promote the use of lower doses of medication in children with ADHD to minimise any potential short term and long term risks. Finally, anecdotal evidence would suggest that parents of ADHD children would also preferentially choose lower doses of psychostimulants for their child over the long term.

An interesting finding in the MTA study was the trend in their data showing that the combined group performed better than the other treatment groups on many of the outcome measures. Although these trends didn't reach statistical significance, the MTA Cooperative Group (1999) advised that lack of statistical significance should not be proof of equivalence of treatments nor should it indicate an absence of clinical significance. Put differently, although not consistently statistically superior, combined therapies have inherent advantages that make them clinically important treatments. For instance the MTA study reported that despite statistically being non-superior to medication alone

management, the parents in the combined treatment group reported significantly higher levels of satisfaction with the multimodal approach, with the implication being that parents felt happier with this mode of intervention. Parents reported that it helped them cope with the multitude of problems associated with ADHD, including problems that were perceived as not being improved with medication alone treatment, such as optimising family functioning. Also, an additive behavioural therapy component in a combined treatment approach can assist in controlling behaviour in the evenings when the effects of medication have perhaps dissipated and may be less effective. Finally, a combined therapy program might be more beneficial to those 30% of children with ADHD reportedly unresponsive to medication alone treatment. The inherent message here is that even if a combined therapy approach is not statistically superior or only modestly superior to medication management alone, it still produces significant improvements that may produce clinically significant changes.

Aside from the various benefits in combined therapy approaches overall, there are also several important aspects and advantages unique to the current study's multimodal approach which may have played an important role in producing significant and modestly superior improvements in the present study. In contrast to past research, the current behavioural program was markedly less intensive and demanding than those implemented previously. The benefits of this design were two fold. Firstly, a brief and simple behavioural therapy program is more ecologically valid, being achievable in the important context of standard clinical practice, not just being feasible in research. Secondly, this design is likely to be more appealing to families due to being minimally demanding on effort and time, in turn increasing adherence to the program. Furthermore, adherence to the behavioural program in the present study is likely strengthened by the individual nature of the treatment. Rather than attending numerous group sessions, each family received brief, but comprehensive, individualised psycho-education and guidance, with specific and relevant recommendations especially for their child. This was conducted within a family systems framework. Therefore not only were parents involved, but primary caregivers were also included to ensure consistency and workability within the family context.

Another unique aspect of the current research was the decision to allow parents some choice over the treatment group their child was assigned to. Although perhaps not ideal from a random assignment point of view, this clinically driven approach parallels the decision making process in clinical practice, where parents ultimately decide upon treatment for their child. Moreover, Barkley (2000) argues that random assignment of children to treatment groups without consideration of parent's choice may result in reduced effectiveness of behavioural programs, attributing this to parents not being ready to change.

In direct contrast, the behavioural components of each multimodal approach in the published literature encompasses an impressively intense and comprehensive program that is both complex and demanding for children, parents and teachers alike. Aside from being extremely intensive, time consuming and exceedingly expensive, the inherent nature of these programs lack sufficient ecological validity to be readily adaptable, let alone replicated, in any standard clinical context in the real world. Therefore these research based multimodal designs are generally incompatible, and not particularly relevant, to normal clinical practice. An additional criticism of many of the combined therapy intervention studies is the implementation of the group therapy "one size fits all" approach not tailored to the needs of the child or the family. To our knowledge, all existing empirical studies investigating multi-component treatments in children with ADHD subscribe to this intensive group therapy approach, a design no doubt useful for treating large numbers in research. However when one considers the heterogeneous nature of the disorder and the multitude of core and associated presenting problems, group therapy, as carried out in these research studies, may not permit a sufficiently individualised program necessary to address the specific needs of each child and family.

