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TIME UNDER-UTILISATION BY CHILDREN WITH ATTENTION-DEFICIT / HYPERACTIVITY DISORDER

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Abstract

The literature review provides an introduction into the highly prevalent, disabling developmental disorder known as Attention-Deficit/Hyperactivity Disorder (AD/HD). It then describes the aetiology behind the condition and current psychological theories that attempt to account for the core features of AD/HD. Three types of psychological theories are described: (a) the theory of behavioural disinhibition and executive dysfunction, (b) theories of premature task disengagement and (c) the state regulation deficit model. Psychological theories are then considered specifically in relation to the AD/HD child's difficulty using time effectively. Finally, areas for future research are highlighted.

The empirical paper aims to explore children with AD/HD's use of time by testing the predictions made by competing theories of AD/HD with regards to children with AD/HD's performance on the Matching Familiar Figures Task; a task that requires children to identify a target from amongst five similar foils. Twenty-five children with AD/HD and 25 controls completed the task under four different trial duration conditions (5-, 10-, 15- and 20-seconds). Control children significantly out-performed AD/HD children on all conditions except the 20-second trial duration condition. AD/HD children's poor performance appeared to be best explained by two factors: (a) their tendency to examine fewer stimuli than their peers and (b) their tendency to be slower to initiate a search. These deficits could reflect (a) poor motivation, (b) a state of underarousal, (c) "slowness" when planning / implementing searches and (d)

difficulties adapting search approach / pace in accordance with the time available. Further research is recommended.

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Literature Review

ATTENTION-DEFICIT/HYPERACTIVITY DISORDER, TIME USE AND TASK PERFORMANCE: WORKING TOWARDS A COMPREHENSIVE THEORETICAL UNDERSTANDING

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Prepared for submission to the Journal of Abnormal Child Psychology (See Appendix A for author instructions)

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Abstract

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Attention-Deficit/Hyperactivity Disorder (AD/HD) is a chronic and pervasive developmental disorder characterised by inattention, impulsivity and hyperactivity. It affects 3-5% of the childhood population and is frequently associated with a range of co-morbid conditions. The precise aetiology of AD/HD remains unknown but a range of genetic / neurological factors and psychosocial factors are widely believed to be implicated.

A number of competing theories attempt to explain the core characteristics of AD/HD. In this review, three types of theories are described: (a) the theory of behavioural disinhibition and executive dysfunction, (b) theories of premature task disengagement (including sustained attention deficit, the need for optimal stimulation and the theory of delay aversion), and (c) the state regulation deficit model. To date, there has been little attempt to unite these different theories. However, Sonuga-Barke (2002b) attempted to test them against one another by comparing their respective abilities to explain AD/HD children's tendency to under-utilise time on the Matching Familiar Figure Task, a simple computer task that requires children to identify a target from amongst five similar foils. The results provided greatest support for the state regulation deficit model, although further research is recommended in terms of analysing AD/HD children's search strategies in order to investigate whether highly context-specific compensatory strategies are being employed.

Introduction

This literature review has five main aims. First, to provide a general introduction into Attention-Deficit/Hyperactivity Disorder (AD/HD) in terms of its core characteristics, diagnostic criteria, prevalence, developmental course and co-morbidity. Secondly, to give a brief overview of its aetiology. Thirdly, to review the leading theories of AD/HD including the theory of behavioural disinhibition and executive dysfunction, theories of premature task disengagement and the state regulation deficit model. Fourthly, to consider how these theories can help to understand and better account for AD/HD children's tendency to under-utilise time. And, fifthly, to identify future areas for research that would allow us to further develop our knowledge of this serious and disabling developmental disorder with respect to time under-utilisation.

General introduction into AD/HD

Diagnosis of AD/HD

Attention-Deficit/Hyperactivity Disorder is a chronic and pervasive developmental disorder characterised by inattention, impulsivity and hyperactivity (Barkley, 1998). Webster-Stratton (2001) provides a typical description of a child with AD/HD-type behaviours:

Cory is six years old and his mother often says, "He's so different from his older brother. If I'd had him first I would have never have had another child!" Although Cory can still watch television, he is otherwise restless and easily distracted, constantly moving from one thing to another. He talks loudly, gets excited easily and is difficult to put to bed at night ... His parents feel exhausted from the constant need to monitor his behaviour ... In kindergarten, the teacher thinks of him as a troublemaker ... he won't listen to instructions or stick with one activity ... Recently, his parents have been even more worried because he says "I'm bad" and his usually sunny disposition has been replaced by a defiant attitude. (p. 232)

The Diagnostic and Statistical Manual of Mental Disorders - Fourth Edition (DSM-IV; American Psychiatric Association, 1994) describes three subtypes of AD/HD: an "inattentive" subtype, a "hyperactive-impulsive" subtype and a "combined" subtype. A diagnosis of the inattentive subtype requires the presence of at least six of the following characteristics: carelessness with details, failure to sustain attention, inability to listen, failure to complete tasks, poor self-organisation, avoidance of tasks requiring sustained mental effort, ready loss of possessions, distractibility and forgetfulness. A diagnosis of the hyperactive-impulsive subtype requires the presence of at least six of the following characteristics: fidgetiness, difficulty remaining seated when required, running or climbing inappropriately, noisiness in play, persistent motor over-activity, blurting out answers before questions completed, failure to wait turn or queue, interrupting conversations or games and talking excessively. A diagnosis of the combined subtype requires the presence of at least six of any of the characteristics described above. Furthermore, for all subtypes, features must be severe or frequent, have been present continuously from the pre-school years, pervasive (in

other words, occur in two or more settings) and impair social, academic and/or occupational functioning. In addition, the symptoms should not be better accounted for by any other disorder.

Attention-Deficit/Hyperactivity Disorder is not classified in the International Classification of Diseases (ICD-10; World Health Organisation, 1990). However, Hyperkinetic Disorder is listed and this represents a similar but more severe form of the disorder (Hill, 2000). Internationally, AD/HD is the more widely used of the two diagnostic labels (Hill, 2000) and thus is the term that will be use here.

Given that the concept of AD/HD is multi-faceted, it is essential that the diagnostic process should consider information provided by parents, teachers, child psychologists and physicians (British Psychological Society, 2000). Nevertheless, it is appropriate that a psychiatrist or paediatrician should be responsible for making the formal diagnosis (British Psychological Society, 2000). Furthermore, diagnosis should only be made after other possible explanations for the existence of the problem have been fully explored and discounted. For example, in some cases a diagnosis of oppositional defiant disorder or autism may be more appropriate (British Psychological Society, 2000).

Prevalence of AD/HD in community samples

The prevalence of AD/HD in community samples ranges from 1.9 percent to 14.4 percent (Scahill & Schwab-Stone, 2000). The highest

estimate (14.4%) comes from Gomez-Benevto, Bonet, Catala, Puche and Vila (1994) who randomly sampled 400 eight-year-old Spanish children using the Kiddle Schedule for Affective Disorders and Schizophrenia. The lowest estimate (1.9%) comes from Costello, Angold, Burns, Stangl et al. (1996) who studied 4067 9-to-13 year old children using the Child and Adolescent Psychiatric Assessment; in other words this study used a considerably larger sample, older children and a different measure than Gomez-Benevto et al's study. One obvious explanation for the difference in documented prevalence rates may be that there is an incremental decline in prevalence with age (Scahill & Schwab-Stone, 2000). Support for this explanation comes from Breton, Bergeron, Valla, Berthiaume and Gaudet (1999) who reported a prevalence of 5.8 percent among six-to-eight year old children, four percent in 9-to-11 year-old children and 2.5 percent in 12-to-14 year olds. However, the consensus of opinion asserts that approximately 3-5% of the childhood population fulfils the diagnostic criteria for AD/HD (American Psychiatric Association, 1994).

Both community and clinical samples show a preponderance of boys with AD/HD. Clinical samples report male-to-female ratios as high as nineto-one (Barkley, 1998). However, in community samples the male-to-female difference is generally not as marked. For example, Jensen et al. (1995) and Szatmari, Offord and Boyle (1989) observed a male-to-female ratio of 1.5-to-1. Breton et al. (1999) and Costello, Angold, Burns, Erkanli et al. (1996) reported slightly higher male-to-female ratios of two-to-one and three-to-one respectively. The over-representation of males with AD/HD in clinical

samples may lie in the fact that boys are most likely to present with the hyperactive-impulsive subtype, whereas girls are most likely to present with the inattentive subtype (Scahill & Schwab-Stone, 2000). Baumgaertel, Wolraich and Deitrich (1995) reported that the inattentive subtype of AD/HD had a male-to-female ratio of two-to-one whereas the hyperactive-impulsive subtype had a ratio of five-to-one. It may be that the hyperactive-impulsive subtype is more likely than the inattentive subtype to lead to a referral as it causes more problems at home and at school in terms of behaviour management. In other words, disruptive behaviour may be perceived to be more unacceptable and less tolerable than inattentive behaviour.

Developmental course of AD/HD

Attention-Deficit/Hyperactivity Disorder should be evident from a very young age and tends to follow a fairly typical developmental course. A good description of the difficulties experienced by children with AD/HD across childhood is provided by the British Psychological Society (2000), as summarised below.

Although AD/HD is very rarely diagnosed during the pre-school years, symptoms should already be present. These may include being unusually active once crawling / walking, experiencing problems with the acquisition and development of speech and language, a tendency towards non-compliance with parental rules and requests, difficulties sustaining interest in play activities and poor impulse control. Other symptoms can also be present but may be better accounted for by other developmental disorders,

such as a lack of interest in interpersonal interactions and a resistance to changes in routine.

Once children with AD/HD start primary school their symptoms typically become more marked and increasingly problematic. They are readily identified as consistently displaying a number of undesirable behaviours including: (a) failure to comply with rules, for example talking in class or running in corridors; (b) an inability to stay on task; and (c) forgetfulness and poor organisation, for example failing to bring correct equipment to lessons. Such behaviours tend to result in disciplinary action and as a consequence adversely impact on the child's academic performance and self-esteem. Children with AD/HD are also likely to experience problems with peer relationships as they struggle to engage in group work or co-operative play.

As children with AD/HD progress through their school career, they tend to encounter repeated experiences of failure both socially and academically due to the inherent characteristics of the disorder. The problems experienced in earlier school years are intensified as the expectations placed on the young person increase and fewer allowances are made for "bad" behaviour. The adolescent with AD/HD is frequently portrayed as defiant and lacking in interest which, combined with academic failure and poor peer relationships, can result in a significant and worrying lack of self confidence. Without appropriate interventions, repeated negative experiences may lead to the emergence of oppositional defiant disorder or

conduct disorder, and to the adolescent leaving school with few or no qualifications.

Many young people cease to display symptoms of AD/HD as they enter into adulthood. Nevertheless, for those individuals who continue to be troubled by the disorder, a number of additional problems may subsequently be encountered including difficulties obtaining and sustaining employment, failed relationships, depression, substance misuse and criminality (Weiss & Hecthman, 1993).

Co-morbidity of AD/HD with other disorders

In both clinical and community samples, children with AD/HD are at high risk of presenting with another psychiatric disorder (Scahill & Schwab-Stone, 2000). Oppositional defiant disorder (disobedient, anti-authoritarian behaviour) and conduct disorder (widespread serious violation of the rights of others) are the most frequently observed (Scahill & Schwab-Stone, 2000). Nevertheless, internalising disorders such as anxiety, depression or somatic complaints are also common (Scahill & Schwab-Stone, 2000). Goldstein and Goldstein (1999) reviewed research indicating that between 16-50 per cent of school children with AD/HD experience co-morbid oppositional defiant disorder or conduct disorder, whilst 30-50 per cent of adolescents with AD/HD experience depressive disorders and anxiety symptoms. It is suggested that co-morbid mental health problems may develop as a consequence of the way in which the social environment reacts to the core problems of AD/HD (British Psychological Society, 2000).

At least half of all children with AD/HD also have specific developmental difficulties such as dyslexia, dyspraxia and/or dysgraphia (Hill, 2000). However, estimating the proportion of children with AD/HD and learning disabilities remains unclear. As described by Hale, Hariri and McCracken (2000), early research tended to report a widely varying prevalence of learning disabilities with AD/HD (15%-92%) while more recent studies, using more stringent criteria, typically indicate a lower prevalence (10%-23%). Hale et al. (2000) suggest that it may be difficult to differential diagnose AD/HD and/or a learning disability due to the potential for shared symptoms and common aetiology.

Finally, specific medical conditions have also been found to be comorbid with AD/HD including foetal alcohol syndrome, fragile-X chromosome disorder, Williams syndrome, hearing impairment, thyroid dysfunction and epilepsy (Taylor, 1994).

Summary

Attention-Deficit/Hyperactivity Disorder is a chronic and pervasive developmental disorder characterised by inattention, impulsivity and hyperactivity. DSM-IV describes three sub-types: inattentive, hyperactiveimpulsive and combined type. ICD-10 does not include AD/HD but describes Hyperkinetic Disorder, which represents a severe form of the same disorder. AD/HD is a prevalent disorder affecting 3-5% of the childhood population. It has the potential to cause a range of difficulties academically, emotionally

and socially. It also places the child at risk of developing a number of other psychiatric disorders, including oppositional defiant disorder, conduct disorder, anxiety, depression and somatic complaints. Furthermore, in the long-term, adults with AD/HD frequently experience problems gaining employment and maintaining relationships. They are also at risk of developing affective disorders, substance misuse and criminal behaviours.

Aetiologies of AD/HD

Historically, brain damage, due to infection, trauma or complications during pregnancy/delivery, was considered to be the fundamental cause of AD/HD (Barkley, 1998). However, most children with AD/HD have no history of significant brain injury (Rutter, 1977). Nevertheless, the similarities between symptoms of AD/HD and those produced by lesions or injuries to the prefrontal cortex are striking (Benton, 1991; Mattes, 1980). Children with prefrontal cortex damage present with deficits in sustained attention, inhibition, regulation of emotions, regulation of motivation and capacity to organise behaviour over time (Grattan & Eslinger, 1991). It may be that whilst children with AD/HD do not have brain damage per se, they have abnormalities in brain development within these particular regions, the causes of which are unknown but likely to be associated with genetic effects (Barkley, 1998). Thus, it seems likely that hereditary factors play a significant role in the aetiology of AD/HD and specifically may result in a smaller and less active prefrontal-striatal network (Barkley, 1998).

Evidence of brain abnormalities in children with AD/HD has increasingly been demonstrated with the advancement of neuroimagining techniques. Neuroimagining techniques provide direct assessment of brain structure and function and are therefore well placed for testing hypotheses concerning the locus of brain dysfunction (Hale et al., 2000). As reported by Hale et al. (2000), evoked response potential (ERP), positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) studies have all repeatedly found abnormal frontostriatal activity to be associated with AD/HD children's inability to successfully perform tasks requiring skills of attention and execution. In addition, fMRI studies of attentional processes in AD/HD have revealed disturbances in the anterior cingulated cortex, a region that has specifically been implicated in stimulus selection and response inhibition (Hale et al., 2000).

Convincing evidence for the hereditary nature of AD/HD comes from twin and family studies. Many large-scale twin studies have consistently shown that as much as 80% of the variance in the trait of hyperactivityimpulsivity is the result of genetic factors; in addition, it appears likely that the genetic contribution may increase the more extreme the scores along this trait (Gjone, Stevenson, Sundet & Eilertsen, 1996; Levy, Hay, McStephen, Wood & Waldman, 1997; Sherman, Iacono & McGue, 1997, Sherman, McGue & Iacono, 1997). Generally, as little as 0-13% of the variance among individuals is accounted for by the shared environment (influences shared by children growing up in the same family) (Levy, Hay, McStephen, Wood & Waldman, 1997; Sherman, Iacono & McGue, 1997). Similarly, family studies

have repeatedly shown an increased incidence of AD/HD in the relatives of AD/HD probands (Biederman, Faraone, Keenan, Knee & Tsuang, 1990; Faraone, Biederman, Keenan & Tsuang, 1991; Perrin & Last, 1996; Schachar & Wachsmuth, 1990).

Molecular genetic studies, using methods of linkage and association to search for aberrant genes that cause disease, also look at the genetic component of AD/HD. Although their results are still tentative, this line of research suggests that three genes may increase the susceptibility to AD/HD: the D4 dopamine receptor gene, the dopamine transporter gene and the D2 dopamine receptor gene (Faraone & Biederman, 1998).

Whilst genetically caused brain abnormalities are the most likely cause of AD/HD, it is suspected that the condition may also be partially caused by pregnancy complications and/or exposure to toxins (Barkley, 1998). Some studies have reported a slightly higher prevalence of short or long labour, foetal distress, toxaemia, eclampsia and low birth weight in children with AD/HD compared to controls (Hartsough & Lambert, 1985; Minde, Webb & Sykes, 1968; Szatmari, Saigal, Rosenbaum & Campbell, 1993). Other studies have found a relationship between AD/HD and elevated body lead burden (Needleman, Schell, Bellinger, Leviton & Alfred, 1990), and AD/HD and prenatal exposure to alcohol or tobacco smoke (Bennett, Wolin & Reiss, 1988; Shaywitz, Cohen & Shaywtiz, 1980; Steissguth et al. 1984).

In terms of psychosocial causes of AD/HD, it is widely believed that AD/HD is not predominantly the result of poor parenting or child mismanagement (Barkley, 1998). Research demonstrates that the overly critical, commanding and negative behaviour of mothers of AD/HD children is most likely to be a reaction to the disruptive behaviour of the child and not the cause of it (Barkley, Karlsson, Pollard & Murphy, 1985; Barkley, Karlsson, Strelecki & Murphy, 1984; Humphries, Kinsbourne & Swanson, 1978). However, some researchers would disagree with this stance. For example, Sonuga-Barke (2002a) suggests that impulsivity may be the result of an aversion to delay acquired through repeated experiences of parents setting unrealistically high standards with respect to appropriate behaviour (particularly self-control) and being unforgiving of children's failures to wait. Furthermore, Sonuga-Barke (2002a) proposes that effective and positive parenting may have the potential to ameliorate the negative effects of genetic and non-genetic predisposing factors.

Whilst there is not a total consensus of opinion with regards to the importance of psychosocial factors in the development of AD/HD, it is nevertheless agreed that the actual severity of symptoms, the persistence of symptoms over age and the development of oppositional difficulties may well be related to the way parents attempt to manage the AD/HD child's behaviour (Campbell, 1987; Campbell & Ewing, 1990; Van den Oord & Rowe, 1997; Weiss & Hechtman, 1993).

In contrast to the idea that all cases of AD/HD are caused exclusively either by brain abnormalities or by psychosocial factors, Sonuga-Barke (2002a) proposed a "dual pathway model" whereby AD/HD may arise out of either of these two quite distinct processes. In other words, it may be that neither neuro-biological factors nor psychosocial factors are adequately able to account for all cases of AD/HD. As suggested by Sonuga-Barke, future research needs to explore the coherence of this model and the extent to which the two pathways act interactively and/or additively.

Summary

Over the years, there has been considerable debate with regards to the aetiology of AD/HD, particularly with respect to whether or not it is fundamentally a neuro-biological or a psychosocial disorder. In support of the neuro-biological argument, there is now considerable evidence that children with AD/HD display brain abnormalities. A variety of genetic and neurological factors are believed to result in or exacerbate the symptoms by impairing the development of the prefrontal cortical-striatal network. With regards to the importance of psychosocial factors, these are no longer widely believed to be the sole cause of the disorder. Nevertheless, they are frequently seen as influencing the severity or persistence of the symptoms and to give rise to co-morbid problems such as oppositional behaviour. However, Sonuga-Barke (2002a) proposed that neither neuro-biological nor psychosocial factors may be able to exclusively account for all cases of AD/HD and a dual pathway model may be more appropriate.

