PROFESSIONAL DOCTORATES

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Ann Lesley Goodson

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Abstract

Attention Deficit Hyperactivity Disorder (ADHD) is a neurobiological disorder traditionally thought to be a disorder of middle childhood. The evidence to date suggests that the contribution of genetic factors is more substantial than that of environmental influences. No one factor is thought to cause ADHD, however, it is accepted that many environmental variables contribute to it and there are many predictable clinical features accompany it.

This thesis reviews the literature regarding ADHD in middle childhood. In addition, it will focus on the growing body of evidence suggesting behavioural, social, familial and academic difficulties are present in preschool children. Symptoms of ADHD have also been shown to persist into adulthood ADHD will be examined in terms of classification, prevalence, developmental course, comorbidity and underlying aetiology. These factors will then be compared and contrasted with ADHD in preschool, adolescence and adulthood.

The empirical paper explores whether similarities in parent and child behaviour influences parental empathetic responding. To investigate this, 152 parents completed measures of current and retrospective ADHD, parenting style, empathic responding, aggression and general mental health for themselves and ADHD screens for their child. The results suggest that mothers with higher symptoms of ADHD, experience lower levels of personal distress when dealing with their ADHD child compared with mothers with low symptoms of ADHD.

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Attention Deficit Hyperactivity Disorder: A Developmental Perspective.

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Key Words: Attention Deficit Hyperactivity Disorder, Preschool children, Adult, Genetics, Aetiology, Environment

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Abstract

Attention Deficit Hyperactivity Disorder is a neurobiological disorder traditionally thought to be a disorder of middle childhood. The evidence to date suggests that the contribution of genetic factors is more substantial than that of environmental influences. No one factor is thought to cause ADHD, however, it is accepted that many environmental variables contribute to it. The literature regarding ADHD in middle childhood will be reviewed, in addition, it will focus on the growing body of evidence suggesting behavioural, social, familial and academic difficulties are present in preschool children and that many symptoms of ADHD have been shown to persist into adulthood. ADHD will be examined in terms of classification, prevalence, developmental course, comorbidity and underlying aetiology. These factors will then be compared and contrasted with ADHD in preschool, adolescence and adulthood.

There is much debate regarding the development of Attention Deficit Hyperactivity Disorder (ADHD). Whilst traditionally ADHD has been conceptualised as a disorder of middle childhood, there is a growing body of evidence suggesting behavioural, social, familial and academic difficulties are present in preschool children (DuPaul, McGoey, Eckert & VanBrackle (2001). There is also literature to suggest comorbid disruptive disorders in this group of children (Keenan & Wakschlag, 2000). In addition, symptoms of ADHD have been shown to persist into adulthood; research has demonstrated that forty percent of children diagnosed with ADHD will continue to meet the criteria for ADHD as adults (Fischer, Barkley, Fletcher & Smallish, 1993a). Many of these adults also display several of the characteristics associated with childhood ADHD, in that they exhibit problems in their interpersonal relationships and in the areas of vocation and cognition (Biederman, Faraone, Knee & Munir, 1990; Dinn, Robbins & Harris, 2001; Murphy & Barkley, 1996; Schweitzer et al., 2000). In this current review, ADHD in middle childhood will be examined in terms of classification, prevalence and developmental course. Comorbidity and underlying aetiology will also be discussed. These factors will then be compared and contrasted with ADHD in preschool, adolescence and adulthood.

ADHD in childhood

ADHD is the diagnostic term used to describe children who are incapable of appropriately modulating attention, impulse control and motor activity. It is a "persistent pattern of inattention and/or hyperactivity" observed more frequently and severely than in a typically developing age-equivalent child. The Diagnostic Statistical Manual of Mental Disorders – Fourth edition (DSM-IV, APA, 1994) lists three specific sub-types of ADHD; inattentive type, predominantly hyperactive-

impulsive type and combined type. In addition, there is the classification of "ADHD not otherwise specified". This is used to describe behavioural presentations that do not meet the full diagnostic criteria for one of the three subtypes of ADHD. The DSM-IV lists 18 symptoms that are classified against two primary areas. The first – inattention, lists carelessness with detail, failure to sustain attention, the appearance of not listening, not finishing instructed tasks, poor self-organisation, avoidance of tasks that require sustained mental effort, losing things, easily distracted and appearing forgetful. Hyperactivity-impulsivity is defined to include fidgeting, leaving seat (when should be seated), excessive and inappropriate running/climbing, noisy in play, persistent motor overactivity unmodified by social context, blurting out answers to questions before question is completed, failure to wait in queues or take turns, interrupting conversations or games and talking excessively in a social context.

Inattention is observed in poor selective attention (attending to irrelevant or distracting stimuli and ignoring relevant stimuli), and lack of sustained attention i.e. the inability to maintain attention over time (Cosgrove, 1997). Children with ADHD are easily distracted and avoid tasks that involve sustained attention over long periods. They also appear to be unable to contain their activity in situations that require them to do so. Hyperactivity, in the context of ADHD, is indicated by increased motor activity. These children are described as moving excessively; they are permanently "on the go" and do not sit still. In addition, they cannot play quietly and often fiddle with their hands or feet. Impulsivity is defined by a lack of control in conditions that require focussed attention. Children with the disorder tend to act without thinking and perform poorly due to a lack of foresight and an absence of planning; they do not give sufficient thought to the effects of their behaviour. These children often find it

difficult to delay gratification of their needs (Schweitzer & Sulzer-Azaroff, 1995) and to resist temptation (Hinshaw, Simmel & Heller, 1995).

ADHD typically begins early in life. It is characterised by developmentally inappropriate behaviours such as observable higher activity levels (Teicher, Ito, Glod & Barber, 1996). Children with ADHD talk more than other children do, either to themselves (Berk & Potts, 1991) or to others (Barkley, Cunningham & Karlsson, 1983). Impairing symptoms must be present for at least 6 months and be apparent by the age of seven for inclusion criteria. Primary impairments must be present in at least two social contexts (e.g. home and school), and there must be an adverse effect on developmentally appropriate achievement in social and academic situations. Exclusions include symptoms occurring within the course of a Pervasive Developmental Disorder, Schizophrenia or other Psychotic disorder. It must also not be better accounted for by an alternative disorder.

Epidemiological studies employing stringent diagnostic criteria suggest that approximately 5% of school age children from a general population meet the diagnostic criteria for ADHD (Scahill & Schwab Stone, 2000, APA, 1994), and it is one of the most common forms of psychopathology in childhood (Ross & Ross, 1982). The prevalence of ADHD in males compared to females is disproportionate; within the general population, ADHD is observed in males at four times the rate for females. This rate increases to nine times in those referred to clinical services (APA, 1994). However, differences in the presentation of the disorder in females may help to explain this gender difference.

It is possible that ADHD occurs because of multiple biologically based aetiological factors (Slomka, 2000). The evidence to date suggests that the contribution of genetic factors is more substantial than that of environmental influences. Heritability estimates in middle-childhood average approximately 70% (Eaves et al., 1997; Goodman & Stevenson, 1989; Rhee, Waldman, Hay & Levy, 1999). Exposure to various early central nervous insults e.g. perinatal and neonatal hypoxia obstetric trauma, intrauterine exposure to infection, (Barkley, 1990); early childhood heavy metal exposure (Thomson et al., 1989); premature birth or low birth weight (Cantwell, 1996) further predisposes some individuals to the development of ADHD. It is because multiple neural systems contribute to attentional functioning and behavioural self-regulation that even slight developmental disruptions can lead to dysfunction of attention.

Comorbidity

More severe forms of ADHD have been associated with psychiatric comorbidity. Szatmari, Offord & Boyle (1989), report that approximately 44% of children with ADHD have a comorbid disorder, almost a third have two comorbid disorders and approximately a tenth have three comorbid disorders. Goldstein & Goldstein (1998) report that CD and ODD are the most common comorbid disorders observed in children with ADHD. In addition, family genetic studies have demonstrated significant rates of CD and affective disorders in children with ADHD and in their families compared with control families (Biederman et al., 1992; Fergusson, Horwood & Lynskey, 1993).

The disruptive behaviour disorders such as ODD and CD are the most comprehensively investigated of the comorbid conditions. ADHD is categorised along with these disorders in DSM-IV. The overlap amongst these disorders is often so extensive that it has been questioned as to whether hyperactivity is synonymous to both ADHD and CD. ODD and CD are found in up to two thirds of children with ADHD. In their longitudinal study, Morrell & Murray (2003) compared the development of children of depressed mothers with children of well mothers. They found that for boys, there was an interaction between 4-month emotional and behavioural dysregulation and rejecting and coercive parenting predictive of emotional and behavioural dysregulation at 9 months.

Coercive parenting may have an adverse influence on attentional processes in infants, the result of which is poor engagement and poor attention/distractibility. This occurs through the intrusive hindering, rather than aiding of the infant's own self-initiated interest in the environment, leading to impaired attentional development (Morrell & Murray, 2003). In addition, it could also be that coercive parenting is affectively aversive, resulting in the development of avoidance and poor attention/distractibility as a learned response to cognitive demands. This theory is supported by data from experimental studies that report that memory processes were impaired in infants at times when learning occurred in the context of infant distress (e.g. Fagan, Ohr, Fleckenstein & Ribner,1985).

Anxiety and mood disorders are found in over 25% of children with ADHD. Adjustment disorder, resulting in dysthymia or major depression may occur because of impaired adaptation to residual, undiagnosed or untreated ADHD symptoms

(Slomka, 2000). The co-occurrence of anxiety disorders needs careful differential diagnosis. Internalising disorder symptoms are more common in the inattentive subtype of ADHD, neuropsychological evaluations are often appropriate for these individuals in order to rule out alternative competing diagnoses.

In summary, no single causal factor has been identified with ADHD, however it is accepted that many environmental variables contribute to it and many predictable clinical features are associated with the disorder. ADHD exists along a continuum of other childhood developmental disorders whereby primary symptoms are observed early in childhood. It is also widely accepted that children diagnosed with ADHD are at increased risk for high rates of comorbid mental health problems, medical difficulties, disrupted familial and peer relationships, academic failure, lower educational attainment and poor work histories (Liebson, Katusic, Barbaresi, Ransom & O'Brien, 2001; Barkley, 1998; Hinshaw, 1994). ADHD is thought to be independent of general intellectual ability and is therefore prevalent in children of all abilities. There is however, an apparent association between ADHD and specific learning disorders (Slomka, 2000).

Subtypes

The inattentive subtype of ADHD is more difficult to detect. This is because the external structure of preschool tends to mollify the expression of symptoms. It is primarily during middle childhood that the demands for self-directed learning increase. There are also greater expectations regarding the amount and the complexity of the academic work required. Children with the inattentive subtype of

ADHD are also more likely to develop internalising behaviour disorders, adjustment disorder problems or both (Biederman, 1999).

Gender differences in the rates of disruptive behaviour disorders are well documented in both clinically referred and community-based samples (Eme & Kavanaugh, 1995). Research has shown that boys manifest aggressive and antisocial actions up to ten times more often than girls (Offord et al., 1987). Comorbidity between ADHD and ODD / CD has also been found to be significantly higher in boys (Faraone et al., 1993). These disorders, in boys have an earlier age of onset and a greater persistence than for girls (Zoccolillo, 1993).

During infancy, the sex differences are minimal; however, from age 5 years onwards, there is an increasing divergence in the frequency of girls' and boys' problem behaviours (Prior, Smart, Sanson & Oberklaid, 1993). Biederman et al. (1999) report that girls are more likely than boys to have the predominantly inattentive type of ADHD; they are also more likely to have more mood and anxiety features in their presentation than are boys. They further suggest that girls may be at a greater risk for developing a substance abuse disorder. However, they are at less risk for comorbid depression, CD and ODD than boys (Biederman et al., 2002).

Many studies of children with subtypes of ADHD have identified the multiple features that distinguish the different subgroups. Dykman & Ackerman (1993) report that children with predominantly hyperactive-impulsive behaviours typically demonstrate more problems related to impulse control and aggressive-defiant characteristics (externalising behaviour disorder characteristics). In comparison,

children who exhibit ADHD of the inattentive type, exhibit more over-anxious, depressed, dysthymic and interpersonal withdrawal symptoms (internalising behaviour disorder characteristics). Barkley (1994) suggests that there is a dissociation between ADHD subtypes that implies that the inattentive subtype may represent a primary deficit in the focus-execute dimensions of attention, whereas the hyperactivity subtype may represent primary deficits in response-inhibition and the ability to sustain focussed or prolonged attention. Therefore, in the subtypes of ADHD, there appears to be a dissociation along the dimensions of both cognition and behaviour.

