CHAPTER TWO

Literature Review

The critical review of literature presented in this chapter serves to provide the theoretical basis for the subsequent research, which examined the current conceptualisation of ADHD and its associated impairments. Initially, the rationale for the current research is presented, and the conceptualisation of ADHD that was established with the publication of DSM-IV (APA, 1994) is discussed. Current information pertaining to diagnostic procedures, developmental course, comorbidity, prevalence, and intervention strategies for ADHD is provided, and the limitations of the DSM-IV formulation are considered. The need for a new theory of ADHD is then examined within the context of the evolving understanding of the disorder. The role of the present study in contributing to the extension and modification of the current theory of ADHD is then illustrated with reference to the impairments of ADHD children that were identified from the literature (i.e., response inhibition, working memory, attention, and the concept of time).

Attention-Deficit/Hyperactivity Disorder: Diagnostic criteria

Attention-Deficit/Hyperactivity Disorder (ADHD) is the current diagnostic label for the cluster of hyperactive, impulsive and inattentive symptoms that is now recognised as one of the most prevalent disorders of childhood (Tannock, 1998). The fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) lists the defining 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" (American Psychiatric Association [APA], 1994, p. 78).

The DSM-IV criteria currently used by professionals were developed through a lengthy process involving field trials, expert consultations, and the examination of published literature (Baxter, 1995; McBurnett, Lahey, & Pfiffner, 1993). Factor analyses conducted on empirical data gathered during these field trials suggested that ADHD comprises "two separate dimensions of symptoms - one composed of inattention symptoms and a second dimension composed of excessive motor activity and impulsivity" (Lahey et al., 1994, p. 1674). From these two symptom clusters, which are thought to be distinct in their etiology, course, response to treatment, and outcome, three ADHD subtypes are delineated: the Predominantly Inattentive Type (ADHD-PI), Predominantly Hyperactive-Impulsive Type (ADHD-HI), and the Combined Type (ADHD-CT) (Tannock, 1998).

The DSM-IV diagnostic criteria, which are reproduced in Table 1, consist of a schedule of nine inattentive symptoms and nine hyperactive-impulsive symptoms. Six (or more) of these inattentive symptoms are required for the diagnosis of ADHD-PI, whereas six (or more) hyperactive-impulsive symptoms are required for the diagnosis of ADHD-HI. At least six symptoms from each cluster are required for the diagnosis of ADHD-CT. In addition, symptoms must cause impairment in two or more different settings (i.e., symptoms must be pervasive), some symptoms must have been present before age seven, and should not be better accounted for by another disorder.

Table 1

DSM-IV diagnostic criteria for Attention-Deficit/Hyperactivity Disorder

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A. Either (1) or (2):

(1) six (or more) of the following symptoms of **inattention** have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Inattention
(a) often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities
(b) often has difficulty sustaining attention in tasks or play activities
(c) often does not seem to listen when spoken to directly
(d) often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions)
(e) often has difficulty organizing tasks and activities
(f) often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)
(g) often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools)
(h) is often easily distracted by extraneous stimuli
(i) is often forgetful in daily activities.

(2) six (or more) of the following symptoms of **hyperactivity-impulsivity** have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity
(a) often fidgets with hands or feet or squirms in seat
(b) often leaves seat in classroom or in other situations in which remaining seated is expected
(c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)
(d) often has difficulty playing or engaging in leisure activities quietly
(e) is often "on the go" or often acts as if "driven by a motor"
(f) often talks excessively

Impulsivity
(g) often blurts out answers before questions have been completed
(h) often has difficulty awaiting turn
(i) often interrupts or intrudes on others (e.g., butts into conversations or games)

Table 1 Continued

B. Some hyperactive-impulsive or inattentive symptoms that caused impairment were present before age 7 years.

C. Some impairment from the symptoms is present in two or more settings (e.g., at school [or work] and at home).

D. There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning.

E. The symptoms do not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder, and are not better accounted for by another mental disorder (e.g., Mood Disorder, Anxiety Disorder, Dissociative Disorder, or a Personality Disorder).

Code based on type:

314.01 Attention-Deficit/Hyperactivity Disorder, Combined Type: if both Criteria A1 and A2 are met for the past 6 months

314.00 Attention-Deficit/Hyperactivity Disorder, Predominantly Inattentive Type: if Criterion A1 is met but Criterion A2 is not met for the past 6 months.

314.01 Attention-Deficit/Hyperactivity Disorder, Predominantly Hyperactive-Impulsive Type: if Criterion A2 is met but Criterion A1 is not met for the past 6 months.

Coding note: For individuals (especially adolescents and adults) who currently have symptoms that no longer meet full criteria, "In Partial Remission" should be specified.

Source: DSM-IV, APA (1994)

Developmental course of ADHD

Whilst the DSM-IV criteria require that some symptoms causing impairment are present before age seven, DuPaul, Guevremont, and Barkley (1994) noted that "the majority of children who will be identified as having ADHD begin to manifest significant overactivity, noncompliance, and short attention span by 3 years of age" (p. 237). In addition, the DSM-IV diagnostic requirements are that the behaviours associated with ADHD must be present to a degree that is maladaptive and inconsistent with the child's developmental level (APA, 1994).

The present study therefore sought to account for these developmental differences by examining the performance of ADHD boys relative to a sample of individually Age-matched Control boys.

In the majority of cases, the symptoms of ADHD persist into adolescence, although there may be some attenuation of symptoms with age (Searight, Nahlik, & Campbell, 1995). Barkley (1997a) cited research that suggested 50%-80% of ADHD children exhibit symptoms into adolescence and between 30%-50% into adulthood (Barkley, Fischer, Edelbrock, & Smallish, 1990; Klein & Mannuzza, 1991; Weiss & Hechtman, 1993). There is also evidence to suggest that ADHD is associated with higher risks for "low academic achievement, poor school performance, retention in grade, school suspensions and expulsions, poor peer and family relations, anxiety and depression, aggression, conduct problems and delinquency, early substance experimentation and abuse, driving accidents and speeding violations" (Barkley, 1997a, p. 65). As adults, children with ADHD are also more likely to experience difficulties with adult social relationships, marriage, and employment (Barkley, 1997a).

In a number of models of ADHD (e.g., Barkley, 1997a; Quay, 1997), the risks associated with ADHD have been linked to the hyperactive-impulsive (HI) symptom cluster, and there is some evidence to suggest that its developmental course might differ from that of inattention (Lahey et al., 1994). Barkley (1997a) has even proposed that the ADHD-PI subtype might represent a separate disorder entirely. A recent study has provided added support for this conjecture, suggesting that the HI and inattentive dimensions might be etiologically distinct. In a community sample of 373 same-sex twins aged between 8 and 18 years, selected because at least one twin showed evidence of learning difficulties, Willcutt, Pennington, and DeFries (1999) concluded that

extreme ADHD scores, as assessed by the Diagnostic Interview for Children and Adolescents, Parent Report Version (DICA; Reich & Herjanic, 1982), "were almost entirely attributable to genetic influences" (p. 154). In addition, while the heritability estimate (h^2, that is, the proportion of the variance in the ADHD symptoms that is attributable to genetic factors) obtained for inattentive symptoms (h^2 = 1.10) was extremely high, the same was not true for HI symptoms without inattention (h^2 = 0.08). However, given that the heritability estimate for inattention exceeded the theoretical limit of 1.00, these results should be interpreted with caution. Nevertheless it is possible this result simply reflects sampling error around the true value (95% confidence interval = 0.65 - 1.55) since heritability estimates calculated in this way are unconstrained (Wilcutt et al., 1999).