Theories of AD/HD

Currently, there are a number of competing theories of AD/HD that all attempt to explain and account for the core features of the condition: inattention, impulsivity and hyperactivity. In this review, three different leading theories will be described: (a) the theory of behavioural disinhibition and executive dysfunction; (b) theories of premature task disengagement (including the sustained attention deficit theory, the optimal stimulation theory and the delay aversion theory); and (c) the state regulation deficit model. Given the extensive number of research studies published in all these areas, it is beyond the scope of this review to exhaustively cover all the relevant literature. Nevertheless, a good, selective overview is intended.

The theory of behavioural disinhibition and executive dysfunction

Barkley (1997) attempted to provide a comprehensive theory of prefrontal lobe functions in order to explain the difficulties experienced by children with AD/HD; the theory of behavioural disinhibition and executive dysfunction. His model proposed that efficient goal-directed behaviour, or "motor control", relies on the application of four key executive functions: nonverbal working memory, verbal working memory, the self-regulation of affect / motivation / arousal and reconstitution. Essentially, executive functions aim to shift behaviour from the control of the immediate environment to the control of internally represented forms of information.

As briefly stated by Barkley (1997), non-verbal working memory enables children to hold in their mind at any one time information about all

aspects of the task at hand (and similar past experiences) in order to plan, organise and modify future behaviour. It is termed representational memory by developmental psychologists. Verbal working memory refers to the capacity of a child to converse with oneself in a quasi-dialogue-like fashion. It is an important skill as it enables children to verbally contemplate an event prior to responding, to self-question and to problem solve. The self-regulation of affect / motivation / arousal reflects the ability to privatise emotions and provide internal motivation. Children must have the capacity to internally motivate themselves in the absence of immediate external rewards if they are to persist successfully with goal-directed behaviour in the pursuit of longer-term outcomes. Reconstitution represents the ability to analyse past behaviours and synthesise new, more effective behaviours. This skill enables children to be flexible and creative with their behaviour in order to obtain the best possible outcome in any given situation.

Barkley (1997) proposed that children with AD/HD struggle to effectively utilise their executive functions due to their tendency to behave impulsively. There is considerable evidence that children with AD/HD show signs of impaired executive functioning (Carte & Treuting, 1998; Chang et al., 1999; Cornoldi, Barbieri, Gaiani & Zocchi, 1999; Frank, Seiden & Napolitano, 1996; Karatekin & Asarnow, 1998; Mariani & Barkley, 1997; O'Neill & Douglas, 1996). Pennington and Ozonoff (1996) reviewed 18 studies investigating executive dysfunction in children with AD/HD. They found that the tasks that most consistently found group differences between AD/HD and control children included: stop tasks, GO / NO-GO tasks, anti-saccade tasks,

conflict motor tasks, the Neuropsychological Assessment for Children, Tower of Hanoi, stroop colour-word test and the Matching Familiar Figures Task. In contrast, tasks that least consistently found group differences were letter and category fluency tasks and the Wisconsin Card Sorting Task.

Barkley's (1997) theory of AD/HD proposes that impulsivity is essentially the result of "behavioural disinhibition". Unlike children without AD/HD, children with AD/HD find it hard to delay the impulsive decision to respond to an event and thus miss the opportunity to utilise executive functions. Barkley (1997) distinguishes three main causes of behavioural disinhibition. First, children with AD/HD struggle to inhibit the initial "prepotent" response to an event. The prepotent response is the one for which the child will receive immediate reinforcement; the reinforcer may be negative, in terms of enabling the child to escape or avoid an aversive consequence, or positive, in terms of enabling the child to gain or access a desired consequence. The inability to inhibit prepotent responses reflects a lack of "self-control". Secondly, children with AD/HD struggle to stop ongoing responses or response patterns even when they prove ineffective. Their motor behaviour lacks flexibility; this is likely to be due, at least in part, to a lack of insight into the effectiveness of current behaviour due to inadequate self-monitoring. Thirdly and possibly most importantly, children with AD/HD are highly vulnerable to both external and internal sources of interference; they display poor "interference control". Interference control is critical in the persistence of goal-directed behaviour and represents a special form of sustained attention.

Evidence in support of AD/HD children's problems with behavioural inhibition is derived from studies using stop signal paradigms, an empirical measure of the ability to interrupt an ongoing response (Logan & Cowan, 1984; Logan, Cowan & Davies, 1984). The stop signal paradigm involves a simple reaction time task where the child needs to respond as fast and accurately as possible to stimuli presented on a computer screen. However, on trials where an auditory stop signal is presented, the participant should withhold from responding; the participant must suppress a response that is already in the process of being executed. The longer the delay between the onset of the stimulus and the onset of the stop signal, the more difficult it is to withhold from responding. The stop signal paradigm has been found to be a reliable and valid measure of response inhibition (Kindlon, Mezzacappa & Earls, 1995; Tannock, Schachar, Carr, Chajczyk & Logan, 1989).

Oosterlaan, Logan and Sergeant (1998) conducted a meta-analysis of eight studies using the stop signal paradigm to investigate response inhibition in children with AD/HD. They found that AD/HD children had flatter "inhibition functions" than the control group, indicating that children with AD/HD were less able than controls to inhibit inappropriate responses. As stated by Oosterlaan et al. (1998):

The inhibition function ... reflects the efficiency of the inhibitory mechanisms controlling for differences in mean reaction time. Most researchers take the slope of this inhibition function as an index of the

subject's capability for response inhibition. The flatter the inhibition function slope, the poorer the capability for response inhibition. (p.413)

Inhibition functions are calculated by plotting the probability of inhibition against the stop signal delay; fitting a regression line to the inhibition function generates the slope (Oosterlaan et al., 1998). It should be noted that Oosterlaan et al. (1998) found that children with conduct disorders showed similar impairments to those children with AD/HD. Hence, the results suggested that poor behaviour inhibition is characteristic of all children with disruptive behaviour and may not be a deficit specific to those with AD/HD.

Kuntsi and Stevenson (2000) describe a number of possible explanations for the flatter inhibition function associated with AD/HD (and possibly also children with conduct disorders); these are described below. First, children with AD/HD may be less likely to trigger the inhibitory process or their inhibitory process may be more variable. Oosterlaan et al. (1998) investigated these possibilities by correcting for the ZRFT (z score of the relative finishing time); correction for the ZRFT controls for the effects of stop signal reaction time and variability of speed on the inhibition function (see Logan (1994) for further details). Interestingly, they found that AD/HD children did not differ significantly from their peers on the ZRFT-slope (slope of the inhibition function plotted as a function of ZRFT). This indicated that children with AD/HD were as likely to trigger the inhibitory process as controls. In addition, their inhibitory processes were no more variable.

Secondly, the flatter inhibition function associated with AD/HD could reflect a slower inhibitory process. In support of this hypothesis, Oosterlaan et al. (1998) reported that AD/HD children were significantly slower than the control group on the reaction time task. Thirdly, the flatter inhibition function could be due to AD/HD children's greater variability in responding on the reaction time task. This possibility was not explored by Oosterlaan et al. (1998). In conclusion, Kuntsi and Stevenson (2000) suggest that "rather than indicating a specific response inhibition deficit, the overall pattern of the findings may suggest a generally slow mode of information processing" (p.14).

It is important to note that studies employing the stop signal paradigm have been criticised. For example, Sonuga-Barke (1995) pointed out that they focus only on <u>momentary</u> inhibition, the ability to suppress a particular response when it is signalled. However, Barkley's theory (1997) proposed that AD/HD involves difficulties with <u>ongoing</u> inhibition, the ability to suppress responding over a period of delay. This would suggest that the stop signal paradigm does not explicitly test the inhibition deficit described by Barkley (1997).

In summary, the deficits described above result in a motor control system that is not under the same degree of control as would be evident in children without AD/HD. Generally, children with AD/HD behave in ways that are less effective than those displayed by their peers because they fail to implement executive functions. In addition, even when they do activate executive functions, they are often less proficient than those found in control

children due to the inferior development of behavioural inhibition. As a rule of thumb, children with AD/HD know what they should do or should have done but this knowledge appears irrelevant when behaving in the moment (Barkley, 1998). Essentially, children with AD/HD encounter problems because their tendency to behave impulsively does not afford them adequate time to plan and organise themselves (Barkley, 1998). Unfortunately, Barkley's (1997) theory does not explain why children with AD/HD are sometimes able to withhold responding and thus apparently display behavioural inhibition.

Theories of Premature Task Disengagement

Unlike Barkley's theory of behavioural disinhibition and executive dysfunction, theories of premature task disengagement suggest that the performance of children with AD/HD is not constrained by problems with behavioural inhibition, but by their style of task-engagement (Sonuga-Barke, 2002a). Children with AD/HD have a tendency to disengage from a task of fixed duration before it has actually come to an end (Alberts & Van der Meere, 1992). This means that children with AD/HD can perform relatively well on short tasks but poorly on lengthy ones (Chee, Logan, Schachar, Lindsay & Wachsmuth, 1989; Rubia, Taylor, Taylor & Sergeant, 1999; Sonuga-Barke & Taylor, 1992). The poor performance on lengthy tasks is explained by the fact that children with AD/HD miss the opportunity to derive added value from additional contact towards the end of a task in the way that children without AD/HD are able to (Conte, Kinsbourne, Swanson, Zirk & Samuels, 1986; Dalby, Kinsbourne, Swanson & Sobel, 1977).

Premature task disengagement may be attributed to (a) difficulties sustaining effort over time in the maintenance of an effective response set (Douglas, 1983), (b) the need for optimal stimulation (Zentall & Zentall, 1976, 1983), and (c) delay aversion (Sonuga-Barke, 1994), as will be discussed below.

(1) Difficulties sustaining attention over time

Douglas (1983) described children with AD/HD as primarily experiencing difficulties with "sustained attention", the ability to maintain attention over time. A few theorists have noted the importance of effort in attention (Broadbent, 1977; Posner, 1978). However, Douglas states that generally there has been a lack of interest in problems concerning the investment and/or organisation of effort, and with respect to the factors within tasks, that make concentration easy or difficult.

Douglas (1983) proposed that difficulties with sustained attention are particularly apparent when children with AD/HD are required to undertake complex tasks. Complex tasks require "careful, organised, perceptual search strategies, or an exhaustive analysis of possible solutions to a logical problem" (p.285), all of which rely on the investment of effort and the inhibition of impulsive responding. Essentially, the inability to maintain effort over time results in premature task engagement, which consequently impairs task performance.

Difficulties sustaining attention are not necessarily considered to reflect pervasive deficits. Douglas recognises that children with AD/HD demonstrate the ability to be highly attentive in certain situations, typically those that are of "high-interest". For example, when playing a computer game or watching a favourite television programme. This would suggest that difficulties sustaining attention are closely associated with poor internal motivation on the part of the AD/HD child.

Evidence in support of a sustained attention deficit comes from AD/HD children's performance on continuous performance tasks (CPT). A CPT requires the participant to respond to target stimuli (typically letters or digits) as they appear on a computer screen whilst refraining from respond to non-target stimuli. Several studies have found that AD/HD children respond more frequently to non-target stimuli and are more likely to fail to respond to target stimuli when compared to control children (Corkum & Siegel, 1993; Losier, McGrath & Klein, 1996). However, Van der Meere and Sergeant (1988) suggest that a group difference in the "overall" CPT performance should not be taken as evidence of a sustained attention deficit. They propose that to demonstrate such as deficit, studies should demonstrate a significant decline in AD/HD children's performance over time. The majority of studies have failed to find such evidence (Van der Meere, 1996).

(2) The need for optimal stimulation

According to the theory of optimal stimulation, the activity of children with AD/HD increases when they are confronted with a stimulus-poor

environment due to their high stimulation threshold (Zentall & Zentall, 1976, 1983). In other words, children with AD/HD readily disengage from tasks in order to search for, or attend to, higher levels of stimulation. The theory is a feedback model based on the assumption that "response output functions to homeostatically regulate the level of stimulus input" (Zentall & Zentall, 1983, The optimal stimulation theory was developed from previous p. 446). theories that hypothesised that all organisms have a biologically predetermined optimal level of stimulation and when this level of stimulation is absent, activity serves as a "homeostatic regulator" (for example, Berlyne, 1960; Duffy, 1962; Hebb, 1955; Pavlov, 1927). In essence, all organisms will initiate stimulation-seeking activity when there is either insufficient or inadequate stimulation in the environment. The curvilinear models of Hebb (1955) and Berlyne (1960) view arousal as the physiological representation of environmental stimulation. Indeed, Berlyne suggests that individuals are aroused by particular stimulus characteristics including size, colour, sensory modality, affective connotations, novelty, complexity, degree or suddenness of change, incongruity and/or uncertainty.

Zentall and Zentall (1983) proposed that children with AD/HD behave as though normal levels of environmental stimulation are insufficient. They appear to suffer from a state of under-arousal. In essence, these children are unusually sensitive to low-stimulation, boring or repetitive environments. The increased activity displayed by children with AD/HD in such environments functions to increase visual or kinaesthetic input (Zentall, 1975, 1977). This increased activity can present as increased locomotor activity,

looking around behaviour and/or excessive talking. Zentall and Zentall (1983) also suggest that children with AD/HD seek additional stimulation when engaged in complex or difficult tasks as they are unable to progress through the task at a rate fast enough to provide adequate stimulation. In other words, during the delay in responding that is inherent in difficult tasks, AD/HD children are likely to engage in stimulus-seeking behaviours.

Evidence to support the optimal stimulation theory comes from research that finds that children with AD/HD are not behaviourally different from controls in high-stimulation settings such as strange, novel or playground settings (Minde et al., 1971; Stewart, 1970; Zentall, 1975). In addition, when activity levels are directly compared in low and high stimulation environments, by either (a) directly manipulating environmental stimulation using either a boring task or no task (Zentall & Zentall, 1976) or (b) observing naturally occurring novel and familiar classroom settings (Zentall, 1980), research finds that increased activity by hyperactive children is shown only in the low-stimulation contexts.

Whilst evidence suggests that children with AD/HD perform well in new situations, since novelty provides adequate stimulation in itself, it should be noted that the novelty of any situation inevitably decreases with time and thus increased activity will gradually be observed (Zentall, 1975). Hyperactivity is believed to provide both proprioceptive and visual stimulation and thus compensates for the natural loss of task novelty over time (Cohen & Douglas, 1972; Reardon & Bell, 1970).

Zentall and Zentall (1983) suggest that temporary increases in stimulation-seeking behaviour by children with AD/HD might not automatically impair task performance. They propose that limited hyperactivity and/or inattention might allow these children to perform more efficiently when they are attending to the relevant task at hand. However, such compensatory strategies are unlikely to be beneficial during tasks that require continuous visual attention. Zentall and Zentall (1983) recommend that these tasks should be made as stimulating as possible for the AD/HD child by adding stimulation directly to the task materials.

In summary, the optimal stimulation theory posits that all organisms work to maintain an optimal level of arousal and for children with AD/HD, the desired level of arousal is unusually high. Large deviations from the optimal level of stimulation will represent an aversive state and consequently, will tend to result in compensatory behaviours. Thus, for children with AD/HD, when in low-stimulating environments, they will be motivated to seek out additional stimulation, typically by becoming hyperactive and apparently inattentive.

(3) <u>Delay aversion</u>

Sonuga-Barke and colleagues (Sonuga-Barke, 1994; Sonuga-Barke, Taylor & Heptinstall, 1992; Sonuga-Barke, Taylor, Sembi & Smith, 1992; Sonuga-Barke, Williams, Hall & Saxton, 1996) proposed that premature task disengagement might be best explained by "delay aversion". In situations

where a delay is enforced on the child, the child with AD/HD will be motivated to disengage from the task and create additional non-temporal stimulation. Non-temporal stimulation alters the subjective experience of delay and typically presents in the form of hyperactive behaviour. The delay-aversion theory represents the most radical departure from the traditional neuro-psychological paradigms (Sonuga-Barke, 1994) since it considers AD/HD behaviours to be "functional expressions of an underlying motivational style rather than the result of dysfunctional regulatory systems" (Sonuga-Barke, 2002a, p.30).

Evidence in support of the delay aversion theory is derived from the Matching Familiar Figures Test (MFFT; Kagan, 1965). The MFFT is a simple computer task that requires children to identify a target from amongst five similar foils. Children with AD/HD tend to respond quickly and inaccurately; they are frequently described as behaving "impulsively" (Barkley, 1998). However, Sonuga-Barke et al. (1996) suggested that early responding may not reflect an inherent problem with response inhibition; it could reflect an attempt to reduce trial length. In other words, it cannot be assumed that children with AD/HD are <u>unable</u> to withhold from responding. They may simply want to finish tasks as quickly as possible, irrespective of the adverse impact this may have on performance.

A study by Sonuga-Barke, Houlberg and Hall (1994) provided support for the delay aversion theory. They compared AD/HD children's performance on the MFFT under two different temporal conditions. In the first condition

(named SMFFT), quick or "impulsive" responding resulted in the task being completed as soon as possible. In the second condition (named RMFFT), all trials were of a fixed length and thus early responding had no impact on the overall length of the task. Sonuga-Barke et al. (1994) found that whilst children with AD/HD were significantly quicker to respond in the SMFFT condition when compared to control children, this was not true in the RMFFT condition. The results appeared to suggest that children with AD/HD were able to withhold from early responding when early responding had no impact on the overall length of the task. Essentially, the children with AD/HD did not appear to be behaving "impulsively" in the RMFFT setting. However, in both conditions, children with AD/HD nevertheless made more mistakes than those without AD/HD. Thus, despite creating more time for stimulus inspection under the RMFFT condition, children with AD/HD still failed to perform as well as control children.

Sonuga-Barke et al. (1996) considered why children with AD/HD did not appear to make more effective use of the extra time afforded by the RMFFT. They suggested that the main factor inhibiting the AD/HD child's ability to use the extra time meaningfully is their overwhelming desire to reduce the perception of delay, irrespective of the costs. Specifically, Sonuga-Barke (1994) suggested that AD/HD children attempt to reduce the subjective experience of time by attending to non-temporal aspects of the environment, despite the fact this may only be achieved by diverting attention away from the task at hand.

Sonuga-Barke et al. (1996) hypothesised that if the high error scores reported on the RMFFT by Sonuga-Barke et al. (1994) were a by-product of delay aversion, then performance would improve if accuracy was subsequently linked to changes in the delay characteristics of the task. To test this hypothesis, Sonuga-Barke et al. (1996) compared AD/HD children's performance under two different testing conditions. In the first, a new trial started immediately after the previous trial irrespective of the accuracy of the previous response given (self-determined trial condition, SDTC). In the second condition, only correct responses were followed immediately by the start of the next trial. If an error was made, a fixed trial length was imposed. In other words, trial length varied as a function of performance (error determined trial condition, EDTC).

The results revealed that AD/HD children gave shorter latencies and made more errors than controls on the SDTC. In contrast, on the EDTC, they withheld from responding on each trial for a longer period and made fewer errors. These findings suggested that AD/HD children were able to withhold from responding, and were willing to do so, when the increased inspection time created reduced the likelihood of a delay being imposed. Sonuga-Barke et al. (1996) concluded that "while all children are aversive to delay to some extent, hyperactive children's aversion to delay is more extreme and they are willing to sacrifice performance and, anything else for that matter, to reduce trial length" (p. 192).