More recent empirical work by Dopfner and colleagues (2004) and van der Ord et al., (2007) have attempted to address the lack of ecologically valid research by adopting designs more analogous to clinical practice. These studies reported beneficial effects of both combined treatment and medication alone in improving ADHD related behaviours,

however they unfortunately failed to explicitly compare the merits of their multimodal approaches directly with the psychostimulant treatment. Furthermore, even though treatment delivery in these studies was intended to more closely reflect those typically employed in normal clinical contexts, their behavioural programs remained group oriented, with the authors themselves advocating for briefer treatment programs more individualised for each child and family.

Even though the results of the current study are quite promising, the findings must be interpreted in light of the small number of participants in each treatment condition of the study. However in the presence of small participant numbers the robustness of the significant group findings and strong effect sizes in light of the stringent statistical control are undoubtedly impressive. Furthermore, it must be kept in mind that there was no group treatment program in this study, all treatment was individualised and performed separately for each child and family. Therefore the small numbers in the present study are somewhat justified by the individualised nature of the treatment program implemented in this study.

An interesting, but non-significant, trend in the data was the tendency for the combined treatment group to be generally performing at a more impaired level across some of the outcome variables at baseline. Although not a statistically significant finding, it certainly could be argued that the children in the combined treatment group were overall more impaired and therefore could potentially be more responsive to treatment or gain more benefit from treatment. Although evidence exists in support of this notion (Rapport & Kelly, 1991), more recent findings emerging in the literature demonstrate that less impaired levels of baseline functioning may be associated with the greatest improvements in response to treatment (Bedard et al., 2002). The influence of baseline levels of performance and the differential effect of treatment need to be investigated further before any conclusions can be confidently made about this effect.

Future research would benefit from using teacher ratings to corroborate and extend the findings of the parent reports. Teacher ratings were not collected in the present study

for two reasons. Firstly, there is often poor compliance to research among teachers due to limited time and extensive workloads, with anecdotal evidence revealing very poor return rates of questionnaires. Secondly, children were followed up over six months, with consecutive assessments often running over into a new school year. This posed a problem of children not being consistently rated by the same teacher, with different raters potentially confounding the results.

Finally, there were no significant between subject effects, suggesting that although the combined group made greater improvements than the medication alone group across a range of outcome measures, there were no significant differences in performance scores at the final six month evaluation. It is extremely tempting to speculate, based on the presence of significant linear and quadratic trends in the data, that there might be an emerging trend for the combined group to demonstrate continued improvements with the medication group evidencing more of a plateau in performance. However without extended duration of follow-up, this proposed divergence in performance between the two groups is purely speculative. Although the treatment follow-up in the present study was comparable or even longer than many studies, it still remains far shorter than the average duration of treatment in standard clinical contexts. This only underscores the importance of extending treatment follow-up in intervention studies to be more comparable to that in normal practice.

8.3 The relationship between behaviour and cognition in ADHD

Based on the assumption of measuring similar underlying constructs, it was expected that the BRIEF parent ratings of behaviour would show strong correlations with performance based measures of executive functions. Although there is little available comparable evidence in the current literature, it was specifically predicted that higher ratings of problem behaviours by parents would be significantly associated with impaired performances on the standardised cognitive measures. In contrast to initial expectations however, only a limited number of weak relationships existed in the expected direction

between the behaviour and cognitive variables. Parent ratings of working memory related problem behaviours were associated with poorer performance based working memory and deficient semantic verbal fluency (relative difficulty component of the CAFT). Furthermore, greater inhibition problems as reported by parents were correlated with inattention as measured by omission errors on the CPT and difficulty with simple and demanding word generation on a semantic fluency measure. Parents ratings of inhibition and working memory problems did not correlate significantly with any other performance based executive function measure.

