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Attention Deficit/Hyperactivity Disorder:

The role of delay aversion and attentional bias

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Abstract

The literature review explores the nature of attention deficit/hyperactivity disorder (AD/HD), and discusses aetiologies, and theoretical models. The focus is upon motivational accounts such as delay aversion, in addition to cognitive deficits in the form of response inhibition. These come together in a Dual Pathway Model proposed by Sonuga-Barke (2001). Attentional bias toward threatening stimuli in anxiety disorders is also discussed. Literature on the impact of motivation and emotion on attentional processes in child psychopathologies is extrapolated to externalising disorders such as AD/HD. Delay aversion theory argues that delay cues in the environment are of motivational significance to AD/HD children.

The empirical study examines the evidence for delay aversion in boys clinically diagnosed with AD/HD. Their performance on a computerised choice-delay task is compared to that of boys with comorbid AD/HD and conduct disorder, boys with conduct disorder only, and normal controls. The study also compares their bias towards delay-related words and social and physical threat words in a modified selective attention paradigm. Results indicate no significant group differences on either task. Methodological issues are discussed in relation to the pattern of findings, suggestions for future research are highlighted, and clinical implications are raised.

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Literature Review

Attention Deficit/Hyperactivity Disorder:

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Attention Deficit/Hyperactivity Disorder:

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Running head

AD/HD, delay aversion, and attentional bias

Attention Deficit/Hyperactivity Disorder:

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Abstract

AD/HD has attracted nearly a century of clinical papers and scientific studies. This review looks at aetiological developments, discusses psychological theories, and introduces research on attentional biases to the AD/HD arena.

Family, adoptee and twin studies indicate a strong role for genetic factors in the aetiology of AD/HD. Evidence is also emerging from molecular genetic studies implicating specific genes that may be involved in the disorder. Psychological accounts have characterised AD/HD as either a neuro-cognitive disorder of regulation or a motivational style. Poor inhibitory control is thought to underpin AD/HD children's dysregulation, while delay aversion is a dominant characteristic of their motivational style. This review particularly discusses evidence for delay aversion, and the possible integration of the two lines of research in a recently proposed dual pathway model of AD/HD (Sonuga-Barke, 2001). The model recognises two quite distinct subtypes of the disorder. Furthermore, research on attentional biases in internalising disorders is discussed, and this is extrapolated to AD/HD children. Studies are emerging that show AD/HD children to have an attentional bias towards delay-related environmental cues, supporting the idea that such cues are of motivational significance to this clinical group.

Introduction

Attention Deficit/Hyperactivity Disorder (AD/HD) is the current label to describe children, adolescents, and adults who display chronic, pervasive, and developmentally inappropriate patterns of inattention, impulsivity, and/or hyperactivity.

Aetiological findings attempt to explain AD/HD at a number of different levels including neurochemical, neuroanatomical and genetic. Over the years, a number of theoretical models have tried to account for the disorder, using biological and psychological conceptualisations. Despite attracting great scientific interest, there are inconsistencies in findings regarding the underlying psychological mechanisms responsible for AD/HD. Such models have mostly focused on the extent to which deficits in inhibitory control are implicated in the impulsivity so often seen in children with AD/HD. In contrast, other theories, such as Delay Aversion (Sonuga-Barke, 1994) take a motivational approach, suggesting that children with AD/HD differ from other children primarily in the way in which they are motivated with regard to time in general, and delay in particular. They see delay as a threat, and their behaviour is an attempt to minimise actual delay as well as the perception of delay.

This paper aims to look at recent etiological findings, review the main theories of AD/HD, particularly the role that cognitive and motivational processes play in the disorder, and possible integration of the two lines of research. If AD/HD children see delay as a threat and, according to theory, have adopted an attentional style to minimise perceived delay at an early stage, they may develop an attentional bias to cues that signal its presence within the environment. This review therefore draws from the literature on selective attention and anxiety in search of support for a motivational approach to understanding AD/HD.

What is AD/HD?

Definitions, historical perspective and conceptual shifts

The history of AD/HD has been reviewed elsewhere (Barkley, 1998; Schachar, 1986) so I only briefly consider it here. Initially, the symptoms were thought to arise out of poor volitional inhibition and defective moral regulation of behaviour. Still (1902) described children who were often aggressive, defiant, resistant to discipline, and excessively emotional, and who showed little 'inhibitory volition'. He proposed the immediate gratification of the self as being the principal quality of these and other attributes of the children. He also noted that insensitivity to punishment characterised many of these cases, as well as problems with sustained attention. Still (1902) attributed such behaviour to a 'defect of moral control', believing it to be a biological defect which was inherited or resulted from some pre- or postnatal injury.

Like Still, Tredgold (1908) believed that such moral deficiency was caused by the inheritance of some brain defect that was being passed from generation to generation. In his view, environmental circumstances played no significant role in its causation. For the first half of the twentieth century, the predominant view regarding the causation of hyperactivity continued to be that of an association with brain damage. Terms such as 'organic driveness', 'minimal brain damage', and 'hyperkinetic disease' were used to describe the disorder.

During the late 50s and early 60s, these terms were replaced by more specific labels such as dyslexia, language disorders, and hyperactivity. New labels were based on the observable and descriptive deficits of children rather than on some underlying unobservable aetiological mechanism in the brain. Problems of hyperactivity were now thought to be the major feature of the disorder (Laufer & Denhoff, 1957; Chess, 1960).

Eventually, Douglas (1972) and her team at McGill University stressed an equal if not greater role for poor sustained attention and impulse control in the disorder. She suggested that motor activity was not the core symptom. This model helped establish research in the study of cognitive processes, and was probably the major reason the disorder was renamed Attention-Deficit Disorder (ADD) with the publication of DSM-III (APA, 1980). She subsequently amended her view to include four major deficits: (a) poor investment and maintenance of effort, (b) deficient modulation of arousal to meet situational demands, (c) a strong inclination to seek immediate reinforcement, along with (d) the originally proposed difficulties with impulse control (Douglas, 1983). Douglas (1988) later concluded that these four deficiencies arise from a more central impairment in self-regulation in AD/HD.

The central importance of attention to the disorder was questioned in the late 1970s as some researchers quoted the situational variability of the symptoms, while others more fully examined the attentional construct (Douglas & Peters, 1979; Rosenthal & Allen, 1978). More recently, the focus has switched to impulsiveness and related concepts (Barkley, 1994).

Over the years, many terms have been used to characterise the syndrome. Although confusing to some, this periodic relabelling has not been without purpose. On the contrary, each diagnostic term has reflected shifts in the way that this disorder has been conceptualised at different points in time. Currently, there are two terms for this disorder: attention-deficit hyperactivity disorder (AD/HD) and hyperkinetic disorder (HKD). Throughout this review preference will be given to the term Attention Deficit/Hyperactivity Disorder (AD/HD), since this is currently the most widely used term. Although there is broadening acceptance of AD/HD into adulthood, this review will focus on children.

Primary symptoms and diagnostic criteria

The specific diagnostic criteria for AD/HD are in the American Psychiatric Association's Diagnostic and Statistical Manual (DSM-IV, 1994) and those for HKD are in the International Classification of Diseases (ICD-10, 1992 and 1993) manual published by the World Health Organisation.

The essential feature of AD/HD is a persistent pattern of inattention and/or hyperactivity-impulsivity that is more frequent and severe than is typically observed in individuals at a comparable level of development. According to DSM-IV, some symptoms that cause impairment must have been present before age 7 years, although many individuals are diagnosed after the symptoms have been present for a number of years. Some impairment from the symptoms must be present in at least two settings (e.g. at home and at school). There must be clear evidence of interference with developmentally appropriate social, academic, or occupational functioning.

Overactivity

All major diagnostic categorisations used at present agree that excessive locomotor activity is part of the disorder. Parents often describe children as 'always on the go', 'acts as if driven by a motor', 'cannot sit still', 'talks excessively'. Observations of such children at school finds them out of their seats, moving about the classroom without permission, restlessly moving their arms and legs while working (Abikoff, Gittelman-Klein, & Klein, 1977; Barkley, DuPaul & McMurray, 1990). Numerous research studies confirm these complaints that AD/HD children are more active, restless, and fidgety than normal children throughout the day and even during their sleep (Taylor, Sandberg, Thorley & Giles, 1991; Teicher, Ito, Glod & Barber, 1996). Such excess of locomotor activity applies across situations from set tasks in the classroom to those

allowing children a choice of activity; from meal times at home to quiet play and watching television.

Inattention

Children rated as hyperactive show inattentive behaviour in many situations; in activities they choose at home as well as in tasks set by teacher at school. Inattention can also be objectively recorded by an outside observer (Taylor, Sandberg, Thorley & Giles, 1991). By 'inattentiveness of behaviour' here is meant lack of persistence in activities ('short attention span'), a great deal of off-task behaviour, frequent changes in activity, and orienting to task-irrelevant aspects of the environment ('distractibility').

However, there appears to be inconsistencies between clinic findings and experimental lab results on the nature of attentional processes in children with AD/HD. As more rigorous and technical studies appeared in the 1980s, an increasing number failed to find evidence of problems with attention under some experimental conditions (see Douglas, 1988 for review; Sergeant, 1988; van der Meere, 1996). Moreover, if attention was conceptualised as involving the perception, filtering, and processing of information, no substantial evidence could be found in these studies for any deficits.

Impulsiveness

Impulsiveness is one of the key characteristics of the DSM-IV diagnostic definition of AD/HD. For a number of reasons, however, it has not been included as part of the ICD-10 definition of hyperkinetic disorder. One of these reasons is that the term impulsiveness means different things to different people; this makes using it in behaviour questionnaires particularly problematic. Even more important are the reasons relating to the fact that at present there is little agreement among clinicians or researchers on what

are the relevant and most significant aspects of impulsiveness, and how to recognise and measure them.

Those forms of impulsivity often associated with undercontrol of behaviour and the inability to delay a response or defer gratification or to inhibit dominant or prepotent responses are the ones most frequently identified in children having AD/HD (Barkley, 1997a). Clinically, these children are often noted to respond quickly to situations without waiting for instructions to be completed or adequately appreciating what is required in the setting. Careless errors are often the result. These children may also fail to consider the potentially negative, destructive or even dangerous consequences that may be associated with particular situations or behaviours, and seem to engage in frequent, unnecessary risk taking. Waiting one's turn in a game or in a group line-up before going to an activity is often problematic for them. They are notorious for taking short cuts in their work performance, applying the least amount of effort and taking the least amount of time in performing tasks they find boring. Blurting out answers to questions prematurely and interrupting the conversations of others are commonplace.

Scientifically, impulsivity has been defined as a pattern of rapid, inaccurate responding to task (Brown & Quay, 1977), poor sustained inhibition of responding (Barkley, 1997a), poor delay of gratification (Campbell, 1987; Rapport, Tucker, DuPaul, Merlo, & Stoner, 1986), or impaired adherence to commands to regulate or inhibit behaviour in social contexts (Barkley, 1985). Studies that have factor-analysed ratings of impulsive behaviour mixed in with ratings of inattention and overactivity (Achenbach & Edelbrock, 1983; Lahey et al., 1994) have failed to differentiate an impulsivity dimension from that measuring hyperactivity – that is, overactive children are also impulsive children and vice versa.

North American versus European concepts of AD/HD

Clinicians and researchers in North America and Europe have differed in the emphasis placed on the clusters of behavioural symptoms: poor sustained attention, impulsiveness, and hyperactivity (See Tannock, 1998 and Swanson et al., 1998). However, DSM and ICD manuals in their most recent versions now recognise the same problem behaviours as the basis of the diagnosis, in almost identical sets of 18 symptoms (see Table 1).

Insert Table 1 about here

In the symptom domain groups (inattention, hyperactivity, and impulsivity) an ICD-10 diagnosis of HKD needs some symptoms in all three groups whereas DSM-IV (AD/HD) does not, but instead specifies partial subtypes if symptoms are only from one domain. Furthermore, ICD-10 makes special provision for a combined diagnosis category if a conduct disorder is present and, because of the high frequency of this combination, uses the presence or absence of a conduct disorder as the basis for the main subdivision of HKD. DSM-IV does not make any special provision for conduct disorder as a comorbid condition but allows its diagnosis as it does other psychiatric disorders.

Another difference between the classifications is the use of other comorbid conditions as exclusion criteria. ICD-10 aims at a single diagnosis and does not recommend the HKD diagnosis in the presence of internalising disorders such as anxiety and depression. DSM-IV aims to recognise as many diagnoses as there are symptom patterns.

Classifications also differ in the criteria for cross-situational pervasiveness. Historically, ICD has placed great emphasis on the stability of the overactivity problems across home and school contexts. In contrast, in the US, this cross-situation stability has not been a core diagnostic criteria within previous editions of the DSM (Hinshaw, 1994). However, both classifications now stipulate that symptoms must be present in two or more settings for a positive diagnosis to be made, although the criteria are more rigorous in ICD-10 than in DSM-IV. The overall result of these differences is that HKD is a subset of AD/HD in ICD-10.

Prevalence and gender ratio

According to Swanson et al. (1998), a diagnosis in the DSM tradition with specific inclusion criteria for symptom onset, duration, pervasiveness and impairment, is found in 5-10% of the general population, whereas this frequency is 1-2% with the ICD-10 tradition, which restricts diagnosis to the full syndrome with limited comorbidity. A behavioural definition based on symptoms shown at one point in time (which does not indicate actual psychiatric disorder), is found in 10-20% of the general population in several countries (Swanson et al., 1998).

Although individual symptoms can be found in a large percentage of normal children, the number and degree of behavioural characteristics must be developmentally appropriate for a child's age and gender before it can be considered a clinical disorder. Using a statistical criterion, a referred child is compared to his or her own peers to determine how deviant they are from same-age, same-gender children. The further the children are from their peers in these behaviours, the greater the odds that they will be impaired in their educational and social adjustment and will eventually be diagnosed as AD/HD.

As is the case for other externalising problems, AD/HD occurs more in boys than in girls. The ratio within clinic samples has been reported to be as high as 6:1, whereas in community samples it occurs in the order of 3:1 (Barkley, 1990). The considerably higher rate of males among clinic samples of children seems to be due to referral bias in that males are more likely than females to be aggressive and antisocial and such behaviour is more likely to get a child referred to child and adolescent services. Even so, males remain more likely to manifest AD/HD than girls in community-based samples, suggesting that there may be some gender-linked mechanism involved in the expression of the disorder.

Additional problems often associated with AD/HD

Besides their primary problems with inattention, impulsivity, and overactivity, children with AD/HD may have a variety of other difficulties. These are considered associated features rather than being diagnostic of the disorder. Children are more likely to be behind in their intellectual development (Faraone et al., 1993; Fischer, Barkley, Fletcher, & Smallish, 1990). They are more likely to be doing poorly at school, typically under-performing relative to their known levels of ability. Such performance is believed to be the result of their inattentive, impulsive and restless behaviour in the classroom. A third associated feature is that children with AD/HD are more likely than normal children to have specific maths and reading difficulties (Safer & Allen, 1976). Another associated problem is children's excessive variability of task or work performance over time. Teachers often report much greater variability in homework and test grades as well as in class performance than is seen in normal children. Lab experiments also find that the standard deviation of performance on multi-trial tasks is considerably larger than seen in normal children (Douglas, 1972). Children with AD/HD display more executive

function difficulties than do normal children, particularly the organisation of material (Amin, Douglas, Mendelson, & Dufresne, 1993). Clinical descriptions of children with AD/HD refer to poor motivation and impaired persistence of effort.

Comorbidity

Between 50% and 80% of children with AD/HD also meet diagnostic criteria for other disorders, with rates of comorbidity varying according to the sample studied and the method of ascertainment (reviewed by Biederman, Newcorn, & Sprich, 1991; Jensen Martin, & Cantwell, 1997). The most frequently observed comorbidity is between AD/HD and other disruptive behaviour disorders, with oppositional defiant disorder and conduct disorder occurring in approximately 40% to 90% of cases (reviewed by Newcorn & Halperin, 1994; Jensen et al., 1997). Data suggest that 15% to 20% of children with AD/HD have concurrent mood disorders, approximately 25% have comorbid anxiety disorders, and about 20% have specific learning disabilities (previously reviewed by Biederman, Newcorn et al., 1991; Jensen et al., 1997).

As mentioned earlier, comorbidity rates will differ in North America and Europe because the two classification systems differ in how they handle co-occurring disorders.

Summary

There is a consensus that AD/HD is a chronic and pervasive condition characterised by developmentally inappropriate levels of inattention, impulsivity, and/or hyperactivity. A long line of diagnostic labels has been used to describe this disorder for nearly 100 years. These labels have reflected its presumed aetiology. One of the more confusing aspects of AD/HD is that its primary features are subject to situational

variation. Although it can occur alone, AD/HD is often accompanied by educational, behavioural, emotional and social complications.

