PROFESSIONAL DOCTORATES

The presentation of attention/deficit hyperactivity disorder (AD/HD) in children with intellectual disabilities (ID)

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The Presentation of Attention Deficit/Hyperactivity Disorder (AD/HD) in Children with Intellectual Disability (ID)

By Katie Bigham

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This thesis is submitted as part of required criteria for the Doctorate of Clinical Psychology

Contents

Abstract

Attention deficit hyperactivity disorder is one of the most common childhood disorders, and is thought to affect more children with intellectual disabilities than those without. It is associated with marked social and educational impairment, and is recognised as a major risk factor for the development of problems throughout adolescence and adulthood such as conduct disorder, educational failure and personality disorder. Although prevalence rates vary, current research suggests that attention deficit/hyperactivity disorder affects between 2% and 5% of typically developing children and up 16% of children with intellectual disabilities. However, there has been little exploration into why this population appear to be at increased risk. This study aimed to examine whether parental reports of hyperactivity were measuring the presence of an underlying liability to AD/HD in their children with intellectual disability, or whether they were being influenced by other factors, such as levels of adaptive behaviour or the presence of other behavioural problems. Parental reports of hyperactivity on a screening questionnaire were compared with semi-structured clinical interviews and an objective measure of AD/HD. Results found that the majority of children, who were reported by parents as being hyperactive on the screening measure, were also found to exhibit AD/HD symptoms on subsequent measures. These results support findings from previous studies that report AD/HD as being commonly exhibited by children with intellectual disability. However, they also highlight a need for more research to be undertaken on the specificity of AD/HD in children with intellectual disability. This would allow advancements to be made in the domains of assessment, diagnosis and intervention for children with intellectual disability and AD/HD.

Acknowledgements.

I would like to thank the following individuals without whom the completion of this study would not have been possible: Dr Dave Daley, who gave me his unfailing support, patience and invaluable guidance throughout the undertaking and completion of this project; Professor Richard Hastings, for his advice and encouragement in the initial development of the study and support thereafter; all the parents that took time out of their incredibly busy lives to complete questionnaires and be interviewed by me during the little spare time they have; all the children who behaved so well, and whose company I thoroughly enjoyed; and to all the schools that so patiently accommodated me throughout the whole project. In addition I would like to thank all the administration staff and the rest of the North Wales Clinical Psychology Programme team for making the last three years an interesting and enjoyable process. Last, but definitely not least, I would like to thank my wonderful family especially Paddy, Anna and Finn for their unfailing patience and support, and for ensuring I maintain a healthy perspective between work and play.

SECTION 1

ETHICS PROPOSAL

School of Psychology Ethics Proposal

1) **Title of project:** Hyperactive behaviours in children with a learning disability.

2) **Name of investigator(s):** Katie Bigham, Trainee Clinical Psychologist (supervised by Dr Richard Hastings, Research Director, UWB and Dr Dave Daley, Research Tutor, UWB).

3) **The potential value of addressing this issue:** The potential value of addressing this issue is primarily to increase our knowledge and understanding of how ADHD presents in children with a mild learning disability (MLD). Although a variety of emotional and behavioural problems have been associated with ADHD in children of average intellect, little is known regarding the concomitant behavioural and emotional difficulties in children with ADHD and a learning disability (LD) (Emerson, 2003). Although more recently psychopathology in children with LD has received increased attention within research and clinical fields, basic information on prevalence rates and the impact of psychological difficulties on children with LD continues to be scarce (Dekker et al., 2003). As part of this study prevalence of ADHD in children with MLD in North Wales will be estimated. Standardised measures (Cookie-Delay Task (C-DT) & Choice-Delay Task (Ch-DT)) will be employed in conjunction with parent and teacher reports in order to estimate prevalence rates and investigate the presentation of increased ADHD symptomatology in children with LD. The core interest will focus on investigating whether the same underlying deficits associated with ADHD are present in children with LD as are seen in children without LD.

4) **Brief background to the study:**

Reviews of epidemiological studies report that prevalence rates for attention deficit hyperactivity disorder (ADHD) range from between 1-19%, depending upon the populations studied and the diagnostic criteria applied (see Cohen et al., 1993; Hinshaw, 1994; McArdle et al., 1995). ADHD had been found to be more prevalent in boys than in girls and in pre-adolescents than in late adolescents (Carr, 1999).

Szatmari et al. (1989) found that children who had a diagnosis of ADHD were 1.5 times more likely to live in urban areas, more then twice as likely to come from single-parent families, and more than three times as likely to be gaining financial support from the state. Furthermore, it was found that ADHD was associated with a psychiatric parental history and family dysfunction.

Clinical features

The core features of ADHD are inattention, hyperactivity, and impulsivity (American Psychiatric Association; DSM IV, 1994).

- ***Inattention***: Children with ADHD have great difficulties with processing instructions and tend to be described as engaging in 'off task' behaviours. They are likely to be easily distractible, disorganised, forgetful, and have a tendency to lose things.

- ***Hyperactivity***: Children with ADHD will appear excessively energetic and undirected. They appear to be constantly fidgety, unable to sit still, restless and rowdy.

- ***Impulsivity:*** Children with ADHD appear to display increased levels of disinhibition. They often interrupt and intrude, unable to wait their turn. Behaviours in a variety of settings appear to be elicited without thought.

According to DSM IV (1994), ADHD is viewed as having two primary factors; inattention and hyperactivity/impulsivity. These two factors create three subtypes of the disorder: Predominantly Inattentive (ADHD-I), Predominantly Hyperactive/Impulsive (ADHD-H), and a ADHD Combined Type.

As a result of the difficulties described above, children with ADHD often experience a wide range of secondary problems including academic and relationship difficulties. They find it hard to conform to parental/educational/societal rules and expectations, which in turn results in chronic conflicting relationships within their environment (Carr, 1999).

Due to the fact that a child with ADHD is frequently at the centre of family and classroom problems, many secondary problems have been associated with the presence of ADHD, including low self-esteem (Mellor et al., 1996). Green and Chee (1994) observed the following secondary difficulties commonly experienced by children with ADHD:

- Insatiability
- Social clumsiness
- Poor co-ordination
- Disorganisation
- Variability of mood
- Specific learning difficulties such as dyslexia or language problems.

These difficulties are likely to be exacerbated if the child also has a mild learning disability.

Aetiology

There are a number of theories that have been developed to try to account for the development of ADHD (Tannock, 1998). These theories fall mainly into two main categories: Biological; and psychosocial theories and will be discussed in more detail, however it is generally thought that it is the interaction between biological and environmental factors that increase the probability of the development of ADHD. (Tannock,1998).

Biological theories: Genetic theorists propose that children who develop ADHD are born with a genetic predisposition for hyperactivity (Carr, 1999). Twin and family studies have found that genetic factors are influential in determining temperament and activity levels in the normal population. However, other environmental factors would need to be present for ADHD to fully develop (Stevenson, 1992; Hinshaw, 1994). It is also reported that minor brain insult caused during the prenatal and perinatal periods is more prevalent among children with ADHD when compared with normal controls (Taylor, 1994; Cantwell and Hannah, 1989; Barkley, 1990). However, this alone would not be enough, an interaction with other factors would again be necessary for ADHD to develop. A further biological hypothesis for the development of ADHD is

the dysregulation of both the dopamine and adrenaline/noradrenaline systems (McCracken, 1991). It is thought to be these systems that are directly affected by effective pharmaceutical therapies in the treatment of the disorder. It has been found that such intervention positively affects approximately 60-90% of sufferers, resulting in a reduction of symptomatology and an improvement in social and academic functioning (Taylor, 1994; Hinshaw, 1994; Gadow, 1992). Recently neuropsychological evidence has suggested the existence of two possible neuropsychological influences on ADHD, namely inhibitory dysfunction and delay aversion (Solanto et al., 2001; Sonuga-Barke, 2002).

Psychosocial factors: These focus largely on the influence that family and social systems have on the development and maintenance of ADHD. It has been found that factors such as parental psychological difficulties including depression and alcohol abuse, marital discord and coercive parent-child relationships are associated with ADHD (Hinshaw, 1994; Taylor, 1994; Anastropoulos et al., 1996). With regard to the wider social context factors such as low SES, institutionalisation, and poor relationships have been associated with the development of ADHD (Taylor, 1994; Barkley, 1990). Some of the best evidence to support psychosocial factors is the fact that psychosocial intervention leads to dramatic reduction in ADHD symptoms (Bot et al., 2002; Sonuga-Barke et al., 2001)

Comorbidity

The characteristics of ADHD are commonly co-morbidly associated with a number of other long-term disorders (Barkley, 1998; Biederman et al., 1992; Mannuzza etal., 1993; Szatmari et al., 1989).

A number of community based studies have investigated the issue of comorbidity between ADHD and other psychiatric disorders (Biederman et al., 1992; Fergusson et al., 1993; Pelman et al., 1992; Szatmari et al., 1989). These studies have found that higher rates of anxiety and affective disorders, conduct disorder, and oppositional defiant disorder were present in children with ADHD when compared with their non ADHD counterparts. These differing patterns of comorbidity indicate that ADHD is a heterogeneous disorder with multiple aetiologies (Scahill et al., 1999).

Given its comorbidity, frequency, and recurrent utilization of health services, ADHD has become an increasing public health issue. Due to this there is a need for continuing research in order to fully understand the aetiology, variability, and maintenance of the disorder in order to provide a more rational mental health policy, preventative strategies/early diagnosis, and effective treatment programmes (Scahill et al., 1999).

ADHD and learning disabilities

The motivation behind this study is the fact that although a variety of emotional and behavioural problems have been associated with ADHD in children of average intellect, little is known regarding the concomitant behavioural and emotional difficulties in children with ADHD and a learning disability (LD) (Emerson, 2003). Although in recent years psychopathology in children with LD has begun to receive increasing attention within research and clinical fields, basic information on prevalence rates and the impact of psychological difficulties continues to be scarce (Dekker et al., 2003). In the limited number of studies conducted to date on the prevalence of ADHD in children with LD estimates range from approximately 0.5% to 11% (Dekker et al., 2003).

Interestingly, it has been found, using psychiatric interview assessment methods that estimated prevalence rates of psychopathy in children with LD are within the same range or less than those detailed in general population studies of children without LD (e.g. Anderson et al., 1987; Verhulst et al., 1997). These figures contradict the three/fourfold increased risk of defiant behavioural and emotional difficulties found in children with LD when compared to non-LD children in studies using standardised rating scales (Dekker et al., 2002; Koller et al., 1982; Linna et al., 1999; Rutter et al., 1970). Dekker (2003) reported that the most likely explanation for this discrepancy is the methods that are employed for data collection. Presently for community based studies of children with LD, a majority of information used for DSM diagnoses is obtained via clinical records. Such records diagnoses have often been recorded without stating how information was gathered. In comparison, in studies that involve mainly non-LD children information is clearly sought and recorded through regular use of standardised measures (e.g. Anderson et al., 1987).

The aim of the present study is to estimate the prevalence of hyperactivity in children attending MLD schools in North Wales and investigate the underlying deficits associated with hyperactive behaviour in children with MLD.

5) Study aims: The presentation of hyperactivity in individuals with a mild learning disability will be investigated. The following research aims will be investigated:

1. Estimate the prevalence of hyperactive behaviours in children attending schools for individuals with mild learning disabilities in North Wales.
2. Investigate how children with MLD and hyperactivity differ from children with MLD without the presence of hyperactivity.
3. Investigate the relationship between subjective reports (parent/teacher ratings) and objective measures (neuropsychological tests) of hyperactive symptoms and behaviour.

6) Recruitment of participants: Between 60-80 children (approximately 30-40 each in high and low ADHD symptoms groups) ranging in age from 6-10 years old with mild learning disability (MLD) will be recruited from schools for children with special needs across North Wales.

Dr Alan Dowey, Clinical Psychologist in Learning Disabilities in Conwy & Denbighshire, has been contacted for guidance in approaching appropriate schools that will be most willing to participate in the study (awaiting conformation of agreeable schools). One school in Wrexham (St Christopher's) has provisionally agreed to take part in the study. Schools in Denbighshire, Flintshire, Conwy, Gwynedd, and Ynys Mon are currently being approached.

Due to the participants' age, consent will be obtained from the participants' parents regarding involvement in the study.

A pilot study will be conducted on a small number of children with MLD in order to evaluate the appropriateness of the tasks included in the proposed study.

7) Research design: The study design is a two-stage procedure.