Genetics

In an attempt to discover genes linked to ADHD, much of the genetic research has focussed on the components of the dopamine system. Studies demonstrating a greater concordance in monozygotic (MZ) than dizygotic (DZ) twins suggest a genetic basis for ADHD. Payton et al. (2001) examined sets of MZ twins and found a trend for an increase in the frequency of the dopamine receptor DRD4 7-repeat allele. The DRD4 has been related to novelty seeking (Benjamin, Patterson, Greenberg, Murphy & Hamer, 1996; Epstein et al., 1996). Individuals with high levels of the novelty-seeking personality trait are impulsive, exploratory, excitable and quick tempered, which when found in excess are also symptoms of ADHD (Faraone et al., 1999). Rowe et al. (1998) examined the relation of the 7-repeat allele to a questionnaire-based diagnosis of ADHD. They found positive findings for the ADHD inattentive subtype. They also found that the 7-repeat allele occurred more frequently in children with the inattentive subtype of ADHD than in controls. They further report that for

the ADHD combined subtype, there was a higher frequency of the 7-repeat allele than in controls (Rowe et al., 1998).

Using a cross-sectional twin design Rietveld, Hudziak, Bartels, Van Beijsterveldt & Boomsma (2003) found that genetic influences on attention problems and overactive behaviour were high across an age span of 3-12 years. Levy, Hay, McStephen, Wood & Waldman (1997) report that MZ twins showed similar ADHD symptom counts across the full range of severity. Furthermore, siblings of hyperactive children are approximately twice as likely as siblings in the general population to have the disorder. It is also possible for one sibling to have predominantly hyperactivity symptoms whilst the other has predominantly inattentive symptoms. Biological parents of children with ADHD have a higher risk for the disorder than do adoptive parents and relatives of individuals with ADHD are six times more likely to have ADHD (Biederman et al., 1992). It is worthy of note that when ADHD is comorbid with CD in a child, there are a higher proportion of alcohol use disorders and antisocial personality disorders in the parents than is found in the general population (Biederman et al., 1992).

These studies are consistent in demonstrating a ratio of genetic to environmental contributions to individual differences of between 3:1 and 4:1 (Barkley, 1997). Gillis, Gilger, Pennington & DeFries (1992) conducted twin research whereby one member of each twin pair (37 MZ and 37 same sex DZ)) had been diagnosed with ADHD. Seventy nine percent of MZ twins compared to 32% of DZ twins were concordant for ADHD. Further evidence comes from Sherman, McGue & Iacono, (1997) who studied MZ and DZ male twins and found that concordance for ADHD ranged from

53% to 67% (as rated by teachers and mothers respectively) in MZ twins compared to 0% to 37% (as rated by teachers and mothers respectively) in DZ twins.

Neurobiology

Although considerable progress has been made regarding the neurobiology of ADHD, the precise aetiology is still not completely understood. It is generally thought that an inherited abnormality of cerebral dopamine functioning underlies most cases. The most common aspect is thought to be an abnormality of the CNS dopamine receptors (Hill, 2000). Converging evidence from studies of neuropharmacology, genetics, neuropsychology and neuroimaging suggest an involvement of fronto-striatal circuitry in ADHD, central to which is poor inhibitory control and deficits in the front-striatal circuitry. However, there is evidence to suggest that more posterior cerebral areas may also be implicated in the disorder (Durston, 2003).

FMRI studies have revealed anterior cingulated cortex dysfunction in ADHD by using a Stroop test. It is recognised that the anterior cingulated cognitive division (ACcd) plays a central role in attentional processing by modulating stimulus selection and/or mediating response selection. Bush et al. (1999) used FMRI and a counting Stroop to demonstrate that individuals with ADHD failed to activate the ACcd during the counting Stroop, supporting the hypothesis of ACcd dysfunction in ADHD.

Data from EEG studies of children with Combined Type ADHD have demonstrated elevated levels of slow wave activity and decreased fast wave activity (Clarke, Barry, McCarthy & Selikowitz, 2002). They report increased theta with deficiencies of beta

activity (indicating cortical hypoarousal) and elevated levels of beta activity, which may represent hyperarousal.

Using functional analysis of event-related potential data, Smith, Johnstone & Barry, (2003), found that for children aged 8-12 years, it was possible to separate those children with ADHD from normal controls with an accuracy of 73%.

Neuropsychology

Psychological accounts of ADHD have traditionally characterised ADHD as either a neuro-cognitive disorder of regulation or a motivational style. Underlying dysregulation is thought to be due to poor inhibitory control. Neuropsychological studies are united in the view that ADHD is associated with problems of executive dysfunction (EDF, Barkley, Grodinsky & DuPaul, 1992). Children with ADHD lack attentional and strategic flexibility. They demonstrate poor planning and working memory and are unable to effectively manage their behaviour (Cepeda, Cepeda & Kramer, 2000; Clarke, Prior & Kinsella, 2000). Although these difficulties are observed in other disorders (Bradshaw & Sheppard, 2000), the primary role of deficient inhibitory control observed in this pattern of dysregulation appears to distinguish ADHD from other disorders (Barkley, 1997a; Bayliss & Roodenrys, 2000; Ross, Harris, Olincy & Radant, 2000). Evidence to support this has come from studies using the "stop signal" paradigm (Schachar & Logan, 1990). This paradigm assesses an individual's ability to inhibit an already initiated pre-potent response to a (typically visual) "go signal" when signalled to do so by a (typically auditory) "stop signal". Research has demonstrated that children with ADHD have a flatter probability of inhibition slope and longer stop signal reaction time (Schachar, Mota,

Logan, Tannock & Klim, 2000; Nigg, 1999). These effects have been reported to be of moderate to large size and to be consistent across clinical samples (Kuntsi & Stevenson, 2000).

Whilst these models of disinhibition and dysregulation tend to dominate the current literature, an alternative hypothesis has moved away from the dominant neuro-psychological paradigm (Sonuga-Barke, 1994). The Delay aversion (DEL) hypothesis states that ADHD behaviours are thought to be the dominant characteristic of motivational style (Sonuga-Barke, 2002). Research on DEL in ADHD has focussed on these children's unwillingness to wait for delayed rewards and events. It appears that under many circumstances, children prefer immediate rather than large delayed rewards (Kuntsi, Oosterlaan & Stevenson, 2001). It has been demonstrated however, (Sonuga-Barke, Williams, Hall & Saxton, 1996), that preference for immediacy is only observed in certain circumstances and there appears to be a double dissociation between preference for delayed rewards and inhibitory control (Sonuga-Barke et al., 1996).

More recently, it has been suggested that DEL and poor inhibitory control are independent co-existing characteristics of Combined Type ADHD (Sonuga-Barke, 2002). These two models (EDF and DEL) have traditionally been thought of as competing, however, it has been suggested that they are in fact complimentary accounts of two psycho-patho-physiological subtypes of ADHD. The two models are hypothesised to have different developmental pathways highlighted by different cortico-striatal circuits and modulated by different branches of the dopamine system (Sonuga-Barke, 2003).

This alternative hypothesis suggests a dual pathway model of ADHD that recognises combined type ADHD as having two separate sub-types distinguishable by their underlying psychological mechanisms, in terms of the context dependant nature of their symptoms, their cognitive motivational profile, both their genetic and non-genetic origins and environmental factors. One type is reported as a dysregulation of thought and action pathway (DTAP) because of poor inhibitory control associated with the meso-cortical branch of the dopamine system that projects in the cortical control centres such as the pre-frontal cortex. This core dysfunction in inhibitory control causes both behavioural symptoms and poor quality task engagement. The appearance of ADHD symptoms is mediated by behavioural dysregulation; effects on task engagement are mediated by cognitive dysregulation. These can be observed in the pattern of difficulties seen in children with ADHD when engaged in tasks that require attentional flexibility, behavioural monitoring, planning and working memory (Sonuga-Barke, 2002). In this model, there is no direct pathway between executive functions and symptoms of ADHD.

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Fig. 1. The Dual Pathway Model of AD/HD. The left-hand pathway represents AD/HD as EDF. The right-hand pathway represents AD/HD as a delay averse motivational style. Reproduced from Sonuga-Barke, 2003).

The second pathway is the motivational style pathway (MSP). This pathway provides an alternative route to ADHD. DEL is not seen to be the core characteristic of the motivational pathway. Instead, it is an acquired characteristic mediating the link between behavioural symptoms, task engagement and a more basic biologically based alteration in reward mechanisms (Johansen, Aase, Meyer & Sagvolden, 2002). It is hypothesised that the link between ADHD behaviours and altered reward mechanisms is mediated by the emergence over time of a generalised aversion to delay (Sonuga-Barke, 2002). The MSP is linked to the meso-limbic dopamine branch associated with the reward circuits such as the nucleus accumbens. The dual pathway model of ADHD predicts that DTAP ADHD will be context independent. It is associated with relatively severe and generalised cognitive dysregulation; it is categorical in nature and less strongly associated with genetic factors. Alternatively, MSP ADHD is context dependent. It has a more limited pattern of cognitive impairment associated

with the provision, protection and utilisation of time. MSP ADHD is likely to be more closely associated with a continuously distributed trait and more closely associated with genetic factors (Sonuga-Barke, 2002).

Environmental influences on ADHD

Childhood ADHD may have an environmental influence observed by an association between hyperactive symptoms, harsh parental discipline and coercive parenting, for ADHD behaviour (Woodward, Taylor & Dowdney, 1998; DuPaul et al., 2001).

Although parenting alone is not considered a significant aetiological factor in the development of ADHD, there is evidence to suggest that negative parenting, in addition to other factors, may compound the developmental risk for the development of the disorder (Barkley, 1990).

Research has revealed that children diagnosed with ADHD are twice as likely to come from single-parent families, 1.5 times more likely to live in an urban area and more than 3 times more likely to be in receipt of public assistance (Szatmari, Offord & Boyle, 1989). Scahill et al. (1999) report an association between psychosocial hardship and ADHD consistent with findings from previous studies (Biederman et al., 1995; Szatmari et al., 1989). They found that higher levels of family dysfunction, low income, overcrowded living conditions and a maternal history of psychiatric treatment were associated with greater ADHD symptom severity. They further report that when adjustments were made for the presence of other variables, only male gender, low income and family dysfunction remained significantly associated with ADHD. This is supported by recent research by Biederman, Faraone & Monuteaux (2002), who

examined the effect of gender as a mediator in the association between environmental adversity and the risk of ADHD in children aged between 6 and 17 years. They found that psychosocial adversity; in particular, low social class, maternal psychopathology and family conflict increased the risk for ADHD in both sexes.

Further evidence for an environmental effect has come from an observed reduction in ADHD symptoms following parent training. Parent training has been associated with both improved child behaviours and with an increase in targeted parent behaviours, for example, a reduction in the use of indirect commands (Barkley, Guevremont, Anastopoulos & Fletcher, 1992a; Anastopoulos, Shelton, DuPaul & Guevremont, 1993). Furthermore, Pelham, Wheeler & Chronis (1998) evaluated psychosocial treatments for ADHD in children and adolescents and concluded that behavioural parent training and behavioural interventions in the classroom are effective in their effect on ADHD.

In summary, ADHD is currently conceptualized as a neurobiological disorder (Faraone & Biederman, 1999; Tannock, 1998; Pliszka, McCracken & Maas, 1996, Voeller, 1991). The evidence to date suggests that the contribution of genetic factors is more substantial than that of environmental influences. ADHD is characterised by developmentally inappropriate behaviours such as observable higher activity levels (Teicher et al., 1996). More severe forms of ADHD have been associated with psychiatric comorbidity; approximately 44% of children with ADHD have a comorbid disorder (Szatmari, Offord & Boyle, 1989c). Currently, no one factor is thought to cause ADHD, however, it is accepted that many environmental variables contribute to it and many predictable clinical features are associated with the disorder. ADHD is

one of the most common psychopathologies in childhood (Ross & Ross, 1982). Even at a young age, children exhibiting high levels of inattention, impulsivity and hyperactivity are likely to experience a multitude of behavioural, social, and academic problems (DuPaul et al., 2001).