Whilst AD/HD children's performance improved in the EDTC when compared to the SDTC, it is important to note that they still made more errors than controls. Therefore, the two groups of children appeared to differ in their fundamental ability to efficiently use the extra time afforded by the EDTC. It would appear that AD/HD children's poor performance on the standard MFFT cannot exclusively be explained by their willingness to tradeoff accuracy for delay reduction. As Sonuga-Barke et al. (1996) states "hyperactive children appear strikingly inefficient at processing information over extended periods of time" (p.193).

The State Regulation Deficit Model

The state regulation deficit model of AD/HD was first proposed by Douglas and Peters (1979) and later developed by Van der Meere (1996) and Sergeant (2000). This model states that AD/HD results from failures to modulate physiological state to meet the demands of tasks and settings and in this sense is similar to the theory of optimal stimulation. Van der Meere (1996) posited that children with AD/HD have an impaired ability to regulate activation-state and maintain effort over time. Activation state is defined as the tonic readiness for motor actions (Van der Meere, Gunning & Stemerdink, 1999). Essentially, "the engine is intact (i.e. the basic information processing capacity is intact) but there is a problem with the supply of petrol" (p.133). However, in contrast to Zentall and Zentall's theory (1976, 1983) problems with state regulation mean that children with AD/HD not only perform poorly on tasks that are too slow, but also on ones that are too fast. The state regulation deficit model proposes that very quick activities lead to state of

over-activation, whereas long tasks result in a state of under-activation. Unlike control children, those with AD/HD are unable to regulate their state in order to counteract a performance decrement; they are unable to inhibit activation on quick tasks or excite activation on long tasks.

Evidence in support of the state regulation deficit model comes from reaction time studies (Van der Meere, Stemerdink & Gunning, 1995). Van der Meere et al. (1995) compared AD/HD children's ability to inhibit a response using a GO / NO-GO task; a GO / NO-GO task allows participants to respond on "GO" trials but requires them to refrain from responding on "NO-GO" trials. Typically, children with AD/HD make more NO-GO responses than children without AD/HD (Grunewald-Zuberbier, Grunewald, Rasche & Netz, 1978; Trommer, Hoeppner, Lorber, & Armstrong, 1988). Van der Meere et al. (1995) varied the presentation rate by increasing the stimulus-interval in three conditions. In the first, the "fast" condition, the stimulus-interval was one second. In the second and third conditions, the "medium" and "slow" conditions, the stimulus-intervals were four and eight seconds respectively.

The results showed that children with AD/HD performed poorly under the fast and slow conditions (in terms of the number of errors made) but as well as controls under the medium paced condition. Van der Meere et al. (1995) concluded that children with AD/HD were easily "over-activated", causing them to respond quickly and inaccurately in the fast condition, and easily "under-activated", causing them to respond slowly and inaccurately in

the slow condition. They considered both difficulties to reflect state regulation deficiencies. In contrast, control children were able to effectively adapt their activation state towards the presentation rate of the stimuli; they were able to reduce their activation-state in the fast condition and increase it in the slow condition. A normative study by Van der Meere & Stemerdink (1999) suggested that the AD/HD child's ability to regulate activation-state is typically delayed by at least two years.

Dalby, Kinsbourne, Swanson and Sobol (1977) revealed similar findings to Van der Meere et al. (1995) in their study on the impact of presentation rate on performance on a paired associate learning task. They found that AD/HD children performed poorly under "fast" presentation rates (four seconds per item) and "slow" presentation rates (12 seconds per item). However, a medium presentation rate of eight seconds per item produced a performance similar to that of the controls.

Borger and Van der Meere (2000) extended the research into state regulation and AD/HD using heart rate measures. Specifically, mean heart rate, heart rate deceleration and acceleration and heart rate variability of AD/HD and control children were compared during a GO / NO-GO task with a fast (two second) and slow (six second) presentation rate of stimuli. Group differences were found with respect to motor activation and effort allocation in the slow presentation rate condition. First, AD/HD children were slower than control children to react to GO signals, indicating poor motor activation. Secondly, AD/HD children had a less pronounced heart rate deceleration

before the onset of GO signals, suggesting less motor preparation. Thirdly, AD/HD children demonstrated a delayed cardiac shift from deceleration to acceleration (indicating response initiation) after GO signals. Finally, AD/HD children showed greater heart rate variability suggesting less effort was being allocated to the task. Interestingly, no group differences were found in the fast presentation rate condition but this may reflect the fact that in this study, the fast presentation rate was set at two seconds whereas in Van der Meere et al's (1995) earlier study, it was one second. Alternatively, the results may provide greater support for the theory of optimal stimulation than the state regulation deficit model; the theory of optimal stimulation would predict that children with AD/HD would only be susceptible to states of under-activation, as would occur in the slow presentation rate condition rate condition.

Summary

The psychological mechanisms that underlie AD/HD have proved very difficult to identify and currently a number of competing theories are available. First, there is Barkley's (1997) theory of behavioural disinhibition and executive dysfunction. This proposes that the core deficit in AD/HD is an inability to delay the impulsive decision to respond to an event. The inability to withhold from responding means that children with AD/HD are frequently unable to adequately consult with executive functions (including non-verbal working memory, verbal working memory, self-regulation of affect / motivation / arousal and reconstitution) and therefore fail to engage in actions that are under the same degree of control as would be evident in their peers.

Secondly, and in contrast to Barkley's theory, a number of authors have proposed that AD/HD reflects a tendency towards premature task disengagement. For example, Douglas (1983) suggested that children with AD/HD essentially experience problems with sustained attention, the ability to maintain attention over time. Whilst Zentall and Zentall (1976, 1983) proposed that inattentive, impulsive and hyperactive behaviours reflect the AD/HD child's need to maintain high levels of optimal stimulation in stimuluspoor environments, due to a tendency to suffer from a state of under-arousal. Finally, Sonuga-Barke and colleagues (Sonuga-Barke, 1994; Sonuga-Barke, Taylor & Heptinstall, 1992; Sonuga-Barke, Taylor, Sembi & Smith, 1992; Sonuga-Barke, Williams, Hall & Saxton, 1996) developed the delay aversion This theory does not consider children with AD/HD to have an theory. inherent deficit. Instead, it posits that they simply have different aims, primarily involving the avoidance of delay.

Thirdly, Van der Meere (1996) and Sergeant (2000) provide yet another account of the psychological mechanism underlying AD/HD, the state regulation deficit model. They posit that AD/HD essentially reflects a susceptibility to over- and under-activation. When under-activated, children with AD/HD need to excite activation whilst, when over-activated, activation must be inhibited. In other words, any non-optimal state must be addressed and changed in the direction of the optimal state.

To date, there has been little attempt to unite the different theories possibly because "theoretical developments tend to be driven by the search

for one underlying mechanism that could provide the basis for a <u>grand theory</u> of AD/HD" (Sonuga-Barke, 2002a, p.29). Findings and counter-findings relating to different theories are regularly published and even where consistent results are reported, the size of the statistical effect is frequently only small to moderate (Sonuga-Barke, 2002a). Consequently, making sense of the wealth of research published in the area is confusing and, in many ways, AD/HD continues to remain among the least well characterised of the developmental disorders. Testing competing theories of AD/HD against one another would appear an essential direction for future research in order to clarify the significance of the different psychological mechanisms currently considered to underlie this disorder.

How do competing theories of AD/HD explain children's under-utilisation of

time?

One area in which there remains considerable confusion with regard to understanding the specific difficulties encountered by children with AD/HD concerns that of <u>time under-utilisation</u>. As previously mentioned, there is convincing empirical evidence that children with AD/HD tend to under-utilise time, as demonstrated by their performance of the MFFT. Children who spend time carefully examining the stimuli before responding make few errors and are described as "reflective". However, AD/HD children's performance on this task is characterised by fast and inaccurate responding (Barkley, 1998). At face value, it would appear that AD/HD children's poor performance is due to their "impulsivity"; their failure to inhibit their "prepotent" response (Barkley, 1998). Interestingly, Sonuga-Barke et al.

(1994, 1996) found that even when children with AD/HD take as long as control children to respond, as a result of fixed trial lengths or error determined trial lengths, they still make more mistakes than their peers. In other words, even when children with AD/HD do not appear to be behaving impulsively, their performance continues to be impaired. Effectively, children with AD/HD appear unable to make efficient use of the extra time afforded by longer trial latencies. Sonuga-Barke (2002b) presented four different competing theoretical explanations for AD/HD children's impaired performance, as described below.

First, poor performance despite additional study time could reflect <u>premature task disengagement</u>. Children with AD/HD may tolerate longer trial lengths by selecting their response early and then use the remaining time to engage in off-task behaviours. As discussed earlier, premature task disengagement might reflect AD/HD children's (a) aversion to the subjective experience of delay (Sonuga-Barke, 1994; Sonuga-Barke, Taylor & Heptinstall, 1992; Sonuga-Barke, Taylor, Sembi & Smith, 1992; Sonuga-Barke et al., 1996); (b) problems with sustained attention (Douglas, 1983) or (c) need to seek out alternative forms of stimulation in low-stimulation environments (Zentall & Zentall, 1976, 1983).

Secondly, poor performance despite adequate trial duration may be due to the <u>cognitive deficits</u> frequently associated with AD/HD. As noted by Sonuga-Barke (2002b), the MFFT is a complex task that requires the application of a range of executive functions such as working memory

(allowing stimuli already viewed to be held in mind and referred to at a later date), planning and monitoring (allowing an organised search of all stimuli), and attentional flexibility (ensuring that previously attended stimuli are not excessively re-referred to). Spatial memory may also be used as this would allow effective serial searches to be implemented. Past research (as outlined earlier in this review) has frequently reported that children with AD/HD experience significant problems in these domains. It is important to note that cognitive deficits would impair AD/HD children's performance on the MFFT under any condition although the deficit might be exacerbated on long trials where executive demands are increased.

Thirdly, the <u>state regulation deficit model</u> would suggest that AD/HD children's time under-utilisation on relatively long trials is the result of state under-activation, due to the slow rate of presentation of information (Van der Meere, Gunning & Stemerdink, 1999). The state regulation deficit model would predict that in addition to problems on relatively long trials, children with AD/HD would experience difficulties on short trials, since the fast presentation rate would lead to a state of over-activation. In contrast to the cognitive deficit model, the state regulation deficit model would not predict difficulties for AD/HD children on trials of intermediate length since, in this condition, physiological state and stimulus setting would be well matched with respect to activation-state and arousal level.

Fourthly, AD/HD children's difficulty making good use of additional time may be best accounted for by the <u>ecological niche model</u> (Sonuga-

Barke, 2002b). According to this model, children with AD/HD would fail to effectively use the extra time afforded by longer trials because these conditions would be out of keeping with the "natural" tempo of their cognitive style. In contrast to their peers, children with AD/HD may have limited experience of processing information over long time periods, which may lead to a skills deficit in this area. The ecological niche model would predict that whilst presenting with impaired performance on long "niche inconsistent" trials, AD/HD children should display superior performance on shorter "niche consistent" trials. In other words, children with AD/HD may be particularly effective at information processing under time pressure (i.e. quick trials).

Sonuga-Barke (2002b) tested the predictions of the cognitive deficit, state regulation deficit and ecological niche models against those derived from the premature task disengagement model using the MFFT presented under fixed presentation conditions. The fixed presentation conditions involved children being given either 5-, 10- or 15-seconds to identify the target-copy. These latencies were selected as they spanned the mean decision latency of the AD/HD children who participated in the self-paced condition of Sonuga-Barke et al. (1996) study.

In line with the premature task disengagement model, a linear interaction between group and trial duration was predicted, but with no difference emerging between the AD/HD and control group on 5-second trials. In line with the cognitive deficit model, a linear interaction was also predicted, however, with a deficit appearing under <u>all</u> trial duration conditions.

According to the state regulation deficit model, an interaction taking a quadratic form was predicted, with control children out performing those with AD/HD on the 5- and 15- (but not 10-second) trials. Finally, based on the ecological niche model, it was predicted that control children would outperform AD/HD children on the longer trials, whilst AD/HD children would outperform control children on shorter trials.

----- Insert Table 1 about here -----

Overall, the results from Sonuga-Barke's (2002b) study showed that AD/HD children displayed different patterns of time use as trial length increased when compared to control children. Specifically, the performance of children with AD/HD was poorer than that of control children at 5- and 15seconds but equivalent at 10-seconds. Poor performance at 5-seconds appeared to support the predictions made by both the cognitive deficit and state regulation deficit models. Hence, it seemed likely that AD/HD children were either (a) slower at initiating their search (as found by Karatekin & Asarnow, 1998), (b) had impaired search strategies and/or (c) were being over-stimulated. In contrast, impaired performance at 15-seconds supported all four models. It suggested that AD/HD children were (a) employing inefficient / disorganised search strategies, (b) prematurely disengaged from the task and/or (c) being under-stimulated. However, the equivalent performance of the two groups at 10-seconds only fitted with the predictions made by the state regulation deficit model.

In conclusion, Sonuga-Barke's (2002b) study appeared to provide greatest support for the state regulation deficit model of AD/HD; this accurately predicted that children with AD/HD would have difficulty performing on short and long trials but not on those of medium duration. Neither the cognitive deficit model nor the premature task disengagement model could easily explain the equivalent performance of the AD/HD and control children on 10-second trials, whilst the ecological niche model failed to predict AD/HD children's poor performance on 5-second trials. Nevertheless, Sonuga-Barke (2002b) proposed that there might be an alternative explanation of his findings other than that offered by the state regulation deficit model. He suggested that children with AD/HD may develop highly context-specific "compensatory strategies that allow them to overcome cognitive deficits under specific circumstances" (p. 262). This hypothesis seems plausible given that considerable research in other areas has demonstrated the use of compensatory strategies by a range of people with other difficulties. For example, Zec (1995) reported that older adults develop specific cognitive strategies in order to enhance their failing memory. Cirstea and Levin (2000) found that people with brain injury can learn to strategically compensate for the inherent difficulties they encounter when undertaking activities of daily living. Similarly, Nation and Snowling (1998) reported that children with reading difficulties are able to develop contextbased compensatory strategies in an effort to overcome encoding deficits.

There is also limited evidence of the use of compensatory techniques by people with AD/HD. For example, Schweitzer et al. (2000) found that

adults with AD/HD spontaneously used compensatory mental and neural strategies in order to overcome deficits in working memory. Whilst, Alberts and Van der Meere (1992) and Borger and Van der Meere (2000) suggested that looking-away behaviour may represent a compensatory strategy that accommodates for the less than optimal state of activation created by tasks lacking in stimulation. This conclusion was arrived at after both sets of researchers found that off-task behaviour did not negatively impact on AD/HD children's task performance. Indeed, it was proposed that off-task behaviours might actually prevent further deterioration in performance by compensating for inadequate stimulation.

A compensatory strategies model would predict that children with AD/HD would perform well under 10-second conditions but poorly under 5and 15-second conditions because the strategies they have developed in settings in tune with their cognitive style are applied "inappropriately and inflexibly to others" (Sonuga-Barke, 2002b, p.263). This explanation would be based on the premise that children with AD/HD develop context-specific compensatory strategies in order to accommodate their cognitive deficits and is in contrast to the state regulation deficit model, which would attribute inferior performance at both 5- and 15-seconds due to under- or overactivation of physiological state (Sonuga-Barke, 2002b).

Summary

Whilst children with AD/HD are generally assumed to under-utilise time due to their tendency towards fast and inaccurate responding, the exact

nature of their difficulties continues to cause considerable theoretical debate. Barkley's theory of behavioural disinhibition and executive dysfunction (1997) would posit that impulsive behaviour reflects a deficit with the ability to inhibit responding. However, Sonuga-Barke et al. (1994, 1996) found that even when children with AD/HD take as long as their peers to respond, and thus appear to inhibit the prepotent response and engage fully with the task, their performance is still impaired. This indicated that children with AD/HD have a fundamental problem making efficient use of the time created by an apparent delay in responding.

Sonuga-Barke (2002b) suggested that there are four competing theoretical explanations for AD/HD children's apparent inability to effectively utilise time: (a) poor performance may reflect cognitive deficits, (b) poor performance may be attributed to a state of under-arousal in line with the state regulation deficit model, (c) poor performance may reflect premature task disengagement and (d) poor performance may be best accounted for by the ecological niche model. He explored these competing theories by testing their specific predictions of how children with AD/HD would perform on the MFFT when given different lengths of time to search stimuli (5-, 10-and 15-seconds). The results showed that children with AD/HD performed as well as controls under the 10-second condition but worse than controls under both the 5- and 15- second conditions, providing greatest support for the state regulation deficit model whereby children with AD/HD are over-activated on very quick trials and under-activated on slow trials. However, it is also possible that AD/HD children's varied performance under different time

conditions may reflect the use of highly context-specific compensatory strategies.

Directions for future research

Sonuga-Barke (2002b) proposed that future research could usefully help further understanding of AD/HD children's varied performance under different time conditions by testing the compensatory strategies model with the state regulation deficit model. Specifically, this could be achieved by investigating the search strategies employed by children under different time conditions. The compensatory strategies model would predict that children with AD/HD would employ different search strategies to control children under the ten-second condition yet still manage to achieve the same level of performance. However, these context-specific strategies would fail to produce a performance equivalent to that of control children when employed under shorter or longer trials. The compensatory strategies model would predict that control children's superior executive functioning would enable them to out-perform children with AD/HD on both quick and long trials by allowing them to modify and adjust their search strategy in a way that would maximise the time available.

For example, in line with the compensatory strategies model, Sonuga-Barke (2002b) posited that children with AD/HD may employ the same average length of inspection per item under all time conditions and therefore fail to inspect all stimuli on the quick trials, whilst inspecting stimuli too quickly on long trials. In contrast, children without AD/HD may be able to modify the length of time they spend looking at each item in accordance with the trial duration; reflecting a more sophisticated approach to the task. Hence, on quick trials, they may spend little time viewing each stimulus in order to ensure all stimuli are viewed before the end of the trial, whilst on long trials, they may slowly and careful view stimuli because adequate time is available and this strategy would ensure a thorough, reflective search.

It would also be pertinent for future research to seek to provide additional support for Sonuga-Barke's (2002b) hypothesis that premature task disengagement is <u>not</u> implicated in AD/HD children's poor use of time. This could be achieved by studying how much time children with AD/HD spend on-task by measuring visual behaviour.

Whenever children with AD/HD perform poorly on a test, it is essential to discover the reasons for their poor performance (Douglas, 1983). Better understanding the nature of the difficulties experienced by children with AD/HD when carrying out tasks will inform the type of strategies they could be taught in order to compensate for their poor motivation, inappropriate state of arousal and/or cognitive deficits, thereby improving their performance and enabling more efficient time-use. Furthermore, it is essential that future research continues to work towards a better understanding of the psychological mechanisms underlying AD/HD in order to develop our knowledge of the aetiology and theory behind this highly prevalent and disabling developmental disorder.

References

Alberts, E., & Van der Meere, J. (1992). Observations of hyperactive behaviour during vigilance. Journal of Child Psychology and Psychiatry, 33, 1355-1364.

American Psychiatric Association (1994). <u>Diagnostic and Statistical Manual</u> of Mental Disorders (4th ed.). Washington, DC: American Psychiatric Association.

Barkley, R. (1997). Behavioural inhibition, sustained attention and executive functions: Constructing a unifying theory of AD/HD. <u>Psychological Bulletin</u>, <u>121</u>, 65-94.