Stage 1: Children with a mild learning disability will be screened for hyperactivity by the SDQ Strengths and Difficulties Questionnaire; Goodman, 1997) (completed by both parents and teachers).

Stage 2: Confirmation of hyperactivity identified by the SDQ will be sought using the PACS (Parent Account of Childhood symptoms; a structured clinical interview; Taylor et al., 1991). Children will subsequently be assigned to one of two research groups (group 1: high hyperactivity scoring children; Group 2: the control or low hyperactivity scoring children).

Each child's level of adaptive behaviour will be sought using the Vineland Adaptive Behaviour Scale – Survey Form (VABS; Sparrow et al., 1984). This semi-structured clinical interview will be performed at the same time as the PACS and will enable the researchers to examine impact of adaptive behaviour on parental reports of hyperactivity, as well as providing an estimate learning disability.

Finally, the presentation of each group (high and low hyperactivity) will be examineded using two neuropsychological tests; the cookie delay task (C-DT) and the choice delay task (Ch-DT).

8) Procedures employed: Two groups of children with mild learning disabilities will be identified. A minimum of 60-70 children in total will be needed. Ideally a diagnostic group consisting of MLD children who fulfil criteria for ADHD would be compared in presentation with a group who do not. Emerson (2003) reported that children with a mild learning disability were at a ten fold risk of developing ADHD; he estimated that between 30% and 40% of children with MLD are also be suffering from comorbid ADHD. Due to this finding it is expected that as a conservative estimate within a total sample of between 110-120 children screened, at least 36 children with MLD and high ADHD symptomatology will be identified. For the purpose of this study children with high hyperactivity scores will be of most interest, as it is not our aim to diagnose children with ADHD.

Firstly, the presence of hyperactivity will be assessed via the completion of the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997). The SDQ will be completed by parents and teachers of children aged between 6-10 years of age (parental consent would have already been sought). The SDQ will be distributed to agreeable schools and hand distributed to parents of participating children via their teachers. Parents will be asked to return the completed forms to the class teacher, through whom the investigator will collect all corresponding parent and teacher completed forms.

The SDQ's will be analysed and children will be assigned to one of two groups depending upon their hyperactivity scores; group 1 will consist of children with high hyperactivity scores and group 2 low hyperactivity scoring children (there will not be a diagnostic ADHD vs. control group). All children with low scores will be invited to participate in the study. But given the time constraints all these children may not contacted to take part. People who were willing to participate but did not get to take part in the end will be sent a letter reminding them that they are entitled to receive a copy of the research summary at the end of the study.

The Parental Account of Childhood Symptoms (PACS), a structured clinical interview (PACS; Taylor et al., 1991) will be used to confirm high hyperactivity scores obtained via the SDQ. The PACS will be undertaken via telephone interaction that will be recorded for later analysis (consent for this will be sought and recording disposed of once an anonymised data set has been created). Levels of adaptive behaviour will be ascertained at the same time through the administration of the VABS (Sparrow et al., 1984).

The children's level of IQ will be estimated so that IQ can be controlled. This will involve undertaking a number of core subtests from the Wechsler Intelligence Scale for Children (WISC); Similarities, Block Design, Arithmetic and Picture Completion.

Finally the two groups' differing presentations will be investigated using two neuropsychological tests; the cookie delay task (C-DT) and the choice delay task (Ch-DT).

The completion of the IQ measures and the neuropsychological tests will be taking place in the school environment with consent from parents and schools alike.

Neuropsychological tests

Cookie Delay Task (C-DT; delay of gratification; Campbell et al., 1982). This involves an experimenter placing 3 transparent cups in front of the child and requesting that they the child can take only take the treat that is under one of them after the experimenter has given the signal which is a clap *first.*

Three practise trials are run in order to establish that the child understands the rule. During each trial the child is reminded not to move before hearing the sound of the clap. If during these practise trials the child is unable to inhibit their behaviour, they are asked to place their hands on their knees at the start of each trial. This enables the experimenter to identify a purposeful move over a twitch.

Up to eight trials are then run with delays of between 5 seconds and 30 seconds randomly. During each trial the experimenter raises their hand approximately midpoint of the time delay period (e.g. at 15s if the delay was 30s) in preparation to clap.

The child is rated on the level of inhibition: 0 = fully inhibited (there was no movement toward the cup); 1 = partially inhibited (any movement towards or touching of the cup); and 2 = not inhibited (the cup was lifted and/or the sweet was taken).

The range of possible scores over 8 trials is therefore 0-16, with higher scores indicating impulsivity. This test has been used successfully with ADHD children in previous research (Sonuga-Barke et al., 2002; Sonuga-Barke, Dalen & Remington, 2003). A study undertaken by Sonuga-Barke et al. (2003) that incorporated all the tests described for the purpose of this study (SDQ; PACS; C-DT; Ch-DT) found that test-re-test reliability was acceptable for all the tests ($r > .66$).

The Choice Delay Task (Ch-DT) (measure of delay aversion; Sonuga-Barke et al., 1992): The Ch-DT involves a response box being used consisting of a choice of 2 buttons: button 1 for a small immediate reward (i.e. after 1 second); and button 2 for a large delayed reward (i.e. two rewards after 20 seconds). On successive trials children will be asked to choose between small immediate rewards or larger later rewards. Children experiencing AD/HD symptomatology have consistently been shown to choose small immediate rewards over larger later rewards under trial constraint (limited number of trials; small reward associated with shorter sessions but less reward overall) but not under time constraints (each alternative having same session length) .

This task evaluates not the child's *inability* to inhibit a response (as in the C-DT), but conceptualises and looks upon impulsivity in ADHD as a *choice* to avoid delay (Solanto et al., 2001).

When validity of the choice delay task was assessed (Solanto et al., 2001) it correlated with teacher ratings of impulsivity, hyperactivity, and conduct problems, and with observations of gross-motor control, physical aggression, and an ADHD composite score. These results propose that delay aversion is associated with a large number of ADHD characteristics.

9) Measures employed: ***Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997):*** The SDQ (Goodman, 1997) is a brief informant-rated measure of behavioural attributes (some positive, some negative) that includes 25 items, which are divided into 5 scales with 5 items each generating a score for conduct problems, hyperactivity emotional symptoms, peer problems, and prosocial behaviour. Apart from the latter, all are summed to produce a total difficulties score. The SDQ will be completed by both teachers and parents.

The form is quick to administer, taking only approximately 5 minutes to complete. The range of test-retest reliability and internal consistency was 0.70-0.58 and 0.51-0.76, respectively.

A copy of the questionnaire can be found in Appendix 1.

Parent Account of Childhood symptoms (PACS – a structured clinical interview; Taylor et al., 1991): The PACS is a structured clinical interview designed for use with children aged between 6 and 11 years. It was developed to assess the core symptoms of ADHD and conduct problems (CP) (Taylor et al., 1991). For the purpose of this study only the AD/HD scale will be administered to parents. They will be asked to describe the severity and frequency of ADHD problems in a variety of settings (e.g. home, school, in public) over the previous six months. Trained interviewers rate these responses using criteria previously validated against clinical judgement.

The PACS takes approximately 20 minutes to administer. Breaks between tasks will be arranged in order to minimise the risk of a reduction in attention across tasks. Trained interviewers rate the responses gained using a criterion that has been previously validated against clinical judgement.

Sample items from this measure can be found in Appendix 2.

Vineland Adaptive Behaviour Scale – Survey Form (VABS; Sparrow et al., 1984)

The VABS is a semi-structured interview measure that contains 297 items in total. Not all 297 items are used during the interview, rather enough specific questions are asked to allow the interviewer to make an accurate rating of adaptive behaviour. The VABS is sub-divided into 4 domains which each give an adaptive behaviour score, and together yield an adaptive behaviour composite score. The composite score can be used as a measure of learning disability. The 4 domains are: Socialisation, Daily Living Skills, Communication, and Motor Skills. The latter is only applicable to children with a developmental age of five years and under, and is not needed to derive the adaptive behaviour composite score.

Administration of the VABS takes approximately 30 minutes and will be performed at the same time as the PACS upon receiving parental consent.

A copy of the questionnaire can be found in appendix 3.

10) Qualifications of the investigators to use the measures: The investigator is currently a third year Trainee Clinical Psychologist and has had regular experience of issuing and interpreting the SDQ and subtest from the WISC in her clinical practice. Training will be undertaken under the guidance of Dr Dave Daley on the administration and interpretation of the C-DT, Ch-DT and PACS.

11) Venue for investigation: All interactions with children involved in the study will occur within the school environment. Interactions involving parents (PACS & VABS) will be performed via telephone communication. The distribution and collection of the SDQ will occur via agreeable schools.

12) The duration of the study:

September 2004	Identify/contact possible MLD schools that are willing to take part in the study.
(Maternity Leave October 2004-April 2005)	
April 2005	Begin screening for appropriate participants for allocation into the two groups using the SDQ and gain consent from parents.
June 2005	Confirm high ADHD symptomatology with the PACS and assign participants to each appropriate group.
July/August 2005	Undertake subtests from the WISC and commence the neuropsychological tests.
Sept-November 2005	Write up findings.

13) Data analysis: A power calculation using Cohen's Power Primer for a two group comparison indicates that 38 participants per group will yield sufficient power at 0.8 to detect differences between the groups at alpha .01.

14) Potential hazards to participants / investigators: The investigator is not aware of any hazards or risks that are likely to be incurred by participants as a result of taking part in this research.

15) Potential offence / distress to participants: The investigator is not aware of any offence or distress that is likely to be incurred as a result of taking part in the study.

Parents will be informed prior to taking part in the study that if for any reason they wish to withdraw their child from the study they can do without having to give reason. If parental concerns are raised throughout any part of the research process regarding the presence of high ADHD symptomatology (hyperactivity) or request advice on how to deal with behavioural difficulties, information on ADHD and a parent training manual will be available (see Appendix 4). Parents may seek information regarding levels of hyperactivity identified in their child. To lessen these anxieties/concerns, parents will be advised that the purpose of the study is not to diagnose any child with ADHD, but to investigate differences in children's behaviour who are eliciting some of the symptoms of the disorder, namely hyperactive behaviours. If a parent specifically asks for individual feedback, a report will be issued outlining general findings.

If a child gets distressed during the neuropsychological tests the procedure will be stopped immediately. Future participation in the tasks will only continue if the parent and school are happy for the child to continue, and if the child is not showing any signs of distress due to their participation.

16) Procedures to ensure confidentiality: All data (consent forms, questionnaires etc) will be stored in a locked cabinet in the Clinical Psychology Department in the UWB. None of the information returned by families will be used in any way that will break confidentiality. Results of the study will describe overall findings and not information about individual children. Once the research has been completed the raw data (consent forms, questionnaires etc.) will be destroyed (shredded) as all the information will then be on an anonymised data file. All anonymised data will be

stored in a locked filing cabinet in the Clinical Psychology Department at UWB for five years after the publication of the research. All data will be destroyed thereafter.

17) *How consent is obtained: Parents will be asked to sign a consent form, after reading the relevant information allowing them to make an informed decision regarding participation in the study and consenting to their child's participation.

18) *Information for participants: Information outlining the study procedures and rationale will be issued to parents enabling them to make an informed choice as to whether they are willing to participate and allow their children to take part. Information will be issued to parents in two stages as not to overwhelm them. Only parents who either fulfil criteria to participate in Stage two, or who are chosen by the researchers to do so, will receive further information on the next stage and further consent forms. Consent for their child to participate will be given by parents upon the basis of parental understanding of procedures and implications. All information will be available bilingually.

19) Approval of relevant professionals: Head teachers of schools will be approached to secure their co-operation in contacting potential participants.

20) Payment to:	**participants:**	No payment
	Investigators:	No payment
	Departments / institutions:	No payment

21) Equipment required and its availability:

Psychological measures:

SDQ	photocopying costs; available.
PACS –	no cost; available.
VABS	£1.25 per answer form; 3-50 needed

WISC Subtests	Response forms easily ordered. The subtests can be borrowed from the WISC at the UWB. Cost of response forms to be sought.
Cookie Delay Task	No cost; available.
Choice Delay Task	A computer will be borrowed from the department to run this task (no cost).
Digital video recorder	Available from the NWCPP. (Booked from April – September 2005).
Telephone recording equipment	Need to purchase at a cost of £90
Audio tape	1 per parent (PACS). NWCPP to order.
Response box	Currently investigating availability from the NWCPP. It is possible that one may have to be purchased.
Lap top	Borrowed from NWCPP (no cost).
Telephone charges	20 minutes a time at 4p per minute = £56
Computer & SPSS	Possessed by the investigator.