Prevalence of ADHD

In a recent report pertaining to the Mental Health of Young People in Australia, Sawyer et al. (2000) found that approximately 14% of 4 to 17 year olds had indications of mental health problems, and that of these ADHD was the most prevalent. However, the determination of accurate prevalence figures for ADHD has been confounded by several factors, including the shifting labels and definitions that have been applied to the disorder, the different diagnostic criteria utilised (e.g., DSM versus ICD-10), and failure to account for comorbidity. Whilst prevalence estimates for ADHD have varied widely, with some studies suggesting rates as high as 15% to 24% (Zentall, Harper, & Stormont-Spurgin, 1993), large scale epidemiological studies have produced more conservative estimates of between 3% and 9% of the school-age population (Barkley 1997a; Mental Health Division of Western Australia, 2000; National Health & Medical Research Council [NHMRC], 1996; National Institute of Health, 2000).

In Western Australia, recent research using the Australian Child and Adolescent Disorders Screening Inventory (ACADSI; Langsford, Houghton, & Douglas, 2000) has estimated the prevalence of ADHD at approximately 7.4% in a sample of 823 school-age children. However, it must be acknowledged that the ACADSI is not a diagnostic instrument, and although it was based on DSM-IV criteria, a positive screen is not necessarily indicative of the presence of a disorder. Nevertheless, the ACADSI was designed to be sensitive to the 20 school-age disorders most commonly referred to school psychologists, and has the particular strength of integrating parent, teacher, and self-report data.

ADHD also affects proportionately more males than females, occurring in approximately three times as many boys as girls (Barkley, 1997a, 2001a; Tannock, 1998). In addition, ADHD is more common in first-degree biological relatives of children with the disorder (APA, 1994, 2000), which is consistent with the notion of genetic heritability. ADHD also occurs across a range of cultures and geographical regions (Tannock, 1998), with figures that suggest it affects 6.7% of children in New Zealand, 4.2% of children in Germany, 6-9% of children in China, 7.7% of children in Japan, and 9.5% of children/adolescents in Puerto Rico (Barkley, 2001b).

Comorbidity of ADHD

Comorbidity refers to the simultaneous occurrence of two or more disorders in the same individual at the same time (Clarkin & Kendall, 1992). Whilst prevalence estimates vary considerably, as many as 50% to 80% of children presenting with ADHD also meet the diagnostic criteria for other disorders (Tannock, 1998). The most frequently documented comorbidity is between ADHD and the other disruptive behaviour disorders, with oppositional defiant disorder and conduct disorder occurring in as many as 40% to 90% of children

with ADHD (Jensen, Martin, & Cantwell, 1997). However, a comprehensive review of the published literature on comorbidity (1997 - 1999) conducted by Langsford (1999) revealed a greater prevalence of ADHD amongst individuals with learning disabilities, disruptive behaviour disorders, mood and anxiety disorders, eating disorders, autistic and tic disorders, than in the general population.

There is also some evidence to suggest that the two ADHD symptom dimensions differ with respect to comorbidity, with the hyperactive-impulsive cluster more likely to be linked with oppositional or antisocial behaviour (Lahey et al., 1994), and the inattentive cluster more likely to be associated with specific learning disabilities (Stanford & Hynd, 1994). The recent findings of a genetic study by Willcutt, Pennington, and DeFries (2000) also appear to provide some support for this claim. Willcutt et al. (2000) again used the DICA (Reich & Herjanic, 1982) to assess ADHD symptoms in a community sample of 313 eight to 16 year-old same-sex twins, who were selected because at least one twin met the criteria for Reading Disability (RD). Willcutt et al. (2000) found that individuals with RD were significantly more likely than individuals without RD to exhibit elevated scores on both the hyperactive-impulsive and inattentive symptom dimensions. However, the bivariate heritability of RD and inattention was significant ($h^2 = 0.39$) whereas the heritability of RD and hyperactivity-impulsivity ($h^2 = 0.05$) was not. Furthermore, the etiology of this overlap appears to differ for the two symptom dimensions with 95% of the overlap between RD and inattention being attributable to common genetic factors, whereas only 21% of the overlap between RD and hyperactivity-impulsivity was attributable to genetic influences.

The presence of comorbid disorders may complicate the assessment, diagnosis, and treatment of ADHD, and is a frequent confounding factor in experimental research. Comorbid conditions are thought to result in increasingly adverse outcomes for children with ADHD, and the recent findings of Langsford (1999) have suggested that ADHD is the most comorbid of the 20 school-age disorders most commonly referred to school psychologists. Langsford subsequently devised and administered a screening device specifically designed to examine the pattern of comorbidity in school-aged students to a sample of 823 school children. The findings revealed that as the number of positive screens for disorders increased, there was a corresponding decrease in the mean level of self-control. The present study therefore sought to control for comorbidity by investigating only those children with ADHD and no diagnosed comorbid conditions. In this manner, any significant differences found between the ADHD and Control participants are more likely to be associated with ADHD itself and not attributable to comorbid influences.

Intervention strategies

Although this thesis is not primarily concerned with intervention strategies for ADHD it is necessary to include a brief description to develop a clearer understanding of the condition. The management of ADHD may be divided into two broad categories consisting of pharmacological and non-pharmacological interventions. Whilst few controlled studies have examined the longer-term efficacy of these interventions, a growing body of literature has until recently suggested that a multi-modal approach was more effective than either method individually (DuPaul et al., 1994; NHMRC, 1996). However, a recent 14-month clinical trial of stimulant medication and/or behaviour therapy found that a combined approach failed to yield significantly greater benefits than medication alone in 579 children with ADHD (MTA Cooperative Group,

1999). Nevertheless, the study also revealed that all four of the treatment groups (including a community care group) showed sizeable reductions in symptoms over time, albeit with significant differences in the degrees of change (MTA Cooperative Group, 1999).

While a range of unproven therapies have been applied to the treatment of ADHD, including diet management, sensory integration training, chiropractic skull manipulation, psychotherapy, EEG biofeedback, self-control (i.e., cognitive) therapies, and social skills training, in many cases there remains little or no empirical evidence to support their use as effective treatments. In contrast, there is considerable empirical evidence to support the use of certain specific medications, parent management training, family therapy, teacher education about ADHD, and classroom behaviour modification, in the treatment of ADHD (Barkley, 2001a). Whilst the medications most commonly used in the treatment of ADHD are the psychostimulants dextro-amphetamine sulphate and methylphenidate hydrochloride (known under the trade names Dexedrine and Ritalin respectively; NHMRC, 1996), noradrenergic medications, tricyclic anti-depressants and anti-hypertensives have also been proven effective (Barkley, 2001a).

The psychostimulant medications act on inefficient or immature neurotransmitter pathways "to mainly influence prefrontal, frontal, and limbic systems with benefits on behavioural inhibition, impulse control, selective attention, active working memory and executive functioning" (NHMRC, 1996, p. 21). Furthermore, "dexamphetamine appears to release newly synthesized dopamine and block uptake postsynaptically, while methylphenidate releases stored dopamine" (NHMRC, 1996, p. 21). Although side effects may include insomnia and loss of appetite (in 50% of cases), headaches and stomach aches

(20%-40%), irritability (10%), nervous habits (10%), mild weight loss, and increased heart rate and blood pressure, stimulant medications may be effective in as many as 90% of children with ADHD (Barkley, 2001a). Unless used improperly, stimulant medications are not addictive and can result in significant benefits including increased concentration and persistence, decreased hyperactivity and impulsivity, increased work productivity, better emotional control, decreased aggression and defiance, and improvements in working memory (Barkley, 2001a).