Having reviewed ADHD in middle childhood, the disorder in preschool children will now be reviewed. The deficits associated with ADHD in older children are pervasive across situations and have a detrimental effect on functioning in many areas. To understand the development of ADHD more fully, it is important to determine whether the difficulties observed in early childhood are similar in their presentation and affect (DuPaul et al., 2001).

ADHD in Preschool Children

Research has shown that impulsive, hyperactive and inattentive behaviours are often observed in young children (Burns, Walsh & Owen, 1997). When extreme, these behaviours have a significant effect on functioning both at home and at school (Hinshaw, 2002). Factor analysis of data from children as young as three has revealed that symptoms of inattentiveness, hyperactivity and impulsiveness cluster (Sonuga-Barke, Thompson, Stevenson & Viney, 1997; Fantuzzo et al., 2001).

Prior to the onset of ADHD symptomatology, young children may demonstrate premorbid behavioural characteristics such as excessive motor activity, behavioural irritability and difficulty in establishing a consistent sleep pattern in infancy (Slomka, 2000). Although not specific to the development of ADHD, these children may be at greater risk if these characteristics continue into the preschool years and there is the

emergence of additional cognitive and behavioural problems. By the age of 3-4, there is an identifiable behavioural phenotype; there are observable difficulties maintaining age-appropriate behavioural self-regulation, excessive motor activity and a limited capacity to adapt to unexpected changes in the environment (Slomka, 2000). When these children enter the educational system, there are greater demands placed on them to conform to rule governed behaviour. For those children who are predisposed to ADHD, a reduced ability to sustain attention results in the expression of an increased number of overt symptoms. It is at this stage that other behavioural disorders or academic difficulties may become apparent because of the increased demand for self-directed learning, independence and autonomy.

Little is known of the validity of the diagnosis of any type of ADHD among preschool aged children. Lavigne, Gibbons & Christoffel, (1996) found that 2% of 2-5 year olds met DSM-III-R diagnostic criteria for ADHD. This was supported by Keenan, Shaw, Walsh, Delinquadri & Giovannelli (1997), who reported that 6% of 5 year olds (from a low-income family community sample), met criteria for DSM –III-R ADHD. Campbell (1994), observed a group of 3-year-old children, identified by their parents as having high ratings of hyperactivity or oppositional behaviour; half the sample still exhibited behaviour problems at age six. It appears that ADHD can be identified in preschool and primary school aged children and that the behaviours exhibited are stable for up to three years (Lahey, Pelham, Stein, et al, 1998). Lahey et al. further report that children who met criteria for each subtype of ADHD (as rated by their teachers) were less popular with their peers and those who met criteria for combined type were reported as being more actively disliked by more of their classmates. Teachers also perceived these children as less prosocial, less cooperative and less

assertive than controls. Lahey et al. found that teachers perceived children who met criteria for the hyperactive-impulsive subtype, but not inattentive subtype, as being more disruptive and less controlled than their age equivalent classmates. Those children who met criteria for each subtype of ADHD also self-reported significantly more problems in friendships than controls. Furthermore, Lahey et al report that standardised testing revealed an academic underachievement in mathematics in those children who met criteria for combined and inattentive subtypes of ADHD. It is likely that young children with ADHD are already behind their age-equivalent classmates in basic mathematics, pre-reading skills and fine motor skills when they enter the education system (Lahey et al., 1998; Mariani & Barkley, 1997; Shelton et al., 1998).

DuPaul et al., (2001) examined behavioural, social, pre-academic and medical functioning in a sample of preschool children with ADHD in comparison with a normative sample. They found that young children with ADHD demonstrated significantly more problem behaviours and were less socially skilled than the control group as rated by parent and teachers. DuPaul et al. further reported group differences between parent and teacher ratings of internalising behaviours such as social withdrawal and anxiety. DuPaul suggests that these differences may indicate that young children with ADHD display multiple behaviour problems across areas of both internalisation and externalisation; although adult perceptions of child behaviour may be negatively biased because the stress associated with parenting a child with ADHD is high (DePaul et al., 2001). DuPaul notes that parents of children with ADHD report higher stress levels associated with both the child's behaviour and dysfunctional interactions. In addition, parent-child interactions were reported as

being more problematic for the children in the ADHD group. In comparison with the control group, these children demonstrated more than twice the level of non-compliance and more than five times the level of inappropriate behaviour when asked to complete tasks by their parents (DuPaul et al., 2001). The parents of the children with ADHD were also observed to exhibit more negative behaviours towards their child, compared to the control group counterparts. Only minimal group differences were found during interactions in low adult attention situations, suggesting that escape from parent-directed tasks was the motivator for non-compliance rather than attention seeking (DuPaul et al., 2001).

Within the preschool classroom setting, a higher frequency of negative social behaviour has been observed in preschool children with ADHD compared to controls (DuPaul, 2001). This is more evident during unstructured free-play activities. DuPaul further highlighted behavioural, social, familial and academic difficulties in pre-school children compared to controls. This is in line with the findings of other research which have reported deficits in working memory (Mariani & Barkley, 1997); mother child disruption, (Barkley, 1998); and comorbid disruptive disorders (Keenan & Wakschlag, 2000), suggesting that pre-academic deficits are exhibited prior to formal school entry. Screening for the disorder among younger children with behavioural difficulties would therefore be beneficial for the prevention of the development of further behavioural difficulties and potential developmental deviance (Brook, Tseng & Cohen, 1996) and comorbid psychopathology (Post, 1992) and would enable evaluation of poor social behaviour, parental stress and family coping problems.

Wilens et al. (2002) evaluated the clinical characteristics, Psychiatric comorbidity and functioning of preschool children referred to a paediatric psychiatric clinic for assessment. They found that referred preschool aged children had distinct similarities to referred school-aged children with regards their ADHD presentation, psychiatric comorbidity and associated impairment. Preschool-aged children were reported as having an earlier onset of their ADHD and comorbid psychopathology than did their school-aged counterparts. Furthermore, preschool children had a similar symptom presentation of ADHD and despite their younger age, they demonstrated prominent academic, social and general dysfunction, as did the older school-aged group with ADHD.

Comorbidity

According to Jenson, Martin & Cantwell (1997), comorbidity may be identified in the preschool years. There is evidence to suggest that the symptoms observed in school age children who develop comorbid disorders such as CD/ODD alongside ADHD are similar to those symptoms displayed during their preschool years (Barkley, 1998; Huesmann & Moise, 1999; Rutter, Giller & Hagell, 1998; Stevenson & Goodman, 2001). There is considerable evidence for comorbidity of ADHD with other disorders, in particular those associated with the antisocial oppositional behaviours; thus mirroring the situation in the school years (Wilens et al., 2002). Early age of onset of ADHD symptoms is associated with higher rates of comorbidity with CD and more severe symptoms and disability by the age of 8-9 years (McGee, Feehan, Williams & Anderson, 1992; Sanson, Smart, Prior & Oberklaid, 1993). A study by Lavigne et al. (2001) reports that preschool children with ODD are likely to continue to exhibit the disorder, with increasing comorbidity with ADHD.

Genetics

There is currently a lack of data on the behavioural manifestations of hyperactivity across development. For this reason, behaviour genetic studies have not yet been possible in infancy (Morrell & Murray 2003).

Neuropsychology

The data on the neuropsychological characteristics of ADHD in the preschool population is limited (Sonuga-Barke, 2003). Current models tend to focus on either cognitive or motivational factors. Barkley (1997a) suggests that ADHD is the result of executive dysfunction, whilst Sagvolden, Aase & Zeiner, (1998) suggest that ADHD is linked to disturbances in motivational processes. The dual-pathway model of Sonuga-Barke (2002) proposes a combination of executive dysfunction and delay aversion. This has been supported with research with school-aged children (Solanto, Abikoff & Sonuga-Barke, 2001). However, little is known about executive function and ADHD in the preschool years. Research by Mariani & Barkley (1997), has demonstrated that preschool ADHD is associated with problems of working memory. More recently, the dual pathway model was tested on a group of children aged between 3 and 5½ years of age (Dalen, Sonuga-Barke & Remington, in press; Sonuga-Barke, Dalen & Remington, 2003).

Preschool children described as "hard to manage" (at risk of developing hyperactivity) have been reported to demonstrate deficits in executive functioning characterised by poor inhibition or planning and responses, in addition to difficulties in emotional regulation and antisocial behaviour (Dunn & Hughes, 2001; Hughes, Dunn & White, 1998; Hughes, White, Sharpen & Dunn, 2000). These studies support

the neuropsychological similarities of school-aged and preschool ADHD and provide further support for the similarity of preschool and school-aged ADHD.

Environmental influences on ADHD in preschool children

As with childhood ADHD, increased problems with comorbidity appear to be related to adverse psychosocial stressors. Atypical maternal-child interactions in addition to disruptive, aggressive social behaviours in preschool settings have been associated with ADHD (Barkley, 1998).

Psychosocial intervention programmes have provided evidence for the influence of the environment on ADHD. Sonuga-Barke, Daley, Thompson, Lever-Bradbury & Weeks, (2001) evaluated two different parent-based therapies (parent training and parent counselling) for parents of pre-school children displaying a preschool equivalent of ADHD. Parents in the training group were taught child-management techniques; the counselling group received non-directive support. At fifteen-week follow-up, only parents in the training group reported reduced symptoms of ADHD. More recently, Bor, Sanders & Markie-Dadds, (2002) found that parent training programmes produced significant reductions in children's co-occurring disruptive behaviours and attentional/hyperactive difficulties. Bor et al. report that 80% of children demonstrated clinically reliable improvement in observed negative behaviour from pre-intervention to follow-up.

Twin studies have further been able to provide information regarding the influence of environmental factors in the development of ADHD. Results have been consistent in finding that the shared environment contributes little, if any, explanation to individual

differences in the trait underlying hyperactive, impulsive and inattentive ADHD. The shared environment typically accounts for less than 5% of the variance among individuals with the disorder (Levy et al., 1997). These environmental factors include social class, and family educational/occupational status, the general home environment, family nutrition, common or shared parental and child-rearing characteristics and apply across all children in the family (Pike & Plomin, 1996).

Morrell & Murray (2003) however, report a link between environment -x - environment interactions and early ADHD. They found that symptoms of hyperactivity were associated with distinct patterns of early parenting that were strongly influenced by infant gender, whereby emotional dysregulation was predicted by a fragile temperament and rejecting and coercive parenting styles. Morrell & Murray propose that such data is indicative of a critical period in early infancy for the development of symptoms of hyperactivity in boys. This is supported by the findings of Biederman, Faraone & Monuteaux (2002) who report that although higher levels of environmental adversity were associated with an increased risk for ADHD and other comorbidity (independent of gender), gender modified the risk for adverse cognitive and interpersonal outcomes, i.e. boys were more vulnerable to the disorder than girls. Just as the shared environment is an important factor in the development of ADHD so is the non-shared environment. An example of this is the unique relationship experienced between a child and his/her parent. Expressed emotion (EE) is a measure that is often used to assess the parent-child relationship among adults and more recently among children with psychological disorders. There is evidence to suggest that caregiver's high scores on EE are associated with the presence of externalising and internalising disorders in children (Daley, Sonuga-Barke & Thompson 2003;

Asarnow, Tompson, Woo & Cantwell, 2001; Stubbe, Zahner, Goldstein & Leckman, 1993; Vostanis, Nicholls & Harrington, 1994). The underlying assumption regarding EE is that the way parents talk about a child is indicative of the way they treat them on a daily basis (Chambless, Bryan, Aiken, Steketee & Hooley, 1999). In a study of boys with ADHD, Marshall, Longwell, Goldstein & Swanson, (1990), found that parents' overall EE towards their child was a good predictor of parents' coercive processes and their negative affective style in interactions. More recently, Daley, Sonuga-Barke & Thompson, (2003), found an increase of EE in the mothers of children with ADHD. They report that with the exception of EE - over-involvement, the remaining components were able to successfully discriminate between parents of preschool children with ADHD and parents of children without ADHD.

It could be that the heritability of hyperactivity in the first year of life is much lower than that observed in older children. This would suggest differential magnitudes of environmental influences at different ages (Morrell & Murray, 2003). Support for this has come from two longitudinal studies that demonstrate that quality of parenting in infancy is a predictor of subsequent hyperactivity. The Minnesota High Risk Project reported that the quality of care giving in infancy, in particular, intrusive maternal interactions, was a more powerful predictor of poor attention/distractibility at age 3-4, and hyperactivity at age 5-6 than was early biological or temperament factors (Carlson, Jocobvitz & Sroufe, 1995). In addition, Sanson et al., (1993) demonstrated that a hyperactive-aggressive group at age 8 had more difficult temperaments and behaviour from infancy, and had received less optimal early parenting environments than a normal control group.