The significant findings of low to moderate correlations between the cognitive variables and the parent ratings of ADHD behaviours somewhat parallel the few studies that have examined the relationship between cognitive and behavioural measures in ADHD. For instance Epstein et al., (2006) and Muir-Broaddus et al., (2002) revealed the presence of significant low to moderate positive correlations across a range of teacher and parent ratings of core ADHD behaviour, such as hyperactivity, inattention and impulsivity with various cognitive variables, including omission errors and reaction times on a CPT. However, neither of these studies utilised specific performance based measures of inhibition nor was the BRIEF employed as the measure of behaviour rating. Interestingly, these previous studies demonstrated more significant correlations than the present study, even though they examined the relationship between general executive and non-executive functions (such as memory ability) and ratings of ADHD behaviour. Rather unexpectedly, the current study revealed generally limited and weak correlations between specific inhibition and working memory measures and behaviours relating to inhibition and working memory.

There are several possible explanations for these overall low correlations. Firstly, despite the BRIEF containing scales specifically evaluating difficulties with various aspects of executive functions, this measure may not actually assess the same constructs as being measured by the objective cognitive tasks. Alternatively, perhaps teacher ratings would have demonstrated more significant correlations between behavioural and cognitive measures, since a previous study suggested that teacher ratings are more highly

correlated with cognitive measures than parent ratings (Epstein et al., 2006). A more feasible explanation might relate to the actual properties of the BRIEF scales used in the correlational analyses. Given that there is a vastly restricted range of options for parents to select in describing their child's behaviour on the BRIEF (ie, never, sometimes and often) it is more likely that a ceiling effect is operating in this entirely clinical sample. Parents tended to strongly endorse problem behaviours which would have clearly reduced variability in responding, thus in turn affecting any relationship between the BRIEF and cognitive measures. Perhaps scales utilising more specific and concretely objectified rating options with less obvious face validity to prevent a biased response set, might increase the correlations between behaviour ratings and performance based measures of executive functions (Muir-Broaddus et al., 2002). Further research should confirm these findings in larger clinical and non-clinical samples.

8.4 Conclusion

The findings from the present study have provided support for the notion that un-medicated children with ADHD exhibit significant weaknesses in various aspects of cognition and behaviour relating to executive functioning. Furthermore, group results provide support for Barkley's theory of a key deficit in inhibition in ADHD, however findings appears to suggest that impairments in inhibition may not be able to account for all deficits observed in all children with ADHD.

The findings also add to the existing literature by establishing the benefits of either medication alone or combined therapy in the treatment of both behavioural and cognitive difficulties in children with ADHD. Impressively, the present study provides a valuable insight into the effectiveness of these treatments, with remarkable rates of normalisation. These have important clinical implications for both short and long term prognosis. This present study's findings are also amongst the first to document evidence, albeit preliminary and modest, to support the superiority of a low intensity, individualised combined therapy approach in the treatment of ADHD over six months of follow-up.

The efficacy and clinically relevant benefits of this multimodal approach supports its use as a valid alternative to a purely psychostimulant treatment program in future intervention research. Furthermore, if these findings are replicated in larger scales studies over an extended period of time, then this low intensity, individualised family centred combined approach may eventually be routinely adopted in standard clinical practice. Although these findings are very encouraging, it is paramount that large scale, longitudinal intervention studies of both medication alone treatment and multimodal therapy of varying intensities are conducted in the future. It would be very relevant and interesting to determine whether these significant improvements in behaviour and cognition persist over time and if they contribute to a brighter and more promising long term future for children, adolescents and adults with ADHD.

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Appendix A: CAFT Instructions

Controlled Animal Fluency Test Instruction Sheet

For each of the three categories allow 60" for the child to complete the task. If the child is silent for 15" or more, repeat basic instructions. Write down each animal name in the order said.

Instructions:

1. Animals (automatic)

- "Tell me as many different animals as you can, in any order and keep going until I say stop".

2. Animals by Size (regulation)

- "I want you to tell me as many different animals as you can, but this time I want you to put them in order of size. That is I want you to tell me the smallest animal you think of first, then one bigger, then a little bigger, then a little bigger again and so on, making sure that each one is bigger than the one before it. Don't get too big too quickly or you'll run out of animals. Keep going until I say stop. Off you go".