The non-pharmacological interventions focus on education and training for parents and teachers of children with ADHD. However, while behaviour management training for parents and behaviour modification training for teachers have proven effective (DuPaul et al., 1994; NHMRC, 1996), the use of cognitive (i.e., self-control) therapies for children with ADHD has proven largely ineffective (Barkley, 2001a). Barkley has argued that the efficacy of cognitive-based interventions for children with ADHD will be undermined by impairments in the underlying cognitive processes. Nevertheless, a number of studies have suggested that cognitive-behavioural interventions may have some (albeit limited) beneficial effects, particularly when used as part of a multi-modal treatment regime (Miranda & Presentacion, 2000; MTA Cooperative Group, 1999; NHMRC, 1996). Other effective non-pharmacological interventions include: parent training (Pelham, Wheeler, & Chronis, 1998), training in problem solving (Robin, 1988), the use of a mentor system (Barkley, 2001a), peer tutoring (DuPaul, Ervin, Hook, & McGoey, 1998), token reinforcement and response costs (McGoey & DuPaul, 2000), and the use of weekly assignment sheets (Barkley, 2001a).

The evolving conceptualisation of ADHD

Since the characteristic cluster of hyperactive, impulsive, and inattentive symptoms that is now recognised as ADHD was first discussed as a behavioural syndrome by Still in 1902 (DuPaul et al., 1994), the understanding of ADHD has continued to evolve. In particular, researchers and clinicians have struggled with the conceptualisation of ADHD as new research findings challenged the prevailing construct (Tannock, 1998). Differences have also emerged between the North American and the European formulations of ADHD, with the European formulation recognising only those children with symptoms of hyperactivity, impulsivity, and inattention, as ADHD (Tannock, 1998). To this point this thesis has reviewed the conceptualisation of ADHD that was established with the publication of DSM-IV (APA, 1994), which represented the culmination of many years of research. However, the understanding of ADHD has continued to develop and as a consequence, the limitations of DSM-IV have become increasingly apparent. In particular, the DSM-IV criteria are descriptive, and fail to account for the many cognitive and behavioural deficits associated with ADHD (Barkley, 1997a). Furthermore, the DSM-IV formulation is largely atheoretical, and provides little insight into the nature of the disorder (Barkley, 1997a), or directions for its treatment (Power & DuPaul, 1996).

Barkley (1997a) subsequently argued that a new theory of ADHD was needed that could: address the findings of previous research; link the disorder to a defect in normal human development; and make explicit predictions about new phenomena that can be tested empirically. Although various theories have been proposed to account for ADHD, most notably Zentall's (1985) optimal stimulation theory, Sergeant's (2000) cognitive-energetic model, and Sonuga-

Barke's notion of "delay aversion" in children with ADHD (Sonuga-Barke, Williams, Hall, & Saxton, 1996), Barkley's (1997a) Unifying Theory appears to be the most scientific conceptualisation to date. Barkley (1997a) drew together literature from the fields of developmental psychology, neuropsychology, and neurology, to construct a theory of ADHD which linked the disorder to an irregularity in the development of self-control. According to Tannock (1998) the recent development of theoretical models that focus on the component processes which underlie ADHD represents a significant advance in the field. Whilst it is beyond the scope of this thesis to provide a full historical account of ADHD, key historical developments will be detailed so that the emerging models can be placed into context of the conceptualisation of ADHD to date.

1900 to 1949: Historical origins

Although the symptoms of ADHD may have been first identified as early as the 1860's (DuPaul et al., 1994), the first clinical descriptions of the disorder were presented by Still in 1902 who described children with what he termed morbid defects in moral control. Using the theories of William James as a basis, Still postulated that the deficits in inhibitory volition, moral control, and sustained attention associated with this condition were due to an underlying neurological deficiency (Barkley, 1990). During the 1930s, links were beginning to be established between brain injuries and a number of cognitive and behavioural impairments such as ADHD, which was then known as organic drivenness or restlessness syndrome (Barkley, 1990). Although several researchers attributed the ADHD symptoms to a frontal lobe dysfunction in the brain, the prevailing consensus during this era was that hyperactivity was the result of psychological causes such as poor parenting or a poor family environment. Another significant development that occurred during this era was the discovery of

amphetamines as an effective intervention for children with the disorder (Barkley, 1990).

1950 to 1959: An era of "Minimal Brain Damage/Dysfunction"

Over the course of the next decade, the conceptualisation of ADHD evolved once again and by the 1950s symptoms were being attributed to minimal brain damage. By the late 1950s and early 1960s, the lack of corroborating evidence, in the form of actual central nervous system damage in children with this condition, forced the concession that although the disorder was neurologically linked, it was not the result of neurological damage (Barkley, 1990). This shift in emphasis was reflected by the change in terminology from minimal brain damage to minimal brain dysfunction. A number of studies conducted during the 1950s also referred to ADHD as "hyperkinetic impulse disorder," postulating that the condition was due to cortical overstimulation, and the result of inadequate sensory filtering. However, no mention was made of ADHD in the inaugural edition of the DSM published in 1957 (McBurnett et al., 1993).

1960 to 1969: Hyperactivity

The understanding of ADHD further evolved in the 1960s when the concept of hyperactive child syndrome appeared. In 1960, Chess emphasised that the key feature of the disorder was hyperactivity, and her conceptualisation of the condition distinguished it from a brain damage syndrome and removed the blame from the child's parents (Barkley, 1990). During this era, the concentration on the hyperactivity component of ADHD to the exclusion of the impulsive and inattentive components may have resulted in the disorder becoming known as hyperactivity or hyperkinesis (DuPaul et al., 1994).

Hyperactivity also began to be viewed as a behavioural syndrome that could occur both in the presence or absence of a biological cause (Barkley, 1990). This view began to gain wider endorsement prior to the publication of the DSM-II in 1968, which was the first edition to incorporate ADHD under the name Hyperkinetic Reaction of Childhood (McBurnett et al., 1993). By the end of this decade, the prevailing view was that although ADHD was still considered a brain dysfunction syndrome, it was less severe in nature than previously suspected and was no longer linked to brain damage. Instead, the focus had shifted to brain mechanisms (Barkley, 1997b).

1970 to 1979: The era of the "Attention Deficit"

The 1970s saw a vast amount of research conducted into the disorder and with in excess of 2000 studies published, the literature abounded. Ironically the increased scientific and professional (as well as public) attention focused on the disorder coincided with the emergence of the concept of the Attention Deficit. Early in the 1970s it became clear that the exclusive focus on hyperactivity had obscured other key characteristics of the disorder such as: impulsivity, distractibility, short attention span, aggressiveness, and low frustration tolerance. Douglas (1972) stressed the role of deficits in sustained attention and impulse control over hyperactivity as the key features of the disorder. Douglas also reported that children with ADHD were not necessarily reading or learning disabled and that they were no more distractible than other children, but linked the observed lack of moral development to the deficiencies in attention and impulse control (Barkley, 1990).