22) What arrangements are you making to give feedback to participants?

There will be no individual diagnostic ADHD feedback given to participants or parents. Diagnoses of ADHD cannot be given; although a diagnosis is possible to achieve via the PACS by running it through a programme called 'Hyperscheme', the researcher will not be trained to do this. If individual feedback is specifically asked for by a parent, a report will be issued outlining high/low symptomatology of ADHD, strengths and weaknesses identified as a result of the IQ and neurological assessments undertaken, and a profile of the PACS assessment (where relevant). The PACS profile will identify specific areas of difficulties identified such as difficulties identified playing/interacting with peers.

If as a result parents express concern regarding their child's behaviour and its management information on ADHD symptomatology and a parent training manual will be available.

At the end of the research a research summary will be issued to the parents and the school discussing group differences.

23) Sign the declaration.

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Investigator – Katie Bigham (Trainee Clinical Psychologist)

***consent forms, demographic sheet and information sheets attached**

References

American Psychological Association. (2000). *Diagnostic and statistical manual of mental disorders.* Washington, DC: American Psychiatric Association.

Anastropoulos, A., Barkley, R., & Shelton, T. (1996). Family based treatment: Psychosocial intervention for children and adolescents with attention deficit hyperactivity disorder. In E. Hibbs and P. Jensen (eds.), *Psychosocial treatments for Child and Adolescent Disorders: Empirically Based Strategies for Clinical Practice* (pp. 267-284). Washington, DC: American Psychiatric Association.

Anderson, J., Williams, S., McGee, R., & Silva, P. (1987). DSM III disorders in pre-adolescent children. *Archives of General Psychiatry,* 44, 69-76.

Barkley, R. (1990). *Attention Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment.* New York: Guildford.

Barkley, R. (1998). *Attention Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment,* 2nd ed. New York: Guildford.

Biederman, J., Faraone, S., & Keenan, K et al. (1992). Further evidence for family-genetic risk factors in attention deficit hyperactivity disorder: patterns of comorbidity in probands and relatives psychiatrically and pediatrically referred samples. *Archives of General Psychiatry,* 49: 728-738.

Cantwell, D. & Hannah, G. (1989). Attention deficit hyperactivity disorder: In A. Tasmin, R. Hales, & A. Frances (eds.), *Review of Psychiatry* (pp. 137-161). Washington, DC: American Psychiatric Association.

Campbell, S. et al. (1982). A multi-dimensional assessment of parent identified behaviour problem toddlers. *Journal of Abnormal Child Psychology,* 10(4), 569-591.

Carr, A. (1999). *The Handbook of Child and Adolescent Clinical Psychology: A contextual approach.* London: Routledge.

Cohen, J. (1992). Quantitative methods in psychology: A power primer. *Psychological Bulletin,* 112(1), 155-159.

Cohen, P., Cohen, J., Kasen, S., Velez, C., Hartmark, C., Johnson, R., Rojas, M., Brook, J., & Streuning, E. (1993). An epidemiological study of disorders in late childhood and adolescence. *Journal of Child Psychology and Psychiatry,* 34, 851-867.

Dekker, M., Koot, H., & Verhulst, F. (2002). Emotional and behavioural problems in children and adolescents with and without intellectual disability. *Journal of Intellectual Disability Research,* 43, 1087-1098.

Dekker, M. & Koot, H (2003). DSM-IV Disorders in Children With Borderline to Moderate Intellectual Disability. 1: Prevalence and Impact. *Journal of the American Academy of Child and Adolescent Psychiatry,* 42(8), 915-922.

Emerson, E. (2003). Prevalence of psychiatric disorders in children and adolescents with and without intellectual disability. *Journal of Intellectual Disability Research,* 47(1), 51-58.

Fergusson, D., Horwood, J., & Lynskey, M. (1993). Prevalence and comorbidity of DSM-III-R diagnoses in a birth cohort of 15 year olds. *Journal of the American Academy of Child and Adolescent Psychiatry,* 32, 1127-1134.

Gadow, K. (1992). Paediatric psychopharmacology: A review of recent research. *Journal of Child Psychology and Psychiatry,* 33, 153-195.

Green, G., & Chee, K. (1994). Management of attention deficit disorder: A personal perspective. *Modern Medicine,* 37, 38-53.

Hinshaw, S. (1994). *Attention deficits and hyperactivity in children.* Thousand Oaks, CA: Sage.

Koller, H., Richardson, S., Katz, M., & McLaren, J. (1982). Behaviour disturbance in childhood and the early adult years in populations who were and were not mentally retarded. *Journal of prev. Psychiatry,* 1, 453-468.

Linna, S., Moilanen, I., & Ebeling, H. et al (1999). Psychiatric symptoms in children with intellectual disability. *European Child Adolescent Psychiatry,* 8, 77-82.

Mannuzza, S., Klein, R., Bessler, A., Malloy, P., & Lapadula, M. (1993). Adult outcome of adolescent boys: educational achievement, occupational rank, and psychiatric status. *Archive of General Psychiatry,* 50, 565-576.

McArdle, P., O'Brien, G., & Kolvin, I. (1995). Hyperactivity: Prevalence and relationship with conduct disorder. *Journal of Child Psychology and Psychiatry,* 36, 274-304.

McCracken, J. (1991). A two part model of stimulant action on attention deficit hyperactivity disorder in children. *Journal of Neuropsychiatry,* 3, 201-209.

Mellor, D., Storer, S., & Brown, J. (1996). Attention deficit hyperactivity disorder: Perceptions, practice and politics. *Journal of Paediatric Child Health,* 32, 218-222.

Pelman, W. & Hoza, B. (1992). Behavioural intervention for attention deficit disorder. In S. Turner, K, Calhoun, & H. Adams (eds.), *Handbook of Clinical Behaviour Therapy* (vol. 2, pp. 259-283). New York: Wiley.

Rutter, M., Tizard, J., & Whitmore, K. (1970). *Education, Health and Behaviour.* London: Longman.

Scahill, L., Schwab-Stone, M., Merikangus, K., Leckman, J., Zhang, H., & Kasl, S. (1999). Psychosocial and Clinical Correlates of ADHD in a Community Sample of School-Age Children. *Journal of the American Academy of Child and Adolescent Psychiatry,* 38(8), 976-984.

Stevenson, J. (1992). Evidence for a genetic aetiology of hyperactivity in children. *Behaviour Genetics,* 22, 337-343.

Solanto, M., Abikoff, H., Sonuga-Barke, E., Schachar, R., Logan, G., Wigal, T., Hechtman, L., Hinshaw, S., & Turkel, E. (2001). The Ecological Validity of Dealy Aversion and Response Inhibition as Measures of Impulsivity in ADHD: A Supplement to the NIMH Multimodal Treatment Study of ADHD. *Journal of Abnormal Child Psychology,* 29(3), 215-228.

Sonuga-Bark, E., Dalen, L., Daley, D., & Remington, B. (2002). Are Planning, Working Memory, and Inhibition Associated with Individual Differences in Preschool ADHD Symptoms? *Developmental Neuropsychology,* 21(3), 255-272.

Sonuga-Barke, Dalen, L., & Remington, B. (2003). Do Executive Deficits and Delay Aversion Make Independent Contributions to Preschool Attention Deficit Hyperactivity Disorder Symptoms? *Journal of the American Academy of Child and Adolescent Psychiatry,* 42(11), 1335-1342.

Szatmari, P., Offord, D., Boyle, M. (1989). Correlates, associated impairments and patterns of service utilisation of children with attention deficit disorder: findings from the Ontario Child Health Study. *Journal of Child Psychology and Psychiatry,* 30, 205-217.

Tannock, R. (1998). Attention deficit hyperactivity disorder: Advances in cognitine, neurobiological and genetic research. *Journal of Child Psychology and Psychiatry,* 39, 65-100.

Taylor, R. (1994). Symdromes of attention deficit and overactivity. In M. Rutter, E. Taylor, and L. Hersov (eds.), *Child and Adolescent Psychiatry: Modern Approaches* (3rd ed., pp. 258-307. Oxford: Blackwell.

Verhulst, F., & van der Ende, J. (1997). Factors associated with child mental health service use in the community. *Journal of the American Academy of Child and Adolescent Psychiatry,* 36, 901-909.

SECTION 2

APPEDICES

APPENDIX A

PARENT INFORMATION SHEET (ENGLISH AND WELSH VERSIONS)

Information for parents – stage 1

Gwybodaeth i Rieni

1. Teitl yr Astudiaeth

Ymddygiadau gorfywiog mewn plant gydag anabledd dysgu ysgafn.

2. Tîm Ymchwil

Katie Bigham	Seicolegydd Clinigol dan Hyfforddiant, Cwrs Seicoleg Glinigol Gogledd Cymru, Bangor, LL57 2DG
Dr Richard Hastings	Cyfarwyddwr Ymchwil, Cwrs Seicoleg Glinigol Gogledd Cymru, Bangor, LL57 2DG
Dr Dave Daley	Tiwtor Ymchwil, Cwrs Seicoleg Glinigol Gogledd Cymru, Bangor, LL57 2DG

3. Beth yw diben yr astudiaeth?

Rydym yn chwilio am deuluoedd plant gydag anableddau dysgu ysgafn i gymryd rhan yn ein project ymchwil. Mae gennym ddiddordeb mewn edrych pa mor gyffredin yw ymddygiad gorfywiog mewn plant gydag anabledd dysgu ysgafn yng Ngogledd Cymru a ph'run a oes angen am fwy o gymorth a chefnogaeth i deuluoedd yn yr ardal. Mae'r astudiaeth mewn dau gam, ac er na ofynnir i'r holl deuluoedd a gymerodd ran yn y cam cyntaf gymryd rhan yn yr ail gam, gwerthfawrogir pob cyfraniad yn fawr. Bydd teuluoedd y gofynnir iddynt gymryd rhan yn ail gam yr astudiaeth yn cael taflen wybodaeth arall bryd hynny yn disgrifio beth y bydd yn ei olygu.

I grynhoi, amcan yr astudiaeth drwodd a thro yw edrych ar dri mater nad ydynt wedi cael fawr o sylw hyd yma:

4. Amcangyfrif pa mor gyffredin yw ymddygiad gorfywiog ymysg plant sy'n mynychu ysgolion i rai gydag anableddau dysgu ysgafn yng Ngogledd Cymru (Cam 1).

5. Edrych ar sut y mae plant gydag anabledd dysgu ac ymddygiadau gorfywiog uwch yn gwahaniaethu oddi wrth blant gydag anabledd dysgu a lefelau is o ymddygiadau gorfywiog (Cam 2).

6. Edrych ar y berthynas rhwng adroddiadau ysgrifenedig o ymddygiad y plentyn ac arsylwadau uniongyrchol o'r plentyn yn yr ysgol (Cam 2).

4. Gwahoddiad i gymryd rhan

Rydym yn chwilio am rieni plant gydag anableddau dysgu ysgafn i lenwi holiadur a fydd yn ein helpu i weld presenoldeb ymddygiadau gorfywiog ac i roi caniatâd i athro/athrawes eu plentyn i lenwi'r un holiadur ar ymddygiad eu plentyn.

Ar ôl hyn gofynnir i nifer o deuluoedd gymryd rhan yn ail gam y project. Bydd hyn yn cynnwys cynnal cyfweliad byr â chi dros y ffôn a Katie Bigham yn ymweld â'ch plentyn yn yr ysgol i gynnal ychydig o dasgau yno. Gêm debyg i 'Beth mae Simon yn ei ddweud', a gemau cyfrifiadurol syml, fydd y tasgau hyn.

Darllenwch weddill y daflen wybodaeth hon yn ofalus a llenwi'r ffurflen ganiatâd amgaeedig os oes gennych ddiddordeb yn ein helpu gyda'r ymchwil hon (mae un ffurflen ganiatâd i chi i'w chadw, tra mae'n rhaid dychwelyd y llall gyda'r holiadur). Os nad yw rhywbeth yn glir i chi, neu os hoffech gael mwy o wybodaeth cyn i chi benderfynu, cysylltwch â ni drwy e-bost os gwelwch yn dda (gan roi eich rhif ffôn cyswllt) a byddwn yn eich ffonio i ateb unrhyw gwestiynau sydd gennych.

Os nad ydych eisiau cymryd rhan, gellwch daflu'r wybodaeth hon – diolch i chi.