3. Animals by Alphabet

- *Pretest* – "Before we start this part I need you to say the alphabet for me, from the start to the end. Off you go" (if the child cant say the alphabet then discontinue).

- *Test* – "Now I want you to tell me as many different animals as you can but this time I want you to order them according to alphabet. That is, the first one is to begin with the letter A, the next with B, then C and so on. Say only one letter for each animal and keep going until I say stop".

Appendix B: CPT Instructions

Continuous Performance Task Instructions

On this computer screen in front of you, you will see a series of letters appear one at a time. Each letter is flashed on the screen fairly quickly so you have to play close attention. What I want you to do is press the "yes button ONLY when you see an X which comes after the letter A. For every other letter I want you to press the "no" button. We'll have a practice first. In the practice you will hear a sound every time you press a button, it will be a different sound for a correct response and an incorrect response. Let's have a go.

PRACTICE

If the child gets a poor score on the practice, repeat instructions

TEST

There will be no beeps this time. This test goes for quite a long time, so it's important to try and concentrate for as long as you can. Ready?

Appendix C: Victoria University Ethics Approval

4.

Victoria University
PO Box 14428
Melboume City MC
VIC 8001 Australia

VICTORIA UNIVERSITY

Human Research Ethics Committee

MEMORANDUM

TO: Dr Alan Tucker, * Monique Roper
Principal Investigators, Psychology

FROM: Rev Dr Christopher Pullin, Acting Chair,
University Human Research Ethics Committee

DATE: December 3, 2004

SUBJECT: **Approval of application involving human subjects**

Dear Alan,

Thank you for your submission detailing amendments to the research protocol for the project titled, ***A comparison of two forms of treatment for children with ADHD: effects on executive functioning and behaviour*** (HRETH.022/04).

The proposed amendments have been accepted by the Human Research Ethics Committee and approval for application HRETH.022/04 has been granted from 3/12/04 to 3/12/06.

Please note that, the Human Research Ethics Committee must be informed of the following: any changes to the approved research protocol, project timelines, any serious or unexpected adverse effects on participants, and unforeseen events that may effect continued ethical acceptability of the project. In these unlikely events, researchers must immediately cease all data collection until the Committee has approved the changes.

If you have any queries, please do not hesitate to contact me via the Ethics Officer on 99195354.

The Committee wishes you all the best for the conduct of the project.

Rev Dr Christopher Pullin, Acting Chair,

University Human Research Ethics Committee

Appendix D: Plain Language Statement: Invitation to Participate and Consent to Participate Forms.

VICTORIA UNIVERSITY
School of Psychology
Invitation to Participate in a Research Study

A comparison of two forms of treatment for children with ADHD: effects on executive functioning and behaviour.

Dear Parent/Guardian,

My name is Monique Roper, and as a postgraduate student of Victoria University, I am undertaking a research project investigating information processing (in particular, executive functioning) and behaviour following intervention in children with ADHD. The aim of the project is to better understand how to improve the effectiveness of treatment for children with ADHD. The project is part of my course for the Doctor of Psychology (Clinical Neuropsychology) degree and is being conducted in conjunction with my supervisor, Dr Alan Tucker, Clinical Neuropsychologist and Co-Director of the Victoria University Psychology Clinic together with collaborating paediatricians.

We invite you and your child to participate in this research project. If participating, your child would complete a cognitive (information processing) assessment, consisting of tasks measuring general intelligence, educational achievement, executive functioning (this includes planning and problem solving) and working memory. The tests will take approximately two hours to complete and the assessment will be conducted in the Psychology Clinic at Victoria University, St Albans campus. Your child will then be randomly assigned to one of two treatment groups. One group will comprise children who will be prescribed stimulant medication alone (by their paediatrician) and the other group will comprise children who will be prescribed stimulant medication (by their paediatrician), in addition to receiving family centered cognitive behavioural therapy, utilising positive reinforcement strategies implemented by key family members. We also need you to complete a questionnaire about your child's behaviour and answer a few questions pertaining to your child's medical history. Your child will be re-assessed at approximately three months after the initial assessment to review progress. Results of your child's assessment will be made available to you, at no cost, in the form of a written report.