The 1970s also saw a profound increase in the use of stimulant medication in the treatment of ADHD. The view that hyperactivity was the result of food additives (and later sugar), and could thus be alleviated by diet modification,

gained popularity which persisted long after these claims were refuted. The views that ADHD was the result of environmental overstimulation or poor parenting also resurfaced. For a time, in the mid 1970s, the research focused on the psychophysiology of children with ADHD, with variables such as galvanic skin response being measured. By the end of the decade, it was clear that inattention and impulsivity were important in explaining the problems of children with the disorder. The focus on the cause of the disorder had shifted from brain damage to other brain mechanisms such as underarousal, underactivity, brain neurotransmitter deficiencies, or neurological immaturity (Barkley, 1997b). The recognition of environmental variables as a causal factor in the disorder led to the ratification of a range of treatment strategies including behaviour modification and parent training.

1980 to 1989: The development of diagnostic criteria

During the 1980s Douglas (1980, 1983, cited in Barkley, 1997a) reformulated her theory of ADHD, arguing that it comprised major deficits in: attention and effort, impulse inhibition, arousal modulation, and an increased need to seek immediate reinforcement. The profound impact of Douglas' research may have been partly responsible for the renaming of the condition as Attention Deficit Disorder (ADD) in the DSM-III. Of further importance, the DSM-III (1980) introduced a multiaxial assessment system comprising two behavioural subtypes: ADD with hyperactivity (ADD/H or ADD/+H), and ADD without hyperactivity (ADD/WO or ADD/-H) (Morgan, Hynd, Riccio, & Hall, 1996). During the 1980s the concept of an underlying attention deficit as the cause of ADHD was replaced by other ideas, including that of a dysfunction in the effort/activation system (Sanders, 1983). Zentall (1985) also proposed an optimal stimulation theory of ADHD, postulating that hyperactivity is a form of self-stimulation used to maintain an optimal arousal level.

However, the main advances in research during this decade were due to the emergence or application of new scientific techniques including regional cerebral blood flow/computed tomography (rCBF/CT) and magnetic resonance imaging (MRI) in the study of ADHD. These new techniques revealed regions of reduced prefrontal lobe activity in the brain and the widely held view that ADHD was the result of neurotransmitter deficiencies was established. The efficacy of psychostimulant medications in the treatment of ADHD was understood and explained in terms of their ability to target inefficient or immature neurotransmitters (NHMRC, 1996). With the advent of the DSM-III-R in 1987 came the current terminology of Attention-Deficit/Hyperactivity Disorder (ADHD) (Morgan et al., 1996). The DSM-III-R returned to a unidimensional syndrome comprising 14 symptoms, the presence of eight of which were required for diagnosis (McBurnett et al., 1993). The additional category of Undifferentiated Attention Deficit Disorder (UADD) was included for children with prominent inattentive symptoms only (Morgan et al., 1996) or attention deficit disorders not specified by the ADHD criteria (McBurnett et al., 1993).

1990 to 1999: The era of the "Executive Functions"

The publication of the DSM-IV (APA, 1994) represented a major advance, with several major changes to the ADHD criteria including the return to a multiaxial assessment and the reinstatement of behavioural subtypes. Whilst the existence of distinct behavioural subtypes of ADHD is now generally acknowledged, there continues to be disagreement as to their composition. The 1990s also saw an increase in the research on comorbidity and a focus on the "executive functions". However, much of the research in these areas was confounded by poorly defined constructs, and a precise definition of executive function (EF) is yet to emerge (Tannock, 1998). While Welsh and Pennington (1988) defined EF

as "the ability to maintain an appropriate problem-solving set for attainment of a future goal (Bianchi, 1922; Luria, 1966)" (p. 201), Eslinger (1996) reported that the National Institute of Child Health and Human Development working group on EF generated 33 different definitions.

However, while there is strong agreement that the construct of EF does not refer to basic cognitive processes such as sensation, perception, motor activation, attention, and memory, a precise definition has proven elusive (Tannock, 1998). Without this, the logic of many EF studies appears almost circular, with the construct under examination effectively being defined by the measures used to assess it. Tannock (1998) stated that EF is typically used to refer to the psychological processes involved in one or more of the following capacities: self-regulation, sequencing of behaviour, flexibility of thinking or responding, response inhibition, planning, and organisation of behaviour. In 1996, Pennington and Ozonoff conducted a review of studies of EF in four developmental psychopathologies: ADHD, conduct disorder, autism, and Tourette's syndrome. Pennington and Ozonoff (1996) concluded that EF deficits were consistently found in both ADHD and autism but not in conduct disorder or Tourette's syndrome. Research by Seidman, Biederman, Faraone, Weber, and Oulette (1997) with 118 boys with ADHD demonstrated significant impairments on the Stroop Task (Trenberry, Crosson, DeBoe, & Leber, 1989) and the Wisconsin Card Sorting Test (WCST; Heaton, Chelune, Talley, Kay, & Curtiss, 1993), both of which are measures of EF according to Pennington and Ozonoff (1996). A subsequent study by Seidman, Biederman, Faraone, Weber, Menin, and Jones (1997) found no significant differences between the neuropsychological performance of ADHD and Control girls. It is worth noting however that neither study controlled for stimulant medication status or comorbidity.

A recent study by Houghton et al. (1999), which controlled for these factors, also reported significant EF impairments amongst a sample of 94 ADHD children compared to non-ADHD Controls. Houghton et al. also found significant impairments on the Stroop and WCST (particularly amongst the ADHD Combined Type), although no gender effects were observed. However, whilst there is sufficient evidence to suggest that EF is impaired in children with ADHD, these deficits do not appear to be specific to ADHD (Pennington & Ozonoff, 1996), or primary to the disorder (Pennington, Bennetto, McAleer, & Roberts, 1996). A new model of ADHD was therefore needed that could drive new research initiatives (Barkley, 1997a), and link the observed impairments in EF to problems with sustained attention, hyperactivity, and impulse control.

Sergeant's Cognitive-Energetic Model

In recent years, Sergeant has utilised the cognitive-energetic model described by Sanders (1983) to direct research into ADHD. The cognitive-energetic model suggests that whilst there may be certain aspects of inhibition that are deficient in children with ADHD, this may also depend on the energetic state of the child (Sergeant, 2000). This model conceptualises information processing as involving three distinct levels: a set of lower cognitive processes (i.e., encoding, central processing, and response organisation); the three energetic pools of arousal, activation and effort; and the management or executive function system (Sergeant, 2000).

To date research conducted at the level of the lower cognitive processes has suggested that there are no apparent deficits in encoding or central processing, but that motor organisation is impaired in children with ADHD (Sergeant & Van der Meere, 1990a, b). At the second level, the primary deficits of ADHD children are thought to be associated with activation (which is concerned with

the control of motor readiness) and effort (which is influenced by motivational factors, such as knowledge of results, and self-regulation) (Tannock, 1998). At the management or executive level, it is suggested that inadequate activation of the inhibitory mechanism in ADHD children results in the secondary deficiencies in the other executive functions described by Barkley (1997a) (Sergeant, 2000). However, the results of a meta-analysis by Oosterlaan, Logan, and Sergeant (1998) suggested that this explanation was not specific to ADHD, but also applied to children with oppositional defiant disorder and conduct disorder.

Sonuga-Barke et al.'s Delay Aversion Model

Alternatively, Sonuga-Barke, Saxton, and Hall (1998) have argued that the impairments seen using standard tests of impulse control amongst hyperactive children might be an artefact of the laboratory situation itself. Sonuga-Barke et al. (1998) observed that in situations where the length of the task is dependent on the delay prior to responding, choosing the more immediate and lesser reward, or responding more quickly, results in shorter trials and less delay overall. Thus according to Sonuga-Barke, "impulsive" behaviour might in fact represent a situation-specific attempt to reduce the subjective perception or experience of delay, and hyperactive children can in fact withhold responses, but choose not to do so in order to minimise time in the laboratory (Tannock, 1998). The delay aversion theory therefore challenges the notion that behavioural inhibition is an underlying impairment in ADHD. Instead, the inhibitory problems are viewed as indicative of a deviation in motivational attitude, and the central construct in the model is a specific aversion to delay or the suppression of responses over time (Tannock, 1998).