More recently, the relationship between parenting coping styles, parent-child interactions and ADHD has been examined by McKee, Harvey, Danforth, Ulaszek & Friedman, (2004). McKee et al. report that maternal use of maladaptive and less adaptive coping styles was related to self-reported lax and over reactive discipline, more observable coercive parenting styles and more observable misbehaviour by the child.

The findings of Lahey et al., (1998) suggest that DSM-IV criteria for the subtypes of ADHD are valid for use with preschool and primary school children. The current data suggest that preschool children have a similar profile to their school-age counterparts and yet they tend to have a less robust response to treatment and a higher side-effect burden (Wilens & Spencer, 2000). It could be that the high rates of comorbidity within this group (particularly mood and anxiety disorders) lead to more complex cases associated with a lower response rate to methylphenidate and a higher side-effect burden reported with this age group (Wilens et al., 2002). However, there is much more research needed regarding the validity of the diagnosis of ADHD in younger children.

ADHD in Adolescence and Adulthood

Despite the assumption that ADHD primarily affects males and is outgrown, recent evidence suggests that ADHD often continues into adulthood with up to 60% of childhood cases still meeting diagnostic criteria as adults (Fischer et al., 1993). For this reason, there has been an increase in the research focussing on ADHD in adolescence and adulthood. This more current research has led to the notion that ADHD is a chronic disorder affecting both sexes (Barkley, 1998; Wender, 1995;

Biederman & Spencer, 2002) that continues into adolescence and often persists into adulthood (Gadow & Weiss, 2001).

A number of researchers have suggested that if childhood ADHD does persist into adulthood, then those adults should demonstrate similar behaviours to children with ADHD. Research has shown similar patterns of impairment in adults to that of their childhood counterparts – including impairments in interpersonal relationships, vocational activities and cognitive domains (Biederman et al., 1990; Dinn et al., 2001; Murphy & Barkley, 1996; Schweitzer et al., 2000). There also appears to be a similar neuro-pathology (Hesslinger, Thiel, Tebartz, Henning & Ebert, 2001; Muglia, Jain, Macciardi & Kennedy, 2000).

As children progress into adolescence there tends to be an improvement in overt symptoms of hyperactivity, impulsivity and inattention (Biederman et al., 2000), however, it does not appear that these declines continue to a level of normalization (Willoughby, 2003). ADHD disorders therefore characterize a class of conditions that affect early development and have the potential to have an adverse effect on social, educational and emotional adjustment in adulthood.

Willoughby (2003) reports that adolescents with ADHD experience many difficulties including increased risk for drug and alcohol problems, antisocial behaviour, peer and self-esteem problems and poor academic performance. Adults with a history of childhood ADHD also completed less education, received poorer marks and had been expelled from school more often in comparison to their same age counterparts (Young, Toone & Tyson, 2003; Biederman et al., 1993; Klein & Mannuzza, 1991). In

employment, adults with ADHD also experience more employment-related problems and reach lower occupational status compared to other family members (Mannuzza, Klein, Bessler, Malloy & LaPadula, 1993; Weiss, Hechtman & Perlman, 1978; Weiss, Hechtman, Perlman, Hopkins & Wener, 1979).

These findings support those of other longitudinal studies. Such studies report that children with ADHD are at an increased risk for many negative developmental outcomes in adolescence and adulthood (Barkley, Fischer, Edelbrock & Smallish, 1991; Barkley, Fischer, Smallish & Fletcher, 2002; Biederman et al., 1996; Fischer, Barkley, Fletcher & Smallish, 1990; Hansen, Weiss & Last, 1999; Liebson et al., 2001). These studies provide more detailed evidence concerning the educational, occupational and psychosocial risks associated with a childhood diagnosis of ADHD, in relation to both normal and clinical controls.

Biederman, Mick & Faraone, (2000) differentiated hyperactive, impulsive and inattentive symptom clusters and considered the developmental course of these symptoms as a function of age rather than "childhood versus adolescence". They found that hyperactivity, impulsivity and inattention significantly diminished with increasing age. This supported the findings of Fischer et al., (1993) who found that young people with ADHD exhibit a pronounced decrease in their hyperactive, impulsive and inattentive behaviours over time. However, adolescents with ADHD continue to exhibit significant hyperactive, impulsive and inattentive behaviours in comparison to normal controls at follow-up.

Comorbidity

There have been a small number of studies to support the suggestion that adult ADHD is associated with psychiatric comorbidity, cognitive deficits (including executive functioning), psychosocial impairments and patterns of treatment response that are similar to childhood ADHD (Biederman et al., 1993; Gallagher & Blader, 2001; Kinsbourne, DeQuiros & Tocci Rufo, 2001). The existence of these comorbid disorders and other alternative psychiatric disorders that share the symptoms of ADHD further confound the diagnosis of ADHD in adults. Shekim, Asarnow, Hess, Zaucha & Wheeler, (1990) report that in a sample of adults with ADHD, only 14% of the sample met DSM-III criteria for ADHD alone. Shekim et al. report that 53% of adults met the criteria for comorbid generalised anxiety disorder, 15% met criteria for comorbid panic disorder and 13% met criteria for comorbid obsessive-compulsive disorder. These high levels of comorbid disorders are further supported by Biederman et al. (1993). Biederman et al. report that 50% of their sample also met criteria for multiple anxiety disorder, 32% met criteria for social phobia, 30% for major depressive disorder and 25% met criteria for substance abuse disorder. Adults with ADHD are also more likely to suffer from cluster B personality disorders and substance use disorders, and have an over representation of mood, anxiety and somatoform disorders (Tzelepsis, Schubriner & Warbase, 1995).

Genetics

Genetic research has implicated the DRD4 gene in ADHD in childhood (Mill et al., 2003; DiMaio, Grizenco & Joober, 2003) and this has been observed in the VNTR polymorphism of DRD4 in adults (Muglia et al. 2000). Genotype analysis

demonstrated that the 7-repeat allele is significantly more common in adults with ADHD than in controls (Muglia et al. 2000).

Neurobiology

There have been a limited number of neuroimaging studies of ADHD with adults. Magnetic Resonance Imaging (MRI) studies (Hynd et al., 1991; 1993) reported reduced volume of the right frontal cortex, the corpus callosum and the left head of the caudate nucleus in individuals with ADHD. Zametkin et al. (1990) used PET to demonstrate reduced glucose metabolism in frontostriatal, somatosensory and occipital areas.

Evidence has also come from EEG studies that have demonstrated abnormalities in the areas of the brain associated with childhood ADHD (Bresnahan, Anderson & Barry, 1999; Bresnahan & Barry, 2002).

Neuropsychology

Neurological impairments found in children have also been observed in adults with ADHD (Epstein, Johnson, Varia & Conners, 2001; Woods, Lovejoy & Ball, 2002). Research has found neurological differences between DSM-IV ADHD subtypes in untreated adolescents with ADHD (Schmitz et al., 2002). They report that adolescents with predominantly inattentive subtype ADHD performed significantly worse on the Digit Span test and Stroop test than did controls. Adolescents with combined type ADHD were reported as performing more poorly on both the Digit Span and the Wisconsin Card-Sorting Test.

There have been several controlled investigations examining adult ADHD and response to medication. There appears to be a response to stimulant drugs (Wilens, Biederman, Spencer & Prince, 1995), with response rates reported as ranging from 25% (Mattes, Boswell & Oliver, 1984) to 78% (Spencer et al. 1995), in comparison with 70% response rate reported in children.

One of the major limitations of the current research is the reliance on self-report. There is evidence to suggest that young adults are often poor informants on their own hyperactive, impulsive, inattentive behaviours (Barkley et al., 2002). There is also evidence to suggest that some young adults, who self-report difficulties with attention, did not have hyperactive, impulsive, inattentive behaviours as children (Schaughency, McGee, Raja, Fehan & Silva, 1994). Furthermore, it is difficult to determine childhood onset of ADHD symptoms for adults in addition to determining whether simultaneous attentional/concentration problems are a result of continuing ADHD or a comorbid disorder (Roy-Byrne et al., 1997; Shaffer, 1994).

The continuation of ADHD symptoms beyond childhood emphasises the increasing need for the development of reliable and valid assessments for adults. An adult diagnosis of ADHD necessitates a childhood history strongly suggestive of the disorder, and assessment needs to focus not only on current symptomology but also any associated comorbidity and/or psychological factors.

Conclusions

Although there are numerous studies that have demonstrated that childhood ADHD is associated with an increased risk for a number of negative developmental outcomes,

we are yet to fully understand the developmental course of ADHD. The current available literature suggests that a significant number of children diagnosed with ADHD will have exhibited these symptoms during their preschool years and that they will continue to exhibit symptoms throughout adolescence and into adulthood. ADHD is one of the most common neurobiological disorders presenting for treatment in young people today (Wilens, Biederman & Spencer, 2002). ADHD often presents as a chronic disorder with observable symptoms and impairment continuing into adulthood. It is often associated with comorbid anxiety, mood and disruptive disorders. Substance abuse is a further risk factor. Evaluation of studies in both childhood and adult ADHD has led to the suggestion that ADHD should be viewed as a symptomatic continuity existing in childhood and continuing throughout the adult years (Biederman et al., 2000). Evidence of comorbidity from family/genetic studies, imaging studies and studies of treatment efficacy combine to point to ADHD as a disorder that spans the lifetime.

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Appendix G

SUBMISSION GUIDELINES FOR

CLINICAL PSYCHOLOGY REVIEW

The influence of parent-child behavioural similarity on parental empathetic responding.

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Abstract

Background: High levels of coercive and negative behaviours have been found to be a maintaining factor in childhood ADHD. As ADHD symptoms are likely to result in negative parenting practices, the parent-child relationships within families of children with ADHD is often conflictual and disturbed in nature. However, an observed interaction between child and maternal ADHD symptoms suggests that parent-child behavioural similarity may lead to higher levels of parental empathetic responding.

Method: One hundred and fifty two parents were recruited from a general population of schoolchildren. Parents completed measures of current and retrospective ADHD, parenting style, empathic responding, aggression and general mental health for themselves and ADHD screens for their child.

Results: Results confirm that both child and maternal ADHD symptoms increase the likelihood of negative parenting. The findings of this study also suggest that mothers with higher symptoms of ADHD, experience lower levels of personal distress when dealing with their ADHD child compared with mothers with low symptoms of ADHD.

Conclusions: This study has found that in ADHD, similarity in mother and child is related to less personal distress in parenting. It would therefore be beneficial to know whether this similarity in parent/child dyads also interact in other positive ways. Although there are many limitations to this study, the exploratory nature of the approach and the initial findings suggest that there is room for further research.

Introduction

Research indicates that Attention Deficit Hyperactivity Disorder (ADHD) occurs because of a marked genetic predisposition for an overactive temperament (Carr, 1999). Exposure to physical and psychosocial factors further adds a high environmental risk. The majority of studies document increased risk for ADHD among family members of children with ADHD, supporting the familiality of this disorder (Faraone & Doyle, 2000; Faraone, Biederman & Friedman, 2000a). However, the aetiology of ADHD in families is a complex interaction of genetic, environmental and gene-environment interactions (Biederman, Faraone & Monuteaux, 2002).

Evidence suggests that high levels of coercive and negative behaviours are a maintaining factor in childhood ADHD (DuPaul, McGoey, Eckert & VanBrackle, 2001). These parenting behaviours are mainly effects of the child's behaviour and are not considered a specific aetiological factor in the development of the disorder (Barkley, 1990). However, these parenting practices may increase the existing developmental risk by dampening down its expression, thus establishing the conditions associated with the development of comorbid antisocial and oppositional behaviour (Barkley, 1990).