We would like to emphasize that we will be collecting group data and at no time will your child's name be reported along with their results. In addition, please be assured that the data your child will contribute will be held in strict confidence by myself, my supervisor and collaborating paediatricians.

Participation in the study is entirely voluntary and you are free to decline participation or withdraw your child at any time and to remove any data your child has contributed. Withdrawal will not in anyway influence the medical treatment your child will receive.

Written consent to your child's participation in this research is needed. If you and your child are willing to participate, or if you have any questions you would like answered before making your decision, please contact Monique Roper on 0421 022 420, or Dr Alan Tucker on 99919 2266.

Thank you for your time,
Monique Roper

VICTORIA UNIVERSITY OF TECHNOLOGY

School of Psychology

Consent to Participate in a Research Study

Certification by Parent/Lawful Guardian of Participant

I, ……..

of……..

certify that I am the parent/lawful guardian of …….

and that I voluntarily give my consent to participate in the research study entitled ***"A comparison of two forms of treatment for children with ADHD; effects on executive functioning and behaviour"***, being conducted at Victoria University by Monique Roper.

I certify that the nature of the research has been fully explained to me by Monique Roper, including any potential risks and safeguards, with an explanatory statement of the research. I certify that I understand the aims and procedures of the study and freely consent for my child to participate in the aforementioned study on the condition that I can withdraw consent at any time without penalty. In addition, I have been guaranteed that the confidentiality and anonymity of my child and his/her data will be preserved at all times.

I also give consent for the researcher to be granted access to this study's clinical data obtained by collaborating paediatricians, only for the purposes of the current research study.

SIGNED.…… *DATE.……*

Witness other than the researcher.……..

Any queries about your participation in this project may be directed to the researcher: Monique Roper (ph. 0421 022 420). If you have any queries or complaints about the way you have been treated, you may contact the Secretary, University Human Research Ethics Committee, Victoria University of Technology, PO Box 14428, MCMC, Melbourne, 8001 (Ph. 9688 4710).

Appendix E: Behaviour Therapy Handout,
Problem Behaviour Questionnaire &
Weekly Progress Diary

BEHAVIOUR THERAPY HANDOUT:
"A comparison of two forms of treatment for children with ADHD: effects on executive functioning and behaviour".

What is Behaviour Therapy?

- A broad set of specific interventions that have a common goal of changing the physical and social environment to change behaviour.

Purpose of Family Behaviour Therapy:

- The aim is for primary caregivers to implement behaviour therapy with their child in order to manage specific problem behaviours. Primary caregivers will receive education about specific behaviour modification techniques and then will act as direct "therapists" for treatment of the child's maladaptive behaviours, with the researcher acting as a "consultant" to the primary caregiver.

General Behaviour Therapy Principles:

- The relationship between the person's behaviour and their environmental surroundings is expressed by the behavioural ABC:
- *Antecedent* = what happens directly before the target behaviour occurs (the trigger for the behaviour). For example: the child is arguing with their sibling.
- *Behaviour* = what the person says and does (the actual behaviour). For example: the child interrupts a conversation.
- *Consequence* = what happens immediately following the behaviour. For example: the child gets told off. The consequence teaches the child whether their behaviour was successful or not and provides the motivation to behave in a similar or different way next time they are in the same situation.
- Behaviour is determined by its antecedents and consequences. We can therefore change behaviour, by either changing what happens before or after a behaviour occurs.

Behaviour develops because it is reinforced:

- Behaviour that is rewarded (with positive reinforcement such as praise, attention or engaging in an enjoyable activity) will increase the behaviour.
- Behaviour that is followed by a negative consequence or one that is not reinforced (such as removal from an enjoyable activity or stopping attention) will decrease the behaviour.