Recently Kuntsi, Oosterlaan, and Stevenson (2001) attempted to address this issue by testing the predictions made by three theories of ADHD: the response inhibition deficit (e.g., Barkley, 1997a; Quay, 1997), working memory/executive impairment (Pennington & Ozonoff, 1996; Pennington, Bennetto, McAleer, & Roberts, 1996), and delay aversion (e.g., Sonuga-Barke et al., 1998). Kuntsi et al. (2001) examined 51 ADHD children and 119 control children using the Maudsley Index of Childhood Delay Aversion (Kuntsi, Stevenson, Oosterlaan, & Sonuga-Barke, in press), the Stop Task (Logan & Cowan, 1984), the Delayed Response Alteration Task (Carpenter & Gold, 1994), and Sentence Span (Siegel & Ryan, 1989). Kuntsi et al. (2000) found that children with ADHD performed worse than controls on the measure of delay aversion (in which participants have to choose between a small immediate reward and a large delayed reward) and some of the working memory tasks (which are reviewed later in this chapter). While no significant differences were found on the measures of inhibition derived from the Stop Task, the ADHD children were found to be more variable than controls in terms of their response speed, and generally slower and less accurate in their responding (Kuntsi et al., 2001).

In recent years, Sonuga-Barke, Williams, Hall, and Saxton (1996) have advocated a modified formulation in which delay aversion and impulsiveness are thought to result from impairments in temporal processing. While this appears to be in conflict with Sonuga-Barke et al.'s earlier non-deficit model (Tannock, 1998), Barkley (1997a) has also suggested that the concept of time might be impaired in children with ADHD. Barkley, Koplowitz, Anderson, and McMurray (1997) examined this issue in more detail in two studies using a time reproduction task. While this (and other) studies of concept of time in children with ADHD are reviewed in more detail later in this chapter, the findings of Barkley et al. (1997) suggest that time reproduction may be impaired in ADHD

children. However, given the small amount of research in this area to date and its acknowledged limitations (including small sample sizes, comorbidity, and issues of motor control and persistence) further research is clearly necessary.

Barkley's Unifying Theory of ADHD

Barkley (1997a) subsequently proposed a theoretical model of ADHD (which is reproduced in Figure 1), designed to apply to the Hyperactive-Impulsive and Combined Types (i.e., those subtypes characterised by Hyperactive-Impulsive behaviour), which posited that the central impairment in the disorder was one of behavioural (or response) inhibition. Whilst the notion of a deficit in behavioural inhibition in ADHD is not new, but builds on earlier work of Douglas (1988), and Quay's (1988) use of Jeffrey Gray's model of anxiety applied to ADHD, Barkley's (1997a) model predicts that it is the central impairment in ADHD. In addition, Barkley (1997a) predicted that secondary impairments in four specific EFs would result from the ADHD child's essential inability to inhibit and postpone responses. These four EFs are: the operation of working memory (including hindsight and forethought); the internalization (or self-direction) of speech; the self-regulation of mood, motivation and arousal; and reconstitution (the ability to analyse and synthesise novel sequences of behaviour).

According to Barkley (1997b) the EFs are those self-directed actions that begin as public behaviours and are gradually privatised over the course of development, becoming increasingly responsible for self-control as the public aspects of these behaviours are inhibited. Thus Barkley (1997b) predicted that the EFs are dependent on behavioural inhibition for their effective development. Furthermore, whilst the successive chain of impairments in the EFs creates the appearance of poor sustained attention in those with ADHD, Barkley (1997b) argued that it actually represents a reduction in the executive control of behaviour (i.e., control by the internally represented information that is afforded by the EFs). Therefore, Barkley predicted that individuals with ADHD will be less proficient in the self-regulation of their behaviour and more susceptible to control by the immediate external environment.

A number of specific predictions have been advanced by the Barkley model that have served to stimulate research (Tannock, 1998). According to Barkley (1997a), poor behavioural inhibition results in secondary deficiencies in working memory and its subfunctions in children with ADHD. In particular, Barkley predicted that impairments in verbal working memory would lead to difficulties with reading comprehension and adherence to verbal rules or instructions, while deficiencies in non-verbal working memory would result in an impaired concept of time. Furthermore, Barkley suggested that as a consequence of this chain of impairments, Barkley (1997b) children with ADHD will manifest difficulties with goal-directed persistence and sustained attention.

The predicted impairments in each of these areas are of particular relevance to the present research since they were also identified in the review of literature and the semi-structured interviews in Study One. The present research sought to contribute to the ongoing development of theory pertaining to ADHD by

extending the findings of previous research in each of these four domains. The following sections therefore review previous and concurrent research relating to each of these four areas of executive functioning.

Response inhibition

Given Barkley's (1997a) contention that response inhibition is the central impairment of children with ADHD, the effective measurement of a deficit in behavioural inhibition in ADHD is crucial to current theories of the disorder (Nigg, 1999). However, there is also considerable evidence to suggest that response inhibition is a multifaceted construct (Barkley, 1997a; Nigg, 2000). In 1997, Barkley described a three phase model of inhibition which comprised three interrelated processes: inhibiting the initial prepotent response; stopping an ongoing response, which permits a delay in the decision to respond; and interference control, which protects this period of delay from disruption by competing events and responses (Barkley, 1999). In contrast, Nigg (2000) identified eight kinds of inhibition that have been applied across different tasks and measurement paradigms, that can be broadly grouped into executive, motivational and automatic inhibitory processes.

Evidence of poor inhibition in ADHD children has been established using paradigms such as the go/no-go task (e.g., Casey et al., 1997; Iaboni, Douglas, & Baker, 1995), the change task (Schachar, Tannock, Marriott, & Logan, 1995), and the stop signal task (Oosterlaan & Sergeant, 1995, 1996; Schachar, Mota, Logan, Tannock, & Klim, 2000). In the go/no-go task, participants are required to respond (e.g., press a key) when a frequent stimulus appears, but to make no response when an infrequent stimulus appears. In contrast, the stop signal task (Logan, 1994) has the advantage of being based on a well-established theory of response inhibition (Nigg, 1999), which unlike other measures permits the

measurement of the underlying inhibitory process (Oosterlaan, Logan, & Sergeant, 1998).

The stop signal paradigm is based on the race model (Logan & Cowan, 1984), in which response inhibition is conceptualised as a race between competing stop and go processes (Nigg, 2000). According to this theory, poor inhibitory control could result from extremely fast response processes or from very slow inhibitory processes (Tannock, 1998). There is also evidence from an increasing body of research that suggests that the latter is in fact the case (Oosterlaan et al., 1998; Pliszka, Borcherding, Spratley, Leon, & Irick, 1997; Schachar et al., 1995). Aman, Roberts, and Pennington (1998), Purvis and Tannock (1997), and Schachar and Logan (1990) have also shown that children diagnosed as ADHD have slower stop signal reaction times than non-ADHD controls. Using a new tracking procedure for assessing stop signal response time, Nigg (1999) also demonstrated that ADHD was associated with slower stop signal response time, and that taken as a whole the results "bolster the idea that deficits in motor inhibition processes are associated with the DSM IV ADHD combined type" (p 399).