Aetiologies

Investigators have attempted to explain AD/HD at different levels. These include neurological, behaviour and molecular genetics, and non-genetic factors such as birth trauma or environmental risks. This review section looks at the multiple aetiologies that may lead to AD/HD, particularly highlighting more recent evidence which seems to support a dual pathway psychological model of the disorder (Sonuga-Barke, in press).

Neurological Studies

Neuroanatomy

Structural imaging studies of children with AD/HD suggest localised abnormalities in the prefrontal cortex, basal ganglia and corpus callosum. Those that measured the anterior frontal region report smaller right prefrontal cortex in AD/HD (Castellanos et al., 1994; Filipek et al. 1997; Hynd et al., 1990). Those that measured basal ganglia report differences in volumes with a corresponding loss of or reversal of the assymetry found in normal controls (Castellanos et al., 1994; Castellanos, Giedd, Marsh et al., 1996; Filipek et al., 1997; Hynd et al., 1993). Decreases in the corpus callosum have also been observed in children with AD/HD (see Tannock 1998 for review). Further support for prefrontal abnormalities in children with AD/HD comes from a study by Casey et al. (1997) who found that these children showed poor performance on response inhibition tasks. This performance correlated only with anatomical measures of frontal-striatal circuitry observed to be abnormal in children with AD/HD. The findings of these studies are concordant with theoretical models of abnormal frontal-striatal function in

AD/HD (e.g. Barkley, 1997a; Benson, 1991) and with attentional network hypotheses (e.g. Mesulam, 1990; Posner & Raichle, 1994). However, little is known about the specificity of the structural abnormalities to AD/HD. Although anatomical studies may provide some insights into the brain basis of AD/HD, functional studies are necessary to determine which anatomical abnormalities have functional sequelae, as well as to demonstrate abnormal cognitive processes inferred from neuropsychological studies.

Neurophysiology

Functional imaging studies of AD/HD have also shown that abnormalities in the frontal-striatal regions probably underlie the development of AD/HD (for reviews see Arnsten, Steere, & Hunt, 1996; Benton, 1991, Mercugliano, 1995; Tannock, 1998).

Quantitative electroencephalograph (QEEG) and evoked response potential (ERP) measures of the frontal lobe taken in conjunction with performance or vigilance tests (Tannock, 1998) suggest problems in central arousal patterns and under-reactivity to stimulation in ERP, particularly wave forms that are likely to be related to subcortical activation. These findings are consistent with an 'energetic' conceptualisation of AD/HD (e.g. Sergeant, 1995) which will be mentioned later. This under-reactivity of AD/HD children to stimulation can be corrected by stimulant medication. Other studies consistently show decreased blood flow to the prefrontal regions and pathways connecting these regions to the limbic system via the striatum and specifically its anterior region (Lou, Henriksen, & Bruhn, 1990; Sieg, Gaffney, Preston, & Hellings, 1995).

According to Tannock (1998), results of functional imaging studies should be interpreted with caution. To date, there is little agreement in terms of identifying the precise nature of the abnormalities and their significance, and it is also unclear whether the observed group differences between AD/HD and controls reflect delayed brain

maturation or developmental deviation. Abnormalities can occur as a result of alterations in normal developmental processes that may be mediated by genetic, hormonal, or environmental effects of a combination of these. Results of physiological studies are complicated even more by the use of different EEG methods and analyses, which are known to influence EEG parameters.

Genetic factors

AD/HD is highly hereditary in nature. This is strongly supported by behaviour genetics using family aggregation studies, adoption research and twin studies. The relationship is also supported by molecular genetic research (see Kuntsi and Stevenson, 2000 for a review).

Behaviour genetics

Family studies investigate the degree to which genetically related individuals are similar phenotypically. If there is no resemblance between family members on a given trait, genetic factors do not influence the phenotypic variance on the trait. Although some family studies suffer from methodological limitations (with regard to diagnostic procedures and nonblind ratings of psychopathology, for example), the general picture strongly suggests that AD/HD runs in families (Biederman et al., 1992; Biederman, Faraone, Keenan, Knee, & Tsuang, 1990; Perrin & Last, 1996; Roizen et al., 1996). According to Biederman and colleagues (1992), parents and siblings of AD/HD probands were five times more likely than relatives of controls to receive a (lifetime) diagnosis of AD/HD themselves. When the relatives were classified based on a broader definition of AD/HD, the proportion of affected individuals increased from 16% to 25%.

Adoption studies can provide a powerful demonstration of genetic influences on behaviour. The logic behind adoption studies is that similarities between adopted-apart relatives suggest genetic influences, whereas similarities between adoptive relatives suggest environmental influences. Only a small number of adoption studies on AD/HD has been carried out. Van den Oord, Boomsma, and Verhulst (1994) found that genetic effects accounted for 47% of the variance on the Attention Problems subscale of the Child Behaviour Checklist (CBCL: Achenbach & Edelbrock, 1983); the effects of the shared environment were very small. According to two studies (Alberts-Corush, Firestone, & Goodman, 1986; Nigg, Swanson, & Hinshaw, 1997), evidence suggests that the genetic resemblance between hyperactive children and their parents need not be limited to the behavioural manifestations of hyperactivity, but may also be found on psychological tests.

Twin studies rely on comparisons between identical or monozygotic (MZ) twins, who share all their genes, and fraternal or dizygotic (DZ) twins, who share approximately half their genes. The increasing number of twin studies shows that there are substantial genetic effects on AD/HD or hyperactivity, with consistency in the findings across different measures used (Kuntsi & Stevenson, 2000). Heritability estimates for the dimension of hyperactivity vary between 55% and 100% for parent-report data and between 50% and 70% for teacher-report data. The remaining variance in hyperactivity not due to genetic effects has been attributed to the nonshared environment (and measurement error).

Molecular genetics

Research using quantitative genetic methods has paved the way for the search for the actual genes influencing complex behaviours, although a definitive mode of

inheritance has not yet been established. Complex behaviours are those that are thought to be influenced by multiple genes and also by environmental factors. The dopaminergic genes have been considered as candidate genes for AD/HD, and pharmacological agents that act on the dopaminergic system have proven efficacy in reducing the effects of the disorder.

First, there is preliminary evidence of an association between one allele (480-bp) of the dopamine transporter locus (DAT1) and AD/HD (Cook et al., 1995). This association has been replicated by Gill, Daly, Heron, Hawi & Fitzgerald (1997), although both studies have small and heterogeneous samples with high levels of comorbidity. The dopamine transporter regulates levels of extra-cellular dopamine. The D1 receptor seems to play a central role in regulating pre-frontal activity (Arnsten, 1997; Robbins, 2000), whilst alterations in the D2 receptor are important for reward processes (Koob, 1992; Blum, Wood, Braverman, Chen, & Sheridan, 1995) and may be directly implicated in moderating the value of delayed rewards (Wade, de Wit, & Richards, 2000).

These research findings support a proposed dual pathway psychological model of AD/HD that recognises two distinct sub-types of the disorder (Sonuga-Barke, in press). In one, AD/HD is the results of the dysregulation of action and thought resulting from poor inhibitory control associated with the meso-cortical branch of the dopamine system projecting in the cortical control centres (e.g. pre-frontal cortex). In the other, AD/HD is a motivational style characterised by an altered delay of reward gradient linked to the mesolimbic dopamine branch associated with the reward circuits (e.g. nucleus accumbens).

Most attention has been focused on the association between AD/HD and the D4 dopamine receptor gene (DRD4). LaHoste et al. (1996) published the first report of an association between this gene polymorphism (located on chromosome 11) and AD/HD.

A further three studies have subsequently replicated the relation of DRD4 to AD/HD (Faraone et al., 1999; Rowe et al., 1998; Smalley et al., 1998), although one study has failed to replicate the finding (Castellanos et al., 1998). The significance of this finding with regard to the dual pathway psychological model (Sonuga-Barke, in press) is unclear.

There are, however, reports of associations between the dopamine D4 receptor gene polymorphisms and a personality trait known as novelty seeking (Benjamin et al., 1996; Ebstein et al., 1996). Novelty seeking refers to characteristics such as impulsiveness, exploration, changeableness and excitability – behaviours similar to those observed in AD/HD. One study demonstrated that adult AD/HD patients score higher than normal controls on a novelty seeking scale (Downey, Stelson, Pomerleau, & Giordani, 1997) although a further three studies have failed to replicate the finding (Jonsson et al., 1997; Malhotra et al., 1996; Vandenbergh, Zonderman, Wang, Uhl, & Costa, 1997). No conclusion can therefore be drawn on the effect of the DRD4 gene on novelty seeking.

Non-genetic aetiology

Non-genetic factors are also likely to be important in the aetiology of AD/HD. A number of studies has found that low birthweight was associated with an increased risk of hyperactivity, inattention, disruptive behaviour, and poor school adjustment (Breslau et al., 1996; Nichols & Chen, 1981; Sykes et al., 1997). However, findings are equivocal: some studies have not found a greater incidence of pregnancy or birth complications in AD/HD compared to normal children (Barkley, DuPaul, & McMurray, 1990). Large scale epidemiological studies have generally not found a strong association between pre- or perinatal adversity and symptoms of AD/HD once other factors are taken into account (Goodman & Stevenson, 1989; Nichols & Chen, 1981). There is evidence that both

executive function performance and AD/HD symptoms are associated with low birth weight (Bylund et al., 2000; Harvey, O'Callaghan, & Mohey, 1999) and that these processes may be mediated by dopamine activity (Brake, Sullivan, & Gratton, 2000) and alterations in the fronto-striatal system (Toft, 1999).

Summary

A variety of genetic and neurological aetiologies can give rise to AD/HD through some disturbance in a final common pathway in the nervous system. That final common pathway appears to be the integrity of the prefrontal cortical-striatal network. It now appears that hereditary factors play the largest role in the occurrence of AD/HD symptoms in children. New evidence from molecular genetic studies has implicated specific genes that may be involved in its aetiology, particularly the dopamine receptor genes, which regulate levels of extra-cellular dopamine. AD/HD may also be exacerbated by non-genetic factors such as pregnancy complications which in turn also implicate the fronto-striatal system and dopamine activity and results in impaired neuropsychological function.

Psychological theories of AD/HD

Over the years, numerous psychological theories have been put forward to explain the manner in which AD/HD affects psychosocial functioning. Many of the early accounts, which did not have the benefit of the aetiological findings, focused almost exclusively on psychological processes that were believed to be at the core of AD/HD difficulties. Among these were theories implicating core deficiencies in the regulation of behaviour in response to situational demands (Routh, 1978), in self-directed instruction (Kendall & Braswell, 1985), in the self-regulation of arousal to environmental demands

(Douglas, 1983), and in rule-governed behaviour (Barkley, 1981). Although differing in their theoretical emphasis, each of these views shared the belief that poor executive functioning was a central problem.

More recent theories have taken on a very distinctive neuropsychological flavour, suggesting patterns of cognitive deficits. These theories emphasise the construct of impulsiveness (poor behavioural inhibition), postulating that a failure to inhibit or delay a behavioural response is the central deficit in AD/HD (e.g. Quay, 1988, 1997; Barkley, 1997a; Schachar et al., 1993). However, the models differ in their formulation of the fundamental impairment. While models emphasising disinhibition dominate the current literature, a number of alternative accounts have been proposed that emphasise the motivational basis of AD/HD (Zentall & Zentall, 1983; Sonuga-Barke, 1994;Van der Meere, 1996).

Response inhibition

Inhibition as a conditioning deficit

Quay (1988, 1997) has proposed that AD/HD stems from an imbalance between two opposing and distinct neuropsychological systems that Gray (1982) suggested control responses to signals of punishment and reward – a behavioural inhibition system and a behavioural activation system. This model states that the impulsiveness characterising AD/HD arises from diminished activity in the brain's behavioural inhibition system (BIS). That system is said to be sensitive to signals of conditioned punishment that, when detected, result in increased activity in the BIS and a resulting inhibitory effect on behaviour. This theory predicts that those with AD/HD should prove less sensitive to such signals (Quay, 1988).

Inhibition as the primary deficit

Barkley (1994; 1996; 1997a) also argues that a deficit in behavioural inhibition is central to understanding the cognitive, behavioural, and social deficits observed with AD/HD. His is one of the most comprehensive and popular models to date. Not only is behavioural inhibition proposed as the primary deficit in AD/HD, but it is also hypothesised to lead to secondary impairments in four executive neuropsychological abilities that are dependent upon behavioural inhibition for their effective execution. Behavioural inhibition refers to three interrelated processes: inhibition of the initial prepotent response to an event; stopping of an ongoing response; and interference control or the protection of this delay from disruption by competing events and responses (Barkley, 1997b). The four executive functions are (1) working memory; (2) self-regulation of affect-motivation-arousal (3) internalisation of speech; and (4) reconstitution. These executive functions permit motor control and fluency, affording effective self-regulation and adaptive functioning.

Barkley's model has face validity because it seems to account for the clinical impression that children cannot inhibit socially inappropriate responses. However, it fails to acknowledge that AD/HD children can withhold responses sometimes.

Inhibition as an inefficient inhibitory control process

Although less comprehensive in its scope, the theoretical view of Schachar, Tannock and Logan (1993) argues that generalised inhibitory deficits are at the heart of AD/HD. Impulsive behaviour is conceptualised as a deficit in the ability to inhibit prepotent courses of action: children who are impulsive have trouble inhibiting action, whereas those who are not impulsive find it easier to do so (Logan, 1994; Schachar & Logan, 1990; Schachar et al., 1993).

This ability to inhibit is presumed to be one of several internally generated acts of control in the repertoire of a higher-order central executive function system that regulates the operations of the human information processing system and permits self-regulation. This approach draws on the race model of Logan (1994) in which environmental stimuli are seen as initiating signals of both activation of responding and inhibition of responding. These signals race against each other to determine whether behaviour towards the stimulus event will be initiated or inhibited. The first signal to reach the motor control system in essence wins the race and determines the nature of the eventual response (approach/responding or withdrawal/inhibition or responding).

According to this theory, the difficulty that children with AD/HD have in inhibiting a prepotent action is attributable to an unusually slow inhibitory process rather than an unusually fast response process (e.g. Schachar & Logan, 1990; Schachar et al., 1993). The most compelling evidence for this view comes from research using the Stop Signal Paradigm, developed by Logan and colleagues (Logan, Cowan, & Davis, 1984) and first applied to childhood behaviour disorders by Schachar and Logan (1990). This paradigm provides an empirical measure of the ability to interrupt an ongoing response.

The Stop Signal Task (SST) is a simple reaction time task: the child responds as fast and accurately as possible to stimuli presented on a computer screen. On some trials an auditory stop signal is presented, indicating that the child should withhold responding. Stop signals are presented at different intervals and many investigators compensate for differences in the primary (go) task reaction times by presenting the stop signals relative to the child's mean reaction time. The longer the delay between the onset of the primary task stimulus and the onset of the stop signal, the more difficult it becomes to withhold responding. Extensive prior research with this paradigm (Logan & Cowan, 1984) has shown that the probability of successful inhibition is a direct function of the length of this

stopping interval: longer intervals are associated with a greater probability of inhibition. Plotting the probability of inhibition against the stop signal delay generates an 'inhibition function'. The slope of the inhibition function is calculated by fitting a regression line to the inhibition function.

Children with AD/HD were shown to have both a flatter slope of inhibition indicating worse performance on the task (see Oosterlaan, Logan and Sergeant, 1998 for meta-analysis) and a longer mean stop signal reaction time (SSRT) (Schachar, Tannock, Marriott, & Logan, 1995) than did normal children.

To examine whether results of the meta-analysis were due to the AD/HD group being less likely to trigger the inhibitory process, or to their inhibitory process being more variable, Oosterlaan et al. (1998) investigated whether the group difference would disappear after statistical transformation to remove the effects of stop signal reaction time and variability of speed on the inhibition function (see Logan, 1994). The meta-analysis showed that the children with AD/HD did not differ significantly from the control children on the transformed slope. In other words, they are neither less likely to trigger the inhibitory process, nor is their inhibitory process more variable on the stop task.

Children with AD/HD are more variable in the speed of their responding and less accurate also on response re-engagement compared to control children (Oosterlaan & Sergeant, 1998). This shows that their pattern of responding is not limited to response inhibition tasks. Rather than indicating a specific response inhibition deficit, the overall pattern of the findings may suggest a generally slow mode of information processing (see also Sergeant, Oosterlaan, and van der Meere, 1999; Tannock, 1998). Furthermore, high variability in reaction times could reflect a lack of motivation or effort on some trials.

Studies conducted in the stop task tradition have been criticised (Sonuga-Barke, 1995) because they focus only on momentary inhibition, the ability to suppress a

particular response when it is signalled. The stop task does not measure ongoing inhibition, the ability to suppress responding over a period of delay, which some accounts (e.g. Barkley, 1997a) view as important in AD/HD.