ADHD symptoms and associated oppositional behaviour are likely to result in negative parenting practices (McKee, Harvey, Danforth, Ulsaszek & Friedman, 2004). Research has demonstrated the conflict ridden and disturbed nature of parent-child relationships within families with a child with ADHD (Morrell & Murray, 2003). Children with ADHD are less compliant to instructions from their parents, they are

unable to sustain their compliance for long periods and they remain on task for shorter periods than their non-ADHD same-age counterparts (Wells et al., 2000). Mothers and fathers of children with ADHD display more directive and commanding behaviour, assistance that is more physical, more disapproval, fewer rewards for prosocial behaviour and more overall negative behaviour towards their child than do parents of children without ADHD (Johnston & Mash, 2001; Barkley, Karlsson & Pollard, 1985; Befera & Barkley, 1984). Subsequent research on mother-child interactions in children with ADHD suggests that greater levels of conflict may occur in the subset of children who have the additional symptoms of oppositional defiant disorder (ODD, Gomez & Sanson, 1994; Johnston, 1996). There is also evidence to suggest that parental stressors, including marital conflict and parental psychopathology, can have an effect on parenting practices and are associated with dysfunctional discipline patterns and an increased children's externalizing symptoms (see Crnic & Low, 2002; Rubin & Burgess, 2002).

Research has examined the extent to which psychosocial variables can play a role in the development of ADHD. Carlson, Jocobvitz & Sroufe (1995) conducted a follow-up study of 191 children from birth to 11 years and found that maternal intrusive care and over-stimulation at 6 months and 42 months respectively, was a significant predictor of children's subsequent risk of hyperactivity during middle childhood. In addition observational research has consistently shown that mothers of children with ADHD are generally more negative and directive, and less responsive to child initiations than mothers of comparison children (Danforth, Barkley & Stokes, 1991; Mash & Johnston, 1982).

Further support has come from studies examining the association of expressed emotion (EE) and child psychopathology (McCarty, Lau, Valeri & Weitz, 2004; Daley, Sonuga-Barke & Thompson, 2003). Plomin (1994) argues that the effect of EE (i.e. its specificity to one child), may be more influential in determining psychopathology than shared factors. The role of parental EE in the development of psychopathology has been investigated in both clinical and community studies (Daley et al., 2003; Hibbs et al., 1991; Stubbe, Zahner, Goldstein & Leckman, 1993; Hirshfeld, Biederman, Brody, Faraone & Rosenbaum, 1997; Peris & Baker, 2000). Baker, Heller & Henker (2000) report a strong relationship between high EE in mothers and the extent of child behaviour problems. A follow-up of their study reported that preschool EE ratings predicted classification of ADHD four years later (Peris & Baker, 2000). Further more, Caspi et al. (2004) report that EE directed towards children plays a causal role in the development of psychopathology.

Further evidence for the influence of parenting deficits on ADHD symptoms comes from the psychosocial intervention literature. Research has demonstrated the efficacy of parent-based interventions with community-based samples for children with the pre-school equivalent of ADHD. Sonuga-Barke, Daley, Thompson, Laver-Bradbury & Weeks (2001) evaluated two different parent-based interventions. Parents were assigned to one of three groups, parent training (PT), parent counselling and support (PCS) or waiting list control (WL). The PT group received education regarding ADHD and were taught a wide range of behavioural strategies designed to increase attention and behavioural organization and to reduce defiant and difficult behaviour. Those in the PCS group were given the opportunity to discuss any concerns they may have regarding their feelings towards their child and the impact of their child's

behaviour on the family. Results indicated that mothers in the PT group had an increased sense of well being and their children displayed a decrease in their ADHD symptoms compared to the PCS and WL groups.

More recently, Bor, Sanders & Markie-Dadds, (2002) report that both an enhanced behavioural family intervention (EBFI) comprising of three elements: parent training, partner support training, and coping skills training and a standard intervention program (Triple P – Positive Parenting Program, Sanders, 1999) were associated with positive outcomes for both parents and children in comparison to the WL group. Both interventions were associated with significantly lower levels of disruptive child behaviour (as reported by mothers) in comparison to the WL group. The EBFI was also associated with significantly less negative behaviour by the children. Both groups also demonstrated a significant improvement in parenting skills and satisfaction. Parenting interventions are therefore being viewed as being as essential as early intervention programmes for children (Bor et al., 2002).

Research into the influence of exposure to parental ADHD in either the transmission of the disorder or its developmental course is still in its infancy. Due to the heritability of the disorder (see Smalley et al., 2000), it is more likely that children with ADHD will have a parent with the disorder. Symptoms of ADHD in a parent are likely to create a chaotic living environment, which in turn is more likely to impact upon subsequent child development (Weiss, Hechtman, &Weiss, 2000).

However, it is possible that the effect of exposure to parental ADHD will be smaller for children with ADHD as compared to children without ADHD (Biederman et al.,

2002). Recent research by Biederman et al (2002) found that ADHD in parents was associated with an increased risk for ADHD in children, regardless of whether or not symptoms in the parents were active. They further found that exposure to ADHD in parents had a differential effect on high-risk offspring, which was contingent on the presence or absence of ADHD in the child, in that, having a parent with active ADHD symptoms did not add additional risk for dysfunction to any offspring with the disorder beyond that associated with ADHD. However, having a parent with active symptoms of ADHD did detrimentally affect children without the disorder (Biederman et al., 2002).

More recently, Psychogiou, Daley, Thompson & Sonuga-Barke, (Submitted), found that both maternal ADHD symptoms and child ADHD symptoms increase the likelihood of negative parental behaviour. However, they also report an interaction between child and maternal ADHD symptoms, whereby, the parenting of high ADHD mothers appeared to be far less affected by symptoms of ADHD in her child than did mothers reporting low ADHD symptoms. High levels of ADHD symptoms in mothers therefore appeared to "protect" children with ADHD against the risk of negative parenting (Psychogiou et al., Submitted). Psychologiou et al suggested that parent-child behavioural similarity might lead to higher levels of parental empathetic responding.

The current study attempts to examine whether the similarity between parental and child ADHD increases parental empathy. The specificity of this finding will also be tested in aggressive parents with children who have high levels of conduct disorder. It is predicted that when either the mother or father and child suffer high ADHD

symptoms, the high ADHD parent will be more empathic towards their high ADHD child. A second prediction is that when both mother/father and child suffer from high levels of aggression or conduct disorder symptoms, the aggressive parent will not be more empathic towards the child with high conduct disordered symptoms.

Method

Participants

One hundred and fifty two parents were recruited from a general population screen of 1297 parents of schoolchildren from the county of Conwy, North Wales, UK. Questionnaire packs were sent to parents of children aged between 5 and 12 years (M = 7.99; SD = 24.19). Returns were received for 152 children (male = 89, female = 63), giving a response rate of 12%.

Measures

For the purpose of this study, eight measures were sent to both parents. A demographic questionnaire provided information regarding gender, occupation and family structure as well as gender and age of the child. Parents completed the Strengths and Difficulties Questionnaire and the Attention Deficit Hyperactivity Disorder-Revised Scale about their child, they completed the Alabama Parenting Questionnaire, the Adult ADHD Rating Scale, The Balances Emotional Empathy Scale, the General Health Questionnaire, the Interpersonal Reactivity Index, the Aggression Questionnaire and the Wender Utah Rating Scale about themselves. The majority of returned questionnaires (96%); were completed by mothers.

1. **Strengths and Difficulties Questionnaire** (SDQ, Goodman, 1997, 1999).

The SDQ is a brief measure of the prosocial behaviour and psychopathology of 3-16 year-olds that can be completed by parents, teachers or youths. The psychometric properties of the five-factor structure (emotional, conduct, hyperactivity-inattention, peer, prosocial) for both the teacher and parent version have been confirmed. Internalising and externalising scales are relatively 'uncontaminated' by one another (Goodman, 2001). Reliability is satisfactory, as judged by internal consistency (mean Cronbach's alpha: 0.73). SDQ scores above the 90th centile also predict a substantially raised probability of independently diagnosed psychiatric disorders (mean odds ratio: 15.7 for parent scales, 15.2 for teacher scales, 6.2 for youth scales). The reliability and validity of the SDQ therefore make it a useful brief measure of the adjustment and psychopathology of children and youth.

2. **ADHD Rating Scale** (ADHD-RS, DuPaul, 1991)

The ADHD-RS consists of 18 items directly adapted from the ADHD symptom list according to the DSM-IV criteria (APA, 1994). Parents are required to select the single response that best describes their child and to indicate the frequency of each symptom on Likert scales ranging from "not at all" (0) to "very much" (3). Higher scores are considered indicative of greater ADHD-related behaviour. Responses on the ADHD-RS are compiled to yield a total score in addition to two subscale scores (Inattention and Hyperactivity-Impulsivity). The total score is computed as the sum of scores on all items; higher scores are indicative of greater symptom severity.

The ADHD-RS has demonstrated good psychometric properties including internal consistency and discriminative validity, (DuPaul, Power, Anastopoulos & Reid, 1998;

Kadesjo, Kadesjo, Haggloff & Gillberg, 2001; Magnesson, Smari, Gretarsdottir & Prandardottir, 1999). Research has shown its factor structure corresponds with DSM-IV diagnostic criteria, with well-supported inattention and hyperactivity-inattention factors (DuPaul et al., 1998; Magnesson et al., 1999). Internal consistency in this sample was measured using Cronbach's Alpha. A high level of internal consistency was found (Cronbach's $\alpha = .93$).

3. **Alabama Parenting Questionnaire** (APQ, Shelton, Frick & Wooten, 1996)

The APQ is a self-report measure for parents examining parenting behaviour practices. The APQ consists of six subscales: Involvement, Positive Parenting, Poor Monitoring/Supervision, Inconsistent Discipline and Corporal Punishment. Ratings of specific items are scored on a 5-point Likert scale ranging from 1 (never)-5 (always). The parent global report version has been found to be reliable, with generally adequate internal consistency (.67 - .80), apart from the CP subscale (.46) and adequate test-retest reliability (.66 - .89). Internal consistency was measured using Cronbach's Alpha. Internal consistency was found in this sample for Involvement (Cronbach's $\alpha = .76$), positive parenting (Cronbach's $\alpha = .77$), poor supervision (Cronbach's $\alpha = .63$), Inconsistent discipline (Cronbach's $\alpha = .79$) and corporal punishment (Cronbach's $\alpha = .61$).

4. **Adult ADHD Rating Scale** (AARS, Barkley & Murphy, 1998).

The AARS consists of 18 self-assessed items. It addresses symptoms listed in DSM-IV criteria, covering symptoms of inattentiveness (9 items), impulsiveness (3 items) and hyperactivity (6 items). Items are scored on a 0-3 Likert-style frequency scale (0

= rarely, 1 = sometimes, 2 = often, 3 = very often). The scale is reported to have good internal consistency and is able to predict concurrent ratings provided by spouse, parents and cohabiting partners regarding the participant (Edwards, Barkley, Laneri, Fletcher & Metevia, 2001; Murphy and Barkley, 1996; Murphy & Schachar, 2000).

5. **General Health Questionnaire** (GHQ, Goldberg, 1970)

The GHQ-12 is a brief measure of current mental health that has been extensively used in a number of different settings. The scale assesses whether the individual has experienced a particular symptom or behaviour recently. Each item on the GHQ is rated on a four-point scale (less than usual, no more than usual, more than usual, much more than usual). It yields a total score of 12 based on a bimodal scoring method (0-0-1-1). The GHQ has been reported to have good internal consistency (0.91) and good construct validity (McCabe, Thomas, Brazier & Coleman, 1996). Test-retest stability has been reported at 0.73 (Goldberg, 1972). Construct validity has been examined in both a GP setting and in a medical outpatient population.

6. **Interpersonal Reactivity Index** (IRI, Davis, 1983)

The IRI measures emotional and cognitive components of an individual's ability to empathise. Empathy is measured on four sub-scales: Perspective Taking (PT), the cognitive tendency to see things from another person's point of view, without necessarily demonstrating affective involvement, Empathic Concern (EC), the tendency to experience the affective reaction of sympathy and compassion for others, Personal Distress (PD), the tendency to experience personal feelings of distress and unease in reaction to another's distress and Fantasy Scale (FS, Davis, 1980; Davis, Luce & Kraus, 94). The FS was not used in this study. The IRI is measured using a

5-point Likert-type scale (0 = does not describe me well – 4 which equals describes me well). Higher scores on the IRI are indicative of greater levels of self-reported empathy. Research has demonstrated good construct validity (Bernstein, & Davis, 1982; Carey, Fox & Spraggins, 1988; Davis, 1983). The individual subscales have also been reported to have satisfactory internal reliability (range 0.71 – 0.77) and test retest reliability (range 0.62 – 0.80, Davis, 1980). Internal consistency for this study was measured using Cronbach's Alpha. Good internal consistency was found for PT (Cronbach's α = .74) and PD (Cronbach's α = .76).