Recently, Leth-Steensen, Elbaz, and Douglas (2000) described this pattern of longer overall response times and increased variability of responding amongst ADHD children as "the most consistent finding in the ADHD cognitive literature" (p. 168). Leth-Steensen et al. subsequently demonstrated that the response time distributions of boys with ADHD were distinguished from those of age-matched Control boys by an increased number of abnormally slow responses, resulting in a larger tail of the distribution. However, given the limited sample size of Leth-Steensen et al.'s (2000) work (n = 17), caution is advised in the interpretation of these findings until such time they can be

replicated with a larger sample. Whilst it is anticipated that the response time data gathered in the present study will be examined using a similar distributional approach, providing a useful extension to Leth-Steensen et al.'s (2000) work, such analyses are beyond the scope of the present research.

However, evidence from research also suggests that children with conduct disorder show similar impairments to those seen in ADHD (Oosterlaan, Logan, & Sergeant, 1998; Oosterlaan & Sergeant, 1996; Schachar et al., 2000), and therefore impairments in response inhibition might not be specific to ADHD. Nigg (2000) argued that if an inhibitory deficit is not specific to ADHD, it cannot be a necessary and significant cause of the disorder. It may be, as suggested by Oosterlaan et al. (1998), that deficits in response inhibition might characterise that wider group of children with disruptive or externalising behaviour problems, although similar findings have also been found in children with reading disability (Purvis & Tannock, 2000). The present study will examine whether deficient inhibition is characteristic of boys with ADHD who have no diagnosed comorbid conditions (including other disruptive or externalising behaviour problems). The individual matching of the ADHD and control group to within three months of age will also address the potential developmental variations in inhibitory functioning suggested by Williams, Ponesse, Schachar, Logan, and Tannock (1999) in their cross-sectional study of inhibitory control across the life span.

Working memory

According to Denckla (1996), working memory refers to the ability to represent and hold in mind visual or verbal information for the duration of a task. Furthermore, Denckla suggested that "working memory entails the ability to behave on the basis of represented rather than immediately presented

information/knowledge" (Denckla, 1996, p. 116). This is consistent with Barkley's (1997a) notion that the development of self-control represents a shift from the external control of behaviour to control by internally represented information (i.e., the EFs, and in particular, working memory). However, whilst impairments in working memory are central to the current theories of ADHD (e.g., Barkley, 1997a; Kuntsi, Oosterlaan, & Stevenson, 2001), systematic investigations of working memory in ADHD are sparse (Tannock, 1998).

The limited research in this area has suggested that children with ADHD perform poorly on tasks of working memory, including repetition of digits forwards and backwards (Barkley, Murphy, & Kwasnik, 1996), mental arithmetic (Zentall & Smith, 1993), the Freedom of Distractibility Scale of the Wechsler Intelligence Scale for Children (Third Edition; Wechsler, 1991) (Anastopoulos, Spisto, & Maher, 1994), and the Tower of Hanoi (Pennington, Grossier, & Welsh, 1993), compared to non-ADHD control children. In addition, children with ADHD appear to have difficulties in adjusting their subsequent responding, despite feedback pertaining to the ineffectiveness of their performance (Houghton et al., 1999; Sergeant & Van der Meere, 1988). In line with this, it has been suggested that failure to adjust performance may reflect an interaction between behavioural inhibition and the retrospective-prospective functions of working memory (Barkley, 1997a).

Kaplan, Dewey, Crawford, and Fisher (1998) examined verbal and non-verbal memory in 53 ADHD, 63 RD, 63 ADHD+RD, and 112 control children using the Wide Range Assessment of Memory and Learning (WRAML; Sheslow & Adams, 1990). The WRAML is a standardised test of memory function in children between five and 18 years of age and consists of nine subtests that between them assess verbal memory, visual memory, learning, and memory

retention. The analyses revealed a multivariate main effect for group on the verbal memory subtests (Story Memory, Sentence Memory, and Number/Letter Memory) which was supported by univariate main effects for Sentence Memory and Number/Letter Memory. A multivariate main effect for group was also observed on the visual memory subtests (Picture Memory, Design Memory, and Finger Windows) which was supported by a univariate main effect for Finger Windows, in which participants must point their finger through a series of "windows" in sequential order. Post hoc comparisons of these univariate effects revealed that the ADHD, RD, and ADHD+RD groups scored significantly lower than the control group on all three of these subtests (Kaplan et al., 1998).

In addition, all three disordered groups performed significantly worse than controls on the overall measure of general memory. Kaplan et al. (1998) also found that the RD and ADHD+RD groups scored significantly lower than either the ADHD or control groups on the overall measures of Verbal Memory and Learning, and had forgotten more information from the Story Memory subtest at delayed recall. However, no significant differences were found on the measures of Visual Learning, or the other measures of memory retention. Kaplan et al. (1998) concluded that these data were consistent with Barkley's (1997a) model of ADHD and provided support for the notion of working memory deficits in ADHD. However, it should be noted that there were significant differences in the mean age, estimated IQ, and socio-economic status of the groups being compared. In addition, while the difference in sex distribution across the four groups was non-significant, the proportion of females ranged between 15.2% to 50%, and 54.7% of the ADHD participants and 49.2% of the ADHD+RD participants were medicated at the time of testing (Kaplan et al., 1998).

In another study, Oie, Sundet, and Rund (1999) compared the memory function of 19 adolescents with schizophrenia (aged 13-18 years), 20 with ADHD (aged 11-18 years), and 30 control children (aged 12-18 years). The WISC-R Digit Span subtest (Wechsler, 1974), California Verbal Learning Test (Delis, Kramer, Kaplan, & Ober, 1987), Kimura Recurring Figures test (Kimura, 1980), and the Digit Symbol Location task (Oie et al., 1999) were chosen to assess visual and verbal memory, short and long term memory, and recall and recognition memory. The results revealed that relative to the controls, the schizophrenia group showed significant impairments in both visual and verbal memory, while the ADHD group only showed significant impairment on measures of verbal memory and learning. While these findings appear to be consistent with Kaplan et al. (1998), Oie et al. failed to control for comorbid RD in the ADHD sample, which was an acknowledged limitation of the study. Thus it is possible that the verbal memory impairments of the ADHD children might have arisen from the known comorbidity with RD (Oie et al., 1999), since Kaplan et al. (1998) also found that children with RD and ADHD+RD performed worse than ADHD children on Verbal Memory and Learning.

More recently Kuntsi et al. (2001) used three working memory measures with a sample of 51 pervasively hyperactive children aged between seven and 11 years. The Delayed Response Alteration Task (Carpenter & Gold, 1994) was utilised, in which participants must choose between two boxes that are presented on a computer screen; each individual is told whether his or her response is correct. The child's task is to determine the rule the computer uses to decide which box is correct. If the child does not find out the rule (which consists of choosing the coloured and uncoloured box on alternate trials), the rule is explicitly taught by the researcher. The second measure utilised was the Sentence Span task (Siegel & Ryan, 1989) in which the child is read several

sentences and then asked to supply the missing last word in each sentence. At the end of each set of sentences, the child is asked to recall all of the words that he or she had supplied, in the correct order. The third measure, the Counting Span task (Case, Kurland, & Goldberg, 1982), is analogous to the sentence span task except that the child is asked to count the number of dots on a series of cards instead of supplying words. Significant group differences were found on the post-instruction phase of the delayed response alteration task and both the sentence span and counting span measures, although these differences became non-significant after controlling for IQ.