<u>Positive Reinforcement:</u>

- A child receives a reward immediately after they engage in appropriate behaviour. For example: they get praise and a smile after waiting their turn in conversation or they get to play games on the computer after they finish their maths homework. This encourages appropriate behaviour whilst also reducing the problem behaviour.

Rewards/Reinforcers:

- Although different things are rewarding for different people, there are common reinforcers that are rewarding for most people:

1. *Social* = praise, smile, attention, clapping, looking interested.
2. *Material* = chocolate, soft drinks, toys.
3. *Sensory* = sounds, warmth.
4. *Activity* = going to the movies, playing on the computer, choosing what's for dinner.
5. *Intellectua*l = solving a puzzle.

**Different children are likely to prefer different reinforcers and it is important to identify what is rewarding for your child.

How to reward your child effectively:

- Choose a reward that your child will actually find rewarding. You will need to figure out what is a good reinforcer for your child. Don't underestimate the effectiveness of positive social reinforcement such as praise or a smile.
- Choose rewards that can be given immediately after your child displays appropriate behaviour, such as a chocolate bar or a trip to the shops.
- Choose rewards that can be given over and over again without your child becoming bored of them (i.e., lollies will not usually be reinforcing to a child who has just eaten some lollies) and use lots of praise, such as "good for you", "well done" and "good job".
- Select as many reinforcers as you can for your child.
- Reward the child *immediately* and *consistently* after your child displays the appropriate behaviour. For example, if you are rewarding a child for not interrupting a conversation you must give them positive reinforcement immediately after they have not interrupted. If you wait, and meanwhile they turn away and engage in another behaviour, you are inadvertently rewarding the other behaviour they are engaging in.
- Explain to your child why you are rewarding them as you are rewarding them (i.e., "you cleaned your room very nicely").
- Focus on a changing a limited number of behaviours.

Timeout:

- If you don't reward the inappropriate behaviour, behaviour will be more likely to decrease.
- Time out on the spot involves not rewarding your child for an inappropriate behaviour. One method is to not pay attention to your child for at least 20 seconds when they misbehave. A second method is to continue to pay attention to your child but to ignore the inappropriate behaviour.
- Timeout should be instituted immediately following misbehaviour.

How to use timeout effectively:

- Explain to your child that they have done something wrong and then immediately ignore their behaviour by placing them on a chair or in the corner for a few minutes.
- To be effective, timeout must occur immediately and without fuss.
- Combine timeout with rewarding your child for an alternative appropriate behaviour. So inappropriate behaviours should be ignored with alternative behaviours being rewarded.

REALISITC EXPECTATIONS!

1. When a behaviour program is introduced, the behaviour often gets worse before it gets better. In fact, things do usually get worse before they get better, but hang in there, doing so will pay off in the long run!
2. Keep in mind how long your child has had the behaviour. Don't expect a major change/improvement to take place all at once!
3. Rewards are only seen as rewards if your child sees them as such.
4. In most families, rewards are usually given haphazardly and inconsistently. It is often inconsistent because at one time a great deal will be given and at another time little will be given for the same response. Be sure that all family members have been prompted to ignore the maladaptive behaviour and to reinforce the desirable alternative behaviour.
5. Usually parents have been doing something for so long that they find it hard to change, which may reduce the effectiveness of behaviour treatment because they are being inconsistent. Often parents will set rules and allow themselves to be coerced by the child to break them. Consistency is the most important aspect of any program.
6. This is an experiment, it may or may not work, but we will work together to help improve your child's behaviour and thinking.