Most models seek a single unitary cause, located within the biological, neurological, and/or genetic substrate – that is, within the individual. Technological advances in human genetics, neuroimaging, and molecular biology account in part for this restrictive focus.

Motivational accounts of AD/HD

In contrast to these theories implicating deficits in inhibitory control, other psychological theories take a motivational approach to the disorder. These accounts do not regard AD/HD as the result of disinhibitory psychopathology but rather as the expression of an altered motivational state that leads to an altered response to reinforcement parameters (especially magnitude and delay). These theories suggest that children with AD/HD have a reduced sensitivity to reinforcement. More immediate, frequent, or intense rewards are required to maintain appropriate performance and behaviour (Haenlein & Caul, 1987). Children with AD/HD are also overly responsive to immediate rewards (Douglas & Parry, 1994; Rapport, Tucker, DuPaul, Merlo, & Stoner, 1986); are less able to delay gratification or resist temptation (Mischel, Shoda, & Rodriguez, 1989); or are higher in "stimulation-seeking" behaviours which are needed to compensate for inherently low levels of central nervous system arousal in AD/HD (Zentall & Meyer, 1987; Zentall & Zentall, 1983).

Optimal stimulation theory

Zentall and Zentall (1983) suggested that a state of underarousal underlies AD/HD. 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 the need to meet their high stimulation threshold. Consequently, children with AD/HD appear to produce more activity than control children when confronted with low levels of stimulation (van der Meere, 1996; Zentall & Meyer, 1987). Zentall and Zentall (1983) reviewed studies which suggest that AD/HD children are indistinguishable from controls in relatively high-stimulation settings (e.g. new game, or playground settings). The effects of stimulants and extraneous distraction, and the sensation seeking behaviours, could be seen as helping to normalise hyperactive children's level of arousal.

State-regulation theory

Van der Meere (1996) emphasises the observations of many parents of AD/HD children that the "deficit" seems only to be present during boring tasks, but disappears when the child is well motivated. He attempts to reduce AD/HD difficulties to a central deficiency in arousal, and draws on information-processing theory and its associated energetic model. The model is critical of the "unitary state" concept of arousal previously mentioned. Van der Meere and his colleagues (Sergeant & van der Meere, 1990a, b) argue for a multi-state model, making a distinction between arousal and activation, which is supported by neurological findings. The arousal system operates by noradrenaline and serotonin and is located in the fronto-limbic forebrain. The primary neurotransmitters in the activation system are dopamine and acetylcholine and is located in the basal ganglia.

State-regulation theory involves a third energetic system, the effort system. In Sander's (1983) model an evaluation system controls the effort system and 'scans' the

individual's arousal and activation state. If the arousal level is nonoptimal, the effort system can compensate for this. Van der Meere (1996) argues that motivation factors such as knowledge of results, absence-presence of the experimenter and pay-off, influence the effort mechanism.

Evidence from information processing studies suggests that AD/HD deficits relate to the motor processing stage rather than the earlier stages of information processing (see Oosterlaan, & van der Meere, 1999; and van der Meere, 1996 for reviews). Instead of showing a pattern of fast, inaccurate responding, AD/HD children show a pattern of slow, inaccurate responding, questioning the validity of impulsivity as fast, inaccurate responding. The emphasis is on the sensation-seeking component of impulsivity. Van der Meere (1996) argues that what appears to be a motor processing deficit could in fact involve an activation-effort dysfunction. In other words, basic information processing capacity is intact; but it is the utilisation of this capacity which depends on state factors such as incentives.

A typical study supporting the theory compared AD/HD and control children's performance on the go-no go task under three different presentation rates (van der Meere, Stemerdink, & Gunning, 1995). AD/HD children made more commission errors only in the slow and fast conditions. In the medium condition they were indistinguishable from the controls. What seems to be poor response inhibition could actually be a difficulty in modulating behavioural state according to task and situation demands. A recent study by Kuntsi & Stevenson (2001) also supported the state regulation theory, although the study was not specifically designed to test the theory. They found that the variable that best discriminated between hyperactive and control groups was the variability in speed on stop task. Hyperactive children were also generally slow and made a high number of errors.

Delay aversion and the dual pathway model

The Delay Aversion hypothesis, developed by Sonuga-Barke and colleagues (Sonuga-Barke, Taylor, Sembi, & Smith, 1992) views inhibitory problems as indicative of deviance, in terms of motivational attitude. So-called impulsive behaviour is not the consequence of a relative inability to inhibit a response, but rather is the result of a rational choice to avoid delay, which the child finds aversive. As with van der Meere's (1996) theory, this formulation is also based on analysis of the situational contexts in which children's impulsive symptoms are found. According to theory, impulsive behaviour represents situation-specific attempts by children to reduce the subjective perception or actual experience of delay, or passage of time (Sonuga-Barke, Taylor, & Heptinstall, 1992).

In their first paper, Sonuga-Barke and colleagues (Sonuga-Barke, Taylor, Sembi & Smith, 1992) carried out two studies to explicitly contrast the predictions of an inhibition deficit/impulsivity, reward maximising and delay aversion hypotheses. Participants were 31 boys, aged 6 and 7 years: 15 boys with pervasive hyperactivity (based on ratings on Rutter's teacher and parent scales) and 16 control boys. The task was a computerised choice-delay task in which the children had to choose, by pressing the appropriate button, either a small reward (1 point) or a large reward (2 points). Children chose across four conditions, and hyperactive children did not differ on three of these. They waited as well as control children for the larger reward when it was the most efficient strategy (post-delay condition). They were not simply attempting to minimise levels of pre-reward delay. There was no group difference in the no-post-delay condition: both groups of children preferred the smaller reward.

A second study tested two competing hypotheses for these findings: either hyperactive children were reward maximisers, or they were averse to delay, thereby giving preference to an immediate reward only if it reduced the overall delay period. This study confirmed that when there was a limited number of trials on which to choose (trials constraint) so that the small reward was associated with shorter sessions but less reward overall, hyperactive children showed a significantly smaller mean preference for the larger delayed reward (18%) than did the control children (48%). This pattern of results suggested that overall delay (as reflected in session length) rather than pre-reward delay or reward size, was the key motivating factor that produced "impulsive" responding.

Sonuga-Barke and colleagues have investigated the extent to which delay aversion may account for the findings of worse performance of children with AD/HD on traditional measures of attention, memory, and impulsivity. Aversion to delay was found to be related to longer serial reaction times (Sonuga-Barke & Taylor, 1992), shorter self-selected stimulus exposure times and worse memory recognition (Sonuga-Barke, Taylor, & Heptinstall, 1992), as well as shorter latency to response on the Matching Familiar Figures Task (MFFT: Sonuga-Barke, Houlberg & Hall,1994) in hyperactive as compared to normal children. The MFFT is one of the measures which Pennington and Ozonoff (1996) identified as sensitive to AD/HD.

Studies on the delay aversion hypothesis have used community-identified children with high parent and teacher ratings of hyperactivity who also meet criteria on assessment questionnaires for oppositional behaviour and conduct problems. It is not known to what extent AD/HD accounts for findings, rather than co-morbid conditions. One study (Kuntsi & Stevenson, 2001) replicated the finding of hyperactive children choosing the small, immediate reward more often than control children in the trials constraint

condition. However, controlling for conduct problems removed the significant group difference, suggesting that co-occurring conduct problems explain most, if not all, of the association between hyperactivity and delay aversion. This issue of whether delay aversion is specific to AD/HD needs further investigation.

Only one study has used clinically diagnosed children with AD/HD to test the delay aversion hypothesis (Solanto et al., in press) and results suggest that executive control deficits and delay aversion might both contribute to AD/HD. Researchers looked at the specific role played by the psychological processes tapped by the stop signal task (SST) and delay aversion paradigm. The SST was used to characterise a primary deficit in inhibitory control in AD/HD, whereas the C-DT conceptualised impulsivity as a choice to avoid delay. Results showed that performance on the two tasks was not correlated, suggesting that the two paradigms tap different components of the AD/HD phenotype. There was an effect of AD/HD on performance of both tasks (with a large effect size) which was not altered when comorbid conduct problems or anxiety disorders were taken into account. A step wise discriminant function procedure showed the two measures together to be highly diagnostic, correctly identifying nearly 90 per cent of cases. This suggests the two paradigms tap different components of the AD/HD phenotype.

The delay aversion and the deficient inhibitory control accounts both represent attempts to develop a 'grand' theory of AD/HD. However, the heterogeneity of its clinical expression and its factorially determined aetiology makes achieving the sort of theoretical unity required by such models of AD/HD unlikely. Results of the study by Solanto et al. (in press) lend support to a proposed dual pathway model of AD/HD (Sonuga-Barke, 2001) that recognises two distinct sub-types of the disorder. In one, AD/HD is the result of the dysregulation of action and thought resulting from poor inhibitory control associated with the meso-cortical branch of the dopamine system

projecting in the cortical control centres (e.g. pre-frontal cortex). In the other, AD/HD is a motivational style characterised by an altered delay of reward gradient linked to the mesolimbic dopamine branch associated with the reward circuits (e.g. nucleus accumbens). The model represents a reconciliation of two philosophically distinct views of behavioural disorder – one that seeks to identify the site of dysfunction in disorder, while the other seeks to explore the role of function.

If, according to delay aversion theory, children with AD/HD are trying to reduce both perceived and actual delay, they first need to detect delay-related cues in their environment. Their attentional style will be specially tuned to detect the threat of delay, in a similar way that socially anxious people are hypervigilant to socially-threatening stimuli. The following section reviews the literature on attentional biases to emotional stimuli, including the only study involving children with AD/HD.

Attentional Bias and AD/HD

The ability to selectively attend to some stimuli while ignoring others is essential for efficient and effective information processing (Broadbent, 1958). Selective attention mechanisms limit the information that is available for later stages of processing. Insofar as cognitive processing influences behaviour, attentional biases have substantial potential to contribute to the development and maintenance of maladaptive behaviour (Crick & Dodge, 1994). Attentional processes have been implicated in the onset and maintenance of emotional disorders (Wells & Mathews, 1994). The idea is that individuals suffering from, or vulnerable to, emotional disorders may selectively attend to emotional information. This attentional bias would then serve to exacerbate their negative mood state, which would potentially lead to further increases in attentional bias for emotional material.

Considerable evidence supports the finding that anxious adults disproportionately attend to emotionally threatening versus neutral stimuli (see Williams, Watts, MacLeod, & Mathews, 1997, for a review). This tendency is found among clinically anxious (e.g. MacLeod, Mathews, & Tata, 1986) as well as high-trait-anxious subjects (e.g. MacLeod & Mathews, 1988).

A variety of tasks has been used to examine biases in selective attention to emotional stimuli. One method is to use tasks in which emotional stimuli are present as distracters, so that if they attract attention this will cause interference with the other task. For example, when searching for a neutral target among distracter words, anxious patients are slowed more by threatening than by neutral distracters, presumably because their attention is captured by the former (Mathews, May, Mogg, & Eysenck, 1990; Mathews, Mogg, Kentish, & Eysenck,1995). By far the most commonly used interference task is the modified Stroop colour naming task (Stroop, 1935). Participants are required to call out the colours in which words are displayed while ignoring their meaning. Words whose meaning matches the emotional concerns of the individual concerned typically cause slowed colour-naming performance. This finding has been reported in patients with anxiety disorders, depression, and eating disorders (for a review, see Williams et al., 1996). Results are typically interpreted in terms of anxious individual's attention being preferentially allocated to the threat content.

The Stroop task leaves the mechanism responsible for colour-naming interference unclear. Anxious subjects may be slowed because their attention is disproportionately drawn to threat words. However, it may be that all subjects attend equally to threatening words, but anxious subjects may experience an emotional reaction to such words that produces interference, or to an attentional shift away from such words (Dalgleish &

Watts, 1990). Given these uncertainties, researchers have turned to other tasks, such as probe detection.

The dot probe method provides a more direct measure of the allocation of visual attention. The task was adapted by MacLeod et al. (1986) from experimental cognitive psychology paradigms, which indicated that spatial attention can be assessed from the speed of manual responses to visual probes. For example, if two words are presented simultaneously for 500 milliseconds, and then one of them is replaced by a probe that subjects are to detect, anxious patients are quicker to detect the probe if it replaces a threatening rather than a neutral word (MacLeod et al., 1986). The implication is that clinically anxious patients are more likely to attend to an emotional stimulus in a perceptual array than are non-anxious control subjects. A bias away from threat is shown by longer latencies for probes that appear in the same position as threat versus neutral words. This selective attention effect is stronger when there is a match between the stimuli presented and the current concerns (e.g. worries) of the individual (Mogg, Mathews, & Eysenck, 1992). Thus, panic disorder patients attend to physically threatening words (e.g. collapse, death) but less so to socially threatening words (e.g. failure, stupid; Asmundson & Stein, 1994).

Recently, researchers have examined attentional processing of emotional information in children with anxiety. Evidence suggests that even young children and infants use attentional disengagement in response to threatening stimuli as a means of regulating fear (see Vasey, Elhag, & Daleiden, 1996). Martin, Horder & Jones (1992) compared children who reported a fear of spiders to children who reported no fear. They all completed a version of the Stroop task that included spider-relevant (e.g. web, crawl) and nonrelevant (e.g. fly, wings) words. They found that spider-relevant words disproportionately disrupted the colour-naming performance of spider-fearful children.

One of the first studies to use a modified dot probe with children was by Vasey, Daleiden, Williams, & Brown (1995). They used a version of the MacLeod et al. (1986) probe detection task in a comparison of clinically anxious and normal children aged 9-14 years. Consistent with adult findings, clinically anxious children showed a significant attentional bias toward threatening words. This means that they detected probes significantly faster when they were preceded by threatening words than when they were preceded by neutral words. The probe detection speed of normal controls in contrast was unrelated to word content. A further study by Vasey et al. (1996) reported similar findings for subclinically anxious children.

Taghavi, Neshat-Doorst, Moradi, Yule, and Dalgleish (1999) extended this research by Vasey and colleagues and examined children with clinical anxiety and also children with a diagnosis of mixed anxiety-depression using the dot probe task. They replicated findings showing that the anxious samples exhibited an attentional bias for threat-related material.

Previous research has focused on the development of attentional biases towards emotionally significant stimuli in internalising disorders. However, one study recently examined attentional biases in children with high parent and teacher ratings of hyperactivity (Sonuga-Barke, Hayes & Bareham, submitted manuscript). Reaction times of hyperactive and control children to probes presented following neutral, threatening (both social and physical) and delay-related words were compared. Results showed that 32 hyperactive children selectively attended to delay-related words. This attentional bias towards delay cues differentiated their performance from that of 32 non-hyperactive controls. Results are in line with the idea that AD/HD children develop an attentional style attuned to the detection of delay (i.e. a delay detection mechanism) within environments, which would be of functional significance given their delay aversion.

Future research needs to establish the extent to which attentional biases for delay-related cues pertain to clinically diagnosed AD/HD children. Furthermore, the extent to which comorbid oppositional problems are related to attentional bias to delay words also needs to be addressed.

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Table 1. Symptom domains for AD/HD (DSM-IV) and Hyperkinetic Disorder (ICD-10)

Inattention	**Hyperactivity**	**Impulsivity**
Fails to attend to details	Fidgets with hands or feet	
Difficulty sustaining attention	Leaves seat in classroom	
Does not seem to listen	Runs about or climbs	
Fails to finish	Difficulty playing quietly	
Difficulty organising tasks	Motor excess ("on the go" - DSM-IV)	
Avoids sustained effort	Talks excessively (DSM-IV)	Talks excessively (ICD-10)
Loses things		Blurts out answers
Distracted by extraneous things		Difficulty waiting turn
Forgetful		Interrupts/intrudes on others

Empirical Paper

Delay aversion and attentional bias to delay-related cues: A comparison of boys with Attention Deficit/Hyperactivity Disorder (AD/HD), Oppositional Defiant Disorder/Conduct Disorder (ODD/CD), AD/HD + ODD/CD, and normal controls.

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Delay aversion and attentional bias to delay-related cues: A comparison of boys with Attention Deficit/Hyperactivity Disorder (AD/HD), Oppositional Defiant Disorder/Conduct Disorder (ODD/CD), AD/HD + ODD, and normal controls.

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Running head

Delay aversion and attentional bias

Delay aversion and attentional bias to delay-related cues: A comparison of boys with Attention Deficit/Hyperactivity Disorder (AD/HD), Oppositional Defiant Disorder/Conduct Disorder (ODD/CD), AD/HD + ODD, and normal controls.