In line with the recent findings of Willcutt et al. (2000) which suggested considerable genetic overlap between inattention and RD, Tannock (2001) postulated that a deficit in working memory might be characteristic of both ADHD and RD. According to Tannock (2001) a common impairment in verbal working memory might also help to explain the frequent comorbidity between ADHD and RD. Although it is not possible to examine this assertion directly in the present study, evidence of impairment on measures of verbal and non-verbal memory, immediate and delayed recall, or working memory, may be provided by the Children's Memory Scale (CMS; Cohen, 1997). This recently developed and validated instrument was utilised in the present research.

Attention

Despite considerable research, attempts to characterise the exact nature of the attention deficit associated with ADHD have proven largely inconclusive. In many cases, the inconsistent research findings might be attributed to methodological limitations such as small sample sizes, substantial variation in diagnostic procedures, and failure to control for the use of stimulant medication, ADHD subtype, and comorbidity (Barkley, 1997b; Houghton et al.,

1999). In addition, whilst there appears to be general agreement that attention is multifactorial, there remains a bewildering array of subdivisions of the attentional construct (Denckla, 1996). Various theories have proposed the delineation of attention into the following components: selective and divided; automatic and effortful; and focus, shift, sustain, and encode (Denckla, 1996). Posner and Peterson (1990) argued that attention consists of at least three separate systems: a selection system responsible for selecting relevant stimuli; a vigilance system, responsible for maintaining readiness to respond in the absence of external cues; and an orientation system, responsible for engaging, moving, and disengaging attention.

Typically measures of selective attention involve the visual search for predetermined targets against competing and irrelevant foils (e.g., locating the knife-and-fork symbols which represent eating facilities on a road map; Robertson, Ward, Ridgeway, & Nimmo-Smith, 1996). In contrast, Continuous Performance Tests (CPTs), which demand sustained attention and vigilance, have been the most widely used measure of sustained attention deficits in children with ADHD (Denckla, 1996; Lin, Hsiao, & Chen, 1999). The CPT is a paradigm in which a series of stimuli (usually digits or numbers) are presented, and participants are required to respond to infrequent, randomly presented targets (Swaab-Barneveld et al., 2000). The dependent measures taken are the number of commission errors (i.e., failures to respond to the target signal, for which a response is required) and the number of omission errors (i.e., failures to withhold a response when no response is required). While commission errors (i.e., missed target signals) are generally attributed to failures of sustained attention (Robertson et al., 1997), omission errors are considered to reflect impulsivity (Swaab-Barneveld et al., 2000).

The results of studies using CPTs, however, have been equivocal with some investigations demonstrating significant deficits in sustained attention and vigilance (e.g., Aylward, Verhulst, & Bell, 1990; Barkley, Grodzinsky, & DuPaul, 1992) while others do not (e.g., Schachar, Logan, Wachsmuth, & Chajezyk, 1988; Van der Meere & Sergeant, 1988a, b). In a meta-analysis of 26 CPT studies, Losier, McGrath, and Klein (1996) found that children with ADHD performed significantly worse than non-ADHD controls in terms of both commission and omission errors. Oades (2000) also reported similar results with a sample of 14 ADHD children, 11 children with a tic syndrome, and 14 healthy controls, using two versions of the CPT (the standard paradigm, and the CPTax in which each target "x" must be preceded by an "a"). However, the limited size of the sample employed in this study would suggest that further research is necessary. Research by DeWolfe, Byrne, and Bawden (1999) has also examined the performance of 25 preschool children with ADHD using visual and auditory forms of the CPT. DeWolfe et al. (1999) found that although the ADHD children made significantly more commission and omission errors on the visual CPT, no significant differences were observed on the auditory CPT.

Swaab-Barneveld et al. (2000) used a visual CPT to study the performance of boys with a range of psychiatric diagnoses (including 52 ADHD boys and 55 healthy controls, 29 boys with oppositional defiant or conduct disorder, 29 boys with anxiety or dysthymia, and 43 boys with pervasive developmental disorder). The results revealed that ADHD children were slower, less accurate, more impulsive, less likely to adjust their behaviour in response to feedback, and showed a larger decrease in vigilance over time compared to normal controls. However, although the ADHD children were the only psychiatric group to be characterised primarily with an "attention deficit", Swaab-

Barneveld et al. (2000) found that deficits in sustained attention deficits were not specific to the ADHD group.

Attentional switching is measured using tasks that require an individual to frequently shift the focus of his/her attention, such as changing the direction of counting, in tasks which in themselves are relatively undemanding (Robertson et al., 1996). However, only a very limited amount of research appears to have examined this construct in ADHD children in recent years. Recent research by Cepeda, Cepeda, and Kramer (2000) for example used the task switching paradigm to examine attentional switching in 16 ADHD and 16 Control children (aged 6-12 years) matched on age and IQ. In the task switching paradigm, participants perform two simple tasks such as deciding whether a letter is a vowel or consonant or deciding whether a number is odd or even. In the baseline condition, participants perform the same task a number of times, whereas in the second condition they must switch between one task and the other. The increase in the response time provides a measure of the time required for the executive control processes to switch from one task to another. Cepeda et al. (2000) found that these "switch costs" were significantly larger in unmedicated ADHD children than in non-ADHD Controls, although their performance normalised on resumption of their normal medication regime. Furthermore, the performance of the ADHD children did not differ significantly from Controls on the non-switch trials regardless of stimulant medication, suggesting that these trials place only minimal demands on the executive processes.

A recent study using the Test of Everday Attention for Children (TEA-Ch; Manly, Robertson, Anderson, & Nimmo-Smith, 1999), found significant differences in sustained attention, attentional switching, and dual task

performance, between 24 ADHD children (mean age 10.0 years) and similarly aged Controls. However, no significant difference were found on the measures of selective (or focused) attention, and Manly et al. (1999) did not examine the relationship between attentional performance and ADHD subtype. Whilst the attentional characteristics of the ADHD subtypes have yet to be examined systematically, research to date intimates that the ADHD Predominantly Inattentive child may have more problems with focused or selective attention, information processing, and memory retrieval. In comparison, the ADHD Combined Type child may have more problems with persistence, working memory, and inhibition (Barkley, 1997b). However, as Barkley (1997b) pointed out, the results of such studies are not sufficiently consistent to conclude unequivocally that these two subtypes have a different attentional disturbance or different patterns of associated cognitive deficits.

The TEA-Ch was used in the present study since it provides measures designed to be sensitive to three types of attention in children (i.e., selective or focused attention, sustained attention, and attentional switching/dual task performance). This permitted the examination of a number of hypotheses pertaining to the nature of the attentional impairment(s) in unmedicated boys with ADHD (and no comorbid conditions), according to subtype and relative to Age-matched Control children. Further information about the TEA-Ch is provided in Chapter Four.

Concept of time

According to Bronowski, the basis for a sense of time derives from the ability to hold a sequence of events in working memory. By comparing these events against each other in any sequence, a sense of time and temporal duration arises (Barkley, 1997a). Barkley (1997b) subsequently predicted that as a consequence

of their hypothesised deficiencies in working memory, children with ADHD should manifest impairments in their sense of time and its associated retrospective (sensory) and prospective (motor) functions.