5. Beth yw manteision cymryd rhan yn yr ymchwil?

Prif fanteision cymryd rhan yn ein project yw cynyddu ein hymwybyddiaeth o ba mor gyffredin yw ymddygiadau gorfywiog ymysg plant gydag anableddau dysgu ysgafn yng Ngogledd Cymru. Ychydig rydym yn ei wybod pa mor gyson yw ymddygiadau gorfywiog ymysg plant gydag anableddau dysgu ysgafn a sut mae eu teuluoedd yn ymdopi â hynny. Trwy gymryd rhan byddech yn cynyddu'r wybodaeth hon a helpu gweithwyr proffesiynol i asesu p'run a oes angen cynyddu'r help a'r gefnogaeth sydd ar gael i deuluoedd.

6. Beth yw peryglon cymryd rhan?

Nid ydym yn credu y byddwch chi na'ch plentyn mewn unrhyw berygl o gael niwed trwy gymryd rhan yn ein project.

7. Oes raid i chi gymryd rhan?

Chi sydd i benderfynu p'run a ydych chi a'ch plentyn yn fodlon cymryd rhan. Os penderfynwch gymryd rhan, a fyddech cystal â llofnodi'r ffurflenni caniatâd amgaeedig a dychwelyd un ynghyd â'r Holiadur Cryfderau ac Anawsterau (HCA) a'r daflen wybodaeth

am y teulu i'ch ysgol. Hyd yn oed wedyn gellwch dynnu'n ôl o'r ymchwil unrhyw bryd a heb roi rheswm. Gellwch gadw'r daflen wybodaeth hon.

8. Beth fydd yn digwydd i chi os cymerwch ran?

Ar ôl i chi ddychwelyd y ffurflen ganiatâd, y daflen wybodaeth am y teulu a'r HCA i'ch ysgol, byddwn yn:

1. Gofyn i athro dosbarth eich plentyn lenwi'r HCA ar ymddygiad eich plentyn.
2. Os cewch eich dewis, cysylltir â chi eto o fewn ychydig wythnosau i ofyn a fyddech yn fodlon cymryd rhan yn rhan olaf yr astudiaeth. Os byddwch, bydd Katie Bigham yn cynnal cyfweliad byr â chi dros y ffôn ac ymwelir â'ch plentyn yn yr ysgol er mwyn cynnal ychydig o dasgau yno.
3. Ar ddiwedd yr ymchwil byddwch chi ac ysgol eich plentyn yn cael crynodeb o ddarganfyddiadau'r ymchwil.

Os na chewch ei dewis ar gyfer cam 2, mae croeso i chi gysylltu â ni trwy lythyr i ofyn am grynodeb o ddarganfyddiadau'r ymchwil pan fydd wedi ei chwblhau.

9. Beth sy'n rhaid i chi ei wneud yn awr?

Os penderfynwch gymryd rhan yn ein hastudiaeth ar ôl i chi ddarllen y daflen wybodaeth hon, dychwelwch un ffurflen ganiatâd wedi ei llofnodi, y daflen wybodaeth am y teulu a'r HCA at athro eich plentyn os gwelwch yn dda. Fodd bynnag, os penderfynwch unrhyw bryd yn ystod yr astudiaeth nad ydych eisiau cymryd rhan ynddi mwyaf, gellwch dynnu'n ôl. Os penderfynwch felly, byddai o gymorth pe gallech roi gwybod i athro eich plentyn, a fydd wedyn yn rhoi gwybod i ni. Os penderfynwch beidio â chymryd rhan o gwbl gellwch daflu'r pecyn hwn. Nid oes angen i chi gysylltu â ni.

Bydd yr holl wybodaeth a roddwch i ni'n cael ei thrin yn hollol gyfrinachol a'i chadw mewn cwpwrdd ffeilio diogel wedi ei gloi ym Mhrifysgol Cymru Bangor.Ni fydd dim o'r wybodaeth a roddwch yn cael ei defnyddio mewn unrhyw ffordd a allai eich adnabod chi fel teulu. Bydd canlyniadau'r astudiaeth yn disgrifio darganfyddiadau cyffredinol ac nid gwybodaeth am blant unigol. Yr unig adeg pryd y byddai'n rhaid torri cyfrinachedd yw pe baech yn rhoi gwybodaeth ynglŷn â niwed posibl i'ch plentyn i'r ymchwilwyr yn ystod y cyfweliad.

Os oes gennych unrhyw bryderon ynglŷn ag ymddygiad eich plentyn o ran gorfywiogrwydd, bydd taflen wybodaeth a llawlyfr hyfforddi rhieni ar gael. Mae'r rhain yn nodi rhai o'r strategaethau ymddygiad y gwyddom sy'n ddefnyddiol i helpu rhieni i ddelio â thrafferthion ymddygiad eu plentyn.

Os mai Cymraeg yw eich dewis iaith, hoffem ymddiheuro mai yn Saesneg y bydd yr holiadur/cyfweliadau. Nid yw'n bosibl eu cyfieithu i'r Gymraeg oherwydd y perygl o golli agweddau pwysig ar eu hystyr trwy gyfieithu. Rydym yn gobeithio y byddwch yn fodlon cymryd rhan yn yr ymchwil hon yn Saesneg ond rydym yn deall os byddai'n well gennych beidio.

10. Manylion pellach

Mae ein manylion isod os hoffech gysylltu â ni:

Katie Harman
Seicolegydd Clinigol dan Hyfforddiant
CSGGC
Prifysgol Cymru
Ffordd y Coleg
Bangor

Dr Dave Daley
Tiwtor Ymchwil (Goruchwyliwr)
CSGGC
Prifysgol Cymru
Ffordd y Coleg
Bangor.

Os oes gennych unrhyw gwynion ynglŷn â'r ffordd y cynhelir yr ymchwil hon, mae croeso i chi gyfeirio pryderon na chawsant eu datrys at:

Yr Athro Fergus Lowe
Pennaeth yr Ysgol Seicoleg
Prifysgol Cymru Bangor
Bangor
Gwynedd
LL57 2AS

Information for Parents – stage 1

1. Study Title

Hyperactive behaviours in children with intellectual disability.

2. Research Team

Katie Bigham	Trainee Clinical Psychologist, North Wales Clinical Psychology Course, Bangor, LL57 2DG
Dr Richard Hastings	Research Director, North Wales Clinical Psychology Course, Bangor, LL57 2DG
Dr Dave Daley	Research Tutor, North Wales Clinical Psychology Course, Bangor, LL57 2DG

3. What is the purpose of the study?

We are looking for families of children with mild learning disabilities to take part in our research project. We are interested in looking at how common hyperactive behaviour is in children with a mild learning disability in North Wales and whether there is a need for increased help and support for families in the area. The study is in two stages, and although not all families taking part in the first stage will be approached for their continued participation in the second, all participation is greatly valued. For the families that are asked to take part in the second stage of the study an information sheet describing what is involved will be issued at this time.

To summarise, the overall study aims to look at three issues that have received little attention to date:

7. Estimate how common hyperactive behaviour in children attending schools for individuals with mild learning disabilities is in North Wales (Stage 1).

8. Look at how children with a learning disability and increased hyperactive behaviours differ from children with a learning disability and lower levels of hyperactive behaviours (Stage 2).

9. Look at the relationship between written reports of the child's behaviour and direct observations of the child in school (Stage 2).

4. Invitation to participate

We are looking for parents of children with mild learning disabilities to complete a questionnaire that will help us identify the presence of hyperactive behaviours, and give permission for their child's teacher to complete the same questionnaire on their child's behaviour.

After this, a number of families will be asked to take part in the second stage of the project. This will involve a short telephone interview with yourself, your child being visited at school by Katie Bigham to undertake a couple of tasks at school. These tasks are a game similar to 'What Simon says', and a simple computer game.

Please read the remainder of this information sheet carefully and complete the consent form enclosed if you are interested in helping us with this research (one consent form is for you to keep, whilst the other must be returned with the completed questionnaire). If there is anything that is not clear, or you would just like more information before you decide, please contact us by mail (including your telephone contact number) and we will telephone you and answer any questions that you may have.

If you do not wish to take part, simply discard this information – thank you.

5. What are the benefits of taking part in the research?

The main benefits of taking part in our project is increase our awareness of how commonly hyperactive behaviours are experienced by children with mild learning disabilities in North Wales. There is little known about how frequently hyperactive behaviours are experienced by children with mild learning disabilities, and coped with by their families. Your participation would help increase this knowledge and help professionals assess whether an increase in the help and supportive available to families is needed.

6. What are the risks of taking part?

We do not believe that you or your child is at any risk of harm as a result of taking part in our project.

7. Do you have to take part?

It is up to you to decide whether you and your child are willing to participate. If you decide to take part, could you please sign the enclosed consent forms, and return one along with the completed Strengths and Difficulties Questionnaire (SDQ) and family information sheet to

your school. You are still free to withdraw from the research at any time, and without giving a reason. This information sheet can be kept by you.

8. What will happen to you if you take part?

After you have returned the consent form, family information sheet and the SDQ to your school, we will:

4. Ask your child's class teacher to complete the SDQ on your child's behaviour.
5. If chosen, you will be contacted again within a few weeks to be asked whether you will take part in the final part of the study. If so, Katie Bigham will conduct a short telephone interview with you, and your child will be visited at school in order to take part in a couple of tasks at school.
6. At the end of the research a research summary will be issued to you and your child's school outlining what we found.

If you are not selected for stage 2, you are welcome to contact us by letter and request to receive the research summary when the research is completed.

9. What do you have to do now?

If when you have read this information sheet you decide to take part in our study, please sign and return one consent form, the completed family information sheet and SDQ to your child's teacher. However, if at any time during the study you decide that you no longer wish to take part in the study, you can withdraw. If this is what you decide to do, it would be helpful if you could let your child's teacher know, who will then inform us. If you decide not to take part at all, please discard this pack. You do not need to make contact with us.

All the information that you give us will be treated as strictly confidential, and will be kept securely locked in a filing cabinet in the University of Wales Bangor. None of the information that you provide will be used in any way that would identify you as a family. Results of the study will describe overall findings and not information about individual children. The only time when confidentiality would have to be broken would be if you gave information, regarding possible harm to your child, to the investigators during the interview process.

If you have any concerns regarding your child's behaviour with regards to hyperactivity, an information sheet and a parent-training manual will be available. These outline some

behavioural strategies that are known to be useful in helping parents deal with their child's behavioural difficulties.

If your preferred language is Welsh, we would like to apologise for the fact that the questionnaire/interviews will be in English. It is not possible to translate them into Welsh due to the risk of losing important aspects of their meaning through translation. We hope that you will be willing to participate in this research using English but understand that you may wish not to do so.

10. Further details

If you want to contact us, our details are below:

Katie Harman
Trainee Clinical Psychologist
NWCPP
University of Wales
University of Wales
College Road
Bangor

Dr Dave Daley
Research Tutor (Supervisor)
NWCPP
University of Wales
University of Wales
College Road
Bangor.

If you have any complaints about the way that this research is being conducted you are welcome to address unresolved concerns to:

Professor Fergus Lowe
Head of the School of Psychology
University of Wales Bangor
Bangor
Gwynedd
LL57 2AS

Information for parents – stage 2

Gwybodaeth i Rieni – Cam 2

1. Teitl yr Astudiaeth

Ymddygiadau gorfywiog mewn plant gydag anabledd dysgu ysgafn.

2. Tîm Ymchwil

Katie Bigham	Seicolegydd Clinigol dan Hyfforddiant, Cwrs Seicoleg Glinigol Gogledd Cymru, Bangor, LL57 2DG
Dr Richard Hastings	Cyfarwyddwr Ymchwil, Cwrs Seicoleg Glinigol Gogledd Cymru, Bangor, LL57 2DG
Dr Dave Daley	Tiwtor Ymchwil, Cwrs Seicoleg Glinigol Gogledd Cymru, Bangor, LL57 2DG

3. Beth yw diben cam 2 yr astudiaeth?

Rydym yn gwahodd nifer o deuluoedd, a gymerodd ran yng ngham cyntaf ein hastudiaeth, i gymryd rhan yn y cam terfynol. Dewiswyd teuluoedd ar hap a'u rhoi mewn dau grŵp: yn dibynnu ar lefelau ymddygiad gorfywiog y rhoddwyd gwybod amdanynt yng ngham 1. Yn aml mae gan blant ag anableddau dysgu ysgafn eisoes fwy o rwystrau ac anawsterau i'w wynebu mewn bywyd bob dydd, heb brofi symptomau gorfywiogrwydd hefyd. Mae gennym ddiddordeb mewn gweld sut mae gorfywiogrwydd yn effeithio ar blant gydag anabledd dysgu ysgafn a ph'run y byddai help a chefnogaeth gynharach o gymorth i leihau anawsterau ychwanegol. Mae'r rhan fwyaf o ymchwil wedi canolbwyntio ar blant heb anabledd dysgu. Trwy gymryd rhan byddwch yn cyfrannu at wella ein gwybodaeth a'n dealltwriaeth o'r maes hwn.