Abstract

This study tested two predictions in support of the delay aversion theory of AD/HD: (1) Boys with a diagnosis of AD/HD will choose fewer large, delayed rewards on a computerised choice-delay task than controls; (2) Boys with a diagnosis of AD/HD will show an attentional bias towards delay-related words. Four groups of children, 8-12 years of age were compared: normal controls (25), boys diagnosed with pure AD/HD (28), boys diagnosed with AD/HD + ODD/CD (25), boys with ODD/CD only (12). They all completed two computerised tasks: choice-delay, and a modified dot probe paradigm. For the latter task, reaction times to probes were recorded following presentation of three word types (delay-related; physically and socially threatening). There were no significant group differences on either the choice-delay or dot probe task. Methodological issues are discussed to account for the pattern of findings and clinical implications are highlighted.

Key words

Attention Deficit/Hyperactivity Disorder, Attentional Bias, Delay Aversion, Dot Probe Paradigm

Abbreviations

RCMAS: Revised Children's Manifest Anxiety Scale; SDQ: Strengths and Difficulties Questionnaire; WISC III-UK: Wechsler Intelligence Scale for Children III-UK; WORD: Wechsler Objective Reading Dimensions;

Delay aversion and attentional bias to delay-related cues: A comparison of boys with Attention Deficit/Hyperactivity Disorder (AD/HD), Oppositional Defiant Disorder/Conduct Disorder (ODD/CD), AD/HD + ODD, and normal controls.

Introduction

The combination of inattentive, hyperactive, and impulsive behaviour in children is recognised as a disorder when these behaviours are severe, developmentally inappropriate, and impair function at home and school. Currently there are two terms for this disorder. Hyperkinetic disorder (HKD) takes its diagnostic criteria from the International Classification of Diseases (ICD-10, 1992 and 1993) published by the World Health Organisation. The more commonly used term of attention deficit/hyperactive disorder (AD/HD) takes its diagnostic criteria from the American Psychiatric Association's Diagnostic and Statistical Manual (DSM-IV, 1994). Prevalence has been estimated as between 3-6% of children from diverse cultures and geographical regions. It predominantly affects boys, in the ratio 3:1 (Tannock, 1998).

There are inconsistencies in findings regarding the underlying psychological mechanisms responsible for AD/HD. Some accounts argue that generalised deficits are at the heart of the disorder (Barkley & Biederman, 1997). The most compelling evidence for this view comes from research using the Stop Signal Paradigm developed by Logan and colleagues (Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984) and first applied to childhood behaviour disorders by Schachar and Logan (1990). The paradigm provides an index of the AD/HD child's ability to inhibit a prepared motor response.

While psychological models emphasising disinhibition and dysregulation dominate the current literature (Barkley, 1994, 1997; Quay, 1988, 1997; Schachar, Tannock, & Logan, 1993), a number of alternative accounts have been proposed that emphasise the motivational basis of AD/HD. These accounts do not regard AD/HD as the result of disinhibitory psychopathology, but rather as the expression of an altered motivational state and an altered response to reinforcement parameters (especially magnitude and delay). Some accounts state that children with AD/HD have a reduced sensitivity to reinforcement such that more immediate, frequent or intense rewards are required to maintain appropriate performance and behaviour (Barkley, 1989; Haenlein & Caul, 1987). Others argue that AD/HD children are overly responsive to immediate rewards (Douglas & Parry, 1994; Rapport, Tucker, DuPaul, Merlo, & Stoner, 1986). Further accounts suggest that AD/HD children are less able to delay gratification or resist temptation (Mischel, Shoda, & Rodriguez, 1986); or are higher in stimulation seeking behaviours which are needed to compensate for inherently low levels of central nervous system arousal in AD/HD (Zentall & Meyer, 1987; Zentall & Zentall, 1983).

The delay aversion hypothesis (Sonuga-Barke, 1994) challenges the dominant neuro-psychological paradigm with its emphasis on psychological dysfunction caused by impairments in specific brain modules (Karmiloff-Smith, 1998). Delay aversion represents a philosophically distinct theory, and is based on the assumption that AD/HD behaviours are functional expressions of an underlying motivational style rather than the result of dysfunctioning regulatory systems. According to the theory, AD/HD children are motivated to escape or avoid delay. Their inattentive, overactive and impulsive behaviours represent functional expressions of what has been termed delay aversion. According to Sonuga-Barke (1994), delay aversion explains all three patterns of behaviour which characterise AD/HD. In situations where AD/HD children have a

choice to reduce delay, which they find aversive, they will act impulsively. When AD/HD children have no choice, they will try to reduce the subjective experience of delay by increasing the level of stimulation through inattentiveness and overactivity or fidgeting.

Experimental evidence for the delay aversion hypothesis comes from research using a computerised choice-delay task (Sonuga-Barke, Taylor, Sembi, & Smith, 1992). Children chose between a large reward, which was associated with a period of delay (30 seconds) and a smaller reward, which was not associated with delay (2 seconds). Fifteen pervasively hyperactive and 16 normal control children (aged 6 and 7) made a choice across four different conditions. These experiments demonstrated that hyperactive children's preference for immediate rewards can only be seen under certain conditions (Sonuga-Barke et al., 1992; Sonuga-Barke, Houlberg, & Hall, 1994; Sonuga-Barke, Williams, Hall, & Saxton, 1996). In some situations, AD/HD children can wait for rewards (to the same extent as controls) even when this involves ongoing inhibition. However, when there was a limited number of trials on which to choose (20 trials as opposed to five minutes) so that the small reward was associated with shorter sessions but less reward overall, the hyperactive children showed a significantly smaller mean preference for the larger delayed reward (18%) than did the control children (48%). In other words, AD/HD children will not wait for rewards if this increases the total amount of delay experienced.

Previous studies have confounded inhibition and delay aversion. However, a key feature of the choice-delay task is the double dissociation between preference for delayed rewards and inhibitory control. After the choice for the delayed option is made, children cannot switch to the small immediate reward during the trial. The preference for the large reward therefore does not involve the inhibition of the response for the small reward, but rather the active initial choice of an alternative.

A recent study (Solanto et al., 2001) supports the view that response disinhibition and delay aversion are independent co-existing characteristics of AD/HD behaviour. AD/HD children chose small immediate over large delayed rewards on a choice delay task (Sonuga-Barke et al., 1992) and had slower reaction times to signals to inhibit on the stop signal paradigm (Schachar & Logan, 1990). Together these measures proved highly diagnostic, correctly identifying almost 90 percent of AD/HD cases. Results lend support to a proposed dual pathway model of AD/HD (Sonuga-Barke, 2001) that recognises two distinct sub-types of the disorder. One subtype implicates the meso-cortical branch of the dopamine system projecting in the cortical control centres (e.g. pre-frontal cortex). In this case, Sonuga-Barke suggests that AD/HD is the result of the dysregulation of action and thought as a consequence of poor inhibitory control. The other sub-type of AD/HD is a motivational style characterised by an altered delay of reward gradient. This implicates the mesolimbic dopamine branch associated with the reward circuits (e.g. nucleus accumbens).

Apart from the Solanto et al. (2001) study, previous research in support of delay aversion has come from community-identified children with high teacher and parent ratings of hyperactivity on traditional scales such as the Connors Parent and Teacher Questionnaire (Connors, 1973). Furthermore, previous research is based on children with high conduct ratings as well as high hyperactivity ratings. No attempts have been made to control for conduct problems. The question is therefore raised whether delay aversion is consistently found in diagnosed children, and whether it is specific to AD/HD. Kuntsi, Oosterlaan, & Stevenson (2001) replicated the finding that hyperactive children choose the small, immediate reward more often than control children in the trials constraint condition. However, statistically controlling for conduct problems removed the

significant group difference, suggesting that co-occurring conduct problems explained most, if not all, of the association between hyperactivity and delay aversion.

Delay aversion theory suggests that AD/HD children may adopt a mechanism to detect delay in an early stage so they can adjust their behaviour to reduce the delay. Such an early detection mechanism can be seen in other psychopathologies. For instance, anxious individuals are thought to have a highly developed 'threat detection system' which leads to selective attending to threatening stimuli within the environment (MacLeod and Mathews, 1988; Vasey, Daleiden, Williams & Brown, 1995). Similar attentional biases have been implicated in a range of other disorders ranging from eating disorders (Boon, Vogelzang & Jansen, 2000), through to specific phobias (Asmundson & Stien, 1997).

The dot probe paradigm has been used to assess selective attention to different classes of stimuli. The original version adapted by MacLeod, Mathews, and Tata (1986) involved a pair of words presented to adults for 500 ms on each trial, one word above the other, and participants read aloud the upper word. On occasional trials, a single dot probe appeared in the position of one of the words, after their offset, and participants had to press a button as quickly as possible whenever they saw the probe. On critical trials, one word of each pair was threat-related and the other neutral. Results indicated that adults with generalised anxiety disorder were faster to respond to probes that replaced threat words rather than neutral words, in comparison with normal controls.

Research on children's attentional biases is based on the task developed by MacLeod et al. (1986) with longer target word presentation times and age appropriate words. The paradigm has been used successfully with both normal and clinical populations of children (Neshat-Doorst, Moradi, Taghavi, Yule & Dalgleish, 2000;

Taghavi, Neshat-Doorst, Moradi, Yule, & Dalgleish, 1999; Vasey et al., 1995, Vasey, Elhag, & Daleiden, 1996).

Previous research has focused on the development of attentional biases towards emotionally significant stimuli in internalising disorders. Only one study has examined attentional biases in externalising disorders such as childhood AD/HD (Sonuga-Barke, Hayes, & Bareham, submitted manuscript). Based on Vasey et al.'s (1995) version of the dot probe), reaction times were compared between children with high teacher hyperactive ratings and control children to probes presented following neutral, threatening (both social and physical), and delay-related words. Results showed that 16 hyperactive children selectively attended to delay-related words. This attentional bias towards delay cues differentiated their performance from that of 32 non-hyperactive controls.

There were a number of limitations to the above study. First, participants were not a clinical sample, and had not received a diagnosis of either AD/HD or Hyperkinetic Disorder. Furthermore, teachers reported that hyperactivity, inattention and overactivity were the main characteristics of all the hyperactive children, but the same children also had behaviours typical of oppositional defiant or conduct disorder. The extent to which attentional biases only related to AD/HD could therefore not be established. A third limitation of the study is the assumption that the words selected by the children as delay-related words actually primarily signified delay. Some words such as *still, remain, behind, follow* had a poor relation to the concept of delay.

The first aim of the present study is to test the delay hypothesis prediction with a group of clinically diagnosed children with AD/HD, and to contrast their performance with that of normal controls, comorbid AD/HD and ODD/CD, and 'pure' ODD/CD children. The first prediction was that children with AD/HD will choose fewer large, delayed rewards on the choice delay task than children in the other three conditions.

The second aim of the present study is to examine the motivational significance of delay cues for AD/HD children by comparing their bias towards delay-related words and social and physical threat words with that of normal controls, comorbid AD/HD and ODD/CD children, and ODD/CD children in a selective attention paradigm. It was predicted that AD/HD children, but not controls, will react faster to probes following delay-related words rather than neutral words.

Method

Participants

There were four groups of participants. Boys only were recruited for two reasons: first, because very few girls were presenting to Child and Family Services for diagnosis of AD/HD; second, because researchers on children's attentional processes have previously found a gender effect (Vasey et al., 1996). Power analysis was based on the effect sizes derived from previous studies by Solanto et al., (2001) and Sonuga-Barke et al. (2001). The necessary sample sizes were 25 in each group.

Normal control group (N = 25). These boys were recruited from two mainstream schools. Class teachers circulated a letter to parents describing the study and asking for volunteers. Nominated children were excluded if either parents or teachers rated them 4 or above, or 7 or above on the conduct and hyperactivity subscales respectively, of the Strengths and Difficulties Questionnaire (SDQ).

AD/HD group (N = 28). This group comprised boys with a diagnosis of AD/HD – Combined type (American Psychiatric Association, 1994) from either a Consultant Psychiatrist or Consultant Paediatrician.

Co-morbid AD/HD and Oppositional Defiant Disorder/Conduct Disorder (ODD/CD) group (N = 25). Boys in this group were identified and diagnosed by a Consultant Psychiatrist based on DSM-IV (American Psychiatric Association, 1994).

ODD/CD group (N = 12). These boys were recruited from three schools for children with emotional and behavioural problems. The original intention was to obtain boys referred to Child and Family Health services in the same way as other clinical groups. However, this was not possible because referred children had comorbid difficulties, and formal diagnoses were not being made. Those with ODD/CD were being managed by the education system.

Participant characteristics are displayed in Table 1.

Insert Table 1 about here

Selection criteria was as follows: boys aged between 8 and 12 at time of testing; Wechsler Intelligence Scale for Children (WISC) short form Full Scale IQ > 70; absence of pervasive developmental disorder, or specific learning difficulties such as dyslexia as indicated by clinicians or case notes.

A one way analysis of variance showed a significant effect of group on age (F = 6.631, df 3, 86, $p < 0.05$), IQ (F = 5.403, df 3, 86, $p < 0.05$) and reading ability (F = 3.967, df 3,86, $p < 0.05$). Scheffe post hoc analyses identified the comorbid and conduct disordered children as being significantly older, and having a lower IQ and reading age than the other two groups.

Eighty one percent of teachers completed the SDQ. There was a significant effect of group on SDQ teacher ratings for hyperactivity (F = 14.415, df 3, 69, $p < 0.05$) and

conduct problems ($F = 20.634$, df 3, 69, $p < 0.05$). Normal controls had the lowest teacher hyperactivity ratings, whereas the other three clinical groups did not differ significantly from each other. Normal controls had the lowest teacher conduct ratings, followed by AD/HD children. There was no statistically significant difference between teacher conduct ratings of comorbid and conduct disordered children.

Ninety four percent of parents completed the SDQ. Groups differed on SDQ parent ratings for hyperactivity ($F = 69.913$, df 3,86, $p < 0.05$). Normal controls had the lowest ratings. Conduct disordered children received lower hyperactivity scores than the AD/HD and comorbid groups. Similarly, there was an effect of group on SDQ parent conduct ratings, with normal controls being rated lower than the three clinical groups. There was no statistically significant difference between parent conduct ratings for the three clinical groups.

Measures

Choice-Delay Task (C-DT).

Scripted instructions were used for this task (Appendix B) which took approximately 20 minutes to complete. The child used one of two buttons to choose between two rectangles, each measuring 6.5cm wide by 3.6cm high, presented side by side on a Toshiba Satellite Pro 4200 notebook computer screen: a green square labelled '1 point' and a blue square labelled '2 points'. The instructions explained to the children that they were about to play a game in which they could earn points, and that each point would be exchanged for 1 pence at the end of the game. In three practice trials they were coached to choose alternating boxes and then were asked to compare the difference in the waiting periods. The relative difference in waiting period was confirmed by the examiner. Before the test trials, each child was instructed that he would have 20 'tries' on

which to earn points, that a chart would show how many tries were left, and that there was no time limit so he could take as much time as he wanted, to choose his points. Children and families received no other monetary compensation for participating in the study.

Task parameters were the same as those used in the 'trials constraint' condition of Experiment 2 in the original paper (Sonuga-Barke et al., 1992). Choices of the 1-point and 2-point rewards were followed by a 'pre-reward' delay of 2 sec or 30 sec, respectively, before displaying on the screen the number of points earned in that trial. One-point vs. 2-point reward choices, and pre-reward delays were chosen because pilot work in a study by Solanto et al (2001) indicated that the 1:2 point ratio was most effective in avoiding floor and ceiling effects and maximising differences between groups. One block of 20 trials was presented. At the start of each trial, the experimenter verbally indicated the number of trials remaining, and showed this on a visible sliding scale. The child was given his monetary reward at the end of the block. The side of presentation of the large reward was counterbalanced between participants. The computer recorded the number of large rewards chosen.

Dot Probe Paradigm.

Various versions of the dot probe task have been developed to examine attentional biases and there is some dispute among researchers regarding their relative merits and sensitivity, with each version appearing to have its own advantages and disadvantages. Based on the published recommendations of Mogg and Bradley (1999), and personally communicated verbal recommendations by the same researchers, the current study modified MacLeod et al.'s (1986) original task to make it more suitable for children with AD/HD.

A forced choice response format similar to that used by Posner and colleagues (Posner, Snyder, & Davidson, 1980, experiment 3) was used. One drawback of MacLeod et al.'s task is that it only obtained data from a small proportion of trials (most were fillers) which is a disadvantage in clinical research with AD/HD children who typically have difficulty sustaining attention over a long tedious task. In the current study, each trial started with a word pair, with one word above the other, as in the original version. A dot probe then appeared in the location of one of the words after the word pair had disappeared and participants pressed either an 'upper' or 'lower' response key to indicate its position (Mogg, Bradley, & Williams, 1995). This is called a probe position task. Reaction time data can be obtained from all trials, which allows various exposure and word type conditions to be examined.