Problem Behaviour Questionnaire

Name of Child:

_____

Gender: MALE / FEMALE **Date:**_____

DOB: _____ **Age:**_____

Grade: _____ **School:** _____

Your relation to the child: (please circle)

Biological Parent	Step Parent
Adoptive Parent	Foster Parent
Grandparent	Other (please specify) _____

1. Please list and describe the behaviour/s that currently concern you most about your child: (i.e., can't sit still for longer than 10 minutes, fails to complete tasks they start, interrupts people's conversation's etc)

_____

_____

_____

_____

_____

_____

_____

_____

2. How often do these behaviours generally occur? (Please circle as many as required for each behaviour).

1. More than once a day
2. Daily
3. Weekly
4. Monthly
5. Other (please specify) _____

3. How often have these behaviours been of concern to you? (Please circle as many as required for each behaviour).

1. One week
2. One month
3. Six months

4. One year
5. More than one year
6. Other (please specify) _____

4. Please identify under what circumstances these behaviours occur: (i.e., when the child is avoiding doing homework, when the child wishes to play their favourite game, when the child is in conversation etc).

_____

_____

_____

_____

_____

5. How are these behaviours currently being managed? (i.e., tell them off, then instruct them to do their homework, do not permit them to play their favourite game etc).

_____

_____

_____

_____

6. Who ordinarily manages/disciplines your child? (i.e., mother, father, grandparents etc)

_____

_____

7. Please list your child's favourite hobbies, activities, games, sports:

_____

_____

_____

WEEKLY PROGRESS DIARY

The purpose of this diary is for you to document and monitor how you are going with the behaviour therapy process. This diary should be filled out **weekly** by any of the primary caregivers involved in the behaviour therapy. There will be approximately 12 diary pages to complete over the three month behaviour therapy period.

Name of Child: _____

WEEK ONE: Dates: __________\ 2005 to __________\ 2005

1. Have you used any behavioural therapy techniques in the past week? YES/NO

2. Have you rewarded your child for appropriate behaviour in the past week? YES/NO

3. How often have you rewarded your child for appropriate behaviour in the past week?

Not at all 1-5 times 6-10 times 11-15 times More than 16 times

4. How well is rewarding your child going?

Very Well Well Average Poorly Very Poorly

5. Please describe an instance of when you have rewarded your child in the past week: (including why they were rewarded, how they were rewarded and how effective it was).

6. Have you used time out or ignored your child's problem behaviour in the past week? YES/NO

7. How often have you used time out or ignored your child's problem behaviour in the past week?

Not at all 1-5 times 6-10 times 11-15 times More than 16 times

8. How well is using time out or ignoring your child's problem behaviour going?

Very Well Well Average Poorly Very Poorly

9. Please describe an instance of when you have used time out or ignored your child's problem behaviour in the past week: (including why time out was used or why they were ignored and how effective it was).

10. Who has been helping with the behaviour therapy program in the past week?

Appendix F: Normative Data for the CAFT

Controlled Animal Fluency Test (CAFT)

Norms for Children and Adolescents

Dr Alan Tucker

School of Psychology Victoria
University Melbourne, Australia

Age		CAFT condition				
		AUTO	SIZE	ALPH	RDsize	N=
7	M	12.29	6.58	6.55	43.03	41
	SD	3.74	1.75	3.55	17.54	
8	M	15.03	8.39	7.45	41.26	31
	SD	4.63	2.81	2.39	17.69	
9	M	16.52	9.24	9.33	41.47	54
	SD	4.45	2.61 .	2.88	16.93	
10	M	17.26	9.70	9.61	42.61	57
	SD	5.04	3.38	3.69	16.64	
11	M	18.13	1 0.43	10.87	39.64	46
	SD	4.86	2.69	3.76	17.53	
13	M	18.75	11.14	11.32 .	40.67	28
	SD	3.66	3.44	2.91	15.28	
15	·M	20.23	12.53	12.20	35.25	43
	SD	4.35	2.82	3.22	19.97	
17	M	22.15	13.07	12.85	39.46	27
	SD	4.69	3.58	4.29	15.92	
19	M	22.50	12.67	11.91	41.98	24
	SD	6.00	3.81	3.90	15.17	
					Total	351