Evidence for impairment in the concept of time of ADHD children has been demonstrated in a number of studies using various paradigms and a variety of time durations. Typically, researchers have required participants to: (a) produce a verbally presented time interval by signalling the start and finish of the interval (such as by turning a light on and then off), (b) verbally report the duration of a previously presented time interval, and (c) reproduce a previously presented time interval in a similar manner. According to Barkley et al. (1997), it is this last paradigm (time reproduction) which is the most difficult to perform and may be the most rigorous means of testing the construct of time (see Barkley et al., 1997, for a more detailed description). Furthermore, these kinds of tasks place heavier demands on working memory (Barkley et al., 1997) and may thus more accurately represent the subjective sense of time (Zakay, 1990).

Cappella, Gentile and Juliano (1977) used durations varying from 7 seconds up to 60 seconds in their earlier time estimation studies with hyperactive and normal children. Results indicated that all children made larger errors as the time durations increased, but that hyperactive children made significantly larger errors in time production than Controls, and that the magnitude of these errors increased with the length of the duration to be reproduced. These findings were subsequently replicated by Walker (1982) who found that boys diagnosed as impulsive made significantly more errors in a time reproduction task.

More recently Barkley et al. (1997) conducted two studies to compare sense of time in children with and without ADHD. In a preliminary study, 32 unmedicated ADHD children aged 8 to 13 years were presented with time reproduction tasks in which they were required to replicate a given time interval using a flashlight. In one type of treatment condition a distractor (a Jack in the Box) was presented since such events have been shown to decrease the accuracy of time reproduction by children in comparison to non distractor periods (Zakay, 1992). The results indicated that ADHD children made significantly larger time reproduction errors than Controls at the 6 and 10 second durations with no distractor and at the 10 and 16 second durations with a distractor. Both groups increased the magnitude of their errors with increasing duration. The Jack in the Box served as both a visual and auditory distractor and while these had an effect on participant's performance no conclusions can be drawn as to which specific component (i.e., visual or auditory) was the effective distractor. The present research therefore attempted to address this issue by employing separate visual and auditory distractors.

Using the same testing procedure over five time durations (12, 24, 36, 48, and 60 seconds) Barkley et al. (1997) tested an additional 12 ADHD children and 26 Controls. Results revealed that the ADHD group made significantly larger errors of time reproduction, and that the magnitude of these discrepancies was increased by the presence of a distractor, particularly at the 12 to 36 second durations. Furthermore, the discrepancies increased with the length of the duration to be reproduced. Barkley et al. (1997) commented that the ADHD children appeared to be making larger time reproduction errors than Controls, but that the direction of these errors was quite variable. In conclusion it was suggested that ADHD children are more variable or erratic in their time reproductions than Controls.

Dooling-Litfin (1997) compared the performance of 16 ADHD children and 14 Controls aged between 8 and 11 years on a simple time reproduction task. During this study, the examiner demarcated six time intervals ranging from 2 to 60 seconds using simple verbal cues at the beginning ("Go") and end ("Stop") of the interval to be reproduced. Participants were then asked to reproduce the interval by saying "Go" and then "Stop" when he or she thought that the same amount of time had passed. Results demonstrated that ADHD children showed significantly larger absolute discrepancies (i.e., the magnitude of errors regardless of direction) than Controls. However Dooling-Litfin qualified these findings by suggestion that the lack of significance in direction of errors (that is, over- versus underproduction) may have been due to the greater variability in accuracy amongst the ADHD group.

More recently, Rubia, Taylor, Taylor, and Sergeant (1999) examined the motor timing synchronisation of boys with ADHD using motor timing anticipation and motor timing synchronisation tasks. In the anticipation task used by Rubia et al. (1999), participants were required to monitor the inter-stimulus interval between a stimulus (an airplane) which appears three times on a computer screen, and press a response button in anticipation of the appearance of the fourth and subsequent airplanes. In the motor timing synchronisation task, participants were required to synchronise their motor response (i.e., a button press) to the appearance of the stimulus airplane, which was presented at regular intervals on the computer screen. Whilst Rubia et al. found no significant differences between the ADHD and Control boys in their perception of time (as measured by the anticipation task), they reported that the boys with ADHD were impaired in the timing of their motor output (as measured by the synchronisation task). In particular, these boys were found to be more inconsistent in anticipating, self-regulating, and synchronising their motor

output to external visual stimuli. However, it is important to note that only 11 ADHD and 11 Control boys participated in this study and that the time discrimination task used simply involved deciding whether a five-second interval was followed another five second interval, or a shorter interval of three seconds' duration.

Although the findings from studies to date have suggested an impaired sense of time in children with ADHD, a number of issues may have confounded these studies and hence these need to be addressed in future research. For example, all of the studies to date appear to have involved small sample sizes which potentially limits their generalisability. Furthermore, as Barkley (1997a) suggested, since ADHD children have problems with motor control and persistence, requiring ADHD children to press and hold an activation button on a flashlight over long time durations might be problematic. In addition, order effects might exist since in most studies time durations were presented in a standard sequence. Finally, since research has suggested extensive comorbidity with ADHD (25%-30%, Barkley, 1997a; 38%, Langsford, 1999) this may confound any explanation linking impairments in sense of time with ADHD.

The present investigation sought to address these issues and to extend the work of Barkley et al. (1997) by using a larger sample of ADHD and Control boys matched for age and with no diagnosed comorbid conditions. The current research also used shorter time intervals to reduce demands on persistence, and used a new measure of time reproduction, which is described in detail in Chapter Four. Furthermore, the accuracy of time reproduction of ADHD and Control boys was evaluated using separate visual and auditory modes of presentation of the time reproduction tasks, and in the presence (and absence) of distractors.

Chapter summary

In summary, this literature review has shown that ADHD is a pervasive and impairing neurobiological/developmental disorder that affects between 3%-5% of the school-age population (although reported prevalence figures have been found to range from 1% to 25%). This literature review has also shown through historical developments that response inhibition and not attention appears to represent the central impairment in ADHD. Evidence from the literature has also suggested that an impairment in working memory might play a significant role in ADHD (e.g., Denckla, 1996; Barkley, 1997a). Barkley (1997c), for example, has suggested that the delayed internalisation of speech and attainment of rule-governed behaviour seen in ADHD children may occur as a result of deficient verbal working memory, and that impairments in sequencing and sense of time may result from impairments in non-verbal working memory. Thus, deficiencies in verbal and/or non-verbal working memory might account for many of the practical difficulties observed in ADHD children, including poor organisation of behaviour with respect to time, problems integrating temporal and spatial concepts, failure to apply past experience and knowledge in new situations, and dual task performance.

In Study One, these issues will be examined in further detail through a series of semi-structured interviews with leading international researchers in the field of ADHD research. These interviews will examine the current conceptualisation(s) of ADHD, the predicted executive impairments of ADHD children, and the types of instrumentation that might be sensitive to these impairments. This information will then guide the current research in a second major study in which the predicted executive impairments of children with ADHD will be identified and systematically examined using instrumentation specifically

designed for this purpose. It is anticipated that the results of this investigation will contribute to and extend current theoretical understanding of ADHD.

Research questions

The present research therefore sought to examine the current conceptualisation(s) of ADHD, arising from the theoretical and research literature and subsequently from interviews with leading international professionals in this field of research. From the literature reviewed and commensurate with the aims of the present research, the following research questions were formulated:

1. What are the current theoretical and clinical conceptualisation(s) of ADHD and its subtypes?

2. What are the predicted executive impairments of ADHD children and their observable manifestations?

3. What types of instrumentation are sensitive to these executive impairments?

4. To what extent can these predicted executive impairments be verified empirically?

5. How might this information be used to contribute to, challenge or extend the current conceptualisation of ADHD?