Barkley, R. (1998). <u>Attention-Deficit/Hyperactivity Disorder: A handbook for</u> <u>diagnosis and treatment</u> (2nd ed.). New York: Guildford Press.

Barkley, R., Karlsson, J., Pollard, S., & Murphy, J. (1985). Developmental changes in the mother-child interactions of hyperactive boys: Effects of two dose levels of Ritalin. <u>Journal of Child Psychology and Psychiatry, 26,</u> 705-715.

Barkley, E., Karlsson, J., Strzelecki, E., & Murphy, J. (1984). The effects of age and Ritalin dosage on the mother-child interactions of hyperactive children. Journal of Consulting and Clinical Psychology, 52, 750-758.

Baumgaertel, A., Wolraich, M., & Dietrich, M. (1995). Comparison of diagnostic criteria for Attention Deficit Disorder in a German elementary school sample. Journal of the American Academy of Child and Adolescent Psychiatry, 34, 629-638.

Bennett, L., Wolin, S., & Reiss, D. (1988). Cognitive, behavioural and emotional problems among school-age children of alcoholic parents. <u>American Journal of Psychiatry, 145, 185-190</u>.

Benton, A. (1991). Prefrontal injury and behaviour in children. Developmental Neuropsychology, 7, 275-282.

Berlyne, D. (1960). Conflict, arousal and curiosity. New York: McGraw Hill.

Biederman, J., Faraone, S., Keenan, K., Knee, D., & Tsuang, M. (1990). Family genetic and psychosocial risk factors in DSM-111 Attention Deficit Disorder. <u>Journal of the American Academy of Child and Adolescent</u> <u>Psychiatry, 29, 526-533.</u>

Borger, N., & Van der Meere, J. (2000). Motor control and state regulation in children with Attention-Deficit/Hyperactivity Disorder: A cardiac response study. <u>Biological Psychiatry, 51,</u> 247-267.

Breton, J., Bergeron, L., Valla, J., Berthiaume, C., & Gaudet, N. (1999). Quebec child mental health survey: Prevalence of DSM-111-R mental health disorders. <u>Journal of Child Psychology and Psychiatry, 40,</u> 375-384.

British Psychological Society. (2000). <u>Attention-Deficit/Hyperactivity</u> <u>Disorder (AD/HD): Guidelines and principles for successful multi-agency</u> <u>working</u>. Leicester: British Psychological Society.

Broadbent, D. (1977). The hidden preattentive process. <u>American</u> Psychologist, 32, 109-119.

Campbell, S. (1987). Parent referred problem three-year-olds: Developmental changes in symptoms. <u>Journal of Child Psychology and</u> <u>Psychiatry, 28,</u> 835-846.

Campbell, S., & Ewing, L. (1990). Follow-up of hard to manage preschoolers: Adjustment at age nine years and predictors of continuing symptoms. <u>Journal of Child Psychology and Psychiatry, 31,</u> 871-889.

Chang, H., Klorman, R., Shaywitz, S., Fletcher, J., Marchione, K., Holahan, J., Stuebing, K., Brumaghim, J., & Shaywitz, B. (1999). Paired associate learning in Attention-Deficit/Hyperactivity Disorder as a function of hyperactivity-impulsivity and oppositional defiant disorder. <u>Journal of Abnormal Child Psychology</u>, 27, 237-245.

Chee, P., Logan, G., Schachar, R., Lindsey, P., & Wachsmuth, R. (1989). Effects of event rate and display time on sustained attention in hyperactive, normal and control children. <u>Journal of Abnormal Child Psychology</u>, 17, 371-391.

Cirstea, M., & Levin, M. (2000). Compensatory strategies for reaching in stroke. <u>Brain, 123, 940-953</u>.

Cohen, N., & Douglas, V. (1972). Characteristics of the orienting response in hyperactive and normal children. <u>Psychophysiology</u>, 9, 238-245.

Conte, R., Kinsbourne, M., Swanson, J., Zirk, H., & Samuels, M. (1986). Presentation rate effects on pair associate learning by attention deficit disordered children. <u>Child Development, 57,</u> 681-687.

Corkum, P., & Siegel, L. (1993). Is the continuous performance task a valuable research children with Attentiontool for use with Deficit/Hyperactivity Disorder? Journal of Child Psychology and Psychiatry, 34, 1217-1239.

Cornoldi, C., Barbieri, A., Gaiani, C., & Zocchi, S. (1999). Strategic memory deficits in attention deficit disorder with hyperactivity participants: The role of executive processes. Developmental Neuropsychology, 15, 53-71.

Costello, E., Angold, A., Burns, B., Erkanli, A., Stangl, D., & Tweed, D. (1996). The Great Smoky Mountains study of youth: Functional impairment and serious emotional disturbance. <u>Archives of General Psychiatry, 53</u>, *1137-1143*.

Costello, E., Angold, A., Burns, B., Stangl, D., Tweed, D., Erkanli, A., & Worthman, C. (1996). The Great Smoky Mountains study of youth: Goals, design, methods and the prevalence of DSM-111-R disorders. <u>Archives of General Psychiatry, 53</u>, 1129-1136.

Dalby, J., Kinsbourne, M., Swanson, J., & Sobel, M. (1977). Hyperactive children's under-use of learning time: Correction by stimulant treatment. Child Development, 44, 1448-1453.

Douglas, V. (1983). Attentional and cognitive problems. In M. Rutter (Ed.), Developmental neuropsychiatry (pp. 280-329). New York: Guildford Press.

Douglas, V., & Peters, K. (1979). Towards a clearer definition of attentional deficit in hyperactive children. In G. Hale & M. Lewis (Eds.), <u>Attention and the development of cognitive skills</u> (pp. 173-247). New York: Plenum Press.

Duffy, E. (1962). Activation and behaviour. New York: Wiley.

Faraone, S., & Biederman, J. (1998). Neurobiology of Attention-Deficit/Hyperactivity Disorder. <u>Biological Psychiatry</u>, 44, 951-958.

Faraone, S., Biederman, J., Keenan, K., & Tsuang, M. (1991). Separation of DSM-111 Attention Deficit Disorder and Conduct Disorder: Evidence from a family-genetic study of American child psychiatric patients. <u>Psychological</u> Medicine, 21, 109-121.

Frank, Y., Seiden, J., & Napolitano, B. (1996). Visual event-related potentials and reaction in normal adults, normal children and children with Attention-Deficit/Hyperactivity Disorder: Differences in short-term memory processing. International Journal of Neuroscience, 88, 109-124.

Gjone, H., Stevenson, J., Sundet, J., & Eilertsen, D. (1996). Changes in heritability across increasing levels of behaviour problems in young twins. <u>Behaviour Genetics, 26</u>, 419-426.

Goldstein, S., & Goldstein, M. (1999). <u>Managing attention disorders in</u> <u>children.</u> Chichester: Wiley.

Gomez-Beneyto, M., Bonet, A., Catala, M., Puche, E., & Vila, V. (1994). Prevalence of mental disorders among children in Valencia, Spain. <u>Acta</u> Psychiatrica Scandanavia, 89, 352-357. Grattan, L., & Eslinger, P. (1991). Frontal lobe damage in children and adults: A comparative review. <u>Developmental Neuropsychology</u>, 7, 283-326.

Grunewald-Zuberbier, E., Grunewald, G., Rasche, A., & Netz, J. (1978). Contingent negative variation and alpha attenuation responses in children with different abilities to concentrate. <u>Electronencephalography and Clinical</u> <u>Neurophysiology</u>, 44, 37-47.

Hale, T., Hariri, A., & McCracken, J. (2000). Attention-Deficit/Hyperactivity Disorder: Perspectives from neuroimaging. <u>Mental Retardation and</u> <u>Developmental Disabilities Research Reviews, 6,</u> 214-219.

Hartsough, C., & Lambert, N. (1985). Medical factors in hyperactive and normal children: Prenatal, developmental and health history findings. <u>American Journal of Orthopsychiatry</u>, 55, 190-210.

Hebb, D. (1955). Drives and conceptual nervous system. <u>Psychological</u> Review, 62, 243-252.

Hill, P. (2000). Attention-Deficit/Hyperactivity Disorder (AD/HD). <u>Primary</u> <u>Care Psychiatry, 6, 119-122</u>.

Humphries, T., Kinsbourne, M., & Swanson, J. (1978). Stimulant effects on co-operation and social interaction between hyperactive children and their mothers. Journal of Child Psychology and Psychiatry, <u>19</u>, 13-22.

Jensen, P., Watanabe, H., Richters, J., Cortes, R., Roper, M., & Liu, S. (1995). Prevalence of mental disorder in military children and adolescents: Findings from a two-stage community survey. <u>Journal of the American</u> Academy of Child and Adolescent Psychiatry, 34, 1514-1524.

Kagan, J. (1965). Reflection-impulsivity and reading abilities in primary grade children. <u>Child Development, 36,</u> 609-628.

Karatekin, C., & Asarnow, R. (1998). Components of visual search in childhood-onset Schizophrenia and Attention-Deficit/Hyperactivity Disorder. Journal of Abnormal Child Psychology, 26, 367-380.

Kindlon, D., Mezzacappa, E., & Earls, F. (1995). Psychometric properties of impulsivity measures: Temporal stability, validity and factor structure. Journal of Child Psychology and Psychiatry, 36, 645-661.

Kuntsi, J., & Stevenson, J. (2000). Hyperactivity in children: A focus on genetic research and psychological theories. <u>Clinical Child and Family</u> <u>Psychology Reviews, 3</u>, 1-23.

Levy, F., Hay, D., McStephen, M., Wood, C., & Waldman, I. (1997). Attention-Deficit/Hyperactivity Disorder: A category or a continuum? Genetic analysis of a large-scale twin study. <u>Journal of the American Academy of</u> Child and Adolescent Psychiatry, 36, 737-744. Logan, G. (1994). On the ability to inhibit thought and action: A users' guide to the stop signal paradigm. In D. Dagenbach & T. Carr (Eds.), <u>Inhibitory</u> <u>processes in attention, memory and language</u> (pp. 189-239). San Diego: Academic Press.

Logan, G., & Cowan, W. (1984). On the ability to inhibit thought and action: A theory of an act of control. <u>Psychological Review, 91,</u> 295-327.

Logan, G., Cowan, W., & Davies, K. (1984). On the ability to inhibit simple and choice reaction time responses: A model and a method. <u>Journal of</u> <u>Experimental Psychology, 10,</u> 276-291.

Losier, B., McGrath, J., & Klein, R. (1996). Error patterns on the continuous performance test in non-medicated and medicated samples of children with and without AD/HD: A meta-analytic review. <u>Journal of Child Psychology and Psychiatry, 37</u>, 971-987.

Mariani, M., & Barkley, R. (1997). Neuropsychological and academic functioning in pre-school boys with Attention Deficit Hyperactivity. <u>Developmental Neuropsychology</u>, 13, 111-129.

Mattes, J. (1980). The role of frontal lobe dysfunction in childhood Hyperkinesis. <u>Comprehensive Psychiatry</u>, 21, 358-369.

Minde, K., Lewin, D., Weiss, G., Lavigueur, H., Douglas, V.; & Sykes, E. (1971). The hyperactive child in elementary school: A five-year controlled follow-up. <u>Exceptional Children. 38</u>, 215-221.

Minde, K., Webb, G., & Sykes, D. (1968). Studies on the hyperactive child:
VI. Prenatal and perinatal factors associated with hyperactivity.
<u>Developmental Medicine and Child Neurology</u>, 10, 355-363.

Nation, K., & Snowling, M. (1998). Individual differences in contextual facilitation: Evidence from dyslexia and poor reading comprehension. <u>Child</u> <u>Development, 69,</u> 996-1011.

Needleman, H., Schell, A., Bellinger, D., Leviton, L., & Alfred, E. (1990). The long-term effects of exposure to low doses of lead in childhood: An 11-year follow-up report. New England Journal of Medicine, 322, 83-88.

O'Neill, M., & Douglas, V. (1996). Rehearsal strategies and recall performance in boys with and without Attention-Deficit/Hyperactivity Disorder. Journal of Paediatric Psychology, 21, 73-88.

Oosterlaan, J., Logan, G., & Sergeant, J. (1998). Response inhibition in AD/HD, CD, comorbid AD/HD and CD, anxious and control children: A metaanalysis of studies with the stop task. <u>Journal of Child Psychology and</u> <u>Psychiatry, 39, 411-425.</u> Pavlov, I. (1927). Conditioned reflexes. London: Oxford University Press.

Pennington, B., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. <u>Journal of Child Psychology and</u> <u>Psychiatry, 37, 51-87.</u>

Perrin, S., & Last, C. (1996). Relationship between AD/HD and anxiety in boys: Results from a family study. Journal of the American Academy of Child and Adolescent Psychiatry, 35, 988-996.

Posner, M. (1978). <u>Chronometric explorations of mind</u>. Hillsdale, New Jersey: Erlbaum.

Reardon, D., & Bell, G. (1970). Effects of sedative and stimulation music on activity levels of severely retarded boys. <u>American Journal of Mental</u> <u>Deficiency, 75,</u> 156-159.

Rubia, K., Taylor, A., Taylor. E., & Sergeant, J. (1999). Synchronization, anticipation and consistency in motor timing of children with dimensionally defined Attention-Deficit/Hyperactivity Disorder. <u>Perceptual and Motor Skills</u>, 89, 1237-1258.

Rutter, M. (1977). Brain damage syndromes in childhood: Concepts and findings. Journal of Child Psychology and Psychiatry, 18, 1-21.

Scahill, L., & Schwab-Stone, M. (2000). Epidemiology of AD/HD in schoolage children. <u>Child and Adolescent Psychiatric Clinics of North America</u>, 9, 541-555.

Schachar, R., & Wachsmuth, R. (1990). Hyperactivity and parental psychopathology. Journal of Child Psychology and Psychiatry, 31, 381-392.

Schweitzer, J., Faber, T., Grafton, S., Tune, L., Hoffman, J., & Kilts, C. (2000). Alterations in the functional anatomy of working memory in adult Attention-Deficit/Hyperactivity Disorder. <u>American Journal of Psychiatry, 157</u>, 278-280.

Sergeant, J. (2000). The cognitive-energetic model: An empirical approach to Attention-Deficit/Hyperactivity Disorder. <u>Neuroscience and Biobehavioural</u> <u>Reviews, 24, 7-12</u>.

Shaywitz, S., Cohen, D., & Shaywtiz, B. (1980). Behaviour and learning difficulties in children of normal intelligence born to alcoholic mothers. Journal of Paediatrics, 96, 978-982.

Sherman, D., Iacono, W., & McGue, M. (1997). Attention-Deficit/Hyperactivity Disorder dimensions: A twin study of inattention and impulsivity-hyperactivity. <u>Journal of the American Academy of Child and</u> Adolescent Psychiatry, 36, 745-753. Sherman, D., McGue, M., & Iacono, W. (1997). Twin concordance for Attention-Deficit/Hyperactivity Disorder: A comparison of teachers' and mothers' reports. <u>American Journal of Psychiatry, 154, 532-535</u>.

Sonuga-Barke. E. (1994). On dysfunction and function in psychological accounts of childhood disorder. <u>Journal of Child Psychology and Psychiatry</u>, <u>35</u>, 801-815.

Sonuga-Barke, E. (1995). Disambiguating inhibitory dysfunction in childhood hyperactivity. In J. Sergeant (Ed.), <u>Eunethydis: European</u> approaches to Hyperkinetic disorder (pp. 209-223). Zurich: Fotorotar.

Sonuga-Barke, E. (2002a). Psychological heterogeneity in AD/HD: A dual pathway model of behaviour and cognition. <u>Behavioural Brain Research</u>, 130, 29-36.

Sonuga-Barke, E. (2002b). Interval length and time-use by children with AD/HD: A comparison of four models. <u>Journal of Abnormal Child</u> Psychology, 30, 257-264.

Sonuga-Barke, E., Houlberg, K., & Hall, M. (1994). When is impulsiveness not impulsive? The case of hyperactive children's cognitive style. <u>Journal of</u> <u>Child Psychology and Psychiatry</u>, 35, 1247-1253.

Sonuga-Barke, E., & Taylor, E. (1992). The effect of delay on hyperactive and non-hyperactive children's response times. <u>Journal of Child Psychology</u> and Psychiatry, 33, 1091-1096.

Sonuga-Barke, E., Taylor, E., & Heptinstall, E. (1992). Hyperactivity and delay aversion 11: The effect of self versus externally imposed stimulus presentation periods on memory. <u>Journal of Child Psychology and</u> Psychiatry, 33, 399-409.

Sonuga-Barke, E., Taylor, E., Sembi, S., & Smith, J. (1992). Hyperactivity and delay aversion 1: The effect of delay on choice. <u>Journal of Child</u> <u>Psychology and Psychiatry, 33, 387-398.</u>

Sonuga-Barke, E., Williams, E., Hall, M., & Saxton, T. (1996). Hyperactivity and delay aversion 111: The effect on cognitive style of imposing delay after errors. <u>Journal of Child Psychology and Psychiatry, 37,</u> 189-194.

Stewart, M. (1970). Hyperactive children. Scientific American, 222, 97-98.

Streissguth, A., Martin, D., Barr, H., Sandman, B., Kirchner, G., & Darby, B. (1984). Intrauterine alcohol and nicotine exposure: Attention and reaction time in 4-year-old children. <u>Developmental Psychology</u>, 20, 533-541.

Szatmari, P., Offord, D., & Boyle, M. (1989). Ontario Child Mental Health Study: Prevalence of Attention Deficit Disorder with Hyperactivity. <u>Journal of</u> <u>Child Psychology and Psychiatry, 30,</u> 219-230.

Szatmari, P., Saigal, S., Rosenbaum, P., & Campbell, D. (1993). Psychopathology and adaptive functioning among extremely low birth weight children at eight years of age. <u>Development and Psychopathology</u>, 5, 345-357.

Tannock, R., Schachar, R., Carr, R., Chajczyk, D., & Logan, G. (1989). Effects of methylphenidate on inhibitory control in hyperactive children. Journal of Abnormal Child Psychology, 17, 473-491.

Taylor, E. (1994). Disorders of attention and activity. In M. Rutter, E. Taylor & L. Hersov (Eds.), <u>Child and adolescent psychiatry: Modern approaches</u> (3rd ed.). Oxford: Blackwell.

Trommer, B., Hoeppner, J., Lorber, R., & Armstrong, K. (1988). The go / nogo paradigm in attention deficit disorder. <u>Annuals of Neurology, 24,</u> 610-614.

Van der Meere, J. (1996). The role of attention. In S. Sandberg (Ed.), <u>Hyperactivity disorders of childhood</u> (pp. 111-148). Cambridge: Cambridge University Press. Van der Meere, J., Gunning, B., & Stemerdink, N. (1999). The effect of methylphenidate and clonidine on response inhibition and state regulation in children with AD/HD. Journal of Child Psychology and Psychiatry, 40, 291-298.

Van der Meere, J., & Sergeant, J. (1988). Controlled processing and vigilance in hyperactivity: Time will tell. <u>Journal of Abnormal Child</u> <u>Psychology, 16, 641-655</u>.

Van der Meere, J., & Stemerdink, N. (1999). The development of state regulation in normal children: An indirect comparison with children with AD/HD. <u>Developmental Neuropsychology</u>, 16, 213-225.

Van der Meere, J., Stemerdink, N., & Gunning, B. (1995). Effects of presentation rate of stimuli on response inhibition in AD/HD children with and without tics. <u>Perceptual and Motor Skills, 81,</u> 259-262.