Bydd cam terfynol yr astudiaeth yn edrych yn benodol ar p'run a ydyw plant gydag anableddau dysgu a gorfywiogrwydd yn dangos yr un symptomau â phlant heb anableddau dysgu. Trwy gynyddu ein dealltwriaeth o orfywiogrwydd mewn plant gydag anableddau dysgu gellir dechrau gwneud gwelliannau o ran yr help a'r gefnogaeth a gynigir i blant a'u teuluoedd.

I grynhoi, byddwn yn edrych ar y canlynol:

1. Sut mae ymddygiad plant gydag anableddau dysgu a lefelau uchel o orfywiogrwydd yn wahanol i ymddygiad plant gydag anableddau dysgu a lefelau isel o orfywiogrwydd.

2. Ymchwilio i'r berthynas rhwng adroddiadau rhieni ac athro ar ymddygiad y plentyn a mesurau ymddygiadol uniongyrchol a ddefnyddir gyda'r plentyn yn yr ysgol.

4. Gwahoddiad i gymryd rhan

Rydym yn chwilio am rieni plant gydag anableddau dysgu ysgafn a gymerodd ran yng Ngham 1 ein hastudiaeth, i gymryd rhan yn y cam terfynol. Bydd hyn yn cynnwys cyfweliad byr (tua 30 munud) ar y ffôn gyda Katie Bigham a fydd yn gofyn cwestiynau i chi am ymddygiad eich plentyn o ddydd i ddydd. Hefyd gofynnir i'ch plentyn wneud ychydig o dasgau yn yr ysgol.

Gêm debyg i 'Beth mae Simon yn ei ddweud', a gemau cyfrifiadurol syml, fydd y tasgau hyn a bydd Katie Bigham yn gweithio gyda'ch plentyn yn unigol. Bydd un arall yn edrych ar y broses hon lle bydd hynny'n bosibl a chaiff ei recordio ar fideo.

Darllenwch weddill y daflen wybodaeth hon yn ofalus cyn penderfynu p'run a ydych am gymryd rhan ai peidio. Os nad yw rhywbeth yn glir i chi, neu os hoffech gael mwy o wybodaeth cyn i chi benderfynu, cysylltwch â ni drwy e-bost os gwelwch yn dda (gan roi eich rhif ffôn cyswllt) a byddwn yn eich ffonio i ateb unrhyw gwestiynau sydd gennych.

Os nad ydych eisiau cymryd rhan, gellwch daflu'r wybodaeth hon – diolch i chi.

5. Beth yw manteision cymryd rhan yn yr ymchwil?

Prif fanteision cymryd rhan yn ein project yw cynyddu ein hymwybyddiaeth o ba mor gyffredin yw ymddygiadau gorfywiog ymysg plant gydag anableddau dysgu ysgafn yng Ngogledd Cymru. Ychydig rydym yn ei wybod pa mor gyson yw ymddygiadau gorfywiog ymysg plant gydag anableddau dysgu ysgafn a sut mae eu teuluoedd yn ymdopi â hynny. Trwy gymryd rhan byddech yn cynyddu'r wybodaeth hon a helpu gweithwyr proffesiynol i asesu p'run a oes angen cynyddu'r help a'r gefnogaeth sydd ar gael i deuluoedd.

6. Beth yw peryglon cymryd rhan?

Nid ydym yn credu y byddwch chi na'ch plentyn mewn unrhyw berygl o gael niwed trwy gymryd rhan yn ein project.

7. Oes raid i chi gymryd rhan?

Chi sydd i benderfynu p'run a ydych chi a'ch plentyn yn fodlon cymryd rhan. Os penderfynwch gymryd rhan, a fyddech cystal â llofnodi'r ffurflenni caniatâd amgaeedig a

dychwelyd un atom ni yn yr amlen a ddarparwyd. Hyd yn oed wedyn gellwch dynnu'n ôl o'r ymchwil unrhyw bryd a heb roi rheswm. Gellwch gadw'r daflen wybodaeth hon.

8. Beth fydd yn digwydd i chi os cymerwch ran?

Ar ôl i chi ddychwelyd y ffurflen ganiatâd, byddwn yn:

1. Cysylltu â chi i gynnal cyfweliad ffôn byr ynglŷn ag ymddygiad eich plentyn gartref.
2. Gyda'ch caniatâd, bydd Katie Bigham yn ymweld â'ch plentyn yn yr ysgol i gynnal tasgau
3. Unwaith y gorffennir yr astudiaeth, anfonir crynodeb atoch chi ac i ysgol eich plentyn yn nodi'r darganfyddiadau cyffredinol. Ni thrafodir unigolion yn fanwl.

9. Beth sy'n rhaid i chi ei wneud yn awr?

Os penderfynwch gymryd rhan yn ein hastudiaeth ar ôl i chi ddarllen y daflen wybodaeth hon, dychwelwch un ffurflen ganiatâd wedi ei llofnodi. Ar ôl derbyn y ffurflen ganiatâd bydd yr ymchwilydd yn cysylltu â chi cyn gynted â phosibl. Fodd bynnag, os penderfynwch unrhyw bryd yn ystod yr astudiaeth nad ydych eisiau cymryd rhan ynddi mwyaf, gellwch dynnu'n ôl ac ni ofynnir unrhyw gwestiynau. Os penderfynwch felly, byddai o gymorth pe gallech roi gwybod i athro eich plentyn, a fydd wedyn yn rhoi gwybod i ni. Os penderfynwch beidio â chymryd rhan o gwbl yn y cam nesaf gellwch daflu'r pecyn hwn. Nid oes angen i chi gysylltu â ni.

Bydd yr holl wybodaeth a roddwch i ni'n cael ei thrin yn hollol gyfrinachol a'i chadw mewn cwpwrdd ffeilio diogel wedi ei gloi ym Mhrifysgol Cymru Bangor.Ni fydd dim o'r wybodaeth a roddwch yn cael ei defnyddio mewn unrhyw ffordd a allai eich adnabod chi fel teulu. Bydd canlyniadau'r astudiaeth yn disgrifio darganfyddiadau cyffredinol ac nid gwybodaeth am blant unigol. Yr unig adeg pryd y byddai'n rhaid torri cyfrinachedd yw pe baech yn rhoi gwybodaeth ynglŷn â niwed posibl i'ch plentyn i'r ymchwilwyr yn ystod y cyfweliad.

Os oes gennych unrhyw bryderon ynglŷn ag ymddygiad eich plentyn o ran gorfywiogrwydd, bydd taflen wybodaeth a llawlyfr hyfforddi rhieni ar gael. Mae'r rhain yn nodi rhai o'r strategaethau ymddygiad y gwyddom sy'n ddefnyddiol i helpu rhieni i ddelio â thrafferthion ymddygiad eu plentyn. Gwaetha'r modd, ni ellir rhoi unrhyw adborth unigol i rieni. Fodd

bynnag, os gofynnir yn arbennig am adborth unigol, gellir rhoi adroddiad yn amlinellu cryfderau ac anawsterau eich plentyn a ganfuwyd o ganlyniad i'r tasgau a wnaeth eich plentyn yn yr ysgol, a'r cyfweliad ffôn byr. Bydd hyn yn cynnwys gwybodaeth am gryfderau ac anawsterau penodol megis chwarae/ymwneud â chyfoedion. Os hoffech gael y wybodaeth hon, cysylltwch â ni drwy'r post a byddwn yn anfon adroddiad atoch cyn gynted â phosibl.

Os mai Cymraeg yw eich dewis iaith, hoffem ymddiheuro mai yn Saesneg y bydd yr holiadur/cyfweliadau. Nid yw'n bosibl eu cyfieithu i'r Gymraeg oherwydd y perygl o golli agweddau pwysig ar eu hystyr trwy gyfieithu. Rydym yn gobeithio y byddwch yn fodlon cymryd rhan yn yr ymchwil hon yn Saesneg ond rydym yn deall os byddai'n well gennych beidio.

10. Manylion pellach

Mae ein manylion isod os hoffech gysylltu â ni:

Katie Harman
Seicolegydd Clinigol dan Hyfforddiant
CSGGC
Prifysgol Cymru
Ffordd y Coleg
Bangor

Dr Dave Daley
Tiwtor Ymchwil (Goruchwyliwr)
CSGGC
Prifysgol Cymru
Ffordd y Coleg
Bangor.

Os oes gennych unrhyw gwynion ynglŷn â'r ffordd y cynhelir yr ymchwil hon, mae croeso i chi gyfeirio pryderon na chawsant eu datrys at:

Yr Athro Fergus Lowe
Pennaeth yr Ysgol Seicoleg
Prifysgol Cymru Bangor
Bangor
Gwynedd
LL57 2AS

Information for Parents – Stage 2

1. Study Title

Hyperactive behaviours in children with a mild learning disability.

2. Research Team

Katie Bigham	Trainee Clinical Psychologist, North Wales Clinical Psychology Course, Bangor, LL57 2DG
Dr Richard Hastings	Research Director, North Wales Clinical Psychology Course, Bangor, LL57 2DG
Dr Dave Daley	Research Tutor, North Wales Clinical Psychology Course, Bangor, LL57 2DG

3. What is the purpose of stage 2 of the study?

We are inviting a number of families, who took part in the first stage of our study, to take part in the final stage. Families have been chosen randomly and are placed into one of two groups; dependent upon reported levels of hyperactive behaviour in stage 1. Children with mild learning disabilities often already have more hurdles and difficulties to overcome in everyday life, without experiencing symptoms of hyperactivity as well. We are interested in how hyperactivity affects children with a mild learning disability and whether earlier help and support could help reduce additional difficulties experienced. Most research about hyperactivity has focussed upon children without a learning disability. Your taking part will contribute to increasing our knowledge and understanding in this area.

Participation in the final stage of the study will focus on looking at whether children with learning disabilities and hyperactivity show the same symptoms as children without learning disabilities do. By increasing our understanding of hyperactivity in children with learning disabilities, improvements can begin to be made regarding the help and support offered to children and their families.

To summarise, we will be looking at:

10. How the behaviour of children with learning disabilities and high levels of hyperactivity is different from the behaviour of children with learning disabilities and low levels of hyperactivity.

11. Investigate the relationship between parental and teacher reports of the child's behaviour and direct behavioural measures undertaken with the child in school.

4. Invitation to participate

We are looking for parents of children with mild learning disabilities who took part in Stage 1 of our study, to take part in the final stage of our research. This will involve a short telephone interview (approximately 30 minutes) with Katie Bigham who will be asking you questions relating to your child's behaviour on a daily basis, and your child completing a couple of tasks at school. These tasks are a game similar to 'What Simon says', and a simple computer game and will involve the investigator, Katie Bigham, to work on a one-to-one basis with your child. Another will observe this process where possible and it will be videoed.

Please read the remainder of this information sheet carefully before deciding whether or not to take part. If there is anything that is not clear, or you would just like more information before you decide, please contact us by mail (including your telephone contact number) and we will telephone you to discuss the research further and answer any questions that you may have.

If you do not wish to take part, simply discard this information – thank you.

5. What are the benefits of taking part in the research?

The main benefits of taking part in our project is increase our awareness of how commonly hyperactive behaviours are experienced by children with mild learning disabilities in North Wales. There is little known about how frequently hyperactive behaviours are experienced by children with mild learning disabilities, and coped with by their families. Your participation would help increase this knowledge and help professionals assess whether an increase in the help and supportive available to families is needed.

6. What are the risks of taking part?

We do not believe that you or your child is at any risk of harm as a result of taking part in our study.

7. Do you have to take part?

It is up to you to decide whether you and your child are willing to take part. If you decide to, could you please sign the enclosed consent forms, and return one to us in the envelope

provided. You are still free to withdraw from the research at any time, and without giving a reason. This information sheet can be kept for your records.

8. What will happen to us if we take part?

After you have returned the consent form, we will:

1) Contact you to undertake a short telephone interview with you regarding your child's behaviour at home.

2) With your consent, your child will be visited by Katie Bigham, on a one-to-one basis, at school to do a couple of tasks.

3) Once the study is completed, a summary will be sent to you and your child's school outlining our general findings, individuals will not be discussed in detail.