Another drawback of MacLeod et al.'s task is that the presence of a threat word serves as a warning cue for probes, and some individuals may vary in the extent to which they detect this covariation between threat and probe stimuli (Mogg and Bradley, 1999). The forced choice paradigm eliminates this covariation between the probe and threat stimuli. It seems more suited to research with clinical children where high error rates and high variance in reaction time data tend to be more problematic than with adults.

A further advantage of the modified task was that the total number of trials (121) was significantly less than the original 160 (MacLeod et al., 1986) or 220 (Vasey et al., 1995), thereby reducing completion time to about five minutes compared to 12 minutes for the original version. This was an important consideration for use with AD/HD children or children with oppositional behaviour. Reducing the task length would increase the likelihood of their engagement and compliance.

Children were presented with 121 trials on a Toshiba Satellite Pro 4200 notebook computer. On each trial a pair of words was presented with one word positioned 3cms

directly above the other. Words were presented in a random order using upper and lower case letters 3 cm high. On each trial children were required to read the upper of the two words out aloud. The experimenter manually recorded the number of reading errors and omissions made. Word pairs were presented for 1250 ms consistent with previous probe detection research on children (Vasey et al., 1995). After each trial, a probe appeared in the form of a small cross 25 ms after the words disappeared, either behind the top or bottom word. This probe remained on the screen until a response was made. Scripted instructions were used for this task (Appendix C). Children were instructed to respond to the probe as quickly as possible by pressing the top button (if the probe appeared where the top word was) or the bottom button (if the probe appeared where the bottom word was). There were 24 trials for each of three conditions: social and physical threat, and delay-related word pairs. The rest were filler word pairs, making a total of 121 trials. Either the active (i.e. threat) or the neutral word was probed. On each of these trials the active or the neutral word appeared in the upper or lower part of the screen with equal probability. The probe could follow in either position with equal probability. This gave three independent factors (i) the type of word probed, (ii) the target threat word location (upper or lower), (iii) the probe position (upper or lower).

All words were taken from previous research (Sonuga-Barke, Hayes & Bareham, submitted manuscript; Vasey et al., 1995). Some of the 13 delay-related words previously used by the former researchers were ambiguous and it was not possible to generate further single words associated with the imposition of delay. Therefore, a pilot study asked children aged between 8 and 12 years how much waiting is implied by each of the 13 words: none at all, a little, or a lot. As far as was known, these children were normal controls from a mainstream school. According to their ratings, the six words most representative of the concept of delay were employed (see Table 2).

Insert Table 2 about here

Following the recommendation of Mogg (personal communication), six words were used from each threat category (i.e. physical, social, and delay-related). These were repeated four times (twice in the top position and twice in the bottom), making a total of 24 exposures, and they were matched with neutral words of an equivalent length. Word pairs were presented in random order.

Wechsler Intelligence Scale for Children III UK (WISC III UK, 1993).

A short form of the WISC III UK provided a measure of IQ. Picture Completion, Similarities, Arithmetic and Block Design were selected because they have been shown to provide the best representative IQ score (Kaufman, Kaufman, Balgopal, & McLean, 1996). This combination was selected on the basis of psychometric, clinical and practical qualities: Correlation with the full scale IQ score was 0.93, they are short to administer, and only Similarities requires some subjectivity to score.

Wechsler Objective Reading Dimensions (WORD: Basic Reading; Rust, Golombok, & Trickey, 1993)

The WORD basic reading subtest was used (Appendix D) because words represented the stimuli in the dot-probe task. It was important that children had a reading age of about 8 years. This would help to ensure that any attentional biases were mediated by stimulus threat value rather than readability of words. The basic reading subtest takes

less than five minutes to administer, and comprises a series of printed words which children are required to read aloud. It is designed for children aged 6 to 16 and produces both a standard and reading age equivalence score. The UK edition was validated on a stratified sample of 794 children. Test-retest stability coefficient for a sample of 367 American children across all age groups was found to be 0.90, and split-half reliability coefficients for ages 8-12 ranged from 0.94 to 0.96 (Rust et al., 1993).

Revised Children's Manifest Anxiety Scale (RCMAS) (Reynolds & Richmond, 1978). The RCMAS is a 37-item self-report measure of the level and nature of anxiety in children and adolescents ages 6-19 years (see Appendix E). It yields a total anxiety score and four subscales of physiological anxiety, worry/oversensitivity, social concerns/concentration, and lie. A high score indicates a high level of anxiety. It takes 10-15 minutes to complete. The scale was standardised on nearly 5,000 American children. Reynolds (1981) reported a 9 month test-retest correlation of 0.68, and Reynolds and Paget (1982) reported Chronbach coefficient alpha reliability estimates by age, race and sex between 0.42 and 0.87, with the majority ≥ 80. The internal consistency and test-retest reliabilities of this measure are therefore within acceptable ranges. The RCMAS has been shown to correlate significantly with other measures of childhood anxiety such as the State-Trait Anxiety Inventory for Children (STAIC) (Spielberger, 1973) and the Fear Survey Schedule for Children – Revised (Ollendick, 1983).

Strengths and Difficulties Questionnaire (SDQ: Goodman, 1997).

This is a brief behavioural screening questionnaire for children aged 4-16 years, available for parents, teachers, others, or children themselves (ages 11-16) to complete (Appendix F). According to Goodman and Scott (1999) the SDQ correlates highly with

the commonly used Child Behaviour Checklist (CBCL: Achenbach, 1991) whose validity and reliability are well established. The SDQ has high internal reliability scores: Cronbach's alpha of 0.76 for total difficulties, 0.75 for inattention-hyperactivity, 0.70 for prosocial behaviour, 0.61 for emotional problems, 0.54 for conduct problems and 0.51 for peer problems. This measure was used instead of the CBCL for a number of reasons. First, it is better at detecting inattention and hyperactivity than the CBCL (Goodman & Scott, 1999). Second, it only takes 5 minutes to complete compared to 30 minutes for the CBCL. Third, the SDQ includes a prosocial scale, giving parents and teachers an opportunity to focus on a positive aspect of each child, rather than solely on problem areas.

Procedure

The research received appropriate local ethical approval (Appendix G). Parents or guardians were sent a letter that outlined the purpose of the study (Appendix H), an informed consent form (Appendix I), and the SDQ questionnaire. If they were willing for their child to participate, they signed the informed consent form and completed the questionnaire. Parents of the clinical groups were assured that declining or withdrawing from the study would not affect any treatment they might receive. Children received no medication for AD/HD up to 12 hours before, or during participation in the current study. They were tested at school, unless specifically requested by parents for this to take place at home. Class teachers were also informed of the study, and asked to complete the SDQ (Appendix J).

Data were collected by one of two trainee clinical psychologists. When boys entered the experimental room, they were first informed of the purpose of the experiment and of the duration and the nature of the tasks that they were going to perform. They

were given the choice of participating, and were told that they could decline participation and/or withdraw at any time (Appendix K).

Four subtests of the WISC III UK were administered in the same order (Picture Completion, Similarities, Arithmetic, Block Design) for all participants. This was followed by the WORD reading test, the Choice-Delay Task, RCMAS, and the Dot Probe task. Two other tasks were administered at the end, which were not connected with this study. The experimenter remained with the child during testing which took about 75 minutes.

Results

Correlations between parent and teacher SDQ hyperactivity ratings (.589) and parent and teacher conduct ratings (.436) were both statistically significant at the 0.01 level (two tailed).

A Pearson Chi Squared test was carried out to determine how accurate were the SDQ hyperactivity and conduct ratings at predicting diagnostic caseness. Both parent and teacher SDQ scores were available for 78% of participants. There was a highly significant association ($\chi^2 = 54.005$, $p < .001$, two tailed) between diagnostic groups (controls, AD/HD, comorbid AD/HD + ODD/CD, and ODD/CD) and groups based on SDQ ratings. Therefore, further statistical analyses compared diagnostic groups.

The SDQ determined groups were based on both parent and teacher ratings as follows: (1) low hyperactivity and conduct problems (i.e. both below or equal to 6 and 3 respectively); (2) high hyperactivity but low conduct disorder; (3) High hyperactivity and conduct disorder; (4) high conduct disorder only.

Insert Table 3 about here

As shown in Table 3, 24 children had low hyperactivity and conduct ratings, 10 of which had received a diagnosis - AD/HD (7) and comorbid (3). Eleven boys were rated by parents and teachers as high hyperactive only, yet four of these had received a comorbid diagnosis. Of the 16 who received both high hyperactivity and conduct ratings, seven received an equivalent comorbid diagnosis, five were diagnosed as AD/HD, and four were represented in the conduct disorder only diagnostic group. Only four boys received high parent and teacher conduct ratings without hyperactivity, yet eight were assigned to this diagnostic group.

Choice-delay task

Mean differences between the four groups for number of large delayed reward chosen are shown in Table 4. A one-way analysis of variance confirmed no significant group difference in choice of the large reward ($F = .720$, $p > .05$, 1-tail). Neither was there any difference when age and IQ were entered as covariates ($F = 1.464$, $p > .05$, 1-tail).

Insert Table 4 about here

A multiple regression analysis was carried out to determine which variables predicted choice of the large delayed reward. The dependent variable was number of large rewards. The following Beta weights resulted from the independent variables: Age (-.192), IQ (.210), RCMAS score (.187), parent hyperactivity rating (.217) and conduct rating (-.121), teacher hyperactivity rating (-.225) and conduct rating (-.066). There were no statistical significant predictors of number of large rewards chosen, and only 19% of variance in choice of the large rewards could be explained by all these seven variables.

Dot-probe task

Reaction time data for one participant was unavailable (AD/HD) due to lack of time to administer the task. In addition, four boys found the task too difficult and failed to complete (1 AD/HD; 2 comorbid; 1 ODD/CD). Ten children were excluded from dot probe analyses because they had reading age equivalence less than 7 years and 9 months (4 controls; 3 AD/HD; 2 comorbid; 1 ODD/CD). Five more sets of data were excluded because participants made reading errors or omissions during the task on more than 15 trials (4 comorbid; 1 ODD/CD). Closer inspection of reading error data for excluded participants confirmed that errors represented a large proportion of the threat trials. Final group numbers for the dot probe task were: 21 controls; 23 AD/HD; 17 comorbid; 10 ODD/CD.

The probe detection latency data were trimmed by dropping scores below 200 ms and above 2,500 ms. This procedure eliminated outliers that may be due to lapses in attention and premature button presses. Although the data was non-parametric, Green, Salkind and Akey (2000) state that ANOVA is robust to violation of the non-parametric assumption of moderate to large sample sizes (15 cases per cell). Because the data was non-parametric, and to minimise further the influence of outliers, the latency data were

log transformed. Subsequently, analyses of covariance were conducted on both the remaining log transformed and untransformed data. As both sets of data yielded the same pattern of results, the untransformed latency data are reported.

The mean latencies by group for the three within-subject conditions are shown in Table 5.

Insert Table 5 about here

The mean probe-detection latencies were subjected to mixed-model analysis of covariance (ANCOVA) for repeated measures, with one fixed between-subjects factor (group) and three fixed within-subjects factors (word type, target location, and probe position) resulting in a 4 x 3 x 2 x 2 (Group x Word type x Target Location x Probe Position) design. Groups did not differ on the number of reading errors or omissions made so this was not introduced as a covariate. Age, IQ, and WORD reading standard scores all represented covariates. In addition, although there was no statistically significant ANOVA group difference on RCMAS scores, nevertheless, RCMAS was used as a covariate because of the extensive research evidence showing a relationship between anxiety and attention.

There were no significant main effects. There were two interactions that reached statistical significance: between target location and group, F (3,63) = 3.82; p = .014; between word type and probe position, F (2,126) = 4.34, p = .015. The first interaction indicated that comorbid group mean RTs were statistically significantly longer when target words were in the top position. Other groups showed a trend in the same direction. The second interaction indicated that mean RTs for delay-related words were statistically

significantly shorter when the top word was probed. There was a trend in the same direction for social and physical threat words. No other interaction was significant.

To clarify the findings, attentional bias scores were calculated for each word type and exposure condition using the following equation (MacLeod and Mathews, 1988; Mogg, Mathews, & Eysenck, 1992):

$$\tfrac{1}{2}\,[(UP/LT - UP/UT) + (LP/UT - LP/LT)]$$

where UP/LT refers to detection times for the probe in the upper area when the threat is in the lower area, and so on. If a child shifts attention towards the position where the threat word appeared, they will detect the probe faster in that area, and the equation will produce a positive value. In contrast, if a child shifts attention away from the position where the threat appeared, they will detect the probe more slowly in that area and the equation will produce a negative value. The bias score reflects the target location x probe position interaction, where positive values reflect a shift of attention towards the spatial location of negative words relative to matched control words, and minus values reflect an attentional bias away from the spatial location of negative words.

Mean attentional bias scores for four groups and the three groups of words are presented in Table 6.

Insert Table 6 about here

A repeated measures ANOVA was carried out with Group (4: controls, AD/HD, comorbid, ODD/CD) as a between subjects variable and word type (3: index of attentional bias scores for physical, social and delay-related) as the within subjects

variable. Results showed that neither the main effect of Group, ($F = 2.16$, $p = .110$), nor the main effect of Word Type, ($F = .116$, $p = .891$) were significant. The interaction between Group and Word Type also did not approach significance ($F = .592$, $p = .736$). Neither were there any significant results when age, IQ, reading standard score, and RCMAS scores were entered as covariates.

Pearson correlations were calculated between the attentional bias scores and anxiety measure. Results showed no significant correlations. Neither the physically threatening mean bias scores (-.103, $p > .05$, two-tailed), nor the socially-threatening mean bias scores (.087, $p > .05$, two-tailed) correlated significantly with anxiety.

Discussion

One purpose of the present study was to examine whether clinically diagnosed children with AD/HD were delay averse as measured by a computerised choice-delay task. Results did not support the hypothesis that AD/HD children would choose fewer large, delayed rewards than normals, comorbid AD/HD + ODD/CD, or children with ODD/CD. These findings are inconsistent with previous research showing hyperactive children to choose the small immediate reward more often than normal controls (Kuntsi, et al., 2001; Solanto et al., 2001; Sonuga-Barke et al., 1992).

The second purpose of the present study was to explore whether AD/HD children have an attentional bias towards delay-related cues. However, there was no support for the hypothesis that AD/HD children would selectively attend to delay-related words more than normals, comorbid AD/HD + ODD/CD, or children with ODD/CD. There are a number of possible reasons to account for the present findings.

Group membership

Although the methodological aim of the present study was to recruit relatively 'pure cases' of AD/HD (without conduct problems), and 'pure cases' of ODD/CD (without hyperactivity), this was clinically impractical as the two typically co-occur. The study reasonably achieved identification of children with more severe symptoms of one disorder rather than the other, consistent with conventional diagnostic criteria. However, group differences on the critical variables of hyperactivity and conduct disorder were not very large.

Normal control children were excluded from the study if they had ratings of 3 and 7 or more on the conduct and hyperactivity subscales of the SDQ. Nevertheless, many of the remaining boys received reasonably high teacher ratings of hyperactivity. These control children all came from two schools and it is possible that they were slightly unrepresentative of the normal population.

Similarly, seven children with a diagnosis of AD/HD received low ratings on both the SDQ parent and teacher hyperactivity subscale, yet still received a diagnosis of AD/HD. A further three children received similar ratings, yet were diagnosed as comorbid AD/HD + ODD/CD. One possible reason for low SDQ hyperactivity ratings is that many children were being successfully treated by medication. Ratings therefore reflected this improvement, and some teachers actually indicated this in additional text on the questionnaire. Future research should ideally use newly-diagnosed clinical samples at time of initial assessment, but this was impractical under the time constraints of the present study.

Another possible reason for low SDQ hyperactivity ratings for AD/HD children is the variability between professionals in assigning a diagnosis. Clinical samples were recruited from different service settings, some of which were possibly more liberal in

granting a diagnosis of AD/HD. This notion was supported at time of testing by a few teachers or headteachers who offered the opinion that there was little difference in terms of hyperactivity between a few clinical participants and their class peers. Previous delay aversion studies by Sonuga-Barke et al. (1994) used boys selected on the grounds of pervasive hyperactivity. A third explanation is that criteria for a diagnosis may have been met two or three years previously, but a review was needed which would possibly question the current validity of the diagnosis. It is also likely that the intended pure AD/HD group was heterogeneous, thereby reducing the distinction between this and the comorbid group. Group mean parent conduct ratings were higher for the AD/HD children than for those with oppositional or conduct problems only.

There is therefore enough evidence to suggest that the limited group differences on critical variables of hyperactivity and conduct problems would dilute any group effects in the analyses of the data, particularly given the relatively small sample sizes. However, analyses carried out on groups determined by SDQ cut-off points also failed to find significant group differences on either the choice-delay or dot probe tasks. This suggests that other factors also account for the pattern of results.