Van den Oord, E., & Rowe, D. (1997). Continuity and change in children's social maladjustment: A developmental behaviour genetic study. Developmental Psychology, 33, 319-332.

Webster-Stratton, C. (2001). <u>The incredible years: A trouble shooting guide</u> for parents of children aged 3-8. Toronto: Umbrella Press. Weiss, G., & Hechtman, L. (1993). <u>Hyperactive children grown up</u> (2nd ed.). New York: Guildford.

World Health Organisation (1990). <u>International Classification of Diseases</u> (10th ed.). Geneva: World Health Organisation.

Zec, R. (1995). The neuropsychology of ageing. <u>Experimental Gerontology</u>, <u>30,</u> 431-442.

Zentall, S. (1975). Optimal stimulation as theoretical basis of hyperactivity. American Journal of Orthopsychiatry, 45, 549-563.

Zentall, S. (1977). Environmental Stimulation Model. <u>Exceptional Children</u>, <u>43</u>, 502-510.

Zentall, S. (1980). Behavioural comparisons of hyperactive and normally active children in natural settings. <u>Journal of Abnormal Child Psychology, 8</u>, 93-109.

Zentall, S., & Zentall, T. (1976). Activity and task performance of hyperactive children as a function of environmental stimulation. <u>Journal of</u> <u>Consulting and Clinical Psychology</u>, 44, 693-697.

Zentall, S., & Zentall, T. (1983). Optimal stimulation: A model of disordered activity and performance in normal and deviant children. <u>Psychological</u> <u>Bulletin, 94, 446-471</u>.

Table 1

Summary of the predictions made by the four different models for the performance of AD/HD and control children under 5-second, 10-second and

15-second trial lengths (Sonuga-Barke, 2002b)

Model	5-Second	10-Second	15-Second
Premature task	Equal	Controls superior	Controls superior
disengagement	performance	to AD/HD	to AD/HD
		children	children
Executive	Controls superior	Controls superior	Controls superior
dysfunction	to AD/HD	to AD/HD	to AD/HD
	children	children	children
State regulation	Controls superior	Equal	Controls superior
deficit	to AD/HD	performance	to AD/HD
	children		children
Ecological niche	AD/HD children	AD/HD children	Controls superior
	superior to	superior to	to AD/HD
	controls	controls	children

Empirical Paper

ATTENTION-DEFICIT/HYPERACTIVITY DISORDER, TIME USE AND TASK PERFORMANCE: WORKING TOWARDS A COMPREHENSIVE THEORETICAL UNDERSTANDING

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Abstract

Predictions made by competing theories of AD/HD with regard to AD/HD children's search strategies, time-use and task performance were tested using a computerised version of the Matching Familiar Figures Test. Twenty-five children with a diagnosis of AD/HD and 25 control children completed the task under four different trial duration conditions (5-, 10-, 15and 20-seconds). The control children were found to out-perform the clinical group on the 5-, 10- and 15-second trials. However, the AD/HD group's performance did improve with time. Furthermore, children with AD/HD were as efficient as their peers at employing logical, exhaustive searches and identifying the target-copy when time spent actually viewing stimuli / number of boxes opened was taken into account. The AD/HD children's poor performance appeared to fundamentally be due to insufficient opening of boxes and, under certain conditions, delays with search initiation. This indicated problems with motivation, a state of under-arousal and/or slowness when planning / pacing / implementing searches.

Key words: Attention-Deficit/Hyperactivity Disorder, Matching Familiar Figures Task

Introduction

There is considerable evidence that children with Attention Deficit/Hyperactivity Disorder (AD/HD) perform poorly on a range of tasks when compared to age and IQ matched controls (for a comprehensive review, see Kuntsi & Stevenson, 2000). However, explaining this impaired performance can be difficult since the underlying psychological mechanisms are often hard to identify. Consequently, AD/HD remains one of the least well characterised of the developmental disorders.

One such task is the Matching Familiar Figures Test (MFFT; Kagan, 1965). This is a simple computer task that requires children to identify a target from amongst five similar foils. Typically, AD/HD children's performance on this task is characterised by fast and inaccurate responding (Barkley, 1998). In contrast, control children are believed to carefully examine the stimuli before identifying the target-copy, make few errors and are described as "reflective". Essentially, it would appear that AD/HD children's poor performance is due to their "impulsivity"; their failure to inhibit the initial "prepotent" response (Barkley, 1997). However, Sonuga-Barke, Houlberg and Hall (1994) demonstrated that under certain conditions, children with AD/HD appear able to inhibit responses and take as long as controls to find the target-copy. Nevertheless, even when they do this, they still make more mistakes than those without AD/HD. In other words, even when children with AD/HD take the same length of time as control children to identify the target-copy, their performance continues to be impaired.

Sonuga-Barke (2002) presented different competing theoretical explanations for AD/HD children's apparent inability to make use of additional study time on the MMFT, as will now be described. First, poor performance may reflect <u>premature task disengagement</u>. Children with AD/HD may tolerate trials of fixed length by selecting their response early on and then engage in off-task behaviours until asked to provide a response. Such behaviour may reflect: (a) their aversion to the subjective experience of delay (Sonuga-Barke, 1994; Sonuga-Barke, Taylor & Heptinstall, 1992; Sonuga-Barke, Taylor, Sembi & Smith, 1992; Sonuga-Barke, Williams, Hall & Saxton, 1996); (b) problems with sustained attention (Douglas, 1983); or (c) their need to seek out alternative forms of stimulation in low-stimulation environments (Zentall & Zentall, 1976, 1983).

Secondly, AD/HD children's poor performance may reflect <u>cognitive</u> <u>deficits</u>. Indeed, some evidence supports the idea that children with AD/HD employ disorganised and inefficient search strategies (Douglas, 1983). These would likely be the result of executive dysfunction, which has frequently been cited as a core feature of AD/HD (Barkley, Grodzinsky & DuPaul, 1992; Houghton et al., 1999; Pennington & Ozonoff, 1996; Seidman, Biederman, Faraone, Weber, Ouellette, 1997). Thirdly, children with AD/HD may perform poorly on tasks if they are either in a state of under-activation, due to the slow presentation rate of information, or over-activation, due to a fast presentation rate of information. The <u>state regulation deficit model</u> (Sergeant, 2000; Van der Meere, 1996) posits that children with AD/HD are highly susceptible to both over- and under-activation.

Finally, the <u>ecological niche model</u> may best account for AD/HD children's impaired performance on the MFFT. According to this model, children with AD/HD may struggle to utilise time in conditions that are not in keeping with the "natural" tempo of their cognitive style. In particular, they are likely to encounter problems when required to process information over long periods of time due to a lack of experience with handling such cognitive demands.

Sonuga-Barke (2002) tested the predictions of the cognitive deficit, state regulation deficit and ecological niche model against those provided by theories of premature task disengagement using the MFFT presented under fixed trials lengths of 5-, 10- and 15-seconds. These latencies were selected as they spanned the mean self-paced decision latency of AD/HD children on this task (Sonuga-Barke et al., 1996). Sonuga-Barke (2002) proposed that theories of premature task disengagement would predict a linear interaction between group and trial duration, with no difference between AD/HD and control children on the 5-second trial. The cognitive deficit model would also predict a linear interaction, but with impaired performance on <u>all</u> trial lengths. In contrast, the state regulation deficit model would anticipate a quadratic interaction with control children outperforming AD/HD children on 5- and 15-second trials but not on 10-second trials. Finally, the ecological niche model would expect control children to outperform AD/HD children on the longer trials and the reverse on shorter trials.

Using a sample of 12 boys with a diagnosis of Hyperkinetic Disorder (ICD-10; World Health Organisation, 1992) and 12 controls, Sonuga-Barke (2002) found that AD/HD children's performance was poorer than controls at 5- and 15-seconds, but equivalent at 10-seconds. Poor performance at 5seconds supported both the cognitive deficits and the state regulation deficit models; AD/HD children's inferior performance could be due to (a) delay initiating searches (as described by Karatekin & Asarnow, 1998), (b) ineffective search strategies or (c) a state of over-activation. Impaired performance at 15-seconds supported all four models; AD/HD children may (a) have inefficient search strategies, (b) be prematurely disengaging from the task, (c) be in a state of under-activation, or (d) be in a "niche" inconsistent setting. In contrast, equivalent performance at 10-second only provides support for the state regulation deficit model.

Overall, the study found greatest support for the state regulation deficit model of AD/HD. However, Sonuga-Barke (2002) posited that the findings could also be accounted for by the use of highly context-specific <u>compensatory strategies</u>. Children with AD/HD may have a rigid approach to the task and struggle to flexibly adjust their search strategy with changes in the trial conditions. On certain trials, these strategies may result in a relatively good performance but on others they may prove relatively ineffective. Compensatory strategies essentially arise out of attempts by the child with AD/HD to overcome their cognitive deficits. On short trials, AD/HD children may not have enough time to implement their compensatory search strategy, whereas control children may be able to arrive at a quick and

effective search method. On longer trials, AD/HD children may continue to use their fixed search strategy, whilst control children are able to develop increasingly sophisticated search strategies.

In order to consider further the possibility that children with AD/HD may employ compensatory strategies when undertaking the MFFT, it is necessary to compare the search strategies used by AD/HD and control children on trials of differing lengths. This could clarify whether or not the state regulation deficit model really is best placed to account for the findings of Sonuga-Barke (2002). In addition, it would be pertinent to provide further evidence that premature task disengagement does <u>not</u> account for poor performance by analysing how much time children with AD/HD actually spend off-task on trials of fixed length.

Hypotheses

The first aim of this study was to replicate the findings of Sonuga-Barke (2002) with respect to AD/HD children's performance on the MFFT when presented under different time conditions using a larger sample of children with a formal diagnosis of AD/HD (combined type). Specifically, it was predicted that AD/HD children would perform as well as control children when given 10-seconds to search the stimuli for the target-copy, but less well when given 5- or 15-seconds. In contrast to the performance of those with AD/HD, it was predicted that control children's performance would improve in a <u>linear</u> fashion as a function of trial length. As an extension of Sonuga-Barke's study, an additional trial length would be included in the present study, namely a 20-second trial. It was predicted that control children would outperform AD/HD children under this condition.

The second aim of this study was to consider whether the state regulation deficit model really was best placed to account for AD/HD children's poor performance under certain time conditions by investigating the <u>search strategies</u> employed by both groups of children. Investigating search strategies enabled a head-to-head test of the state regulation deficit model against the compensatory strategies model. The state regulation deficit model would predict that children with AD/HD would employ the same search strategies as control children. Namely, ones that are responsive to trial duration and well organised. In contrast, the compensatory model would anticipate significant differences in the search strategies used by the two groups. Specifically, children with AD/HD would be expected to use the same search strategy irrespective of trial length whilst control children would display sophisticated and context-mediated strategies.

To assess the search strategies employed by AD/HD and control children, both qualitative and quantitative factors should be investigated. "Qualitative" factors refer to the type of search strategy employed in terms of (a) the order in which children searched the stimuli, (b) whether an exhaustive search was carried out, and (c) whether the search appeared logical or disorganised in nature. "Quantitative" factors refer to (a) the number of boxes opened, (b) the length of time spent viewing individual boxes and boxes overall, and (c) the time taken to initiate a search.

The third aim of this study was to assess AD/HD children's ability to remain on-task, in order to clarify whether premature task disengagement could account for impaired performance.

Method

Participants

A total of 50 boys between the ages of eight and 12 years participated in this study, 25 with AD/HD and 25 controls. The mean age of participants with AD/HD was 10.75 years (<u>SD</u>=1.45), while for the controls it was 9.73 years (<u>SD</u>=0.89). The age difference between the two groups was significant and, as such, age was controlled for during all subsequent analyses (see "Results"). No participants were recruited until appropriate ethical approval was given (see Appendix B).

The clinical group was recruited with the help of a consultant paediatrician and a consultant psychiatrist covering three different geographical areas. The consultants originally identified 75 boys who had received a diagnosis of AD/HD (combined type) using the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV, American Psychiatric Association, 1994). Whilst all these children had received a formal diagnosis of AD/HD, none had any other formal co-morbid diagnoses. In other words, no child was known to have any form of learning difficulty (including dyslexia, dyspraxia and dysgraphia), autism, oppositional defiant disorder, anxiety, depression or somatic complaints. Parents of all potential participants were sent a recruitment pack; this contained an information sheet and consent form (see Appendix C), the Strengths and Difficulties Questionnaire (SDQ, see "Measures") and a stamped-addressed envelope. Parents who agreed to their child participating were invited to return the signed consent form along with the completed SDQ.

Of the 75 parents of children diagnosed with AD/HD who were approached, 35 (47%) responded; no information was available on nonrespondents. All participants were subsequently screened for learning disabilities using four sub-scales of the Wechsler Intelligence Scale for Children (WISC-III; Wechsler, 1992); see "Measures" for further details. Any child with an IQ < 80 was excluded from the study. In the end, ten children were excluded from the clinical group. Three children were excluded due to a low IQ and seven because they scored below the required inattentionhyperactivity sub-scale cut-off on either the parent or teacher completed SDQ (see "Measures"). This left a final clinical sample of 25.

The control group was recruited through a local state primary school situated in a middle socio-economic status area. The head-teacher originally identified 50 children who were not known by their teacher to have any psychiatric diagnoses or learning difficulties, and were considered to be free of AD/HD-type behaviours. The parents of these children were sent out a similar recruitment pack to the clinical sample (the wording of the information sheet and the consent form were slightly different, see Appendix D). Of the 50 parents who were approached, 26 (52%) provided consent; no information

was available on non-respondents. One child was subsequently excluded as he failed to meet the control group's inclusion criteria; he scored above the cut-off on the inattention-hyperactivity sub-scale on the teacher-completed SDQ (see "Measures"). As with the clinical group, all control children were screened for learning disabilities using four sub-scales of the WISC-III. All control children were found to have an IQ > 80 and therefore none needed to be excluded from the sample on the basis of a low IQ score.

<u>Measures</u>

As mentioned above, all children were screened for learning disabilities prior to participation using four sub-scales of the WISC-III including: Similarities and Vocabulary (providing an estimate of Verbal IQ), and Block design and Object assembly (providing an estimate of Performance IQ). The four subtests were pro-rated and an estimate of the child's Full Scale IQ obtained. Any child who appeared to have a Full Scale IQ of less than 80 was excluded from the study. The mean IQ of the AD/HD and control groups were 104 (<u>SD</u>=13.12; range 83-127) and 109 (<u>SD</u>=16.63; range 83-146) respectively.

The parents and teachers of all participants were asked to complete the SDQ (Goodman, 1997; see Appendix E). The teachers of clinical participants were written to (see Appendix F), whilst the teachers of control children were asked in person. The SDQ contains 25 items and assesses inattention-hyperactivity, emotional symptoms, conduct problems, peer problems and prosocial behaviour. All children included in the AD/HD group

scored above the cut-off for "abnormal" inattention-hyperactivity (a score of 7-10) on both parent and teacher completed versions. All children included in the control group scored below the cut-off on the inattention-hyperactivity sub-scale on both parent and teacher completed versions. The SDQ was chosen as it is a brief measure yet has good psychometric properties and has been found to be significantly better than the Child Behaviour Checklist (Achenbach, 1991) at detecting inattention / hyperactivity and at least as good at detecting emotional and conduct problems (Goodman & Scott, 1999).

----- Insert Table 1 about here -----

As can be seen in Table 1, parents of all children provided a completed SDQ, as did all the teachers of control children. Twenty-one teachers of children in the AD/HD group provided a completed SDQ, however, one teacher provided incomplete responses to the items on the emotional symptoms sub-scale (hence \underline{n} =20) and only nine teachers felt able to complete the inattention-hyperactivity sub-scale. Twelve teachers who returned the SDQ for children included in the clinical group stated that they were unable to accurately answer questions concerning inattention and/or hyperactivity as they had never seen the child off the medication they took in order to control their symptoms of AD/HD (hence n=9).

Procedure

All children were tested in a quiet room at their school or the local child and family guidance centre. Each testing session began with a short general conversation in order to engage the child. The purpose of the meeting was then explained. All children were made fully aware that their attendance was not compulsory and that they were free to leave at any time. All children were given the option of opting out, without penalty, before being asked to sign a consent form (see Appendix G). All children provided consent although one child in the clinical group did not want to be videoed and this was respected. Once a child had given consent, he first completed the computer task and then, the four sub-tests from the WISC-III (the sub-tests from the WISC-III were always administered in the same order). The child was videoed whilst completing the computer task. Each session lasted approximately one hour. All children in the clinical group were asked to refrain from taking methylphenidate in the 24-hours prior to testing. A check that this instruction had been followed was made at the beginning of all sessions.

A debriefing statement was sent to the parents following their child's participation (see Appendix H). The debriefing statements invited parents to return a response slip if they wished to receive a copy of the results, when available. Sixteen parents subsequently requested a summary of the results.

The experimental task

The MFFT was presented on a portable laptop and consisted of two practice trials followed by four randomly presented blocks of ten trials. At the start of each trial the target stimulus was presented in a box measuring 5.5 x 6.5 cm in the centre at the top of the screen. This box was permanently open and thus could be viewed constantly by the participant. Six other boxes of the same dimensions were presented in two rows of three under the test stimuli. These contained the six test stimuli, including five foils and one copy of the target. All stimuli were black and white line drawings. The test stimuli boxes were permanently closed unless the participants "clicked" on the box using the computer's mouse. No more than one test stimuli could be open at anyone time so each box had to be closed, by "clicking" on it using the mouse, before the next one could be viewed. The position of the copy of the target was varied randomly from trial to trial.

In the four blocks, children were given 5-, 10-, 15- and 20-seconds to view the stimuli. The participants were instructed to view the stimuli and find the one that was identical to the stimulus at the top of the screen. They were told that after a set amount of time, the words "please choose now" would appear at the top of the screen and when this happened, they should click on the box that they thought contained the target-copy.

The computer software recorded a number of aspects of the child's performance on the experimental task including: (a) the number of correct responses made under each time condition, (b) the time taken to initiate the

search, (c) the number of boxes opened in each trial, (d) the length of time each box was opened for, and (e) the order in which the boxes were opened (all boxes were assigned a number; the stimuli in the top row were numbered 1-3, left to right, the stimuli in the bottom row were numbered 4-6, left to right). The search order was subsequently analysed by hand and categorised as one of the following: (a) an exhaustive, logical search, (b) an exhaustive but apparently disorganised search, (c) a logical search that was initiated but not completed, (d) an apparently disorganised search, (e) no search, or (f) only one box opened. A logical search included all searches where the participant started with any box (position 1-6) and then searched up and down all three columns or across rows in a sequential fashion. The tester and an independent child clinical psychologist had previously generated a list of all possible "logical" search orders; there was 100% agreement on what constituted a "logical" search. In other words, two independent raters generated an exhaustive list of all possible logical search orders. This list was then used to classify all computer generated search orders as logical or otherwise.

The experimental task used here was adapted from that used by Sonuga-Barke (2002). The same range of stimuli was used and the box layout was identical. However, in Sonuga-Barke's study, all the stimuli could be viewed all of the time (the boxes were permanently open) and the task included three blocks of 14 trials as opposed to four blocks of ten trials (the difference in the number of blocks being due to the addition of the 20-second condition in the present study).