9. What do we have to do now?

If after reading this information sheet you decide to continue your participation in our study, please sign and return one consent form. On receipt of the consent form, the investigator will be in touch as soon as possible. However, if at any time you wish to withdraw from the process, you can do so with no questions asked. If this is what you decide to do, it would be helpful if you could let your child's teacher know, who will then inform us. If you decide not to take part at all in the next stage please discard this pack. You do not need to make contact with us.

All the information that you give us will be treated as strictly confidential, and will be kept securely locked in a filing cabinet in the University of Wales Bangor. None of the information that you provide will be used in any way that would identify you as a family. Results of the study will describe overall findings and not information about individual children. The only time when confidentiality would have to be broken would be if you gave information, regarding possible harm to your child, to the investigators during the interview process.

If during the process of the study you have any concerns regarding your child's behaviour with regards to hyperactivity, an information sheet and a parent training manual will be available. These outline some behavioural strategies that are known to be useful in helping parents deal with their child's behavioural difficulties. Unfortunately, no individual feedback

can be given to parents. However, if individual feedback is specifically desired, a report can be given outlining your child's strengths and difficulties found as a result of the tasks done by your child at school, and the short telephone interview. This will include information about specific strengths and difficulties such as playing/interacting with peers. If this information is required please contact us by post and we will forward a report as quickly as possible.

If your preferred language is Welsh, we would like to apologise for the fact that the questionnaire/interviews will be in English. It is not possible to translate them into Welsh due to the risk of losing important aspects of their meaning through translation. We hope that you will be willing to participate in this research using English but understand that you may wish not to do so.

10. Further details

If you want to contact us, our details are below:

Katie Bigham
Trainee Clinical Psychologist
NWCPP
University of Wales
University of Wales
College Road
Bangor

Dr Dave Daley
Research Tutor (Supervisor)
NWCPP
University of Wales
University of Wales
College Road
Bangor.

If you have any complaints about the way that this research is being conducted you are welcome to address unresolved concerns to:

Professor Fergus Lowe
Head of the School of Psychology
University of Wales Bangor
Bangor
Gwynedd
LL57 2AS

APPENDIX B

CONSENT FORMS
(ENGLISH AND WELSH VERSIONS)

Consent form –stage 1

Ffurflen Caniatâd Ymchwil

Teitl y Project: Ymddygiadau gorfywiog mewn plant gydag anabledd dysgu ysgafn.

Llenwch y canlynol a dileu fel bo'r angen os gwelwch yn dda:

1) Ydych chi wedi darllen y Wybodaeth i Deuluoedd – taflen 1? DO/NADDO

2) Ydych chi wedi cael digon o wybodaeth am gam cyntaf yr astudiaeth?
DO/NADDO

3) Ydych chi'n deall y gellwch dynnu'n ôl o'r astudiaeth hon:

..unrhyw bryd
..heb roi rheswm am dynnu'n ôl
..heb i hynny effeithio ar unrhyw driniaeth rydych yn ei chael?
YDW/NAC YDW

4) Ydych chi'n rhoi caniatâd i ni ofyn i athro/athrawes eich plentyn lenwi'r Holiadur Cryfderau ac Anawsterau ynglŷn â'ch plentyn? YDW/NAC YDW

Rwy'n fodlon cymryd rhan yn yr astudiaeth hon. YDW/NAC YDW

Rwy'n rhoi caniatâd i chi gysylltu â mi ar gyfer cam 2 yr astudiaeth os bydd angen.
YDW/NAC YDW

Llofnod _____

Dyddiad _____

Enw mewn priflythrennau_____

Rhif Ffôn _____

Enw'r plentyn_____

Ysgol y plentyn_____

Diolch yn fawr.

Research Consent Form

Title of Project: Hyperactive behaviours in children with a mild learning disability.

Please complete the following and delete as necessary:

2) Have you read the Information for Families leaflet 1? YES/NO

4) Have you received enough information about the first stage of the study? YES/NO

5) Do you understand that you are free to withdraw from this study:

..at any time
..without giving a reason for withdrawing
..without affecting any treatment you receive? YES/NO

4) Do you give us permission to ask your child's teacher to complete the Strengths and Difficulties Questionnaire regarding your child? YES/NO

I am willing to participate in this study. YES/NO

I consent to being contacted for stage 2 of the study, if required. YES/NO

Signature_____

Date_____

Name in block letters_____

Telephone Number_____

Child's name_____

Child's school_____

Thank you.

Consent form – stage 2

Taflen Gwybodaeth Teuluoedd

Mae'r cwestiynau canlynol yn gofyn am wybodaeth gefndir amdanoch chi, eich plentyn gydag anabledd dysgu, a'ch teulu. Ticiwch y blychau priodol neu ysgrifennwch yn y mannau gwag a ddarparwyd. Llenwch bob adran mor onest ag y gellwch, os gwelwch yn dda, hyd yn oed os nad ydych yn hollol sicr neu os yw'r eitem yn swnio'n hurt!

1. Ydych chi'n wryw neu'n fenyw? Gwryw ☐ Benyw ☐

2. Faint oedd eich oed ar eich pen-blwydd diwethaf? _____

3. Beth yw eich statws priodasol ar hyn o bryd?

Priod, ac yn byw gyda gŵr/gwraig..... ☐

Byw gyda phartner ..... ☐

Wedi Ysgaru/Gwahanu/Sengl a DDIM yn byw gyda phartner..... ☐

4. Faint o bobl sy'n byw yn eich tŷ ar hyn o bryd? _____ Oedolion _____ Plant

5. Ticiwch y blychau wrth ochr y cymwysterau addysgol sydd gennych

Dim cymwysterau addysgol ffurfiol ..... ☐

TGAU, TAU, Lefel O neu gyfwerth ..... ☐

Lefel A, HNC, GNVQ neu gyfwerth ..... ☐

HND, Diploma arall, neu gyfwerth ..... ☐

Gradd gyffredin neu anrhydedd o Brifysgol/Polytechnig ..... ☐

Gradd Meistr neu Ddoethurol..... ☐

6. Oes gennych chi swydd y tu allan i'r cartref ar hyn o bryd? Oes ☐ Nac oes ☐

7. Beth yw eich perthynas â'ch plentyn ag anabledd dysgu (e.e. mam, tad, llysfam, nain, rhiant mabwysiadol)?

8. Faint yw oed eich plentyn ag anabledd dysgu? _____ blynyddoedd _____ misoedd

9. Ydi'ch plentyn ag anabledd dysgu yn wryw neu'n fenyw? Gwryw ☐ Benyw ☐

10. Ticiwch y blychau isod i ddangos unrhyw ddiagnoses/cyflyrau sydd hefyd yn berthnasol i'ch plentyn ag anabledd dysgu:

Awtistiaeth..... ☐

Parlys yr Ymennydd..... ☐

Syndrom Down ..... ☐

Syndrom arall (nodwch beth ydyw) _____

11. Oes gan eich plentyn ag anabledd dysgu nam ar y synhwyrau sy'n ymyrryd â'i fywyd/bywyd o ddydd i ddydd?

Oes ☐ Nac oes ☐

Os oes, beth yw'r nam hwn?

Nam ar y golwg? Oes ☐

Nam ar y clyw? Oes ☐

12. Ydi'ch plentyn ag anabledd dysgu yn dioddef oddi wrth ffitiau epileptig ar hyn o bryd?

☐ ☐

Ydi N Nac ydi

13. Oes gan eich plentyn ag anabledd dysgu broblemau gyda symud sy'n ei gwneud yn anodd iddynt symud o gwmpas yn annibynnol (e.e. angen defnyddio cadair olwyn)? Oes ☐ Nac oes ☐

14. Oes gan eich plentyn ag anabledd dysgu unrhyw broblemau iechyd eraill na nodwyd yn barod? Oes ☐ Nac oes ☐

Os oes, nodwch os gwelwch yn dda _____

15. Beth yw cod post eich cartref? _____

Diolch i chi am gymryd rhan ac am eich gonestrwydd.

Research Consent Form (2)

Title of Project: Hyperactive behaviours in children with a mild learning disability.

Please complete the following and delete as necessary:

3) Have you read the Information for Families leaflet 2? YES/NO

6) Have you received enough information about the second stage of the study? YES/NO

7) Do you understand that you are free to withdraw from this study:

..at any time
..without giving a reason for withdrawing
..without affecting any treatment you receive? YES/NO

4) Do you give us permission to visit your child in school to undertake a couple of tasks involved in the research (outlined in the Information for families' leaflet 2)? YES/NO

I am willing to participate in this study. YES/NO

Signature_____

Date_____

Name of child_____

School attended_____

Home postcode_____

Home telephone number_____

Please let us know the best time to telephone you to undertake the short telephone interview (please tick):

	Morning	Afternoon	Early evening	After 8.00pm
Monday				
Tuesday				
Wednesday				
Thursday				
Friday				

APPENDIX C

MEASURES AND MATERIALS

Family Information sheet

The following questions ask for background information about you, your child with a learning disability, and your family. Please tick the appropriate boxes or write in the spaces provided. Please complete each section as honestly you can, even if you are not absolutely certain or the item seems daft!

1. Are you male or female? Male ☐ Female ☐

2. What was your age in years on your last birthday? _____

3. What is your current marital status?

Married, and living with spouse..... ☐

Living with partner..... ☐

Divorced/Separated/Single and NOT living with a partner..... ☐

4. In total how many people currently live in your house? _____ Adults _____ Children

5. Please tick the boxes next to all of the educational qualifications that you hold

No formal educational qualifications..... ☐

GCSE, CSE, GCE, O Levels or equivalent..... ☐

GCE, A Levels, HNC, GNVQ or equivalent..... ☐

HND, other Diploma, or equivalent..... ☐

Polytechnic/University ordinary or honours degree..... ☐

Masters or Doctoral degree..... ☐

6. Do you currently have a job outside of the home? Yes ☐ No ☐

7. What is your relationship to your child with a learning disability (e.g., mother, father, stepmother, grandmother, adoptive parent)?

8. How old is your child with a learning disability?_____ years _____ months

☐

9. Is your child with a learning disability male or female? Male ☐ Female ☐

10. Please tick the boxes below to indicate any diagnoses/conditions that also apply to your child with a learning disability:

Autism..... ☐

Cerebral Palsy..... ☐

Down Syndrome..... ☐

Other syndrome (please specify) _____

11. Does your child with a learning disability have sensory impairment that interferes with his/her day to day living?

Yes ☐ No ☐

If yes, what is this impairment?

Visual impairment? Yes ☐

Hearing impairment? Yes ☐

12. Does your child with a learning disability currently suffer from epileptic fits?

Yes ☐ No ☐

13. Does your child with a learning disability have problems with mobility that means it is difficult for them to move around independently (e.g. needs to use a wheelchair)?

Yes ☐ No ☐

14. Does your child with a learning disability have any other health problems not already mentioned?

Yes ☐ No ☐

If yes, then please specify_____

Thank you for your participation and honesty.

Demographic sheet

Taflen Gwybodaeth Teuluoedd

Mae'r cwestiynau canlynol yn gofyn am wybodaeth gefndir amdanoch chi, eich plentyn gydag anabledd dysgu, a'ch teulu. Ticiwch y blychau priodol neu ysgrifennwch yn y mannau gwag a ddarparwyd. Llenwch bob adran mor onest ag y gellwch, os gwelwch yn dda, hyd yn oed os nad ydych yn hollol sicr neu os yw'r eitem yn swnio'n hurt!

1. Ydych chi'n wryw neu'n fenyw? Gwryw ☐ Benyw ☐

2. Faint oedd eich oed ar eich pen-blwydd diwethaf? _____

3. Beth yw eich statws priodasol ar hyn o bryd?

Priod, ac yn byw gyda gŵr/gwraig..... ☐

Byw gyda phartner ..... ☐

Wedi Ysgaru/Gwahanu/Sengl a DDIM yn byw gyda phartner..... ☐

4. Faint o bobl sy'n byw yn eich tŷ ar hyn o bryd? _____ Oedolion _____ Plant

5. Ticiwch y blychau wrth ochr y cymwysterau addysgol sydd gennych

Dim cymwysterau addysgol ffurfiol ..... ☐

TGAU, TAU, Lefel O neu gyfwerth ..... ☐

Lefel A, HNC, GNVQ neu gyfwerth ..... ☐

HND, Diploma arall, neu gyfwerth ..... ☐

Gradd gyffredin neu anrhydedd o Brifysgol/Polytechnig ..... ☐

Gradd Meistr neu Ddoethurol..... ☐

6. Oes gennych chi swydd y tu allan i'r cartref ar hyn o bryd? Oes ☐ Nac oes ☐

7. Beth yw eich perthynas â'ch plentyn ag anabledd dysgu (e.e. mam, tad, llysfam, nain, rhiant mabwysiadol)?

8. Faint yw oed eich plentyn ag anabledd dysgu? _____ blynyddoedd _____ misoedd

9. Ydi'ch plentyn ag anabledd dysgu yn wryw neu'n fenyw? Gwryw ☐ Benyw ☐

10. Ticiwch y blychau isod i ddangos unrhyw ddiagnoses/cyflyrau sydd hefyd yn berthnasol i'ch plentyn ag anabledd dysgu:

Awtistiaeth..... ☐

Parlys yr Ymennydd..... ☐

Syndrom Down ..... ☐

Syndrom arall (nodwch beth ydyw) _____

11. Oes gan eich plentyn ag anabledd dysgu nam ar y synhwyrau sy'n ymyrryd â'i fywyd/bywyd o ddydd i ddydd?

Oes ☐ Nac oes ☐

Os oes, beth yw'r nam hwn?