A further limitation of the present study concerns its power. Based on Cohen's power convention (Cohen, 1992), a four-group ANOVA with 22 participants per cell would yield more than adequate power at 0.01 level to detect group differences. Although in excess of 120 children were tested by two researchers, many did not meet full inclusion criteria, or failed to successfully complete all the tasks, thereby reducing final group sizes to 21, 23, 17, and 10. It remains possible that with a much larger sample, delay aversion and attentional bias effects may have been found, but it is unlikely given the results in the present study, and given other methodological difficulties.

Another explanation for the failure to find several of the predicted effects in this study is that groups differed markedly on IQ, age and reading ability, which is likely to limit the sensitivity of the tasks. According to Vasey and colleagues (Vasey et al., 1995), the tendency to bias attention in the upper position on a dot probe detection task increased with age and reading recognition scores. An improved design would match these variables across groups. However, this would prolong data collection, and was not possible within the time constraints of the present study. Although these variables were controlled as covariates in statistical analyses, there is an argument for not including IQ as a covariate because the lower IQ of AD/HD children is part of the disorder and so is part of any difficulties. Future studies must also explore the impact of factors other than anxiety status (e.g. age or reading ability) on the probe detection task.

Dot probe task issues

The present study modified the dot probe task that had previously been used with children (Sonuga-Barke et al., submitted manuscript; Vasey et al., 1995). It is possible that the modifications rendered the measure insensitive to attentional biases in children. Fuelling this conclusion is the finding that there were no statistically significant differences in children's response towards socially- or physically-threatening, delay-related or neutral words. Children responded to threatening words as if they were neutral.

Another indication that the dot probe measure was probably insensitive is the finding that none of the mean threat bias scores correlated with anxiety levels. This is inconsistent with previous findings that have shown an association between attentional threat bias and state anxiety with adults (Mogg, Bradley, de Bono, & Painter, 1997) and children (Vasey et al., 1996), and with clinically anxious children (Vasey et al., 1995). However, results are equivocal because predicted biases have not been consistently found

in both probe positions (e.g. MacLeod & Mathews, 1998, Mogg, Mathews, & Eysenck, 1992, Vasey et al., 1995).

The forced choice, probe position task has not been used with children before. This method has an additional requirement of deciding location of the probe (top or bottom) which may have exerted high cognitive processing demands for children, and even more so for clinically diagnosed children. Support for this notion comes from mean reaction time differences between this and other studies. Mean RTs to threat words for control children commonly lie between 470 (97) and 550 (201) ms (Sonuga-Barke et al., 2001; Vasey et al., 1995). In the current study they were approximately 100 ms slower. This does not seem to be due to sample differences, because ages were roughly comparable. Slower RTs for this probe position task therefore seems to reflect its increased complexity relative to a detection-only task involving the pressing of one response button. Whilst the task has been shown to be a sensitive measure of attentional biases in adults (Mogg et al., 1995; Mogg & Bradley, 1999), it may be too complex for young children. It is clearly essential, in order to facilitate the investigation of potential cognitive biases in children, to develop reliable, robust and sensitive tasks. We also need to determine how biases may develop with increasing age, and which tasks are suitable for particular age groups. Longitudinal studies are required.

A further requirement adding to the complexity of the dot probe task was one of reading the top word out aloud. For adults using the forced choice paradigm, reading has become a fast and automatic process, consuming limited cognitive capacity. However, reading is an effortful task for children as young as 8 years, or for clinical children whose reading skills are limited. They may have insufficient time or attentional resources to adequately process the word pairs, and they would show no evidence of attentional bias in such a task. In the current study, many participants found reading out aloud difficult and

anxiety provoking, especially in conjunction with other dot probe task requirements. Nevertheless, it is important to determine that they can read threat words; if they are unable to do so, a search for threat bias would be meaningless.

Use of words as stimuli introduced a number of other difficulties. It necessitated a rigorous exclusion criterion in the form of reading age equivalence that considerably reduced the final sample. In particular, it left a small number of children with ODD/CD because they commonly have associated learning or reading difficulties. This significantly reduced statistical power of the study. Furthermore, it may have excluded clinical children with more severe difficulties, thereby diluting results, and reducing the study's generalisability.

The use of words as stimuli introduced another methodological problem, namely a confound between stimulus threat value and subjective familiarity. Words are ambiguous and have multiple meanings for individuals. For example, words like 'stop' are also likely to reflect the sorts of negative interactions with authority figures experienced by oppositional and defiant children. Children may have been responding to the punitive element of the word rather than, or as well as, their relation to delay. Delay-related words are also likely to have a subjective frequency of usage in children with AD/HD who are often told by parents and teachers to 'wait'. This raises the question of whether potential attentional biases for such material are mediated by the effects of threat or subjective familiarity. A way to test this would be to measure biases before and after treatment of AD/HD. A reduction in attentional bias would suggest that familiarity may not be a critical confound. Another issue relating to the words chosen involves the distinction between delay and inhibition. Many of the words chosen represent instructions to inhibit as well as to wait.

In an attempt to minimise ambiguity, the current study reduced the number of delay-related words previously used by Sonuga Barke and colleagues (Sonuga Barke et al., submitted manuscript) to six. Another reason for doing this is that it was impossible to generate enough single words that typified the concept of delay, or the passage of time. These words, in addition to six from each of the social and physical threat categories were presented four times. This made a total of 18 threat words compared to 44 threat words used in the study by Vasey and colleagues, 1995. No other study of attentional bias has repeated stimulus words four times. It is possible that children habituated with each repetition of a threat word, thereby reducing its threat value, and minimising effects. This would account for the finding that children responded to threat words in the same manner as responses to neutral words. Further statistical analysis could help to clarify whether habituation had occurred. It is possible to re-order the database and run a repeated measures ANOVA, comparing participants' performance at each presentation rate (i.e. four levels). If increased repetition produced longer reaction times, this would support the habituation hypothesis.

Future research should avoid the use of words altogether. One recent children's study has addressed this issue (de Ruiter & Sonuga-Barke, unpublished manuscript) by adopting a classical conditioning paradigm. Using a computerised dot probe task, children were taught to associate a particular colour with a period of delay. Compared to 25 normal controls, 25 children with AD/HD responded more quickly on trials in which the dot replaced a delay-related cue than on trials where the dot replaced a neutral cue. Their results showed that AD/HD children had a significantly greater attentional bias towards delay-related cues. They support the notion that these children are motivated to detect delay in the environment.

Clinical implications

The present study was unable to provide further support for delay aversion theory, the dual pathway model (Sonuga-Barke, 2001) or AD/HD children's attentional bias towards delay-related cues. However, previously published findings have a number of implications for clinical research and practice. The first relates to assessment and diagnosis of the condition. Scores on lab measures of impulsivity correlate poorly both among themselves and with parent and teacher ratings of 'impulsive' behaviour in natural settings (Barkley, 1991; Milich & Kramer, 1985). If it can be shown that the choice-delay task has consistently demonstrated utility in the characterisation of AD/HD difficulties, this could contribute towards assessment by providing a sensitive and specific tool.

A second implication relates to the clinical utility of previous delay-aversion research and conclusions. As mentioned before, this has mostly consisted of homogenous groups of children within a narrow age range, based on high parent and teacher ratings of hyperactivity. The question is raised whether findings and theoretical conclusions are generalisable to the clinic where the pattern of the disorder often presents as heterogeneous.

According to the dual pathway model (Sonuga-Barke, 2001), the motivational pathway of AD/HD is moderated by the environment, for example, parents or teachers setting unrealistically high standards which might create more failures to wait. Delay-rich settings come to acquire aversive properties through association with negative emotions related to failures to wait. If it can be consistently shown that AD/HD children have an attentional style tuned to the detection of delay, then intervention could incorporate this knowledge in a similar manner as anxiety treatment. For example, it would seem reasonable to consider the child, parent and teacher awareness of the

connection between excessive requirements to wait and the AD/HD child's behaviour and emotional reaction. Attention can be directed to explaining how certain situational consequences can exacerbate the condition. Emphasis could also be placed on lowering the child's affective response to delay. One way might be to encourage parents and teachers to gradually increase the length of time a child spends in a delay situation, which might be posing a threat to the child, and by giving positive feedback on tolerance of delay.

Another clinical implication relates to the rules and instructions provided to AD/HD children as part of their behavioural intervention programme. These are clear, brief, and often delivered through more visible and external forms of presentation. AD/HD children are also encouraged to repeat them out aloud, or utter them softly to themselves whilst carrying out the instruction. Words such as 'stop', 'listen', and 'wait' are typically used (Barkley, 1998). If AD/HD children's attentional style is sensitive to detection of delay cues, then procedures using delay words may be aversive and counter productive.

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Table 1. Participant characteristics: Means and standard deviations (*SD*s) by Group

Group		**Age (Mths-yrs)**	**WISC IQ**	**WORD Standard score**	**SDQ-P Hyp**	**SDQ-P Conduct**	**SDQ-T Hyp**	**SDQ-T Conduct**	**RCMAS Anxiety**
Controls:	Mean (SD)	9,9 (0,8)	104.5 (12.4)	99.9 (14.4)	3.1 (1.7)	.74 (1.0)	2.8 (2.0)	.41 (.71)	47.5 (10.1)
	N	25	25	25	23	23	17	17	25
ADHD:	Mean (SD)	10,1 (1,2)	101.2 (16.6)	100.2 (14.3)	9.0 (1.4)	5.6 (3.2)	6.7 (2.9)	2.5 (2.2)	52.1 (13.2)
	N	28	28	28	28	28	23	23	28
ADHD +	Mean (SD)	10,7 (1,2)	93.5 (10.7)	89.8 (11.9)	9.2 (1.1)	6.8 (1.7)	8.0 (2.2)	4.4 (2.4)	55.1 (10.9)
ODD/CD:	N	25	25	25	22	22	23	23	24
ODD/CD:	Mean (SD)	11,2 (1,0)	88.8 (11.3)	91.7 (9.5)	6.7 (2.6)	4.9 (2.0)	6.2 (3.3)	5.6 (1.5)	48.1 (7.8)
	N	12	12	12	12	12	10	10	12

WISC = Wechsler Intelligence Scale for Children – III UK; WORD – Wechsler Objective Reading Dimensions – reading age equivalence score;
SDQ = Strengths and Difficulties Questionnaire (parent or teacher version); Hyp = hyperactivity subscale of the SDQ; Conduct – conduct subscale of the SDQ;
RCMAS = Revised Children's Manifest Anxiety Scale

Table 2. Emotionally threatening words presented in the dot probe task

	Word Type	
Delay	Physical	Social
Afterwards	bleeding	dumb
halt	danger	fool
later	death	hated
slow	hurt	lonely
stay	injury	stupid
stop	painful	teased

Table 3. Chi Square crosstabulation to show the degree of association between diagnostic groupings and SDQ hyperactivity and conduct score determined groups

	Groups determined by both parent and teacher SDQ ratings				
	Low hyperactivity and conduct disorder ratings	High hyperactivity ratings only	High hyperactivity and conduct disorder ratings	High conduct disorder ratings only	
Diagnostic groups					Totals
Control	14	0	0	0	14
AD/HD	7	7	5	0	19
AD/HD + ODD/CD	3	4	7	0	14
ODD/CD	0	0	4	4	8
Totals	24	11	16	4	55

Table 4. Group mean differences for large rewards chosen on the choice-delay task

Group	**Mean**	**SD**
Controls (N = 25)	11.0	5.7
AD/HD (N = 28)	11.2	6.1
AD/HD + ODD/CD (N = 25)	12.0	5.7
ODD/CD (N = 12)	9.2	3.0

Table 5. Mean RTs in Msec to probes for each group (controls, AD/HD, AD/HD + ODD/CD, ODD/CD) as a function of word type (physical, social, delay) and target location and probe position (top or bottom)

	Position of Target (T) and Probe (P) (top/bottom)			
	TtopPtop	TtopPbot	TbotPbot	TbotPtop
Word/threat type and group				
Delay				
Controls	650 (228)	630 (159)	645 (167)	601 (160)
AD/HD	689 (246)	748 (254)	773 (259)	682 (236)
AD/HD + ODD/CD	676 (210)	709 (205)	639 (163)	633 (198)
ODD/CD	550 (171)	579 (234)	615 (304)	624 (222)
Social				
Controls	645 (187)	637 (170)	608 (178)	609 (147)
AD/HD	772 (287)	728 (242)	702 (180)	674 (232)
AD/HD + ODD/CD	642 (233)	678 (204)	634 (170)	642 (195)
ODD/CD	549 (156)	604 (193)	632 (200)	604 (186)
Physical				
Controls (N = 21)	618 (161)	612 (165)	608 (173)	603 (123)
AD/HD (N = 23)	691 (249)	708 (195)	729 (261)	721 (263)
AD/HD + ODD/CD (N = 17)	690 (260)	675 (238)	589 (142)	608 (173)
ODD/CD (N = 10)	544 (127)	616 (275)	565 (184)	530 (160)

Note: Figures in brackets represent standard deviations.

Table 6. Mean attentional bias scores in Msec for each group (controls, AD/HD, AD/HD + ODD/CD, ODD/CD) as a function of word/threat type (delay, social, physical)

	Word/threat Type		
	Delay	Social	Physical
Group			
Controls	-32.0060	-3.5925	-6.1262
	(77.9895)	(57.4129)	(70.2648)
AD/HD	-16.3167	-35.8428	5.0380
	(104.2141)	(89.5500)	(122.1679)
AD/HD + ODD/CD	13.4289	21.8735	1.8377
	(50.2984)	(68.4799)	(102.0271)
ODD/CD	19.1750	13.4025	18.8492
	(141.8043)	(45.8139)	(107.2022)

Note: Figures in brackets represent standard deviations.

Appendices

Appendix A

The Journal of Child Psychology and Psychiatry and Allied Disciplines

Instructions to Authors

Notes for Contributors

General

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References in running text should be quoted as follows: Smith and Brown (1990), or (Smith, 1990), or (Smith, 1980, 1981a, b), or (Smith & Brown, 1982), or (Brown & Green, 1983; Smith, 1982).

For up to five authors, all surnames should be cited the first time the reference occurs, e.g. Smith, Brown, Green, Rosen, and Jones (1981) or (Smith, Brown, & Jones, 1981). Subsequent citations should use "et al." (not underlined and with no period after the "et"), e.g. Smith et al. (1981) or (Smith et al., 1981).

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Full references should be given at the end of the article in alphabetical order, and not in footnotes. **Double spacing** must be used.

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Kiernan, C. (1981). Sign language in autistic children. *Journal of Child Psychology and Psychiatry, 22*, 215–220.

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Whilst every effort is made by the publishers and editorial board to see that no inaccurate or misleading data, opinion or statement appear in this journal, they wish to make it clear that the data and opinions appearing in the articles and advertisements herein are the sole responsibility of the contributor or advertiser concerned. Accordingly, the publishers, editorial board and editors, and their respective employees, officers and agents accept no responsibility or liability whatsoever for the consequences of any such inaccurate or misleading data, opinion or statement.

Appendix B

Instructions for the choice-delay task

In the game that you are about to play, you will have the chance to win some money. You can do this by scoring points on the computer by pressing the red button – here, or the black button – here. When you press the red button you will win 2 points and when you press the black button you will win 1 point. For every point you win I will give you 1 pence. So, if you win 20 points, I will give you 20 pence; if you win 30 points I will give you 30 pence.

If you press the black button to get 1 point, it will be delivered straight away, and you can then make another choice. If you press the red button to choose the large reward, you will have to wait some time before you earn your points and can make your next choice – a wait of 30 seconds.

You have 20 goes to win your money. This means that after the game starts, you can only choose 20 times by pressing the buttons and then the game will stop and the computer will add up your points.

Each time that you make a choice by pressing a button I will tell you how many goes you have left, and I will show you on this scale.

Do you understand what you have to do?

(If yes) … let's have a practice.

(If no) … *repeat instructions*

Appendix C

Instructions for the dot probe task

You are going to see words shown on the computer screen, two at a time. The words will appear one above the other in the middle of the screen, like this (participant is shown the first screen of the 5 practice trials). You must read the top word out aloud as soon as it appears. The words will disappear after about one second.

Then, a small cross will appear where the top word was, or where the bottom word was. When you see the cross, you must press a button as fast as you can – the top button if the cross appeared where top word was, the bottom button if the cross appeared where the bottom word was. The computer programme will time how long it takes you to press the button.

The programme will stop after all the words have been shown. But you will need to watch the screen carefully for about 5 minutes. I will be sitting here, but I will just be watching.

So, you have two things to do: (1) read the top word out aloud, and (2) press the button as fast as you can after you have seen the cross.

Do you understand? Let's have a practice.

(5 practice trials)

Appendix D

Wechsler Objective Reading Dimensions (WORD)

WORD Record Form

WECHSLER OBJECTIVE READING DIMENSIONS

Name .....

School .....

Examiner .....

Date of National Curriculum assessment ..... National Curriculum level .....