Analyses of video material

The video of the child's behaviour whilst completing the experimental task was used to assess the total length of time the child spent off-task when boxes were open under each time condition. The child was considered to be off-task whenever he was not looking directly at the computer screen, as gauged by visual behaviour. This data was used not only with respect to time spent off-task but also in order to calculate the actual amount of time spent viewing stimuli in all time conditions. The length of time children spent viewing stimuli was calculated using the length of time all boxes were opened for, minus the length of time spent off-task when boxes were open. It should be noted that the tester who analysed the videos was not blind to which participants belonged to which group. In order to assess the accuracy of the measurements of off-task behaviour, inter-rater reliability was performed on 20% of all the data (10% from clinical sample and 10% from control group). Unlike the first rater, the second rater was not aware which group which child belonged to. A good level of inter-reliability was achieved in all cases (M=98%, maximum=100%, minimum=86%).

Results

Performance

To establish if there were significant differences between the age and/or IQ of the AD/HD and control groups, in order to clarify whether these factors should be included in subsequent analyses as covariates, independent sample t-tests were carried out. As can be seen in Table 2,

there were no significant difference between the two groups in terms of IQ, however, there was a significant difference with respect to <u>age</u>. The participants in the AD/HD sample were significantly older compared to those in the control sample. It was therefore decided that age would be included as a covariate in subsequent analyses.

Independent sample t-tests also revealed that there were significant differences between the AD/HD and control groups on both the emotional problems and conduct problems sub-scales of the SDQ, as completed by both parents and teachers (see Table 2). In all cases, the AD/HD group obtained higher scores, indicating that both parents and teachers rated these children as presenting with more severe emotional and conduct problems when compared to control children. It was decided that these factors would not be introduced as covariates in subsequent analyses as these difficulties are frequently regarded as an inherent feature of AD/HD.

----- Insert Table 2 about here -----

The mean numbers of correct responses made by the AD/HD and control groups both with and without age as a covariate are presented in Table 3. In all time conditions, the control group appeared to outperform the AD/HD group.

----- Insert Table 3 about here -----

The mean number of correct responses, with age introduced as a covariate, given in each time condition by the two groups were submitted to a mixed-design ANCOVA with group as the between-subject factor (AD/HD vs. control) and time condition as the within-subject factor (5-, 10-, 15- and 20-seconds). A very significant difference between the performance of the AD/HD and control group was revealed ($\underline{F}(1,47)=8.88$; $\underline{p}<.01$); the control sample significantly outperformed the AD/HD group. Furthermore, linear interactions between group and time approached significance ($\underline{F}(1,47)=3.65$; $\underline{p}=.06$); the performance of both groups tended to improve in a linear fashion with time. No significant quadratic interactions were found ($\underline{F}(1,47)=1.25$; $\underline{p}=.27$).

In order to investigate how the performance of the AD/HD and control groups compared under different time conditions, post-hoc one-way ANCOVAs were carried out with performance as the dependant variable, group (AD/HD vs. controls) as the fixed factor and age as the covariate. The control group significantly out-performed the clinical group under the 5-, 10- and 15-second conditions. The greatest difference in the performance between the clinical and control groups occurred on the 5-second (E(1,47)=11.16; p<.01) and 10-second trials (E(1,47)=8.23; p<.01), then the 15-second trials (E(1,47)=4.61; p=.04). No significant difference was found between the performance of the two groups on the 20-second trials (E(1,47)=0.35; p=.56). This suggested that AD/HD children's performance was significantly impaired on trials of short or intermediate length, but equivalent to that of control children's on long trials.

Factors influencing performance

In order to analyse whether there was a difference in the quantitative characteristics of the search strategies used by the two groups, the following factors were submitted to mixed-design ANCOVAs: (a) length of time spent off-task when boxes open, (b) delay initiating search, (c) failure to initiate a search, (d) length of time spent viewing all stimuli, (e) number of boxes opened, (f) number of times only one box was opened, and (g) the average length of time spent viewing a stimulus. Similarly, in order to analyse whether there was a difference in the qualitative characteristics of the search strategies used by the two groups, the following factors were also submitted to mixed-designs ANCOVAs: (a) number of exhaustive searches completed (logical or otherwise), (b) number of exhaustive logical searches completed, (c) number of exhaustive but apparently disorganised searches completed, (d) number of logical searches initiated but not completed, and (e) number of apparently disorganised searches initiated but not completed. In all analyses, group was used as the between-subject factor (AD/HD vs. control), time condition as the within-subject factor (5-, 10-, 15- and 20- seconds) and age as the covariate. Table 4 summarises the results.

----- Insert Table 4 about here -----

As can be seen from Table 4, there were a number of significant differences with respect to the quantitative characteristics of the search strategies used by the two groups. Firstly, there were significant differences

between the lengths of time the two groups spent off-task; AD/HD children spent significantly more time looking away from opened boxes than control children. There was also a significant linear interaction between group and time with the AD/HD group spending more time off-task the longer the trial. In other words, the more time available to the AD/HD children, the more time they spent off-task instead of studying the stimuli. Secondly, in terms of delays with search initiation, there was a significant main effect of group with the AD/HD children taking significantly longer than controls to initiate a search. This suggested that children with AD/HD were generally slower to start the task when compared to control children and consequently lost valuable study time. In contrast, control children were generally quicker to initiate their search and thus created the opportunity to open more boxes and/or spend more time studying stimuli than their peers. A significant quadratic interaction was also found since AD/HD children took significantly longer to initiate their searches on the 10- and 15-second trials when compared to the 5-second and 20-second trials. Hence, for some reason, the children in the clinical group were guicker to initiate searches on the short and long trials than on trials of intermediate length. In comparison, the control group were relatively consistent in the length of time they took to commence their search across all conditions.

Thirdly, there were significant differences between children with and without AD/HD with respect to the number of boxes opened and the length of time spent studying stimuli in each trial. Children with AD/HD opened significantly fewer boxes per trial and spent less time viewing stimuli overall.

However, there was no significant difference between the mean length of time AD/HD and control children spent viewing individual boxes. This suggested that both groups of children spent roughly equivalent time periods studying stimuli once a box had been opened. In other words, it appeared that control children opened more boxes and spent more time overall studying stimuli but both groups opened and viewed individual boxes for similar time periods. Finally, there were significant differences between the two groups with respect to the number of times no search was initiated or only one box was opened. Children with AD/HD were significantly more likely than those without AD/HD to fail to initiate a search or to open only one box. However, it is important to note that for AD/HD children a significant linear interaction was found, indicating that the clinical group were less likely to fail to initiate a search or to open only one box as trial length increased. In other words, the more time available, the more likely children with AD/HD were to fully commence their search. In contrast, a significant quadratic interaction was found for the control group with respect to the number of times only one box was opened. The control children were most likely to open only one box on 5-second trials but the next most likely condition was, surprisingly, the 20-second trials. Hence, the control group were least likely to open only one box on 10- and 15-second trials.

As can be seen from Table 4, there were few significant differences between the qualitative characteristics of the search strategies used by the two groups. Interestingly, there was no significant difference between the two groups with respect to the number of exhaustive searches carried out,

including logical and/or disorganised exhaustive searches. Nevertheless, a significant quadratic interaction was revealed between the number of exhaustive logical searches completed by the control group under different time conditions. The control children completed an increasingly greater number of exhaustive, logical searches with time, up to and including the 15second trial condition. However, on 20-second trials, the number of these types of searches being performed levelled off and was equivalent to that found in the AD/HD group. Thus, by 20-second trials, the clinical group's performance, in this area, was equivalent to that of their peers. Similarly, there were no significant group differences in terms of the number of disorganised searches initiated but a significant quadratic interaction was found with regards to the control group's use of disorganised searches over time. Specifically, the control children initiated increasingly large numbers of disorganised searches until the 20-second trial condition, when a decrease was then found. Interestingly, the control children were significantly more likely to initiate a logical search than AD/HD children.

Efficiency at studying stimuli and identifying the target-copy

In order to compare the efficiency of the two groups at correctly identifying the target-copy when considering (a) the actual time spent studying the stimuli and (b) the number of boxes opened, two types of "efficiency scores" were calculated. The efficiency of both groups at correctly identifying the target-copy on the basis of time available was computed by dividing the time spent viewing all stimuli by the number of target-copies correctly identified. The efficiency of both groups at correctly identifying the

target-copy on the basis of the number of boxes viewed was computed by dividing the total number of boxes opened by the number of correctly identified target-copies. A mixed-design ANCOVA compared the efficiency of AD/HD and control children, with group as the between-subject factor (AD/HD vs. control), time condition as the within-subject factor (5-, 10-, 15and 20- seconds) and age as the covariate. There were no significant differences between the efficiency of the two groups when considering the time available to study stimuli (F(1,46)=1.44; p=.24) and number of boxes opened (F(1,47) < 0.01; p=.95). So, when considering the performance of children with and without AD/HD on the basis of the actual amount of time spent studying the stimuli and number of boxes opened, no difference was found. This suggested that when on-task and opening adequate numbers of boxes, both groups were equally as efficient at correctly identifying the targetcopy. There were also no significant linear interactions for efficiency between group and time on the basis of viewing time (F(1,46)=0.01; p=.92) or number of boxes opened (F(1,47)=0.47; p=.50). Similarly, there were no significant quadratic interactions between group and time on the basis of either viewing time (F(1,46)<0.01; p=.98) or number of boxes opened (F(1,47)=0.22; p=.64).

Factors associated with performance

Pearson Correlations were calculated between performance and all qualitative / quantitative factors in order to highlight which factors were significantly associated with outcome. The results for the 5-second condition are presented in Table 5.

----- Insert Table 5 about here -----

On 5-second trials, significant <u>positive</u> relationships were found between performance and the number of logical searches initiated but not completed and the number of boxes opened. Thus, children generally performed better when they opened more boxes and initiated a logical search, even if they were unable to complete it. In contrast, there was a significant <u>negative</u> relationship between performance and only opening one box. Therefore, as would be anticipated, children performed poorly if they only viewed one box. For the control group specifically, a significant <u>positive</u> relationship was found between performance and the number of logical searches initiated, and a significant <u>negative</u> relationship between performance and only opening one box.

The results for the 10-second condition are presented in Table 6.

----- Insert Table 6 about here -----

On 10-second trials, significant <u>positive</u> relationships were again found between performance and number of logical searches initiated but not completed and the number of boxes opened. The association between performance and total length of time spent viewing stimuli approached significance. Therefore, as on the 5-second trials, children generally performed better the more boxes they opened, the longer they spent viewing the stimuli and the more logical searches they initiated, even if they remained

incomplete. Significant <u>negative</u> relationships were found between performance and delay initiating a search, only opening one box and the length of time spent viewing individual stimuli. In other words, children tended to perform poorly if they were slow to start their search, only opened one box or spent too long viewing individual boxes. In terms of the clinical group specifically, significant <u>positive</u> relationships were found between performance and the number of boxes opened and the number of logical searches initiated. Significant <u>negative</u> relationships were found between performance and only opening one box or spending too long viewing individual stimuli. The negative association between performance and delay initiating a search approached significance. In terms of the control group, a significant <u>negative</u> relationship was demonstrated between performance and delay initiating a search.

The results for the 15-second condition are presented in Table 7.

----- Insert Table 7 about here -----

On 15-second trials, significant <u>positive</u> relationships were generally found between performance and number of exhaustive, logical searches completed and the number of boxes opened overall. A significant <u>negative</u> relationship was found between performance and delay initiating a search. In summary, children performed better the more boxes they opened and if they searched all stimuli in an orderly fashion. However, their performance was impaired if they were slow to initiate their search. With specific consideration

given to the AD/HD group, a significant <u>positive</u> relationship was found between performance and the number of exhaustive, logical searches completed; whilst a significant <u>negative</u> relationship was found between performance and the number of logical searches initiated but not completed.

The results for the 20-second condition are presented in Table 8.

----- Insert Table 8 about here -----

On 20-second trials, a significant <u>positive</u> relationship was again found between performance and the number of boxes opened. A significant <u>negative</u> relationship was found between performance and only opening one box. The negative relationship between performance and delay initiating showed a trend towards significance. In other words, the more boxes opened and the quicker the search was commenced, the better the child's performance.

Discussion

In line with previous research, the results revealed that the performance of AD/HD children on the MFFT was significantly impaired when compared to that of control children. Control children out-performed children with AD/HD on the 5-, 10- and 15-second trial conditions, with the greatest difference appearing to occur on 5-second and 10-second trials. However, both AD/HD and control children tended to display similar patterns of time use namely, a linear relationship between trial length and performance.

Thus, for all children, performance tended to improve with time. Taken together, these findings would appear to provide greatest support for the cognitive deficit model. As described in the introduction, the cognitive deficit model predicted a linear interaction between group and trial duration, with control children outperforming AD/HD children at all durations.

At first, a number of quantitative and qualitative factors appeared to differentiate the clinical group's performance from that of control children. Firstly, children with AD/HD were significantly slower to initiate a search. Secondly, they were significantly more likely to fail to initiate a search or to only open one box. Thirdly, they were significantly less likely to initiate a logical search. Fourthly, they spent significantly less time viewing stimuli. Fifthly, they opened significantly fewer boxes. Finally, they spent significantly more time off-task, particularly on longer trials. However, it is important to note that Pearson Correlations between performance and off-task behaviour revealed that under no conditions was poor performance associated with offtask behaviour, as such, this factor can not be considered to explain AD/HD children's impaired performance. Similarly, poor performance was also not associated with the total length of time spent viewing stimuli. Thus, AD/HD children's impaired performance can not be attributed to a lack of time spent examining stimuli. In contrast, the other four factors described were all found to be significantly associated with the number of target-copies correctly identified by children, although not necessarily under all time conditions. For example, a delay with search initiation was only associated with poor performance on trials lasting at least 10-seconds. The number of boxes

opened was the only factor that was associated with good performance on all trial lengths and thus appears critical to good performance under all conditions. The more boxes opened, the more opportunities the child has to assess whether or not certain stimuli are alike or different to the target and consequently, are more likely to be able to correctly identify the target-copy by the end of the trial.

The fact that children with AD/HD were less likely than controls to initiate a logical search is probably due to their tendency to open only one box or, indeed, to fail to initiate a search. There was no evidence of a fundamental problem with undertaking an orderly search since there was no difference between the two groups in terms of the number of exhaustive, logical searches completed. The AD/HD children's ability to carry out exhaustive, logical searches to the same extent as control children does not support the idea that children with AD/HD experience cognitive deficits in this area; they appear well able to fundamentally organise and carry out an efficient search of all stimuli displayed. Similarly, both groups of children were equally as efficient at studying the stimuli and identifying the target-copy when the number of boxes opened and total viewing time was taken into consideration. Again, this suggests that both those with and without AD/HD are equally proficient at examining and comparing stimuli. The ability to both complete orderly searches and efficiently examine stimuli demonstrates that children with AD/HD do not display obvious information processing difficulties.

Given that the number of boxes opened and a delay initiating a search (under trials of intermediate and long length) appear to explain AD/HD children's poor performance on the MFFT used in the present study, it is important to consider why children with AD/HD display these characteristics. Possible reasons and suggestions for future research are discussed below.

A delay with search initiation could reflect (a) poor motivation. (b) a state of under-arousal or (c) slowness with search planning / pacing / organisation. In terms of poor motivation, it may be that children with AD/HD have difficulties with the provision of internal motivation, which negatively impacts on their ability to engage with the task. Future research could explore the importance of poor internal motivation by offering external rewards dependent on task performance. If children with AD/HD were offered external rewards on the basis of good performance, this would overcome the need to activate sources of internal motivation and thus would compensate for any difficulties in this area. This, in turn, may increase the speed with which children with AD/HD commence their search in order to maximise the time spent on-task and thus opportunities to identify correctly the target-copy. With respect to a state of under-arousal, it could be that the task does not provide adequate stimulation for the child with AD/HD and consequently they are slow to start their search. The task itself may lack sufficient "cognitive-energetic factors" (Sergeant, 2000). Future research could enhance the stimulation and therefore arousal potential of the MFFT by changing certain characteristics of the task, as recommended by Zentall and Zentall (1983). For example, more colourful stimuli may be of more interest

than the black and white line drawings used in the present study. Similarly, the addition of sound effects may add greater stimulation. In addition, trials of differing lengths could be randomly presented as opposed to being randomly presented within blocks; this may add a greater degree of uncertainty and thus increase arousal. Finally, with regard to slow search planning or organisation, it may be that children with AD/HD are slower than their peers to decide which box to open first or spend more time studying the target before commence any search. It would be interesting for future research to explore whether control children are more consistent in opening the same box across all trials compared to those with AD/HD since this would reduce the time taken to decide which box to open first. Similarly, it would be useful for future research to investigate how long control and AD/HD children spend studying the target before opening any boxes.

Opening an <u>insufficient number of boxes</u>, and thus missing opportunities to study stimuli, appears to suggest that children with AD/HD are less efficient than their peers at utilising all the time available during the course of a trial. Control children appear better at maximising all the time afforded by a trial in terms of opening as many boxes as possible, and thus create as many opportunities as possible to identify correctly the target-copy. AD/HD children's tendency to be less efficient at opening the maximum number of boxes afforded by any given trial length may reflect: (a) poor motivation, (b) a state of under arousal and/or (c) slow search planning or an excessive need to re-refer to the target after opening each box.

As described earlier, poor motivation would most likely reflect poor internal motivation in the absence of powerful external reinforcers, whilst a state of under arousal would reflect AD/HD children's sensitivity to lowstimulation tasks. In contrast, slow search planning and/or the need to excessively re-refer to the target could suggest difficulties with certain aspects of the search organisation and problems making the decision where to search next. Slow search planning would reflect AD/HD children's difficulty adjusting the tempo / pace of their search to the time available. It would suggest that children with AD/HD are less able to adjust flexibly the pace of their search when compared to their peers and to this extent would provide support for Sonuga-Barke (2002) compensatory strategy model. Whereas control children may have the ability to quickly decide which box to open after the previous box has been closed when time is at a premium, those with AD/HD may need more time to action their next search and thus are less able to accommodate short trials. They may be unsure which box to open next, or rigidly keep re-visiting the target instead of holding its image in their mind, and may be unable to speed up either process on short trials.

To explore whether children are slow to decide which box to open, it may be useful to track the eye movements of children whilst completing the task. Children with AD/HD may spend more time scanning all the boxes that could be opened next, whereas control children may immediately move their eyes to the next box. Children with AD/HD could also take more time to implement their next search as they employ different types of search orders whereas their peers without AD/HD may consistently employ the same

search order, thereby reducing the time needed to plan a search with every new trial. To investigate whether children with AD/HD need to refer more frequently to the target than control children, it would be necessary to hold the target stimuli in a closed box and record the number of times it is opened during a trial. If children with AD/HD were found to re-visit the target more often than controls, this would indicate either an impaired working memory or problems adjusting their search strategy to compensate for the limited amount of time available.

If future research found that children with AD/HD consistently referred to the target after studying each new stimulus, even on short trials where time is of the essence, this would provide convincing support for the compensatory strategy model proposed by Sonuga-Barke (2002). It would suggest that whilst children with AD/HD are able to search all stimuli in an organised manner, they are unable to adjust flexibly the frequency with which they refer to the target in line with the amount of time available. On longer trials, this compensatory strategy would prove most effective since the length of the trial would accommodate the excessive need to re-visit the target when searching the stimuli and, indeed, may result in a high success rate at accurately identifying the target-copy.

It is interesting to note that the present study found no evidence of impulsivity in children with AD/HD. First, an impulsive child would be expected to initiate their search quickly whereas the reverse was found in this study. Secondly, an impulsive child would fail to complete an exhaustive

search of stimuli, as a decision would be made early on in the search process. Thirdly, an impulsive child would spend less time studying stimuli than their peers without AD/HD; a rapid "yes/no" decision would be made as to whether or not the stimulus being viewed at any particular moment was the target-copy. In contrast, the present study found that children with AD/HD were as efficient and reflective as control children with respect to time spent studying boxes once opened.