7. **The Aggression Questionnaire** (AQ, Bryant & Smith, 2001).

The AQ is based on the 29-item, self-report aggression questionnaire of Buss & Perry (1992). It is 12-item measure of a respondent's self-perceived levels of aggression and anger. The AQ uses four scales of assessment: Physical aggression, Verbal aggression, Anger and Hostility. Generalizability for the British population has been confirmed (Archer, Holloway & McLoughlin, 1995; Goodness-of-fit = .93). Analyses have provided evidence in support of the model's construct validity and have demonstrated discriminant validity for the Hostility factor (Bryant & Smith, 2001).

8. **Wender Utah Rating Scale** (WURS, Ward, Wender & Reimherr, 1993).

The abbreviated WURS consists of a 25-item scale that can be used with adults to retrospectively diagnose ADHD. The WURS reflects the distinct features of ADHD in adults and includes the emotional aspects of the disorder. Scores on the WURS correlated with retrospective findings of childhood symptoms by the participants' mothers and were linked with positive response to stimulant medication in adults; supporting its validity (Ward et al., 1993a). Research has shown the WURS to have

excellent internal consistency (alpha + 0.91, Reitz-Junginger et al., 2003). The WURS is therefore sensitive in identifying childhood ADHD and may be useful in recognising individuals with unclear psychopathology (Ward et al, 1993a).

Demographic Information

Parents were asked to provide background information regarding age and gender of the child, relationship to the child, family structure and occupation of respondent. They were asked for informed consent to participate in the study and consent for their child's teacher to complete an SDQ (Goodman, 1999).

Teacher Ratings

Where permission was granted, teachers were asked to complete an SDQ (Teacher version) for hyperactivity and conduct scales only.

Procedure

Ethical approval was sought and granted from the University of Wales, Bangor. Verbal permission was given from seven state schools in the county of Conwy, North Wales for information regarding the study (provided in English and Welsh), questionnaires and a consent form to be sent home to parents. If parents wished to take part, they were asked to return them to the researcher in the self-addressed envelope provided.

Two information letters, questionnaire packs, consent forms and self-addressed envelopes (SAE) for each child who fulfilled the age criteria (age 5 – 12) were given to the schools to be taken home for mothers and fathers to complete. An e-mail

address and telephone number were provided for parents who required additional clarification or information. The information sheet informed parents that participation in the study was voluntary and that their school would receive £1 for each questionnaire returned to the school. The "return-by" date was set by the school. Permission was sought from parents for their child's teacher to complete the SDQ.

For parents who consented to teachers completing the SDQ, the same format as above was followed (i.e., teachers received an information sheet, a copy of the parents' signed consent form, a questionnaire, a consent form and an SAE. The information sheet, again informed the teacher that participation was voluntary.

Results

Data Preparation

Prior to data analyses, exploratory analysis examined the distribution of the data and suitability for parametric analysis, means were calculated and all data examined for outliers (this was set at 2sd from the mean). Outliers were removed; the new mean was calculated and used to replace any outliers. A Kolmogorow-Smirnov (KS) test was performed to assess the normality of the distribution of scores. A non-significant result (alpha > 0.05) indicates normality. The results indicated that some of the data were parametric; these were the IRI (PT), the IRI (PD), the APQ (Involvement) and the APQ (Inconsistent Discipline), therefore, $Z \leq .1.16$, $P \geq .05$. The KS test indicated that the non-parametric data were the AQ, the AARS, the WURS, the ADHD-RS, the SDQ (Conduct), the SDQ (Hyperactivity), the GHQ, the IRI (EC), the APQ (positive Parenting), the APQ (Poor Supervision) and the APQ (Corporal Punishment), therefore, $Z \geq 1.54$, $P \leq .05$. Correlation analysis in this study will therefore employ

Spearman Rho correlations and alpha adjustments will be made to control for multiple measurement, with 225 correlations, the new alpha level will be 0.05 / 225 = 0.0002. Analysis of variance (ANOVA) will still be used as it is robust to violation of the non-parametric assumption with moderate to large sample sizes of more than 15 cases per cell (Green, Salkind & Akey, 2000). Z-scores were calculated to create interaction terms for use in linear regression. Any missing data were dealt with using the default SPSS approach.

Analysis Strategy

1. One-way ANOVA will be used to explore the influence of demographic factors on both IV's and DV.
2. Correlations will be used to examine associations between IV's and DV.
3. Linear regression will be used to examine the linear association between parent and child ADHD symptoms and parental empathy and parenting. A simple regression model will be constructed with empathy and parenting variables as dependent variable and, child ADHD symptoms, maternal ADHD symptoms and an interaction term of child ADHD x Maternal ADHD all entered together into a final model.

One-way ANOVA

Total scores for all measures were entered into an analysis of variance (ANOVA) to conduct further analysis on the effects of child gender and family structure (i.e. one or two parents in the home). An examination of Table 1 demonstrates that male children were rated by their mothers as being significantly more hyperactive than female children. There was an influence of family structure on levels of reported ADHD

symptoms in children; more symptoms of ADHD were reported in families where only one parent was present. There was also some influence of family structure on psychopathology i.e. mothers in single parent families reported more mental health problems and greater levels of personal distress when dealing with their child suggesting a possible burden of care. Less adult hyperactivity was observed in two parent families.

[Insert Table 1 here]

Associations between variables

Spearman's Rho Correlations were used to examine associations between all variables. Table 2 shows a significant correlation between the WURS, the SDQ hyperactivity scale, and the ADHD-RS. This suggests there is a correlation between adults reporting retrospectively on their own ADHD and children's symptoms of ADHD. Very high correlations between related measures such as the ADHD-RS and the SDQ supported the concurrent validity of the measures.

[Insert Table 2 here]

A linear regression model was used to examine the effect of parent and child ADHD symptoms on maternal empathy. This was achieved by using the three predicting variables of child psychopathology, adult psychopathology and child psychopathology x adult psychopathology with the IRI.

No main effect was found for the ADHD-RS on personal distress, suggesting that child ADHD symptoms do not predict personal distress in parents. A main effect was observed for adult ADHD suggesting that adults with more symptoms of ADHD experience more personal distress. The interaction term was also significant with a negative beta weight, indicating that the higher the ADHD symptoms in both the parent and the child, the lower the mother's level of personal distress when responding to their child (see Figure 1). This interaction was plotted for ease of interpretation. Participants were divided into two groups based on a median split on maternal ADHD symptoms. The scores relating to child ADHD and Personal Distress were then plotted and a line fitted through the relevant points. An examination of Figure 1 shows that mothers with high ADHD symptoms that have children with high symptoms of ADHD, report lower levels of personal distress than the corresponding group of mothers with low ADHD symptoms who have children with high symptoms of ADHD.

[Insert Figure 1 here]

A second linear regression was used to examine the influence of ADHD symptomatology on maternal perspective taking. A main effect was found for adult ADHD and perspective taking suggesting that the higher the levels of ADHD symptomatology, the less able they are to engage in perspective taking. There was no interaction term observed.

A third linear regression was used to examine the influence of ADHD on empathic concern. Table 3 illustrates a main effect observed for child ADHD, indicating that

the higher the levels of ADHD symptomatology in the child, the less empathic the parent. There was no main effect for adult ADHD and no significant interaction term.

[Insert Table 3 here]

A second set of linear regressions were used to examine the influence of parent, child and parent x child ADHD symptoms on maternal parenting style. These can be seen in Table 4.

A marginal effect was observed for childhood ADHD symptoms on corporal punishment suggesting mothers used more corporal punishment for children with higher ADHD symptoms. There was no main effect for adult ADHD and no interaction.

With respect to inconsistent discipline, a main effect was found for child ADHD symptoms suggesting that the higher the child's ADHD symptomatology, the more inconsistent the discipline. There was no main effect for adult ADHD and no significant interaction.

A main effect was observed for child ADHD symptoms suggesting the higher the levels of ADHD, the lower the parental involvement. A negative beta weight suggests the higher the levels of parental ADHD symptomatology the less able they are to be involved with their child. There was no significant interaction.

A trend was observed towards an association between higher levels of adult ADHD and higher levels of poor supervision, however, there were no main effects and no interaction. No main effects or interactions were observed for positive parenting or poor supervision with child or adult levels of ADHD.

[Insert Table 4 here]

A second set of analyses was conducted using a retrospective measure of parental ADHD in parents rather than the AARS measure of concurrent symptoms. Two significant main effects existed, the first for perspective taking where retrospective ADHD symptoms were predictive of perspective taking, $R^2 = 0.07$, $\beta = -1.94$, $p=0.03$, showing that adults with higher retrospective ADHD symptoms had higher levels of perspective taking. The second main effect was found for inconsistent discipline where retrospective ADHD symptoms predicted inconsistent discipline, $R^2 = 0.07$, $\beta = 0.27$, $p=0.00$, demonstrating that adults with more retrospective ADHD symptoms had higher levels of inconsistent discipline.

A replication of the study was conducted using child conduct and parental aggression and empathic responding. This was to determine whether the findings of study one were specific to ADHD.

Total scores were entered into an ANOVA to conduct an analysis on the effects of child gender and family structure and child and maternal aggression. No significant findings were found for the effects of gender; however, there was an effect of family structure on child conduct and levels of generic aggression and hostility in mothers.

An examination of Table 5 shows that more mothers from single families tend to report higher levels of personal aggression and hostility and more conduct problems in their children.

[Insert Table 5 here]

A linear regression model was used to examine of the effect of maternal aggression and child conduct symptoms on maternal empathy. This was achieved by using the three predicting variables of child psychopathology, adult psychopathology and child psychopathology x adult psychopathology with the IRI. Results of this analysis are presented in Table 6.

A main effect was observed for child conduct suggesting that conduct problems in children predicts parental perspective taking. The main effect was significant with a negative beta weight for the SDQ conduct, indicating that the higher the conduct problems in the child, the lower the perspective taking in the parent. A main effect was also found for adult aggression demonstrating that aggression in parents is predictive of parental perspective taking. A negative beta weight for the AQ, indicated that the higher the aggression in the parent, the lower their perspective taking when dealing with their child.

A second linear regression was used to examine the influence of child conduct and maternal aggression on maternal levels of personal distress. A main effect was observed for adult aggression suggesting that aggression in parents is predictive of personal distress. A positive beta weight for the AQ indicated that the higher the

aggression in the mother, the higher her personal distress when responding to her child.

A second set of linear regressions were used to examine the influence of child conduct and maternal aggression on maternal parenting styles. This was achieved by using the three predicting variables of child psychopathology, adult psychopathology and child psychopathology x adult psychopathology with the AQ.

A main effect was observed for child conduct suggesting that conduct problems in children are predictive of parental involvement. A negative beta weight for the SDQ conduct, indicated that the higher the conduct problems in the child, the lower the parental involvement.

A second linear regression was used to examine the influence of child conduct and maternal aggression on maternal levels of supervision. A main effect was observed for child conduct suggesting that conduct problems in children predicts poor supervision/monitoring. A positive beta weight for the AQ, indicated that the higher the conduct problems in the child, the lower the maternal supervision/monitoring.

[Insert Table 6 here]

Discussion

Results from this study suggest that both child and maternal ADHD symptoms increase the likelihood of negative parenting. Symptoms of ADHD in mothers' were

related to both poor supervision and less involvement with the child. Symptoms of ADHD in children were related to discipline that was more inconsistent in nature.

The findings of this study also suggest that mothers with higher symptoms of ADHD, experience lower levels of personal distress when dealing with their ADHD child compared with mothers with low symptoms of ADHD. These findings support those of Biederman et al., (2002), who found that exposure to ADHD in parents, had a differential effect on high-risk offspring, which was dependent on the whether or not ADHD was present in the child. Therefore, having a parent with active ADHD symptoms did not serve as an additional risk for dysfunction to any offspring with the disorder, beyond that associated with ADHD. More recently, Psychogiou et al., (submitted), found that when maternal ADHD symptoms were high, parenting seemed to be less affected by the child's ADHD (compared to parents with low symptoms of ADHD).

This study has found a relationship between children's ADHD symptoms and their mothers' ability to perspective take when responding to their child. It could be that mothers with high ADHD symptoms are more empathic towards their child with ADHD and so are more able to understand things from their child's perspective. This is in line with the reports of Weiss, Hechtman & Weiss (2000) who suggests that parents with ADHD might be more tolerant and understanding of their ADHD children.