School year .....

Gender .....

First language (if not English) .....

Handedness .....

	Year	Month	Day
Date tested			
Date of birth			
Age			

Behavioural observations

Ability–achievement discrepancy analysis

Ability test ..... Date of ability testing .....

Predicted achievement method (using Wechsler FSIQs)
① Predicted WORD standard scores (Tables C.1–C.3)
② Actual WORD standard scores
③ Subtract ② from ①
④ Significance of D (Table C.4)
⑤ Frequency of D (Tables C.5–C.7)

Predicted achievement method (using other ability tests)
Refer to pp. 79–80 in the Manual for the necessary formulae

	① Predicted WORD standard score	② Actual WORD standard score	③ Difference (D)	④ Significance (circle one)	⑤ Frequency (%)
Basic Reading				0.05 0.01 NS	
Spelling				0.05 0.01 NS	
Reading Comprehension				0.05 0.01 NS	

 ISBN 0 7491 0293 4

Test scores

	Raw score	Standard score	Conf. inter. ____ %	Percentile	Stanine	Age equivalent	WISC-III^{UK} scaled score
Basic Reading			–				
Spelling			–				
Reading Comprehension			–				
WORD Composite			–				

Profiles

WORD scaled scores

BR Sp RC Comp

For WISC-III^{UK} users only

WISC-III^{UK} index scores

VCI POI FDI PSI

WISC-III^{UK} IQ scores

FSIQ VIQ PIQ

160 150 140 130 120 110 100 90 80 70 60 50 40

Basic Reading

About 10 seconds for each item

If the child scores 0 on any of the first 5 items administered, administer preceding items in reverse sequence until child scores 1 on each of 5 consecutive items

6 consecutive scores of 0

	Item	Notes			Score 0 or 1
5–7					
1.	fun	ask	**sit**	girl	
2.	park	next	for	**card**	
3.	**duck**	but	did	can	
4.	stop	**push**	box	walk	
5.	call	**cow**	could	come	
6.	has	had	**hat**	hot	
7.	**sheep**	shop	sleep	street	
8–9					
8.	the				
9.	up				
10.	into				
11.	so				
12.	said				
13.	then				
14.	animal				
15.	because				
10+					
16.	slow				
17.	again				
18.	any				
19.	fruit				
20.	know				
21.	shut				
22.	instead				
23.	enough				
24.	sight				
25.	photograph				
26.	completely				

Item	Notes	Score 0 or 1
27.	courage	
28.	comforting	
29.	jealous	
30.	responsibility	
31.	dozing	
32.	ajar	
33.	ruin	
34.	useless	
35.	pier	
36.	ideally	
37.	chord	
38.	acquire	
39.	governmental	
40.	abrupt	
41.	pathetic	
42.	cleanse	
43.	unique	
44.	sparse	
45.	accordion	
46.	poise	
47.	ridicule	
48.	indomitable	
49.	catastrophe	
50.	conscience	
51.	reminisce	
52.	coerce	
53.	euphemism	
54.	antithesis	
55.	hierarchical	

Child's behaviour when presented with unfamiliar words (tick where applicable)

- **A.** Used decoding skills .....
- **B.** Was persistent when decoding .....
- **C.** Gave up easily when decoding .....
- **D.** Used no strategy (guessed) .....
- **E.** Made no attempt .....

Max=55

RAW SCORE _____

Spelling

Items 1–6: About 10 seconds to begin writing and as much time as needed to complete the response
Items 7–50: About 15 seconds

If the child scores 0 on any of the first 5 items administered, administer preceding items in reverse sequence until child scores 1 on each of 5 consecutive items

6 consecutive scores of 0

	Item	Notes	Reg.	Irreg.	Hom.	Score 0 or 1
5–6	1.					
	2.					
	3.					
	4.					
	5.					
	6.					
7–9	7.					
	8.					
	9.					
	10.					
	11.					
	12.					
	13.					
	14.					
	15.					
10–12	16.					
	17.					
	18.					
	19.					
	20.					
13+	21.					
	22.					
	23.					
	24.					
	25.					

Item	Notes	Reg.	Irreg.	Hom.	Score 0 or 1
26.					
27.					
28.					
29.					
30.					
31.					
32.					
33.					
34.					
35.					
36.					
37.					
38.					
39.					
40.					
41.					
42.					
43.					
44.					
45.					
46.					
47.					
48.					
49.					
50.					

Max=50

RAW SCORE

Reading Comprehension

About 15 seconds for each item

If the child scores 0 on any of the first 5 items administered, administer preceding items in reverse sequence until child scores 1 on each of 5 consecutive items

4 consecutive scores of 0

	Item	Response	Score 0 or 1
5–8	1.What does the bird do?		
	2. Why is the girl sad?		
	3. What do the people want to do?		
	4. What does the girl want to do?		
	5. Why was the dog running?		
	6. Whose book did the cat sit on?		
	7. When did the lion laugh?		
	8. What is in the box?		
9–10	9. What did Lee see first?		
	10. What animal is this story about?		
	11. Why did the milk fall down?		
11+	12. What animal is this story about?		
	13. What makes this boat different?		
	14. Why did Mr Clark want a second job?		
	15. Which dog has the same name as a cat?		
	16. What will probably happen at the next game?		
	17. What in the popcorn makes it pop?		

CONTINUE

Reading Comprehension

About 15 seconds for each item

If the child scores 0 on any of the first 5 items administered, administer preceding items in reverse sequence until child scores 1 on each of 5 consecutive items

4 consecutive scores of 0

Item	Response	Score 0 or 1
18. What will probably happen next?		
19. What do the Mexican Indian women do to their hair that men do not?		
20. How can you get your body to burn fat while you sleep?		
21. Why are tigers rarely studied in the wild?		
22. What makes one flute sound different from another?		
23. Why should you be prepared before you begin assembling the model?		
24. Why was the warden sleeping when the phone rang?		
25. How did cardamom come to Europe?		
26. What is likely to happen to the lemurs?		
27. Why have efforts to stop dumping been unsuccessful?		
28. Before sulphur is heated with rubber, what is done to make the rubber stronger?		

CONTINUE →

Reading Comprehension

About 15 seconds • for each item

If the child scores 0 on any of the first 5 items administered, administer preceding items in reverse sequence until child scores 1 on each of 5 consecutive items

4 consecutive scores of 0

Item	Response	Score 0 or 1
29. What is likely to happen when prices decrease?		
30. How are mammals and saurians different?		
31. According to the passage, what happens before cloth is made?		
32. What is the most likely reason for the changes in the prices of peaches during the year?		
33. Why is Jellinek's disease receiving more attention?		
34. When are you most likely to remember a dream?		
35. How was the innocence of the accused established?		
36. Why is Hawaii the only state in America to produce coffee commercially?		
37. What word or phrase in this sentence is a trope?		
38. Explain why a string of beads and a rubber band are examples of concatenation or synthesis.		

Max=3

RAW SCORE

Spelling

Name .....

1. .....
2. .....
3. .....
4. .....
5. .....
6. .....
7. .....
8. .....
9. .....
10. .....
11. .....
12. .....
13. .....
14. .....
15. .....
16. .....
17. .....
18. .....
19. .....
20. .....
21. .....
22. .....
23. .....
24. .....
25. .....
26. .....

Spelling

Continued

27. .....

28. .....

29. .....

30. .....

31. .....

32. .....

33. .....

34. .....

35. .....

36. .....

37. .....

38. .....

39. .....

40. .....

41. .....

42. .....

43. .....

44. .....

45. .....

46. .....

47. .....

48. .....

49. .....

50. .....

WORD
WECHSLER OBJECTIVE READING DIMENSIONS

Appendix E

Revised Children's Manifest Anxiety Scale (RCMAS)

"WHAT I THINK AND FEEL"

(RCMAS)

Cecil R. Reynolds, Ph.D., and Bert O. Richmond, Ed.D.

Published by
WESTERN PSYCHOLOGICAL SERVICES
wps® 12031 Wilshire Boulevard
Los Angeles, CA 90025-1251
Publishers and Distributors

Name: _____

_____

Age: _____ **Grade:** _____

Sex ***(circle one):*** Girl Boy

Today's Date: _____

School: _____

Teacher's Name ***(optional):*** _____

_____

DIRECTIONS

On the back of this form, there are some sentences that tell how some people think and feel about themselves. Read each sentence carefully. Circle the word *Yes* if you think the sentence is true about you. Circle the word *No* if you think it is *not* true about you. Circle an answer for every sentence, even if it is hard to choose one that fits you. Do not circle both *Yes* and *No* for the same sentence. If you want to change an answer, draw an X through your first answer and then circle your new choice.

There are no right or wrong answers. Only you can tell us how you think and feel about yourself. Remember, after you read each sentence, ask yourself, "Is it true about me?" If it is, circle *Yes*. If it is not, circle *No*.

W-199D

"WHAT I THINK AND FEEL"
(RCMAS)

Cecil R. Reynolds, Ph.D., and Bert O. Richmond, Ed.D.

Published by
WESTERN PSYCHOLOGICAL SERVICES
wps®
12031 Wilshire Boulevard
Los Angeles, CA 90025-1251
Publishers and Distributors

SCORING INSTRUCTIONS

1. Refer to the scoring grid on the other side of this page. To the right of every item for which *Yes* is circled, place a check mark in each of the corresponding boxes. If both *Yes* and *No* are circled for any item, and neither response is crossed out, exclude that item.
2. Tally the number of check marks you have entered in each column and record these column totals in the spaces provided at the bottom of the scoring grid. The column totals are the final raw scores for the five scales of the RCMAS.
3. Transfer the raw scores you have obtained to the appropriate spaces in the "Raw Score" column below.
4. In the RCMAS Manual, refer to the "Scoring" section of chapter 2 and to Appendixes A through F. Using the norms tables found in the appendixes, locate the percentile and *T*-score that correspond to the Total Anxiety raw score, and the percentile and scaled score that correspond to the raw score for each of the four subscales. Record these values in the spaces provided below.

RCMAS Scale	Raw Score	Percentile	*T*-Score or Scaled Score
Total Anxiety	_____	_____	_____
I. Physiological Anxiety	_____	_____	_____
II. Worry/Oversensitivity	_____	_____	_____
III. Social Concerns/Concentration	_____	_____	_____
Lie	_____	_____	_____

W-199D

			Total Anxiety	I. Physiological Anxiety	II. Worry/ Oversensitivity	III. Social Concerns/ Concentration	Lie
Yes	No	1.	☐	☐			
Yes	No	2.	☐		☐		
Yes	No	3.	☐			☐	
Yes	No	4.					☐
Yes	No	5.	☐	☐			
Yes	No	6.	☐		☐		
Yes	No	7.	☐		☐		
Yes	No	8.					☐
Yes	No	9.	☐	☐			
Yes	No	10.	☐		☐		
Yes	No	11.	☐			☐	
Yes	No	12.					☐
Yes	No	13.	☐	☐			
Yes	No	14.	☐		☐		
Yes	No	15.	☐			☐	
Yes	No	16.					☐
Yes	No	17.	☐	☐			
Yes	No	18.	☐		☐		
Yes	No	19.	☐	☐			
Yes	No	20.					☐
Yes	No	21.	☐	☐			
Yes	No	22.	☐		☐		
Yes	No	23.	☐			☐	
Yes	No	24.					☐
Yes	No	25.	☐	☐			
Yes	No	26.	☐		☐		
Yes	No	27.	☐			☐	
Yes	No	28.					☐
Yes	No	29.	☐	☐			
Yes	No	30.	☐		☐		
Yes	No	31.	☐			☐	
Yes	No	32.					☐
Yes	No	33.	☐	☐			
Yes	No	34.	☐		☐		
Yes	No	35.	☐			☐	
Yes	No	36.					☐
Yes	No	37.	☐		☐		
	Raw Scores ▶		_____ Total Anxiety	_____ I. Physiological Anxiety	_____ II. Worry/ Oversensitivity	_____ III. Social Concerns/ Concentration	_____ Lie

Circle one answer for each sentence.

Yes	No	1. I have trouble making up my mind.
Yes	No	2. I get nervous when things do not go the right way for me.
Yes	No	3. Others seem to do things easier than I can.
Yes	No	4. I like everyone I know.
Yes	No	5. Often I have trouble getting my breath.
Yes	No	6. I worry a lot of the time.
Yes	No	7. I am afraid of a lot of things.
Yes	No	8. I am always kind.
Yes	No	9. I get mad easily.
Yes	No	10. I worry about what my parents will say to me.
Yes	No	11. I feel that others do not like the way I do things.
Yes	No	12. I always have good manners.
Yes	No	13. It is hard for me to get to sleep at night.
Yes	No	14. I worry about what other people think about me.
Yes	No	15. I feel alone even when there are people with me.
Yes	No	16. I am always good.
Yes	No	17. Often I feel sick in my stomach.
Yes	No	18. My feelings get hurt easily.
Yes	No	19. My hands feel sweaty.
Yes	No	20. I am always nice to everyone.
Yes	No	21. I am tired a lot.
Yes	No	22. I worry about what is going to happen.
Yes	No	23. Other people are happier than I.
Yes	No	24. I tell the truth every single time.
Yes	No	25. I have bad dreams.
Yes	No	26. My feelings get hurt easily when I am fussed at.
Yes	No	27. I feel someone will tell me I do things the wrong way.
Yes	No	28. I never get angry.
Yes	No	29. I wake up scared some of the time.
Yes	No	30. I worry when I go to bed at night.
Yes	No	31. It is hard for me to keep my mind on my schoolwork.
Yes	No	32. I never say things I shouldn't.
Yes	No	33. I wiggle in my seat a lot.
Yes	No	34. I am nervous.
Yes	No	35. A lot of people are against me.
Yes	No	36. I never lie.
Yes	No	37. I often worry about something bad happening to me.

Appendix F

Strengths and Difficulties Questionnaire (SDQ)

Strengths and Difficulties Questionnaire

T4-16

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 the child's behaviour over the last six months or this school year.

Child's Name ..... Male/Female

Date of Birth .....

	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 tempers	☐	☐	☐
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	☐	☐	☐

Do you have any other comments or concerns?

Please turn over - there are a few more questions on the other side

Appendix G (i)

University of Southampton ethical approval

University of Southampton

Department of Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ
United Kingdom

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

21st July 2000

[redacted]

Dear [redacted],

Re: Application for Ethical Approval

I am writing to confirm you that your ethical application titled "ADHD children's response to delay, and delay-threatening words" has been given approval by the departmental ethics committee.

Should you require any further information, please do not hesitate in contacting me on (023) 80 593995.

Yours sincerely,

[redacted]

[redacted]
Ethical Secretary

DEPARTMENT OF PSYCHOLOGY

OUTLINE OF PROPOSED RESEARCH TO BE SUBMITTED FOR ETHICAL APPROVAL

PLEASE NOTE: You will need to discuss this form with your Supervisor. In particular, you should ask him/her for any departmental guidelines relating to this area of research which you must read and understand. You should also read and understand the Ethical Principles for Conducting Research with Human Participants published by the British Psychological Society. ***You must not begin your study until ethical approval has been obtained. Failure to comply with this policy will affect the viability of your research***

To obtain ethical approval it may take up to one week for undergraduates and up to two weeks for staff and postgraduates.

1. Name(s): Sharon Pettit
   Supervisor: Professor Edmund Sonuga-Barke

2. How may you be contacted?
   - Department of Clinical Psychology
   - By post to [redacted]
   - By telephone at home [redacted] (including answerphone)

3. Into which category does your research fall?

Year 1 Practical	
Year 2 Practical	
Year 3 Project	
Intercalated Medical	
MSc Ed Psy	
MSc/Diploma Health	
DClin Psy	X
PhD Research	
Intercalated Medical Student	
Staff Research	

4. Provisional Title of Project:
   ADHD children's response to delay, and delay-threatening words.

5. ANSWER THE FOLLOWING QUESTIONS.
Give full details where necessary.

a) What are the aims, hypothesis or research questions of this project?
To support the delay-aversion theory of ADHD. It will do this in three ways. First, a behavioural delay task previously used by Sonuga Barke et al (1992) will identify children who are concerned to reduce overall delay. Second, clinical populations will be used who have presented to Child and Family Services in Swindon, and who have received a diagnosis of either pure ADHD, Conduct Disorder, or Co-morbid. Third, a revised dot-probe task will determine whether ADHD children find the list of delay-associated words more threatening than neutrals or socially- and physically-threatening words.

b) What measurement procedures will be employed?
(If a questionnaire/test protocol/structured interview is to be used, a copy should be attached).
(1) 4 subscales from the Wechsler Intelligence Scale for children WISC-R
(2) Wechsler Objective Reading Dimension
(3) Strengths and Difficulties Questionnaire (Goodman, 1997) for home and school
(4) Revised Children's Manifest Anxiety Scale (Reynolds & Richmond, 1978).