Nam ar y golwg? Oes ☐

Nam ar y clyw? Oes ☐

12. Ydi'ch plentyn ag anabledd dysgu yn dioddef oddi wrth ffitiau epileptig ar hyn o bryd?

Ydi ☐ Nac ydi ☐

13. Oes gan eich plentyn ag anabledd dysgu broblemau gyda symud sy'n ei gwneud yn anodd iddynt symud o gwmpas yn annibynnol (e.e. angen defnyddio cadair olwyn)? Oes ☐ Nac oes ☐

14. Oes gan eich plentyn ag anabledd dysgu unrhyw broblemau iechyd eraill na nodwyd yn barod? Oes ☐ Nac oes ☐

Os oes, nodwch os gwelwch yn dda _____

Diolch i chi am gymryd rhan ac am eich gonestrwydd.

Strengths and Difficulties Questionnaire

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

Male/Female

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

Signature ..... Date.....

Parent/Teacher/Other (please specify:)

Pre-PACS Interview ADHD Subscale

2. Now I would like to ask about some of the things s/he enjoys doing.
Has she watched <u>television</u> this week?
When was the last time you saw her doing this?

<u>How long did she watch for</u>?
If the answer is vague: **Would it be more or less than half an hour?**

More than 20 mins	0	
More than 15 mins but less than 20 mins	1	
From 6 to 15 mins	2	
No more than 5 mins	3	
Situation not arisen, unrateable or missing	9	____ h1

What that be a typical time for him when s/he likes the programme? ____ h2

Those particular times she was watching TV, was she <u>up and down out of her seat</u> at all?
How many times during that particular period?
If answer is vague: **Would it be every 15 minutes or less?**

Not at all/sits still	0	
About once every 15 mins	1	
Once every 5 mins	2	
More than once a minute but less than 5 times a minute	3	
More than 5 times a minute	4	
Situation not arisen, unrateable or missing	9	____ h3

What would be her typical rate of getting up and down out of her seat during a programme she enjoys watching? ____ h4

That particular time of watching that we were talking about, was she <u>fidgeting</u> at all?
(Like swinging legs, tapping fingers or fiddling with an object?)

How much?
If the answer is vague: **Would it be all the time, more than half the time or less than half the ti**

Not at all	0	
Less than half of the time	1	
More than half of the time but not throughout	2	
Continuous, never stopped	3	
Situation not arisen, unrateable or missing	9	____ h3

What would be her particular pattern during watching a Television programme she enjoys? ____ h4

3. Has she done anything which she enjoys doing on her own recently?
Such as painting, drawing, modelling, jigsaws etc.
When was the last time you saw her doing that?
Was this typical of her playing on her own?
(If not), what would be a more typical situation?
When did you last see her doing that?

That particular time, how long was she playing for?
If the answer is vague: **Could he play on his own for 30 minutes, or would it usually be less than that**

More than 30 mins	0	
16 to 30 mins	1	
6 to 15 mins	2	
No more than 5 mins	3	
Situation not arisen, unrateable or missing	9	____ h1

How long would be typical for her to play on her own like this? ____ h2

(Notes: If the attention span differs according to activity, rate the longest duration. Do not include ac shared with a parent or another child.)

The time that you have just described of her playing on her own, was she up and down out of her seat at all?

How many times during that period?

Not at all	0	
About once in every 15 mins	1	
More than once per 15 mins but less than once per 5 mins	2	
Every 5 mins or more	3	
Situation not arisen, unrateable or missing	9	____ h3

What would be the typical rate of her getting up and down while playing on her own? ____ h4

The time that we have just talked about of her playing on her own, was she fidgeting about at all?
If the answer is vague: **Would it be all the time, or more/less than half?**

How much?

Not at all	0	
Less than half of the time	1	
More than half of the time but not throughout	2	
Continuous, never stopped	3	
Situation not arisen, unrateable or missing	9	____ h3

What would be her typical pattern of fidgeting during that kind of activity on her own? h4

4. Has she played indoors with other children like her brothers and sisters or friends recently?
When was the last time you saw her?
What did they play with/ what sort of game was it?

That time she was playing with other children/someone else,
how long did she stick to one activity for?

More than 30 mins	0	
16 to 30 mins	1	
6 to 15 mins	2	
No more than 5 mins	3	
Situation not arisen, unrateable or missing	9	____ h1

Was this typical of her playing with other children?
How long would she usually spend on one activity? ____h2

That particular time you have just described of her playing with another child/other children, was s/he running around unnecessarily in and out of rooms during the time they played; or was she staying in one place?
How often did s/he do that?

Not at all	0	
About once in every 15 mins	1	
More than once per 15 mins but less than once per 5 mins	2	
Every 5 mins or more	3	
Situation not arisen, unrateable or missing	9	____ h3

Would that be her typical pattern during a similar activity? ____ h4

5. Have you seen her at a mealtime during the last week?
When was the last time?
Was that a meal that she was supposed to sit down at the table with you?

(Note: If not, choose a meal time during which the child was supposed to sit down at the table and which is also well remembered.)

That particular time, did she get up and leave the table at all?
(Note: Do not rate getting up to fetch a glass of water etc., unless parent states these are excuses to get up.)

How often did she do that?

Not at all	0	
Once	1	
2x to 5x	2	
more than 5 times	3	
Situation not arisen, unrateable or missing	9	____ h1

Would that be usual for her during mealtimes over the past 6 months? ____ h2

6. Has she been with you to the shops in the last week?
When was the last time?

That particular time, did she run away from you at all?

If so, how much of the time was she with you between running away?

Not running away at all	0	
Running away every 5 mins or less	1	
Running away every 2 to 5 mins	2	
More than every 2 mins	3	
Situation not arisen, unrateable or missing	9	____ h1

What would be her usual pattern when she is in a shop with you? ____ h2

(Notes: Include disturbing other shoppers by pushing the trolley in an uncontrolled way. If parent keeps chil restrained in trolley due to past experience of repeated running off, or has stopped taking the child shopping fo the same reason, rate severity the last time in shops if within the last month.)

7. You have told me about ..... (behaviours stated e.g. not sticking to activities, or fidgeting, or rushing around - refer to any above)

Do you regard this as a problem?

No problem	
Minor problem	
Serious problem	

APPENDIX D

COPY OF SCHOOL OF PSYCHOLOGY ETHICS COMMITTEE APPROVAL LETTER

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June 13, 2005

Professor R. Hastings, Dr. D.Daley
Katie Bigham, Trainee Clinical Psychologist
North Wales Clinical Psychology Programme
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Dear Colleagues

Hyperactivity disorders in children with a mild learning disability

Your research proposal, (referred to above and on the attached sheet) has been reviewed by the School of Psychology Research Ethics Committee and they are satisfied that the research proposed accords with the relevant ethical guidelines.

If you wish to make any substantial modifications to the research project, please inform the committee in writing before proceeding. Please also inform the committee as soon as possible if participants experience any unanticipated harm as a result of taking part in your research, or if any adverse reactions are reported in subsequent literature using the same technique elsewhere.

Good luck with your research.

Kath Chitty

Kath Chitty
Coordinator -School of Psychology Research Ethics Committee

C Fergus Lowe PhD, FBPsS
Athro a Phennaeth yr Ysgol • Professor and Head of School

SECTION 3

LITERATURE REVIEW

Attention Deficit Hyperactivity Disorder (AD/HD) in children with and without Intellectual Disabilities (ID) – A review

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Literature review written in the style appropriate for the Journal of Intellectual Disability Research. For author's instruction, please see attached material.

Abstract

Attention deficit/hyperactivity is a relatively common, debilitating childhood disorder that is responsible for up to 50% of total referrals to child services. Existing research suggests that children with intellectual disabilities (ID) are at an increased risk of developing AD/HD compared to their non-ID counterparts. Much research has investigated the aetiological explanations and associated risk factors thought to account for the development of AD/HD in children without ID, such as the role of genetics and environmental influences. Although no genetic studies, neuroimaging studies, or psycho-social intervention studies have been undertaken with children with ID, a number of biological and socio-economic factors known to increase AD/HD in the non--ID population continue to be relevant to children with ID. AD/HD is associated with a number of co-morbid disorders and long-term psychosocial difficulties for both populations, however, there appears to be a disparity between available interventions for children with and without ID and AD/HD. This paper reviews the current literature on these issues and indicates the need for further research.

Selection criteria

For the purpose of this review the following methodology was employed:

Databases: *Web of Science and PsychInfo.*

Publication dates: *1994-2006. No articles published prior to 1994 were sought due to this being the most recent revision of DSM criteria for AD/HD.*

Key words: *Attention deficit hyperactivity disorder; children; childhood; intellectual disability; mental retardation; psychopathology, intervention; methylphenidate; psychosocial.*

Attention deficit/hyperactivity disorder (AD/HD; American Psychiatric Association, 1994) is a complex neurobiological disorder that is characterised by deficits in three main areas of observable behaviour: inattention, impulsivity, and hyperactivity (Swanson et al., 1998). In order to fulfil DSM IV criteria (APA, 1994), a child has to display six out of nine symptoms of inattention 'that have persisted for at least 6 months, to a degree that is maladaptive and inconsistent with their developmental level', and six or more symptoms of hyperactivity-impulsivity (there are three symptoms of impulsivity and six of hyperactivity). However, there are also two subtypes of AD/HD, the predominantly inattentive type, where only inattentive criteria are met and the predominantly hyperactivity-impulsivity type where only hyperactive-impulsive criteria are met (Daley, in press).

The closely related disorder Hyperkinesis (a more severe form of AD/HD) is based on alternative diagnostic criteria in the International Classification of Mental and Behavioural Disorders Manual (ICD-10, World Health Organisation, 1992 & 1993). Although more recently both the DSM and ICD manuals recognise the majority of the same problem behaviours as being core to a diagnosis of AD/HD, there continue to be three major differences between the two taxonomies (Swanson et al., 1998).

Unlike DSM-IV criteria, an ICD-10 diagnosis of Hyperkinesis cannot be given unless symptoms in all three groups are present (inattention, hyperactivity and impulsivity). Secondly, ICD-10 criteria include conduct disorder as a combined diagnosis category, and due to the high usage of this combination, use its presence or absence as its main subdivision; DSM-IV does not make any special provision for the presence or absence of a co-morbid disorder. Finally, unlike DSM-IV, ICD-10 uses

other co-morbid internalising disorders (such as depression and anxiety) as exclusion criteria (Seagar & O'Brien, 2003).

Prevalence of AD/HD in children with and without ID

Most prevalence estimates of mental health problems experienced by children vary widely according to populations studied and diagnostic criteria applied (Strømme & Diseth, 2000). It remains the case that due to the lack of well-designed epidemiological studies, detailed information on the prevalence rates and impact of mental health problems on children remain inadequate (Emerson, 2003). However, the most recent population studies have estimated that as many as 40% of children in the general population will experience a mental health problem of one sort or another during childhood (Enfield & Tonge, 1996*a,b*; Emerson 2003).

In 1999 the Social Survey Division of the Office for National Statistics (ONS) carried out a survey of the mental health in children and adolescents in England and Wales on behalf of the Department of Health, the Scottish Health Executive and the National Assembly for Wales (see Meltzer, Gatward, Goodman, & Ford, 2000). Out of the 10,000 children surveyed, it was found that 6% had clinically significant conduct disorders, 1% had hyperactive behaviour, and 4% were experiencing an emotional disorder (Meltzer et al., 2000).