It is important to consider the implications of the finding that whilst children with AD/HD spent more time off-task than controls, this behaviour was not associated with impaired performance. As suggested by Alberts and Van der Meere (1992) and Borger and Van der Meere (2000), it may be that children with AD/HD actually <u>prevent</u> deterioration in task efficiency over time by engaging in looking away behaviour. This special form of self-stimulation may effectively compensate for the low level of stimulation provided by the task, thereby enabling children with AD/HD to continue engaging with the task at hand to the best of their ability.

In contrast to Sonuga-Barke (2002), <u>no</u> evidence was obtained in support of the state regulation deficit model and the prediction that children with AD/HD would perform poorly on short trials due to over-activation and on long trials due to under-activation. If anything, the present study appeared to suggest that children with AD/HD may be inherently <u>under-aroused</u> by the task, as they were consistently slow to initiate their search. The different findings between the present study and that of Sonuga-Barke (2002) may

reflect the different sample sizes used. Sonuga-Barke used a clinical sample of 12 whereas this study used a clinical sample of 25. The larger sample used here may have revealed more generaliseable and reliable findings. To confirm this possibility, the current study should be replicated. Alternatively, the version of the MFFT used in this study may have produced different results to that used by Sonuga-Barke. The modifications made to the MFFT used here arguably increased the cognitive demands placed on participants, which may have consequently revealed the cognitive and/or motivational deficits in the AD/HD group. The version of the MFFT used here required the children to hold in their working memory all information concerning stimuli already searched and still to be searched, as only one stimulus could be viewed at any one time. In contrast, Sonuga-Barke's study allowed children to view all of the stimuli all of the time.

When considering the results from this study, it is important to remember that there are a number of factors inherent in the testing condition that may have influenced the performance of all children; namely the presence of an unfamiliar, "authority" figure and the administration of tests in a one-to-one setting with few or no external distractions. These factors may have particularly benefited children with AD/HD by minimising the effects of any attentional, inhibitory and arousal problems (Douglas & Peters, 1979). To overcome these possible performance-enhancing factors, it would be useful for future research to replicate this study in a natural setting, such as the classroom, and using the teacher or parents as the facilitator. It may be

that in real-life situations, the problems identified in this study would be magnified.

It should also be noted that this study is not without its limitations. Firstly, the clinical and control groups were not perfectly matched; children in the clinical group were significantly older than children in the control group and thus age had to be introduced into statistical analyses as a covariate. Introducing a covariate results in a loss of power in analyses of variance. Secondly, completed inattention-hyperactivity SDQ sub-scale scores were available for only 9 children in the clinical sample; many teachers felt unable to respond to salient questions as they had never seen the child off the medication used to control symptoms of AD/HD. Consequently, it is possible that 16 children included in the clinical group potentially would not have met the inclusion criteria had this information been available. Thirdly, only boys were included in the current study; little is known about the possible gender differences in AD/HD. Fourthly, all children completed the experimental task at the beginning of the testing session. This may have particularly benefited children with AD/HD. If the experimental task had been administered after the WISC-III sub-tests, children with AD/HD may have struggled with sustaining attention / concentration and their performance may have been significantly more impaired. In order to have controlled for potential order effects, the order in which the experimental task and WISC-III sub-tests were administered could have been randomised. Finally, in terms of statistical analyses, a large number of different tests were carried out due to the large number of variables that needed to be analysed. Although these tests were

all necessary, it is important to recognise that significant results may have been found by chance alone. When a large number of tests are performed, it is possible that a Type 1 error will result (Cone & Foster, 1997).

In terms of future research, it would be interesting to replicate the current study with adolescents with AD/HD. It is possible that older children with AD/HD learn to partially overcome the cognitive and/or motivational deficits evident in their younger counterparts.

In summary, this study revealed some interesting differences between the way children with and without AD/HD approach the MFFT but further research is essential in order to consider the precise nature of these differences and thus targets for real-life interventions. Better understanding the nature of the difficulties experienced by children with AD/HD when carrying out timed tasks will inform the type of strategies they could be taught within the classroom in order to compensate for their lack of motivation, state of arousal and/or higher cognitive deficits. Teaching children with AD/HD to develop more tailored approaches to the task at hand and/or to learn how to overcome poor motivation may enable them to improve their performance on tasks requiring efficient time use, and thus provide additional opportunities to experience academic success.

References

Achenbach, T. (1991). <u>Manual for the Child Behaviour Checklist 4-18 and</u> <u>1991 profile</u>. Burlington, VT: University of Vermont, Department of Psychiatry.

Alberts, E., & Van der Meere, J. (1992). Observations of hyperactive behaviour during vigilance. Journal of Child Psychology and Psychiatry, 33, 1355-1364.

American Psychiatric Association (1994). <u>Diagnostic and Statistical Manual</u> of Mental Disorders (4th ed.). Washington, DC: American Psychiatric Association.

Barkley, R. (1997). Behavioural inhibition, sustained attention and executive functions: Constructing a unifying theory of AD/HD. <u>Psychological Bulletin</u>, <u>121</u>, 65-94.

Barkley, R. (1998). <u>Attention-Deficit/Hyperactivity Disorder: A Handbook for</u> <u>Diagnosis and Treatment</u> (2nd ed.). New York: Guildford Press.

Barkley, R., Grodzinsky, G., & DuPaul, G. (1992). Frontal Lobe functions in attention deficit disorder with and without hyperactivity: A review and research report. Journal of Abnormal Child Psychology, 20, 163-188.

Borger, N., & Van der Meere, J. (2000). Motor control and state regulation in children with AD/HD: A cardiac response study. <u>Biological Psychiatry, 51,</u> 247-267.

Cone, J., & Foster, S. (1997). <u>Dissertations and theses from start to finish</u>. Washington, DC: American Psychological Association.

Douglas, V. (1983). Attentional and cognitive problems. In M. Rutter (Ed.), <u>Developmental neuropsychiatry</u> (pp. 280-328). New York: Guildford Press.

Douglas, V., & Peters, K. (1979). Towards a clearer definition of the attentional deficit of hyperactive children. In G. Hale & M. Lewis (Eds.), <u>Attention and the development of cognitive skills</u> (pp. 173-247). New York: Plenum Press.

Goodman, R. (1997). The Strengths and Difficulties Questionnaire: A Research Note. Journal of Child Psychology and Psychiatry, 38, 581-586.

Goodman, R., & Scott, S. (1999). Comparing the Strengths and Difficulties Questionnaire and the Child Behaviour Checklist: Is small beautiful? <u>Journal</u> of Abnormal Child Psychology, 27, 17-24.

Houghton, S., Douglas, G., West, J., Whiting, K., Wall, M., Langsford, S., Powell, L., & Carroll, A. (1999). Journal of Child Neurology, 14, 801-805. Kagan, J. (1965). Reflection-impulsivity and reading abilities in primary grade children. <u>Child Development, 36,</u> 609-628.

Karatekin, C., & Asarnow, R. (1998). Components of visual search in childhood-onset Schizophrenia and Attention-Deficit/Hyperactivity Disorder. <u>Journal of Abnormal Child Psychology, 26,</u> 367-380.

Kuntsi, J., & Stevenson, J. (2000). Hyperactivity in children: A focus on genetic research and psychological theories. <u>Clinical Child and Family</u> <u>Psychology Reviews, 3,</u> 1-23.

Pennington, B., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. <u>Journal of Child Psychology and</u> Psychiatry, 37, 51-87.

Seidman, H., Biederman, J., Faraone, S., Weber, W., & Ouellette, C. (1997). Towards defining a neuropsychology of Attention-Deficit/Hyperactivity Disorder: Performance of children and adolescents from a large clinically referred sample. Journal of Consulting and Clinical Psychology, <u>65</u>, 150-160.

Sergeant, J. (2000). The cognitive-energetic model: An empirical approach to Attention-Deficit/Hyperactivity Disorder. <u>Neuroscience and Biobehavioural</u> <u>Reviews, 24, 7-12</u>.

Sonuga-Barke. E. (1994). On dysfunction and function in psychological accounts of childhood disorder. <u>Journal of Child Psychology and Psychiatry</u>, <u>35</u>, 801-815.

Sonuga-Barke, E. (2002). Interval length and time-use by children with AD/HD: A comparison of four models. <u>Journal of Abnormal Child</u> <u>Psychology, 30,</u> 257-264.

Sonuga-Barke, E., Houlberg, K., & Hall, M. (1994). When is impulsiveness" not impulsive? The case of hyperactive children's cognitive style. <u>Journal of</u> <u>Child Psychology and Psychiatry, 35,</u> 1247-1253.

Sonuga-Barke, E., Taylor, E., & Heptinstall, E. (1992). Hyperactivity and delay aversion 11: The effect of self versus externally imposed stimulus presentation periods on memory. <u>Journal of Child Psychology and Psychiatry, 33,</u> 399-409.

Sonuga-Barke, E., Taylor, E., Sembi, S., & Smith, J. (1992). Hyperactivity and delay aversion 1: The effect of delay on choice. <u>Journal of Child</u> <u>Psychology and Psychiatry, 33,</u> 387-398.

Sonuga-Barke, E., Williams, E., Hall, M., & Saxton, T. (1996). Hyperactivity and delay aversion 111: The effect on cognitive style of imposing delay after errors. Journal of Child Psychology and Psychiatry, 37, 189-194.



Van der Meere, J. (1996). The role of attention. In S. Sandberg (Ed.), <u>Hyperactivity disorders of childhood</u> (pp. 111-148). Cambridge: Cambridge University Press.

Wechsler, D. (1992). <u>Wechsler Intelligence Scale for Children – Third</u> Edition UK. Sidcup: The Psychological Corporation Limited.

World Health Organisation (1992). <u>The ICD-10 classification of mental and</u> <u>behavioural disorders.</u> Geneva: World Health Organisation.

Zentall, S., & Zentall, T. (1976). Activity and task performance of hyperactive children as a function of environmental stimulation. <u>Journal of</u> Consulting and Clinical Psychology, 44, 693-697.

Zentall, S., & Zentall, T. (1983). Optimal stimulation: A model of disordered activity and performance in normal and deviant children. <u>Psychological</u> <u>Bulletin, 94, 446-471</u>.

SDQ ratings for the AD/HD and control group

Sub-scale	AD/I	HD gro	oup	Con	trol gro	pup
	M	<u>SD</u>	<u>n</u>	M	<u>SD</u>	<u>n</u>
Parent-version						
Inattention-hyperactivity	9.28	0.94	25	2.52	1.91	25
Emotional symptoms	5.40	2.74	25	1.76	2.01	25
Conduct problems	6.00	1.91	25	1.20	1.32	25
Teacher-version						
Inattention-hyperactivity	9.33	1.11	9	1.92	2.00	25
Emotional symptoms	3.60	2.80	20	0.96	1.57	25
Conduct problems	3.29	2.81	21	0.24	0.60	25

Results from the independent samples t-tests comparing the age, IQ and

Factor	<u>t</u>	df	p
IQ	48	-1.02	.31
Age	48	2.97	<.01*
Parent SDQ			
Emotional problems	48	5.36	<.01*
Conduct problems	48	10.31	<.01*
Teacher SDQ			
Emotional problems	28	3.77	<.01*
Conduct problems	22	4.87	<.01*

SDQ ratings of the two groups

* significant at 99% level or less

Mean number of correct responses with and without age introduced as a

<u>covariate</u>

	<u>Age no</u>	Age not introduced as covariate			Age introduced as covariate			variate
Time	Control	children	AD/HE	D children	Control	children	AD/HD	children
condition	M	<u>SE</u>	M	<u>SE</u>	M	<u>SE</u>	M	<u>SE</u>
5 sec	3.68	2.27	2.16	1.40	3.95	0.38	2.10	0.38
10 sec	5.40	2.31	3.76	2.11	5.67	0.44	3.81	0.44
15 sec	5.76	2.60	4.56	2.29	6.02	0.51	4.42	0.51
20 sec	5.28	2.32	5.12	2.52	5.51	0.48	5.09	0.48

	Grou	o effe	<u>ots</u>	Inter	actions	s betwe	en gro	up and	<u>d time</u>
					<u>Linea</u>	<u>r</u>	<u>C</u>	<u>)uadra</u>	tic
Factor	<u>F</u>	<u>df</u>	p	E	<u>df</u>	p	<u>F</u>	<u>df</u>	p
Quantitative									
Time off-task	11.26	1,46	<.01*	14.11	1,46	<.01*	0.01	1,46	.94
Delay initiating	6.04	1,47	.02*	0.77	1,47	.77	7.59	1,47	< .01*
No search	8.33	1,47	<.01*	10.81	1,47	<.01*	3.51	1,47	.07
Viewing time	7.60	1,46	<.01*	0.07	1,46	.79	0.72	1,46	.40
Number boxes	10.43	1,47	<.01*	0.18	1,47	.67	13.05	5 1,47	<.01*
One box only	14.79	1,47	<.01*	4.10	1,47	.05*	4.81	1,47	.02*
Av. stimulus time	2.86	1,46	.10	1.05	1,46	.31	8.14	1,46	<.01*
Qualitative									
Exhaustive	1.41	1,47	.24	0.01	1,47	.96	5.46	1,47	.02*
Exh. logical	1.88	1,47	.18	0.03	1,47	.02	5.91	1,47	.02*
Exh. disorganised	0.64	1,47	.43	0.53	1,47	.47	0.01	1,47	.94
Initiated logical	27.38	1,47	<.01*	3.69	1,47	.06	2.76	1,47	.10
Initiated disorgan.	1.65	1,47	.21	0.15	1,47	.70	6.43	1,47	.02*

Group comparisons on quantitative and qualitative search characteristics

Table 4

* significant at 95% level or less

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Factors	All p	artici	pants	٨D	HD (Group	 Cor	itrol	Group
correlated with									
performance	<u>r</u>	<u>df</u>	р	<u>r</u>	<u>df</u>	p	<u>r</u>	<u>df</u>	р
Quantitative							 		
Time off-task	05	47	.71	.01	22	.96		-	
Delay initiating	22	48	.12	12	23	.56	34	23	.09
No search	23	48	.10	11	23	.60	17	23	.42
Viewing time	.21	47	.15	94	22	.94	.35	23	.09
Number boxes	.36	48	.01*	.25	23	.23	.31	23	.13
One box only	39	48	<.01*	20	23	.34	41	23	.04*
Av. box open	17	47	.24	16	22	.46	10	23	.63
<u>Qualitative</u>									
Exhaustive		-			-			-	
Exh. logical		-			-			-	
Exh. disorganised		-			-			-	
Initiated logical	.48	48	<.01*	.35	23	.09	.41	23	.04*
Initiated disorgan.	14	48	.34	16	23	.44	.05	23	.83

Correlation matrix for 5-second condition

Table 5

* significant at 95% level or less

- could not be computed because at least one of the variables was constant

Factors	All participants	AD/HD Group	Control Group
correlated with			
performance	<u>r df p</u>	<u>r df p</u>	<u>r df p</u>
Quantitative	ан <u>ал у Колон</u> алияния на 1900 4 ,000 4 ,000 4 ,000 годинали	uyu yilin da araa ahaa ahaa ahaa ahaa ahaa ahaa a	
Time off-task	19 47 .20	25 22 .25	.14 23 .49
Delay initiating	44 48 <.01*	38 23 .06	47 23 .02*
No search	10 48 .50	19 23 .37	.14 23 .49
Viewing time	.27 47 .06	.22 22 .30	.33 23 .11
Number boxes	.53 48 <.01*	.66 23 <.01*	.24 23 .25
One box only	50 48 <.01*	61 23 <.01*	22 23 .28
Av. box open	38 47 <.01*	56 22 <.01*	06 23 .79
Qualitative			
Exhaustive	.17 48 .26	.29 23 .17	.09 23 .67
Exh. logical	.17 48 .26	.29 23 .17	.09 23 .67
Exh. disorganised	-	-	-
Initiated logical	.37 48 .02*	.56 23 <.01*	21 23 .33
Initiated disorgan.	.12 48 .40	.27 23 .19	.10 23 .63

Correlation matrix for 10-second condition

Table 6

* significant at 95% level or less

- could not be computed because at least one of the variables was constant

Factors	All participants	AD/HD Group	Control Group
correlated with			
performance	<u>r df p</u>	<u>r df p</u>	<u>r df p</u>
Quantitative			
Time off-task	18 47 .22	36 22 .09	.24 23 .25
Delay initiating	37 48 <.01*	36 23 .08	31 23 .13
No search	-	-	-
Viewing time	02 47 .89	08 22 .70	.07 23 .74
Number boxes	.28 48 .05*	.38 23 .06	.01 23 .97
One box only	15 48 .31	13 23 .53	.14 23 .49
Av. box open	20 47 .16	28 22 .19	.02 23 .91
Qualitative			
Exhaustive	.31 48 .03*	.44 23 .03*	.13 23 .55
Exh. logical	.30 48 .04*	.42 23 .04*	.11 23 .59
Exh. disorganised	.25 48 .09	.38 23 .06	.14 23 .50
Initiated logical	25 48 .10	40 23 .05*	08 23 .70
Initiated disorgan.	.05 48 .76	.20 23 .34	20 23 .34

Correlation matrix for 15-second condition

Table 7

* significant at 95% level or less

- could not be computed because at least one of the variables was constant

Factors	All participants	s AD/HD Group	Control Group
correlated with			
performance	<u>r df p</u>	<u>r df p</u>	<u>r df p</u>
Quantitative			
Time off-task	.02 47 .91	08 22 .70	.19 23 .36
Delay initiating	27 48 .06	14 23 .49	38 23 .06
No search	03 48 .85	.06 23 .77	12 23 .58
Viewing time	.13 47 .38	.05 22 .83	.26 23 .21
Number boxes	.28 48 .05*	.18 23 .39	.38 23 .06
One box only	31 48 .03*	28 23 .18	35 23 .09
Av. box open	16 47 .26	09 22 .67	24 23 .17
Qualitative			
Exhaustive	.13 48 .39	05 23 .80	.34 23 .10
Exh. logical	.13 48 .38	06 23 .78	.35 23 .08
Exh. disorganised	.01 48 .96	.03 23 .89	02 23 .93
Initiated logical	07 48 .66	.02 23 .93	15 23 .47
Initiated disorgan.	.12 48 .12	.34 23 .10	.12 23 .58

Correlation matrix for 20-second trial

* significant at 95% level or less

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Appendixes

Appendix A

Journal of Abnormal Child Psychology: Author Instructions

Journal of Abnormal Child Psychology

An official publication of the International Society for Research in Child and Adolescent Psychopathology

1. Manuscripts should be submitted to the Editor:

Susan B. Campbell Department of Psychology University of Pittsburgh 210 South Bouquet Street MPAC Bldg, 3rd Floor Pittsburgh, Pennsylvania 15260

e-mail: <u>sbcamp+@pitt.edu</u>

Copies of the manuscript will *not* be returned by the Editor to the author unless the manuscript submission is accompanied by a stamped, self-addressed envelope.