If a standard questionnaire is to be used, have you obtained permission to duplicate this questionnaire or purchased sufficient copies?" Yes

c) Who are the participants?
Four groups of 20 boys between ages 8 and 11. Three groups will be clinical samples who have received a diagnosis of either ADHD, Conduct Disorder, or Comorbid. The fourth group will be normal controls between 8 and 11.

d) How will they be recruited?
(1) From existing clients at Swindon Child and Family Service where a weekly ADHD clinic takes place.
(1) From a local mainstream school.

e) If participants are under the responsibility of others (such as teachers, nurses or medical staff) have you obtained permission for the participants to take part in the study? YES

f) Is there reason to believe participants will experience discomfort during your study?
No

g) How will you obtain the consent of participants?
Parents will sign a consent form after reading an information sheet. Children will be asked whether they want to participate. For controls, teachers will be asked for their assistance in contacting parents of children in their class.

h) How will it be made clear to participants that they may withdraw consent to participate at any time?
It is written in the parents' information sheet and consent form. It will be verbally stated to children.

i) Will the procedure involve deception of any sort? NO

j) Do you propose to debrief participants and/or provide them with information about the findings of your study?

YES. They will receive an information sheet before the study on which it states they can request details of findings. If they so, I will send them an A-4 sheet with brief details of results at a later stage.

k) How will information obtained from or about participants be protected?
Participants will be kept anonymous. Details will be locked in a secure office at a clinical psychology department. Data Protection Act rules will be applied.

l) Experimental apparatus employed must be approved for safety by Martin Hall or Bryan Newman. Has this approval been given?
Yes, Martin Hall is assisting with provision of computer hardware and software.

m) Do you intend to make a submission to the medical ethical committee?
(Certain projects may need medical ethical approval, please check with your supervisor)

Yes, to Swindon Medical Ethics Committee.

6. Outline any other information you feel relevant to this submission.
None.

I endorse the following statement: "I confirm that I have a copy of, have read and understand the Ethical Principles for Conducting Research with Human Participants published by the British Psychological Society".

Signature(s) [redacted]

If you have received additional written guidelines from your supervisor please endorse the statement; "I have received, read and understood departmental ethical guidelines issued to me by my Supervisor relating to this work"

Signature(s) [redacted] Date 20.6.00

7. To be completed by the Supervisor

Do you foresee any ethical problems with this research? YES/NO

If YES, please detail.

Signature of Supervisor [redacted] Date 20/6/00

8. Ethical Authorisation given by

Name(s) .....

Signature(s) .....

Date .....

9. If not Authorised, give reason for transmission to Full Ethics Committee

10. Decision of Full Ethics Committee

11. Points to be noted at the end of year meeting of the Ethics Committee

When full approval has been given, please pass this form to the Ethics Committee Secretary in the Psychology Department General Office (room 4041).

Appendix G (ii)

NHS ethical approval (Swindon)

Wiltshire NHS
Health Authority

Ref: kp GF SW 24/2000

9 September 2000

Southgate House
Pans Lane
Devizes
Wiltshire
SN10 5EQ

Tel: 01380 728899
Fax: 01380 722443
DX 121831
www.healthywiltshire.org.uk

[redacted]

Dear [redacted]

SW 24/2000 **(this number must be quoted on all correspondence)**
ADHD Children's Response to Delay and Delay-Threatening Words

Thank you for your recent letter clarifying the points of concern which were raised by the Committee and confirming that the amendments will be made. I also note the addition to the protocol and confirm that this is approved. I confirm that this study may now proceed.

Any changes or extensions to the protocol, or additional investigators, should be notified to the Committee for approval. Adverse events should also be reported to the Committee. May we remind you of the Data Protection Act 1984, and the need to conduct the trial in accordance with the Good Clinical Practice guidelines.

The Committee is required to audit progress of research, and to produce a yearly report to the Wiltshire Health Authority and Department of Health. You are therefore required to provide a brief yearly report and a short final report.

The Swindon Research Ethics Committee is fully compliant with the International Conference on Harmonisation/Good Clinical Practice (ICH) Guidelines for the Conduct of Trials Involving the Participation of Human Subjects and undertakes to adhere to the relevant clauses of the guidelines for clinical practice adopted by the European Union in January 1997.

Yours sincerely

[redacted]

pp [redacted] (Mr)
Chairman - Swindon Research Ethics Committee

Appendix G (iii)

NHS ethical approval (Salisbury)

Wiltshire NHS
Health Authority

SL KP SA 43/2000

18 December 2000

Southgate House
Pans Lane
Devizes
Wiltshire
SN10 5EQ

Tel: 01380 728899
Fax: 01380 722443
DX 121831
www.healthywiltshire.org.uk

[redacted]

Dear Ms Pettit

SA 43/2000 *(This number must be quoted in all correspondence)*
ADHD Children's Response to Delay and Delay-Threatening Words

At its meeting on 29 November 2000 the Salisbury Research Ethics Committee received your letter dated dated 7 November 2000 in response to our letter of 2 October. This study was now approved subject to you confirming that on the Information Sheet for Head Teachers a paragraph is inserted stating that taking part is not compulsory.

Any changes or extensions to the protocol, or additional investigators, should be notified to the Committee for approval. Adverse events should also be reported to the Committee. May we remind you of the Data Protection Act 1984, and the need to conduct the trial in accordance with the Good Clinical Practice guidelines.

The Committee is required to audit progress of research, and to produce a yearly report to the Wiltshire Health Authority and Department of Health. You are therefore required to provide a brief yearly report and a short final report.

The Salisbury Research Ethics Committee is fully compliant with the International Conference on Harmonisation/Good Clinical Practice (ICH) Guidelines for the Conduct of Trials Involving the Participation of Human Subjects and undertakes to adhere to the relevant clauses of the guidelines for clinical practice adopted by the European Union in January 1997.

Yours sincerely

[redacted] (Dr)
Acting Chairman - Salisbury Research Ethics Committee

Appendix H (i)

Letter of introduction to parents of normal control children

University of Southampton

Department of Psychology

Doctoral Programme in Clinical Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ
United Kingdom

Telephone +44 (0)23 8059 5321
Fax +44 (0)23 8059 2588
Email

Dear parent/guardian,

A research study investigating why some children with Attention Deficit Hyperactivity Disorder (ADHD) or Conduct Disorder (CD) find it difficult to wait. Boys between 8 & 12 years are required.

As part of my doctoral degree in clinical psychology, I am conducting a research study that looks at how some children respond to delayed situations. Professor Edmund Sonuga-Barke (University of Southampton) is supervising the project. I am writing to ask if you would be prepared to give permission for your child to take part, along with about 80 others. **Your son will enable us to look at how children without these difficulties approach these tasks.** The Swindon Research Local Ethics Committee has reviewed the study.

He will be required to complete four puzzles, read a short passage, answer a few questions, and then perform 2 easy computer games which should take no longer than 75 minutes. He will suffer no discomfort.

Results from this study will shed light on the impulsivity, inattention and hyperactivity shown by some children. Information will be stored on a computer - none of the children's names will be used.

You or your child can withdraw permission for involvement at any time.

If you agree for your son to take part, please indicate by signing and returning the enclosed consent form and questionnaire. I will be at _____ school from ___to __, when I will carry out the study. If you agree, your child's teacher will also be asked to complete a questionnaire.

Please let me know if you have any queries, require further information, or wish to know the results of the study. I can be contacted on [redacted] Tuesday-Thursdays.

Many thanks for your help.
Yours sincerely,

[redacted]

Att.

Appendix H (ii)

Letter of introduction to parents of AD/HD and comorbid children

University of Southampton

Department of Psychology

Doctoral Programme in Clinical Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ
United Kingdom

Telephone +44 (0)23 8059 5321
Fax +44 (0)23 8059 2588
Email

Dear parent/guardian,

A research study investigating why some children with Attention Deficit Hyperactivity Disorder (ADHD) or Conduct Disorder (CD) find it difficult to wait. Boys between 8 & 12 years are required.

As part of my doctoral degree in clinical psychology, I am conducting a research study that looks at how some children respond to delayed situations. Professor Edmund Sonuga-Barke (University of Southampton) and Dr. Dick Eyre, (Consultant Psychiatrist, Swindon Child and Family Consultation Service) are supervising the project. I am writing to ask if you would be prepared to give permission for your child to take part, along with about 80 others. The Swindon Research Local Ethics Committee has reviewed the study.

He will be required to complete four puzzles, read a short passage, answer a few questions, and then perform 2 easy computer games, which should take no longer than 75 minutes. He will suffer no discomfort. **If your child is on medication for ADHD, it is important that he be drug-free on the day of testing.**

Results from this study will shed light on the impulsivity, inattention and hyperactivity shown by some children. Information will be stored on a computer - none of the children's names will be used.

You or your child can withdraw permission for involvement at any time. It will not affect any service you might receive from Marlborough House.

If you agree for your child to take part, please indicate by signing and returning the enclosed consent form and questionnaire in the stamped, addressed envelope provided. I will then contact you to arrange participation either at home, or at school with school's permission. If you agree, your child's teacher will also be asked to complete a questionnaire.

Please let me know if you have any queries, require further information, or wish to know the results of the study. I can be contacted on [redacted] Tuesday-Thursdays.

Many thanks for your help.
Yours sincerely,

[redacted]

Att:

Appendix H (iii)

Letter of introduction to parents of ODD/CD children

University of Southampton

Department of Psychology

Doctoral Programme in Clinical Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ
United Kingdom

Telephone +44 (0)23 8059 5321
Fax +44 (0)23 8059 2588
Email

Dear parent/guardian,

A research study investigating why some children with behaviour difficulties find it difficult to wait. Boys between 8 and 12 years of age are required.

As part of my doctoral degree in clinical psychology, I am conducting a research study that looks at how children respond to delayed situations. Professor Edmund Sonuga-Barke (University of Southampton) is supervising the project. I am writing to ask if you would allow your child to take part, along with about 80 others. The Swindon Research Local Ethics Committee has reviewed the study.

Your son will be asked to do four puzzles, read a short passage, answer a few questions, and then perform 2 easy computer games, which should take no longer than 75 minutes.

The results will help us understand more about why some children seem to be impulsive, find it difficult to pay attention, or seem overactive. Information will be stored on a computer - none of the children's names will be used.

If you agree for your son to take part, please sign and return the enclosed consent form and questionnaire in the stamped, addressed envelope provided. I will then arrange to meet your son, probably at Stratton Education Centre. If you agree, your child's teacher will also be asked to complete a questionnaire.

Please let me know if you have any queries, require further information, or wish to know the results of the study. I can be contacted on [redacted] Tuesday-Thursdays.

Many thanks for your help.
Yours sincerely,

[redacted]

Encs:

Appendix I

Informed Consent Form

Consent Form

Children's response to delayed situations

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 _____

School address _____

Teacher's name _____

Please complete the following:

	Please circle either yes or no
I have read the information sheet	Yes / No
I know who to contact if I have further questions, or want to discuss the study	Yes / No
I have received satisfactory answers to any questions I had	Yes / No
I have received enough information about the study	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 from Marlborough House/ Stratton Education Centre	Yes / No

Signed _____ Date _____

Please return to Sharon Pettit in the envelope provided.

Appendix J (i)

Letter of introduction to headteacher and teacher of normal control children

University of Southampton

Department of Psychology

Doctoral Programme in Clinical Psychology

University of Southampton
Highfield
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SO17 1BJ
United Kingdom

Telephone +44 (0)23 8059 5321
Fax +44 (0)23 8059 2588
Email

Dear Headteacher and Class teacher,

A research study investigating why some children with Attention Deficit Hyperactivity Disorder (ADHD) or Conduct Disorder (CD) find it difficult to wait. Boys between 8 & 12 years are required.

As part of my doctoral degree in clinical psychology, I am conducting a research study that looks at how children respond to situations of delay. Professor Edmund Sonuga-Barke (University of Southampton) is supervising the project.

I am writing to ask for your assistance in enabling us to look at how **boys without these difficulties approach some tasks.** This will involve your help in a number of ways:

- Circulating the enclosed letters to parents of boys in your class. Attached is a copy of what we are sending to parents (together with a return envelope) in order to receive their consent.
- Once parental consent has been obtained, you will be asked to complete a Strengths and Difficulties questionnaire for each child (example enclosed).
- I also wonder if it is possible for me to use the school for testing purposes. Children will be required to complete four puzzles, read a short passage, answer a few questions, and then perform 2 easy computer games on a portable computer supplied by me, which should take about 75 minutes per child. Times and dates will obviously be scheduled to suit your convenience. Taking part is not compulsory.

Results from this study will shed light on the impulsivity, inattention and hyperactivity shown by some children. Information will be stored on a computer - none of the children's names will be used.

If you have any queries or require further information, I can be contacted on the above telephone number.

Many thanks.
Yours sincerely,

[redacted]

Encs:

Appendix J (ii)

Letter of introduction to headteacher and teacher of children with AD/HD and comorbid

University of Southampton

Department of Psychology

Doctoral Programme in Clinical Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ
United Kingdom

Telephone +44 (0)23 8059 5321
Fax +44 (0)23 8059 2588
Email

Dear Headteacher and Class teacher,

A research study investigating why some children with Attention Deficit Hyperactivity Disorder (ADHD) or Conduct Disorder (CD) find it difficult to wait. Boys between 8 & 12 years are required.

As part of my doctoral degree in clinical psychology, I am conducting a research study that looks at how children handle delay. Professor Edmund Sonuga-Barke (University of Southampton) and Dr. Dick Eyre (Consultant Psychiatrist, Swindon Child and Family Consultation Service) are supervising the project. The Swindon Local Research Ethics Committee has reviewed the study.

I have received parental permission for_____to take part in the study, whom I believe is a student in your class. I wonder if you could kindly complete and return the enclosed questionnaire which will give us valuable information. Mr(s) ___ _____ is aware that I am approaching you and has given consent.

I also wonder if it is possible for me to use the school for testing purposes. _____ _____ will be required to complete some puzzles, read a short passage, answer a few questions, and then perform two easy computer games on a portable computer supplied by me, which should all take about 75 minutes. I will telephone you shortly to discuss this further. Taking part is not compulsory.

Results from this study will shed light on the impulsivity, inattention and hyperactivity shown by some children. Information will be stored on a computer - none of the children's names will be used.

If you have any queries or require further information, please let me know. I can be contacted at Marlborough House (Tel No.) on Tuesdays - Thursdays.

Many thanks for your help.
Yours sincerely,

[redacted]

Encs.

Appendix J (iii)

Letter of introduction to headteacher and teacher of children with ODD/CD

University of Southampton

Department of Psychology

Doctoral Programme in Clinical Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ
United Kingdom

Telephone +44 (0)23 8059 5321
Fax +44 (0)23 8059 2588
Email

Dear (Head teacher),

A research study investigating why some children with Attention Deficit Hyperactivity Disorder (ADHD) or Conduct Disorder (CD) find it difficult to wait. Boys between 8 & 12 years are required.

I am writing to ask if you would assist me with a Doctoral research study as part of my Clinical Psychology training. I am nearing completion, and need access to **boys between ages 8 and 12 years** who have conduct difficulties (without ADHD). Most of my participants so far have come from Marlborough House referrals, and they have been selected because they have a degree of ADHD. In addition, 20 children without difficulties have participated. However, I am struggling to identify boys **with** conduct problems, yet **without** ADHD symptoms, and I wonder if you can help. Taking part is not compulsory.

My project is looking at the behaviour of children under situations of delay. It is being supervised by Professor Edmund Sonuga-Barke (University of Southampton), and has been facilitated by Dr. Dick Eyre (Consultant Psychiatrist, Swindon Child and Family Consultation Service). The Swindon Research Local Ethics Committee has reviewed the study.

There is a standard procedure. Parental consent is sought first, together with children's consent. Children are then required one at a time, to complete four puzzles, read some words, answer a few questions, and then perform two computer games, which takes about 75 minutes. Results will shed light on the impulsivity, inattention and hyperactivity shown by some children.

I hope you can help, and I will telephone you in the next couple of days to discuss further.

Many thanks.
Yours sincerely,

[redacted]
[redacted].

Appendix K

Introductory instructions given to participants

My name is Sharon Pettit. I work as a clinical psychologist. As part of my job I am doing a project about some children who find it difficult to wait for things, and I wonder if you will help me. Your parent(s) have already said they are happy for you to help if you want to.

You can help by answering some questions, doing a few puzzles which most children enjoy, and doing two easy computer games. All these tasks will take between an hour and an hour-and-a-quarter. Your name, answers and scores will be kept confidential, which means they will only be seen by me and other people working on the project at my college.

You don't have to take part if you don't want to. Even if you say yes, and do some tasks, you can stop at any time if you change your mind. Do you understand? Have you any questions to ask me? Would you like to take part?

(If no – discontinue).

If yes – let's start. Stop me if you need a break, or if you decide you no longer want to continue. OK?