It is important to note that participants in this study were from the general population and had mild to moderate levels of ADHD. It is unclear if the same effects would be observed within a clinical sample.

One of the main limitations of the study was the number of participants (n =152). The low response rate meant that factor analysis was unable to be conducted on the data. Data was therefore analysed at an observed level. Despite this, statistical power was still observed in the effect for personal distress.

The adjustment to the P value resulted in only very conservative correlations. If a smaller adjustment were made, it is possible that there would be more correlations that would be worthy of further investigation.

Recruitment of fathers was particularly difficult and therefore the response rate was particularly low resulting in insufficient data to analyse. The reasons for this are unclear. Non-participating fathers of children with disruptive behaviour, generally range from 20% (Biederman, Munir & Knee, 1987; Schachar & Wachsmuth, 1990), up to 75% (e.g. Tapscott, Frick, Wooten & Kruh, 1996). Alternatively, research has shown that studies that target fathers of children with antisocial behaviour often find that the fathers are no longer a part of the family (Gagnon, Craig, Tremblay, Zhou & Vitaro, 1995; Loeber & Hay, 1997).

Lack of data therefore meant it was not possible to investigate any potential main effects between fathers and children with high ADHD symptoms, and levels of parental empathy. It may be that mothers and fathers have different emotional

responses to their children. Few studies have examined the influence of father involvement on parenting of children with ADHD (see Arnold, Leary and Edwards, 1997), and therefore very little is known about fathers of these children (Phares & Compas, 1992). Arnold et al., (1997) found an interaction between paternal ADHD and the amount of father involvement in predicting self-reported overreactivity. However, comparisons were not made between child ADHD symptoms, paternal symptoms of ADHD, and levels of parenting.

Recruitment of teachers was also problematic. It was therefore not possible to base the analysis on data from multiple informants. This limitation should be taken into consideration. Data was only available from mothers and there are concerns that parents of children with ADHD may report more of the symptoms in themselves because of their familiarity with the disorder; thus possibly influencing the data by giving false positives. However, Faraone, Biederman, Monuteaux & Cohan, (2003) found that parental ADHD did not affect maternal reports of their child's ADHD. It is also important to note however, that both the measures used in this study, the AARS and the ADHD-RS both have good cross-informant ratings.

Time constraints did not allow for the use of multiple sources of information; only questionnaires were used. It may be beneficial to use other sources of information such as interviews and observation. Limited time meant that a decision had to be made whether to use multiple measures or higher power. Based on the time constraints, a decision was made to use higher power.

Attempts were made to look at the generalizability of the findings from this study, i.e. were these findings specific to ADHD? Similarities between aggression in mothers and conduct problems in their children were therefore investigated. Initial findings suggest that the main effects found in this study are not observed in parental aggression and child CD. Further research is needed to explore whether this possible "safeguard" against the risk of negative parenting is specific to ADHD, possibly using other psychopathologies such as anxiety or depression. Alternatively, it might be beneficial to look at similarities between parents and children with physical health problems such as asthma or diabetes.

Although there are many limitations to this study, the exploratory nature of the approach and the initial findings suggest that there is room for further research.

Assessments in Child and Adolescent Mental Health Services currently investigate parental variables that may influence child psychopathology; however, it is important to examine any similarities between parents and children. The results of this study suggest that similarity can be either a positive or a negative factor in the development of child psychopathology.

Previous studies have stressed the importance of considering maternal ADHD symptoms in the design and implementation of training programmes for parents of children with ADHD (Sonuga-Barke et al., 2001). The present study has found that mothers with ADHD report less personal distress when parenting their children with ADHD. This has significant implications for models of parent/child interactions and

the subsequent impact on child development. It could be that the most appropriate therapy/treatment is determined based on the parents style and behaviour.

Furthermore, with limited resources available to clinical services, it may be beneficial to target psychosocial interventions to parents and children who are dissimilar because the mothers who are least like their children are those who appear to have more problems understanding their child's behaviour. Intervention outcomes might prove to be more successful for dissimilar parent/child dyads.

These findings suggest that in ADHD, similarity in parent and child is related to less personal distress when parenting, it would therefore be beneficial to know whether these similarities in parent/child dyads interact in other positive ways such as being able to keep their ADHD child on task or to cope with delay.

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Appendix H

RESULTS TABLES

Table 1: Effects of gender and family structure

Measure	Gender		F	P	Family Structure		F	P
	Male Mean (Standard deviation)	Female Mean (Standard deviation)			Two Parent Mean (Standard deviation)	One Parent Mean (Standard deviation)		
ADHD-RS	14.63 (11.28)	9.78 (7.76)	8.14	**0.01**	10.90 (9.08)	17.59 (11.76)	11.96	**0.00**
WURS	46.34 (19.35)	41.61 (15.24)	2.55	0.11	42.61 (15.72)	49.39 (22.40)	4.16	**0.04**
AARS	7.69 (6.00)	6.95 (4.44)	0.67	0.41	6.34 (4.28)	10.34 (7.00)	17.15	**0.00**
SDQ Hyperactivity	3.91 (2.99)	2.50 (2.49)	9.13	0.00	2.90 (2.66)	4.52 (3.13)	9.59	**0.00**
IRI (EC)	25.38 (2.20)	25.37 (2.07)	0.00	0.95	25.43 (2.10)	25.25 (2.28)	0.18	0.67
IRI (PT)	20.79 (0.44)	21.21 (4.40)	0.35	0.55	21.15 (4.32)	20.39 (3.95)	0.89	0.35
IRI (PD)	10.97 (5.21)	12.01 (6.07)	1.26	0.26	10.73 (5.39)	13.46 (5.76)	6.90	**0.01**
GHQ	1.02 (1.76)	1.16 (2.08)	1.98	0.16	0.98 (1.62)	1.85 (2.47)	6.19	**0.01**
APQ (Poor supervision)	11.16 (1.73)	11.10 (1.70)	0.05	0.83	11.15 (1.77)	11.10 (1.54)	0.02	0.88
APQ (Inconsistent discipline)	12.31 (3.61)	11.68 (3.03)	1.26	0.26	12.08 (3.55)	11.95 (2.90)	0.04	0.84
APQ (Corporal punishment)	3.84 (0.84)	3.72 (0.90)	0.66	0.42	3.84 (0.88)	3.66 (0.84)	1.24	0.27
APQ (Involvement)	42.30 (3.94)	42.98 (3.63)	1.14	0.29	42.59 (3.89)	42.58 (3.64)	0.00	0.98
APQ (Positive parenting)	27.14 (2.54)	27.24 (2.42)	0.06	0.81	27.13 (2.45)	27.34 (2.59)	0.21	0.65

Table 2: Correlation Matrix

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
WURS	1.0														
1	0														
AQ	.40**	1.0													
2	(.00)	0													
AARS	.56**	.35**	1.0												
3	(.00)	(.00)	0												
SDQ Hyp.	**.41****	**.27****	**.26****	1.0											
4	**(.00)**	**(.00)**	**(.00)**	0											
ADHD –RS	**.40****	**.30****	**.38****	**.75****	1.0										
5	**(.00)**	**(.00)**	**(.00)**	**(.00)**	0										
IRI (PD)	.23**	**.31****	**.31****	**.30****	**.25****	1.0									
6	(.01)	**(.00)**	**(.00)**	**(.00)**	**(.00)**	0									
IRI (PT)	-.28**	-.34**	-.23**	-.21*	-.25**	-.28**	1.0								
7	(.00)	(.00)	(.01)	(.01)	(.00)	(.00)	0								
IRI (EC)	-.03	.01	-.02	.16*	.06	-.08	.24**	1.0							
8	(.77)	(.92)	(.77)	(.05)	(.51)	(.31)	(.00)	0							
GHQ	.31**	.32**	.28**	**.32****	.25**	.18*	-.29**	-.07	1.0						
9	(.00)	(.00)	(.00)	**(.00)**	(.00)	(.03)	(.00)	(.38)	0						
APQ (Corp Punish)	-.01	.03	-.04	.13	.08	.03	-.06	.06	-.06	1.0					
10	(.95)	(.74)	(.63)	(.13)	(.35)	(.69)	(.46)	(.49)	(.50)	0					
APQ (Inc Discipline	.07	.12	.10	.16	.23**	.17*	-.36**	.03	.09	.18*	1.0				
11	(.43)	(.15)	(.26)	(.06)	(.01)	(.05)	(.00)	(.74)	().27	(.03)	0				
APQ (Poor Supervisn)	.14	-.04	.18*	.09	.14	.04	-.18*	-.21*	.14	-.02	.14	1.0			
12	(.10)	(.67)	(.04)	(.29)	(.10)	(.66)	(.03)	(.01)	(.10)	(.84)	(.10)	0			
APQ (Pos Parenting)	-.07	.00	-.16	.02	-.16	.07	.30**	.25**	-.15	.05	-.30**	-.22**	1.0		
13	(.38)	(.98)	(.06)	(.78)	(.05)	(.40)	(.00)	(.00)	(.07)	(.58)	(.00)	(.01)	0		
APQ (Involvement)	-.11	.03	-.19*	-.07	-.13	-.09	.31**	.07	-.07	-.01	-.30	-.20	.35**	1.0	
14	(.20)	(.74)	(.02)	(.40)	(.15)	(.31)	(.00)	(.38)	(.40)	(.94)	(.00)	(.02)	(.00)	0	
SDQ conduct	**.38****	**.31**	.18	**.55****	**.43****	.23**	**-.31****	.05	.29**	.15	.26**	.16	.02	-.15	1.0
15	**(.00)**	**(.00)**	(.03)	**(.00)**	**(.00)**	(.01)	**(.00)**	(.56)	(.00)	(.07)	(.00)	(.06)	(.82)	(.08)	0

Table 3: Linear Regression for Empathy and Child and Adult Psychopathology.

Variable	Predictors	R^2	Beta	P
IRI				
Personal	Child ADHD	0.13	0.13	**0.18**
Distress	Adult ADHD		0.32	0.00
	Child x Adult		-0.23	0.02
Perspective	Child ADHD	0.12	-0.17	**0.08**
Taking	Adult ADHD		-2.60	0.10
	Child x Adult		1.08	0.28
Empathic	Child ADHD	0.04	0.20	**0.04**
Concern	Adult ADHD		-1.33	0.19
	Child x Adult		-0.88	0.38

Table 4: Linear Regression for Parenting Style and Child and Adult Psychopathology

Variable	Predictors	R^2	Beta	P
APQ				
Corporal Punishment	Child ADHD	0.03	0.18	**0.07**
	Adult ADHD	0.03	-0.12	0.23
	Child x Adult	0.03	-0.05	0.64
Inconsistent Discipline	Child ADHD	0.07	0.26	**0.01**
	Adult ADHD	0.07	0.02	0.87
	Child x Adult	0.07	0.00	0.98
Involvement	Child ADHD	0.05	-0.07	0.49
	Adult ADHD	0.05	-0.20	0.46
	Child x Adult	0.05	0.21	0.82
Poor Supervision	Child ADHD	0.04	0.07	0.49
	Adult ADHD	0.04	0.15	0.15
	Child x Adult	0.04	0.02	0.85
Positive Parenting	Child ADHD	0.04	-0.11	0.25
	Adult ADHD	0.04	-0.10	0.30
	Child x Adult	0.04	-0.02	0.82

Table 5: Child Gender and Family Structure and Child and Maternal Aggression.