- 2. Submission is a representation that the manuscript has not been published previously and is not currently under consideration for publication elsewhere. A statement transferring copyright from the authors (or their employers, if they hold the copyright) to Plenum Publishing Corporation will be required before the manuscript can be accepted for publication. The Editor will supply the necessary forms for this transfer. Such a written transfer of copyright, which previously was assumed to be implicit in the act of submitting a manuscript, is necessary under the U.S. Copyright Law in order for the publisher to carry through the dissemination of research results and reviews as widely and effectively as possible.
- 3. Type double-spaced on one side of 8 $1/2 \times 11$ inch white paper using generous margins on all sides, and submit the original and four copies (including copies of all illustrations and tables).
- 4. A title page is to be provided and should include the title of the article, author's name (no degrees), author's affiliation, and suggested running head. Academic affiliations of *all* authors should be included. The affiliation should comprise the department, institution (usually university or company), city, and state (or nation) and should be typed as a footnote to the author's name. The suggested running head should be less than 80 characters (including spaces) and should comprise the article title or an abbreviated version thereof. For office purposes, the title page should include the complete mailing address, telephone number, and fax number of the one author designated to review proofs.
- 5. An abstract, preferably no longer than 150 words, is to be provided as the second page.
- 6. A list of 4–5 key words is to be provided directly below the abstract. Key words should express the precise content of the manuscript, as they are used for indexing purposes.
- 7. Illustrations (photographs, drawings, diagrams, and charts) are to be numbered in one consecutive series of Arabic numerals. Photographs should be large, glossy prints, showing high contrast. Drawings should be high-quality laser prints or should be prepared with india ink. Either the original drawings or high-quality photographic prints are acceptable. Artwork for each figure should be provided on a separate sheet of paper. Identify figures on the back with author's name and number of the illustration. Each figure should have an accompanying caption. The list of captions for illustrations should be typed on a separate sheet of paper. Electronic artwork submitted on disk should be in the TIFF or EPS format (1200 dpi for line and 300 dpi for half-tones and gray-scale art). Color art should be in the CYMK color space. Artwork should be on a separate disk from the text, and hard copy *must* accompany the disk.
- Tables should be numbered and referred to by number in the text. Each table should be typed on a separate sheet of paper and should have a descriptive title. Center the title above the table, and type explanatory footnotes (indicated by superscript lowercase letters) below the table.
- 9. List references alphabetically at the end of the paper and refer to them in the text by name and year in parentheses. References should include (in this order): last names and initials of *all* authors, year published, title of article, name of publication, volume number, and inclusive pages. The style and punctuation of the references should conform to strict APA style illustrated by the following examples:

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Journal Article

Peyrot, M. (1996). Causal analysis: Theory and application. *Journal of Pediatric Psychology*, *21*, 3--24. *Book*

Hembree-Kigin, T. L., & McNeil, C. B. (1995). *Parent-child interaction therapy.* New York: Plenum Press.

Contribution to a Book

Melamed, B. G., Meyer, R., Gee, C., & Soule, L. (1993). The influence of time and type of preparation on children's adjustment to hospitalization. In M. C. Roberts, G. P. Koocher, D. K. Routh, & D. J. Willis (Eds.), *Readings in pediatric psychology* (pp. 223--236). New York: Plenum Press.

- 10. Footnotes should be avoided. When their use is absolutely necessary, footnotes should be numbered consecutively using Arabic numerals and should be typed at the bottom of the page to which they refer. Place a line above the footnote, so that it is set off from the text. Use the appropriate superscript numeral for citation in the text.
- 11. The 1994 *Publication Manual of the American Psychological Association* (Fourth Edition) should be used as the style guide for the preparation of manuscripts, particularly with respect to such matters as the citing of references and the use of abbreviations, numbers, and symbols. Manuscripts departing significantly from Fourth-Edition style will not be reviewed until a corrected manuscript has been received.
- 12. After a manuscript has been accepted for publication and after all revisions have been incorporated, manuscripts should be submitted to the Editor's Office as hard copy accompanied by electronic files on disk. Label the disk with identifying information software, journal name, and first author's last name. The disk *must* be the one from which the accompanying manuscript (finalized version) was printed out. The Editor's Office cannot accept a disk without its accompanying, matching hard-copy manuscript.
- 13. The journal makes no page charges. Reprints are available to authors, and order forms with the current price schedule are sent with proofs.

Appendix B

Letters confirming ethical approval



University of Southampton

Department of Psychology University of Southampton Highfield Southampton SO17 1**B**J United Kingdom

Telephone +44 (0)23 8059 5000 Fax +44 (0)23 8059 4597 Email

4 May 2001

Sarah Elgie 20 Glebe Court Highfield Lane Highfield Southampton SO17 1RH

Dear Sarah,

Re: Visual search in attention deficit hyperactivity disorder: An exploratory study

The above titled application - which was recently submitted to the departmental ethics committee, has now been given approval.

Should you require any further information, please do not hesitate in contacting me on 023 8059 3995.

Yours sincerely,

Kathryn Smith Ethical Secretary

cc. Janet Turner

NORTH AND MID HAMPSHIRE LOCAL RESEARCH ETHICS COMMITTEES North and Mid Hampshire Health Authority Harness House Aldermaston Road, Basingstoke Hampshire RG24 9NB Tel: 01256 312248 Fax: 01256 312299 Email: sandra.tapping@nm-ha.nhs.uk www.hants.gov.uk/nmhha/ethics.html

Chair of Biomedical Committee: Chair of Qualitative and Non-invasive Committee: Ethics Committee Co-ordinator: Mrs Jane Ogden-Swift Rev'd Dr Rosemary Baker Mrs Sandra Tapping

Our ref: SKT/mk/316B/L4233

25th October 2001

Ms Sarah Elgie 20 Glebe Court Highfield Lane Highfield Southampton Hants

Dear Ms Elgie

316/B – Visual search in children with Attention Deficit Hyperactivity Disorder : An exploratory study

Decision - Approval

Thank you for your letter of 21st October 2001 which dealt with minor amendments. I am satisfied with your response and am empowered to grant you full approval.

I must emphasise that whilst the committee look at work on ethical grounds, it is up to the Trust to finally sanction the work, taking into account financial and other implications.

To comply with good practice a list of members at the July meeting is enclosed.

The committee wish you every success with the study. The following conditions apply to all approvals:

- (a) that you notify the LREC immediately of any information received or of which you become aware which would cast doubt upon, or alter, any information contained in the original application, or a later amendment application, submitted to the LREC and/or which would raise questions about the safety and/or continued conduct of the research.
- (b) you need to comply with the latest Data Protection Act and Caldicott Guardian issues.

- (c) you need to comply throughout the conduct of the study, with good clinical research practice standards, including obtaining informed consent.
- (d) you need to refer proposed amendments to the protocol to the LREC for further review and to obtain LREC approval thereto prior to implementation (except only in cases of emergency where the welfare of the subject is paramount).
- (e) you must supply an annual summary of the progress of the research project and of the conclusion and outcome of the research project and inform the LREC should the research be discontinued.
- (f) that satisfactory indemnity arrangements agreed with the Trust are in place before the study commences.

The committee is fully compliant with the International Committee on Harmonisation/Good Clinical Practice (ICH) Guidelines for the Conduct of Trials involving the participation of human subjects as they relate to the responsibilities, composition, function, operations and records of an independent Ethics Committee/Independent Review Board. To this end it undertakes to adhere as far as is consistent with its Constitution, to the relevant clauses of the ICH Harmonised Tripartite Guideline for Good Clinical Practice, adopted by the Commission of the European Union on 17 January 1997.

Yours sincerely

Jill Parnham Vice Chair – Qualitative and Non-invasive Committee

cc Maureen Larkin – RHCH

enc July meeting members

logel Research Ethics Committees

West Dorset General Hospitals

GP/DM

Your Ref: (01305) 254646 (Secretary)

Direct Dial 5 July 2001

Our Ref:

E-mail: Miss S Elgie 20 Glebe Court Highfield Lane Highfield **SOUTHAMPTON** Hampshire SO17 1RH

Dear Miss Elgie

Title:

Visual search in children with ADHD

Date of Submission: 2 May 2901

Date of Approval: 5 July 2001

Research Worker: Miss Elgie

Ethical approval is given for this project to be conducted to the submitted protocol in West Dorset for a period of two years. If the project is not started within this time, further approval should be sought.

You are required to notify us if the questionnaire changes significantly after the pilot.

You are required to keep raw data in hard copy for a period of ten years to avoid the fraudulent use of any data collected.

You must notify the NHS body under whose auspices the research will take. In the case of the West Dorset General Hospitals NHS Trust, this notification should be made to Ms S Mooney, Research & Development Manager. Your research must not proceed until the Research & Development Committee has given you their agreement if your study involves patients within this Trust. Your study will also be registered in the National UK Research Register. You should agree to make your results publicly accessible.

We wish you well with your project. You are required to provide this Committee with a brief report on progress of the project at least once a year.

NHS Trust

Dorset County Hospital Williams Avenue Dorchester Dorset DT1 2JY

Telephone: 01305 251150 Fax: 01305 254155 Minicom: 01305 254444



Appendix C

Sample information sheet and consent form for parents of clinical

participants

Dear Parent / Guardian

Re: Information regarding a study investigating how children with Attention Deficit / Hyperactivity Disorder (AD/HD) utilise study time

I am Sarah Elgie, a Trainee Clinical Psychologist at the University of Southampton. As part of my doctoral degree in clinical psychology, I am conducting a research study that looks at how children use study time. This project will be supervised by Professor Sonuga-Barke (University of Southampton) and (name and address of relevant Consultant Psychiatrist or Paediatrician). I am writing to ask whether you would give permission for your child to take part, along with about 60 others. The (name of relevant LREC) has reviewed this study.

If you agree to your child taking part, he will be required to complete some puzzles and perform four short computer games. This should take about one hour. Your child should experience no distress. If your child is on medication for AD/HD, it is important that he does not take these particular drugs on the day of testing. Your child's responses will be videotaped. This videotape will be analysed and then destroyed on completion of the study in September 2002, it will be used for no other purposes. All your child's responses will be kept anonymous and personal information will not be released to, or viewed by, anyone other than the researchers in this project. Results of this study will not include your child's name or any other identifying characteristics.

You or your child can withdraw permission for involvement at any time, without affecting any services you receive.

If you agree to your child taking part, please read and sign the attached consent form and complete the enclosed questionnaire, and return them to me using the stamped addressed envelope provided. When I hear back from you, I will get in contact to arrange a time for your child to meet with me at (name of local child and family guidance centre). At this time, I will also ask you if you would agree to me contacting your child's teacher, to ask if they would complete a questionnaire.

If you are happy to be contacted by telephone, please could you fill in the attached slip and return it to me along with the consent form and questionnaire.

If you have any questions, please do not hesitate to get in contact.

Many thanks for your help.

Yours faithfully

Sarah Elgie Trainee Clinical Psychologist I am happy to be contacted by telephone

Child's name:.....

Contact telephone number:.....

If there are specific times when it would be more convenient to be telephoned please indicate below:

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Consent Form

Children with AD/HD and study time use

I hereby consent for my child to take part in the above clinical research about which I have received written information.

Child's full name:

Parent/Guardian's full name:

Please answer the following questions by circling Yes or No as applicable:

I have read the information sheet	Yes / No
I know who to contact if I have any questions or want to discuss the study	Yes / No
I have received satisfactory answers to all my questions	Yes / No
I agree to my child's responses being recorded on video tape	Yes / No
 I understand that we are free to withdraw from the study: at any time without having to give a reason why without affecting any service we might receive. 	Yes / No Yes / No Yes / No
Signed:	
Date:	

Please return using the stamped addressed envelope provided.

Appendix D

Sample information sheet and consent form sent to parents of control

participants

Dear Parent / Guardian

Re: Information regarding a study investigating how children with Attention Deficit / Hyperactivity Disorder (AD/HD) use study time.

I am Sarah Elgie, a Trainee Clinical Psychologist at the University of Southampton. As part of my doctorate in clinical psychology, I am conducting a study that looks at how children with and without problems with attention, impulsivity and hyperactivity use study time. Professor Sonuga-Barke, Head of Psychology, is supervising this project at the University of Southampton.

I am writing to ask if you would be prepared to give permission for your child to take part in this study, along with about 30 others from Ludlow Junior School. Your child will enable us to look at how children **without** attention, impulsivity and hyperactivity difficulties use study time. Children with these difficulties are being recruited via the Children's Centre, Dorset County Hospital, Friarsgate Medical Centre, Winchester and Family Consultancy, Andover.

If your child takes part, he will be asked to complete some puzzles and perform four short computer games. This should take about one hour and would take place during the school day. Your child should experience **no** distress. Your child's responses will be videotaped. This videotape will be analysed and then destroyed on completion of the study in September 2002, it will be used for no other purposes. All your child's responses will be kept anonymous and the results of this study will not include your child's name or any other identifying characteristics. In addition, you or your child can withdraw permission for involvement at any time.

If you **agree** to your child taking part, please could you sign the enclosed consent form and complete the enclosed questionnaire and then return them to me, via your child's teacher, using the envelope provided. If, and when, I hear back from you, I will arrange a time to meet with your child at school. On receiving consent from you, I will also ask your child's teacher to complete a questionnaire.

If you have any questions or queries about this study please do not hesitate to get in contact with me at the above address and I will get back to you as soon as possible. Just to reassure you, (name of head teacher) has seen all the puzzles and computer games your child would be asked to complete should you agree to him or her participating and is aware that I am contacting you.

Many thanks for your time and help.

Yours faithfully

Sarah Elgie Trainee Clinical Psychologist

Consent Form

Children with AD/HD and study time use

I hereby consent for my child to take part in the above clinical research about which I have received written information.

Child's full name:

Class:

Parent/Guardian's full name:

Please answer the following questions by circling Yes or No as applicable:

I have read the information sheet Yes / No				
I know who to contact if I have any Yes questions about the study				
I have received satisfactory answers to Yes / No all my questions				
I agree to my child's responses being Yes / No recorded on video tape				
 I understand that we are free to withdraw from the study: at any time Yes / No without having to give a reason why Yes / No without penalty or loss of benefit Yes / No to my child or myself. 				
Signed:				
Date:				

Please return using the envelope provided.

Appendix E

Strengths and Difficulties Questionnaire

Strengths and Difficulties Questionnaire

For each item, please mark the box for Not True, Somewhat True or Certainly True. It would help us if you answered all items as best you can, even if you are not absolutely certain or the item seems daft! Please give your answers on the basis of your child's behaviour over the last six months.

Is your child	Not True	Somewhat True	Certainly True
Considerate of other people's feelings			
Restless, overactive, cannot stay still for long			
Often complains of headaches, stomach-aches or sickness			
Shares readily with other children (treats, toys, pencils etc)			
Often has temper tantrums or hot temper			
Rather solitary, tends to play alone			
Generally obedient, usually does what adults request			
Many worries, often seems worried			
Helpful if someone is hurt, upset or feeling ill			
Constantly fidgeting or squirming			
Has at least one good friend			
Often fights with other children or bullies them			
Often unhappy, down-hearted or tearful			
Generally liked by other children			
Easily distracted, concentration wanders			
Nervous or clingy in new situations, easily loses confidence			
Kind to younger children			
Often lies or cheats			
Picked on or bullied by other children			
Often volunteers to help others (parents/teachers/other children)			
Thinks things out before acting			
Steals from home, school or elsewhere			
Gets on better with adults than with other children			
Many fears, easily scared			
Sees tasks through to the end, good attention span			

Appendix F

Information sheet for teachers of clinical participants

Dear (name of teacher)

Re: Information regarding a study investigating how children with Attention Deficit / Hyperactivity Disorder (AD/HD) use study time.

I am Trainee Clinical Psychologist at the University of Southampton and am currently carrying out research into how children with Attention Deficit/Hyperactivity Disorder use study time. Professor Sonuga-Barke (University of Southampton) and (name and address of local Consultant Paediatrician or Psychiatrist) are supervising this project. It has been approved by the (name of relevant LREC).

(Child's name) is taking part in this research and I understand that you are his form teacher. As part of the study, I would like to ask you to complete the *Strengths and Difficulties Questionnaire*, which you will find enclosed. (Child's name)'s mother is aware that I am approaching you and has given her consent. This questionnaire provides valuable information about a range of difficulties that may be experienced by children with Attention Deficit/Hyperactivity Disorder. When you have completed the enclosed questionnaire, I would be extremely grateful if you could return it to me using the stamped addressed envelope provided.

If you have any questions or would like to know more about this study, please do not hesitate to get in contact with me via the above address / telephone number.

Thank you very much for your time and help.

Yours sincerely

Sarah Elgie Trainee Clinical Psychologist Appendix G

Consent form for children

Consent Form

Children with AD/HD and study time use

I am Sarah Elgie and I work at the University of Southampton. I would like to ask you a few questions, ask you to do a few puzzles and then ask you to play some games on the computer. While you are playing on the computer, I would like to video you so that I can look at how you play later.

I won't tell anyone about your answers; it's just for me to find out about you. If you don't want to answer a question, do one of the puzzles or play any of the computer games just let me know and we'll move onto the next. Also, if you want to stop at any point just tell me. You don't have to finish anything if you don't want to.

Do you have any questions?

Shall we start?

Name:

Signed:

Date:

Appendix H

Debriefing statements

Debriefing statement for parents of clinical participants

Dear (name of parent / guardian)

Re: Research study investigating how children with AD/HD use study time

Following your recent meeting with me, I would like to thank you for allowing (child's name) to participate in the above research. The main aim of the project is to try and better understand why children with Attention Deficit/Hyperactivity Disorder (AD/HD) often struggle to make efficient use of study time and therefore tend to under perform on tasks. It is exploring several different theories that all potentially account for why this may be the case. Your child made a valuable contribution to my study by providing important information on how children with AD/HD utilise study time, along with about 30 others from (names of participating clinics).

Once again, I would like to reassure you that the results of this study will not include your child's name or any other identifying characteristics. Also, the video of (child's name) will be destroyed in September 2002, when the research is completed.

As you are aware, I will not need to meet with your child again. However, if you or (child's name) have any questions following his participation, please do not hesitate to contact me at the above address. If you would like a summary of the results, please return the slip below to me. A summary should be available by September 2002.

Thank you very much for your help with this research.

Yours sincerely

Sarah Elgie Trainee Clinical Psychologist

If you have any questions about your rights as a participant in this research, or if you feel that your child has been placed at risk, you may contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ.

Please send me a summary of the results of the research study on how children with AD/HD use study time.

NAME:	
ADDRESS:	· · · · · · · · · · · · · · · · · · ·
	·····

Debriefing statement for parents of control participants

Dear (name of parent / guardian)

Re: Research study investigating how children with AD/HD use study time

I would like to thank you for letting your son participate in the above research. The main aim of the project is to try and better understand why children with Attention Deficit / Hyperactivity Disorder (AD/HD) often struggle to make efficient use of study time and therefore tend to under perform on tasks. It is exploring several different theories that all potentially account for why this may be the case. Your child made a valuable contribution to my study by providing important information on how *children without AD/HD* utilise study time, along with about 30 others from Ludlow Junior School.

Once again, I would like to reassure you that the results of this study will not include your child's name or any other identifying characteristics. Also, the video of your child participating will be destroyed in September 2002, when the research is completed.

I will not need to meet with your child again. However, if either you or your son have any questions, please do not hesitate to contact me via the above address / telephone number. If you would like a summary of the results, please return the slip below to me. A summary should be available by September 2002.

Thank you very much for your help with this research.

Yours sincerely

Sarah Elgie Trainee Clinical Psychologist

If you have any questions about your rights as a participant in this research, or if you feel that your child has been placed at risk, you may contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ.

Please send me a summary of the results of the research study on how children with AD/HD use study time.