Measure	Family structure		F	P
	Two parents Mean (Standard deviation)	One parent Mean (Standard deviation)		
SDQ (Conduct)	1.04 (1.26)	1.85 (2.43)	6.99	**0.01**
AQ (Aggression)	3.86 (1.74)	5.11 (3.68)	7.62	**0.01**
AQ (Hostility)	5.27 (3.11)	8.13 (4.97)	17.34	**0.00**

Table 6: Linear Regression for Parenting Style, Empathy and Child and Adult Psychopathology

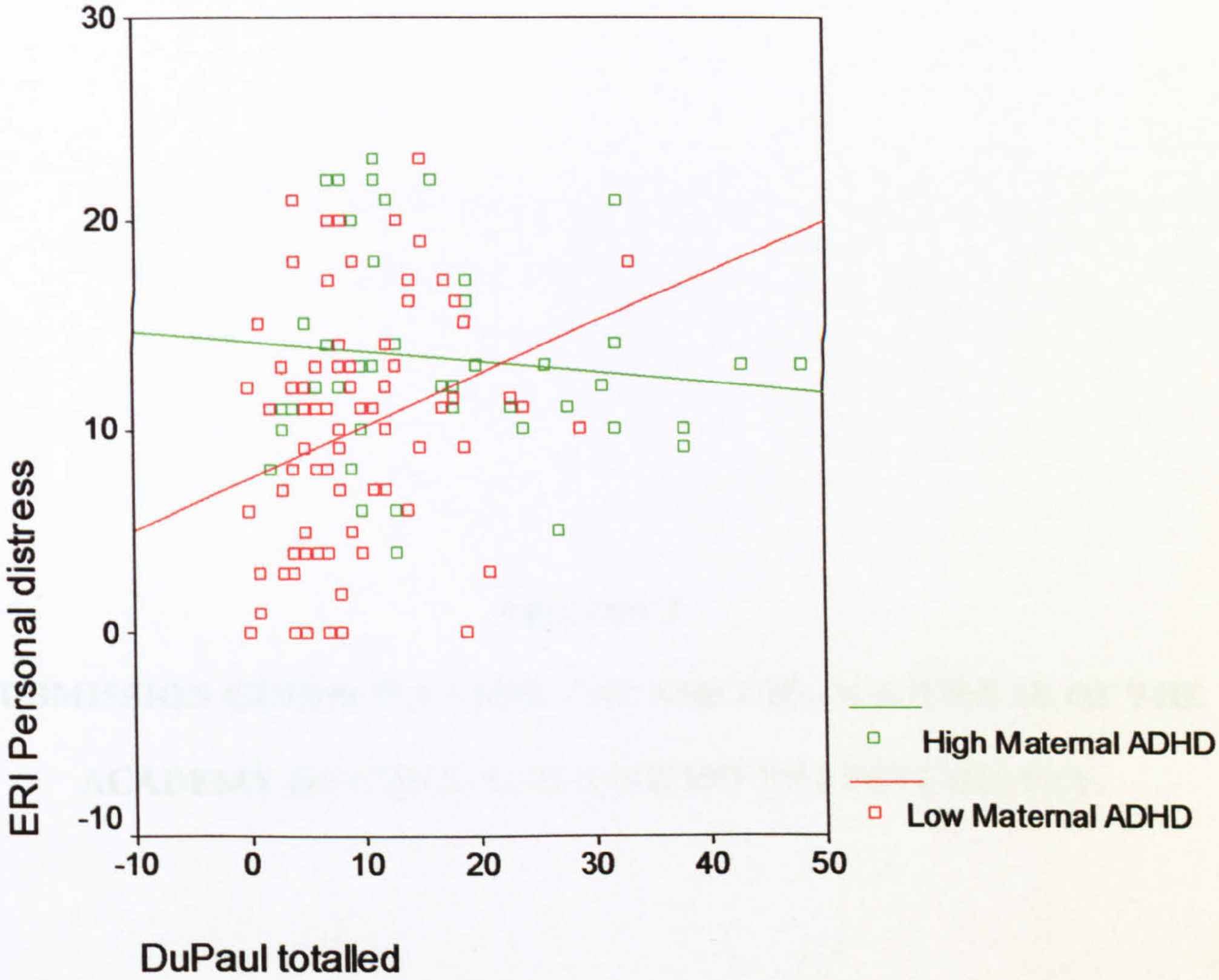
Variable	Predictors	R^2	Beta	P
IRI				
Perspective Taking	Child Conduct	0.18	-0.27	**0.00**
	Adult Aggression	0.18	-0.29	**0.00**
	Child x Adult	0.18	0.10	0.20
Personal Distress	Child Conduct	0.15	0.14	0.10
	Adult Aggression	0.15	0.34	**0.00**
	Child x Adult	0.15	-0.12	0.13
APQ				
Involvement	Child Conduct	0.03	-0.18	**0.05**
	Adult Aggression	0.03	0.06	0.48
	Child x Adult	0.03	0.05	0.54
Poor Supervision	Child Conduct	0.05	0.23	**0.01**
	Adult Aggression	0.05	-0.08	0.38
	Child x Adult	0.05	-0.09	0.30

Appendix I

FIGURES

Figure 1: Interaction between levels of child and parental ADHD and personal distress

Figure 1


30
20
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ERI Personal distress
-10
0
10
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30
40
50
DuPaul totalled
High Maternal ADHD
Low Maternal ADHD

Appendix J

SUBMISSION GUIDELINES FOR THE AMERICAN JOURNAL OF THE ACADEMY OF CHILD AND ADOLESCENT PSYCHIATRY.

Contributions to Theory, Clinical Practice and Learning

Implications for future research and theory

The findings of this study suggest that adults with higher symptoms of ADHD, experience lower levels of personal distress when dealing with their ADHD child than adults without symptoms of ADHD supporting the findings of research by Biederman et al., (2002). Biederman et al. found that exposure to ADHD in parents had a differential effect on high-risk offspring, contingent on the presence or absence of ADHD in the child. More recently, Psychogiou, Daley, Thompson & Sonuga-Barke, (submitted), also found that the parenting of mothers with high ADHD symptoms appeared to be far less affected by child ADHD than it was for parents with low symptoms of ADHD.

This study has found a relationship between ADHD symptoms in children and their mothers' ability to take their child's perspective when responding to their child; it could be that mothers with high ADHD symptoms are more empathic towards their child with ADHD and so are more able to understand things from their child's perspective. Weiss, Hechtman & Weiss (2000) reported that parents with ADHD might be more tolerant of their ADHD children. This is based on current symptoms and not retrospective childhood symptoms.

Response rate from fathers was particularly low and there was insufficient data for analysis. It was not therefore possible to see if there was a main effect between fathers and children with high ADHD symptoms and levels of parental empathy. It could be that mothers and fathers have different emotional responses to their children. Fathers tend to be absent from research and clinical settings related to ADHD (Singh,

2003) and few studies have examined the influence of father involvement on parenting of children with ADHD (see Arnold, Leary & Edwards, 1997), therefore very little is known about fathers of these children (Phares & Compas, 1992). Singh (2003) reported that fathers tend to fall into one of two categories: "reluctant believers" and "tolerant misbelievers". According to Singh, this may account for the absence of fathers in both research and clinical domains. Alternative methods are possibly needed to recruit fathers into research. Anecdotal evidence suggests that fathers defer responsibility for filling in questionnaires. Perhaps more could be done to try to recruit fathers, possibly sending out text reminders, e-mailing questionnaires to work or using public posting. For example, Pasto & Baker (2001) report an increase in seat belt use among front seat passengers through the use of public posting of performance feedback i.e. the proportion of passengers observed wearing seat belts on the previous observation day, and distribution of an information flyer to all cars in the target car park. Seat belt use was reported to increase from 64% to 71%.

Recruitment of teachers was also problematic and it has therefore not been possible to make a comparison between multiple respondents to enable data that are more robust. Mitsis, McKay, Schulz, Newcorn & Halperin, (2000) report that using the Diagnostic Interview Schedule for Children, concordance for DSM-IV ADHD by parents and teachers was only 74%. Agreement on a particular subtype was particularly poor, with only 17 cases out of 55 cases agreed on. It should be noted however, that both the measures used in this study, the AARS and the ADHD-RS, both have good cross-informant ratings (Edwards, Barkley, Laneri, Fletcher & Metevia, 2001; Murphy and Barkley, 1996; Murphy & Schachar, 2000; DuPaul, Power, Anastopoulos & Reid,

1998; Kadesjo, Kadesjo, Haggloff & Gillberg, 2001; Magnesson, Smari, Gretarsdottir & Prandardottir, 1999).

It may be beneficial to use other sources of information such as interviews and observation. The inclusion of other sources of information may have resulted in further interactions for parenting practices with higher maternal and child ADHD symptoms. Psychogiou et al., (submitted) observed maternal behaviour during mother/child interactions during play, the coding was drawn from Daley, Sonuga-Barke & Thompson, (2003), and Maternal Negative Expressed Emotion, using the Pre-school Five Minute Speech Sample (Daley et al., 2003). Psychogiou et al. found that negative expressed emotion and distant and/or controlling parenting was associated with maternal ADHD. Furthermore, they found interactions similar to those found in this study using observational data and EE speech samples.

Attempts were made to look at the generalizability of the findings from this study, i.e. whether or not these findings are specific to ADHD. Initial findings suggest that the main effects observed in this study are not apparent in parental aggression and child CD. Further research is needed to explore whether this possible "protection" against the risk of negative parenting is specific to ADHD, using other psychopathologies such as anxiety or depression. Alternatively, it might be possible to look at similarities between parents and children with physical health problems such as asthma or diabetes. For example, if parent-child similarity in ADHD improves parental empathetic responding, might this also occur for parent-child asthmatic dyads? It might be expected that asthmatic mothers would find it easier to engage in perspective taking for their asthmatic child than non-asthmatic mothers.

The evidence from this study suggests that the higher the maternal and child ADHD the lower the personal distress experienced by the mother when dealing with her child. It might be possible to verify this through alternative measures such as observation. It would also be beneficial to compare parenting practices with siblings. Whilst Psychogiou et al., have used observation to assess parent-child relationships, their observations were not specifically focussed on parenting practices. Further observations of parenting practices could help demonstrate the influence of parent-child similarity in parenting, for example, do high ADHD mothers help their high ADHD children to adjust and accommodate better to stressful situations, such as experiencing delay, having to wait their turn or situations requiring sustained attention.

It might also be interesting to research further whether the observed effect is specifically familial. Children spend a great part of their day with teachers. Might teachers with symptoms of ADHD be more empathic towards their ADHD pupils? If this were true, then pupils with ADHD may function better within classes run by teachers with higher ADHD symptoms, although this may have a detrimental effect on other children in the classroom. However, as many children with ADHD require help from Special Education Needs Coordinators and classroom assistants at school, school management and educational psychologists might like to consider issues of similarity before assigning coordinators to specific children. It could be that teachers with ADHD might be able to provide good individual tuition for children with ADHD; alternatively, they might make good home tutors for those children with ADHD who have been excluded. Although there are many limitations to this study,

the exploratory nature of the approach and the initial findings suggest that there is room for further research.

Implications for clinical practice

Assessments in Child and Adolescent Mental Health Services currently investigate parental variables that may influence child psychopathology (Carr, 1999); results from this study suggest that it might also be important to explore the interactions or similarities between these variables in parent and child. The results of this study illustrate that similarity can be either a positive or a negative factor in the development of child psychopathology.

The present study has found that mothers with ADHD demonstrate more positive emotional response towards children with ADHD. This has significant implications for models of parent/child interactions and the subsequent impact on child development. It could be that the most appropriate therapy/treatment is determined based on the parents style and behaviour. With limited resources available to clinical services, it may be beneficial to target psychosocial interventions to parents and children who are dissimilar because the mothers who are least like their children are those who appear to have more problems understanding their child's behaviour. Intervention outcomes might also prove to be more successful for dissimilar parent/child dyads, who would have a higher hill to climb.

This study has found that in ADHD, similarity in parent and child leads to less personal distress when responding to the child. If similarities and interactions between children with ADHD and teachers with symptoms of ADHD are similar to

those between maternal and child ADHD then they too may result in a more positive effect on the child with ADHD symptoms.

Obtaining data from both parents and teachers would also benefit the ongoing monitoring of a child's response to treatment. This is crucial in determining whether the treatment is being successful or in need of modification.

Process/personal issues arising from the conduct of the research.

This study was started just before Christmas 2003. A previous study, for which data collection had started in October 2002, was stopped due to low recruitment numbers and a very low chance of recruiting enough participants to analyse data on. For this reason, a new research project was started with only 6 months to collect data and write up. The timescale for data collection did not allow for extra time for alternative methods of recruitment to be considered in order to try to boost the numbers of fathers' and teachers' responses. The limited timescale has also meant less time than would have been preferred for write-up and for building up a more solid knowledge base of the subject.

Confidentiality was assured to all participants in the study. However, there was the potential for the disclosure of information that could have raised concerns regarding child protection. It is important to note that child protection laws take priority over confidentiality, therefore had any responses on the questionnaires given cause for concern, then confidentiality would have had to be broken.

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Appendix J

WORD COUNTS

Word counts

Title	10
Abstract	214
Ethics Proposal	1951
Literature Review	7999
Empirical Paper	6011
Critical Review	1583
Total	17768
References and appendices	13462