In terms of investigation into specific disorders such as AD/HD, research with typically developing children has found an AD/HD prevalence rate of between 2% and 5% of school aged children (Scahill et al., 1999), and up to 2% of pre-school children (DuPaul, McGoey, Eckert, & Vanbrackle, 2001). In addition, AD/HD is reported to be becoming increasingly more prevalent, with up to 50% of total referrals to children's services being AD/HD related (Faraone, Sergeant, Gillberg, & Biederman, 2003; Mellor, Storer, & Brown, 1996). This apparent increase in the

prevalence of AD/HD is thought to be due mainly to an increase in awareness by clinicians of AD/HD symptomatology, and its long-term impact (Mellor, Storer, & Brown, 1996).

Mental health problems in children with ID have received more attention in recent years, with a number of studies attempting to compare the prevalence of general childhood psychopathology in children with and without ID (Linna et al., 1999; Dekker & Koot, 2001; Wallander, Browne, & Stankovic, 2002). In general, it is reported that children with ID face between a three-fold and seven-fold risk of experiencing childhood mental health problems (Wallander, Dekker, & Koot, 2003).

Although there are relatively few studies that present prevalence estimates of specific disorders, such as AD/HD, in children with ID (Dekker & Koot, 2003), from those that do, AD/HD has been found to be consistently more common among children with ID than it is in their non-ID counterparts (Dekker & Koot, 2003; Linna et al., 1999; Strømme & Diseth, 2000). Dekker and Koot (2003) conducted a study whereby a representative sample of children with ID were recruited from Dutch special schools and underwent a DSM-IV diagnostic assessment using a standardised clinical interview. They found an AD/HD prevalence rate of 14.8%. Similarly, in a population-based study Strømme and Diseth (2000) found that 16% of the total population with ID were experiencing Hyperkinesis (according to ICD-10 criteria). More recently, Hastings, Beck, Daley, & Hill (2005) reported that 60% of their ID sample scored above the level of clinical concern on a questionnaire of AD/HD/hyperactivity, compared to only 2.7% of non-ID siblings. More importantly the significantly increased prevalence of AD/HD remained, once chronological and mental age differences had been controlled.

There are many problems associated with the accurate assessment and diagnosis of mental health problems, such as AD/HD, in children with ID. Primarily, assessment of mental health problems in children with ID generally relies upon information being gathered from behaviour-rating scales (as generally used with their non-ID counterparts). However, the most reliable method for obtaining accurate, detailed information in order to make reliable diagnoses has been found to be via clinical interviews (Dekker & Koot 2003). Given this, it is likely that studies that have only used behaviour rating scales as a means of estimating prevalence are reporting estimates that are lower than they actually are.

An example of the underestimation of childhood psychopathology can be seen where studies have undertaken large population surveys of case files where clinical judgement was relied upon to describe and define psychopathology. For example, in a study undertaken by Koskentausta, Iivanainen, & Almqvist (2002) it was found that the percentage of children originally diagnosed with a psychiatric disorder increased from 11% to 33% when case files were carefully re-examined and additional information, from observations and clinical interviews were gained. It is reported that the most accurate diagnoses of mental health problems in children with ID occur when psychiatric diagnoses have been sought using behaviour rating scales *in addition* to structured clinical interviews (Koskentausta, Iivanainen, & Almqvist, 2002).

Further factors that exacerbate the lack of consensus regarding prevalence rates in ID and non-ID populations include differing sampling methods and sizes, varying definitions of ID and psychiatric disorder, and varying age ranges and level of ID (Enfield & Tonge, 1996*a*, Emerson, 2003). These differences in methodologies applied in studies result in an inability to make valid study comparisons to reach a

'best' estimate of prevalence. This in turn potentially hampers both the understanding of childhood mental health problems in children with ID, and the provision of adequate service provision. More research into prevalence rates of AD/HD in children with ID is much needed using methodologies, such as clinical interviews that yield more accurate information.

Aetiology of AD/HD in children without ID, and associated 'risk factors'

There are a number of aetiological explanations and associated risk factors that aim to account for the development of AD/HD in the general population (Tannock, 1998); however its precise aetiology continues to remain unclear (Durston, 2003). To date explanations tend to be categorised under two main headings: biological and socio-environmental risk factors. However, it is generally thought that the interaction *between* both is likely to increase the development and maintenance of, AD/HD (Larsson, Larsson, & Lichtenstein, 2004; Tannock, 1998). Biological theories include the role of genetics and brain structure, and socio-environmental risk factors include the role of parenting and demographic variables.

Biological theories: In recent years, due to information gained from studies on the genetics of AD/HD and the rise of new neuroimaging technologies, the theoretical understanding of the development of AD/HD has moved forward a great deal (Voeller, 2004).

Twin and family studies report that genetic factors are influential in determining temperament and activity levels, proposing that there is little doubt that genetics play an important role in the development of AD/HD (Stevenson et al., 2005). It is generally accepted that AD/HD is a highly heritable disorder, with an increased likelihood of the disorder developing if it is present in a parent (Smalley et al., 2000;

Thapar, Holmes, Poulton, & Harrington, 1999). Twin studies report the highest heritability estimates, which range from 0.87 to 0.98 (Levy et al., 1997; Thapar, 1995). It is estimated that there is a genetic basis in 80% of cases (Voeller, 2004; Taylor, 1994), with the remaining 20% due to a brain insult. Some individuals are thought to have a combination of genetic and acquired forms (Voeller, 2004).

Associations have been found between particular genes and AD/HD. The genes associated with the regulation of the dopamine system have been of particular interest and DNA variants of one of the dopamine transporter genes (DAT1; Cook et al., 1995) and the D4 receptor gene (La Hoste et al., 1996) have been associated with diminished dopamine levels (Ashgari et al., 1995) and increased sensation seeking (Benjamin et al., 1996; Ebstein et al., 1996). The marked response to stimulant medication (such as methylphenidate) gives rise to many of the theories about the genetic basis to AD/HD (Taylor, 1999). Methylphenidate directly affects the dopamine systems, and has been found to be clinically efficacious in the treatment of AD/HD symptoms (Paule et al., 2000). In addition, neuroimaging techniques, such as Positron Imaging Topography (PET) and Magnetic Resonance Imaging (MRI), have associated an area of the brain, normally rich in dopaminergic activity (frontostriatal circuitry) with symptoms of AD/HD (Taylor, 1999).

With regards neuropsychological research, there have traditionally been two predominant theoretical models that have sought to separately explain the development and maintenance of AD/HD. They independently focussed on two single, neuropsychological deficits: executive dysfunction caused by deficient inhibitory control mechanisms (Barkley, 1997) and disturbances in motivational processes (Sagvolden, Aase, Zeiner, & Berger, 1998), the delay aversion hypothesis (Sonuga-Barke, 1994).

The first theoretical model, deficient inhibition, states that a child's AD/HD behaviour results from cognitive dysregulation and deficits in executive function (EF) (Schachar et al., 2000). This model is supported by evidence from a number of studies that have found poorer executive function performance in children with AD/HD, compared to non-AD/HD matched controls (Pennington & Ozonoff, 1996; Sergeant, Abikoff, & Sonuga-Barke, 2002). The second theoretical model sees AD/HD, not as a disorder of executive dysfunction, but as a motivational style (e.g. delay-aversion; Sonuga-Barke, Houlberg, & Hall, 1994). The delay-aversion hypothesis suggests that due to extreme delay-aversion, AD/HD symptomatology is a *functional* behavioural strategy that provides a child with the ability to cope with delay. This alternative theoretical model followed investigations by Sonuga-Barke et al. (1994) into the findings of previous studies that reported AD/HD as a disorder of cognitive dysfunction; Sonuga-Barke et al., (1994) found that these results were confounded by delay. A number of studies investigated the delay aversion hypothesis, and found that even when inhibitory deficits were not present, children with AD/HD continued to exhibit hypersensitivity to delay (Kuntsi, Ooserlaan, & Stevenson, 2001, Neef, Bicard, & Endo, 2001, Schweitzer and Sulzer-Azaroff, 1995, Tripp & Alsop, 2001). The delay aversion hypothesis characterised the influence of delay on behaviour as dependent upon whether the child has control over their environment or not. When the child is in control of their environment they can choose to minimise delay by acting impulsively, for example skipping the queue at the end of the slide! When the child is not in control of their environment, or at least where they are expected to behave in a certain way or face sanctions, the child will choose to distract themselves from the passing of time. For example, in a classroom context the child could achieve this by either daydreaming (inattention) or by

fidgeting (hyperactivity). To summarise, the former theory states that AD/HD is a disorder of cognitive dysregulation whereby the relationship between biology and behaviour is mediated by inhibitory dysfunction, leading to increased impulsivity. The latter suggests that the disorder is a motivational style whereby the relationship between biology and behaviour is mediated by delay aversion, leading to increased inattention and hyperactivity (Sonuga-Barke, 2002).

Following the findings of Solanto et al. (2001), which found that there were independent associations between AD/HD and inhibitory deficits, and AD/HD and a delay aversion, Sonuga-Barke (2002) proposed a dual-pathway model of AD/HD. The dual-pathway model incorporates *both* previous theoretical models (Sonuga-Barke, Dalen, & Remington, 2003). One pathway is mediated by deficits in EF and inhibition dysfunction, the other by delay aversion.

Socio-environmental risk factors: These focus largely on the influence family and social systems have on the development, and maintenance, of AD/HD. Some of the best evidence to support psychosocial factors is the fact that psychosocial intervention works. In a number of intervention studies, childhood AD/HD symptoms greatly improved when parents were taught alternative parenting approaches (Sonuga-Barke et al., 2001; Bor, Sanders, & Markie-Dadds, 2002). Parents and their children with AD/HD can get into a vicious circle whereby negative aspects of the child's behaviour negatively influences the behaviour of the parent, and negative aspects of the parent's behaviour, in turn, negatively influence the behaviour of the child.

Additional factors such as parental psychological difficulties (depression and alcohol abuse), and marital discord and divorce are further associated with AD/HD (Hinshaw, 1994; Taylor, 1994; Anastropoulos, Barkley, & Shelton, 1996). With regard to the wider social context factors, low SES, institutionalisation, and poor peer

relationships have further been associated with the development of AD/HD (Taylor, 1994; Meltzer et al., 2000).

Further environmental influences found to be associated with AD/HD include dietary factors such as food additives and colourings, preservatives, refined sugars and fatty acid deficiencies (Bateman et al., 2004; Schnoll, Burshteyn, & Cea-Aravena, 2003). Eigenmann & Haenggeli (2004) stated that although improvements in dietary intake may positively affect general health and overall behaviour, there remains no specific evidence to confirm the clinical significance of dietary change as a way of mediating AD/HD symptomatology.

Aetiology of AD/HD in children with ID, and associated 'risk factors'

To date there have been no genetic studies, neuroimaging studies (e.g. fMRI, PET, MRI), or psychosocial intervention studies undertaken involving children with AD/HD and ID. However, many of the biological and socio-economic factors that are known to increase risk of AD/HD developing in children without ID continue to be relevant to children with ID. In addition, there are a number of factors that are thought to further increase the development, and maintenance, of AD/HD in children with ID. There is thought to be a relationship between the presence of AD/HD symptoms and the underlying causes of ID. Socio-environmental factors specifically associated with ID may increase the susceptibility of the disorder developing, and there is also the possibility that AD/HD itself may be the cause of lowered IQ (Seager & O'Brien, 2003).

Genetic factors: There is evidence that AD/HD is associated with a number of specific, genetically based syndromes that have ID as part of their phenotype. For example, AD/HD symptoms are found in William's syndrome and velocardiofacial syndrome (Carlson, Papolos, & Pandita, 1997), and Smith-Magenis syndrome (Udwin

& Dennis, 1995). In Smith-Magenis syndrome, hyperactivity is reported in approximayely 75% of cases (Udwin & Dennis, 1995). In a study by Berney, Ireland, & Burn (1999) 59% of 5 year olds with tuberous sclorosis were rated as 'hyperkinetic' and 40% of participants with Cornelia de Lange's syndrome displayed clinical hyperactivity levels.

Acquired brain injury: Genetic studies report that up to 20% of cases of AD/HD are caused as a direct result of an acquired brain insult during the prenatal and/or perinatal period (Voeller, 2004; Taylor, 1994). Brain injury in the perinatal stage and early childhood is also a known cause for ID (Seager & O'Brian, 2003; Whitaker et al, 1997). Associations have also been made linking AD/HD and ID to extreme prematurity (Lou, 1996), and prenatal exposure to alcohol (Aronson, Hagberg, & Gillberg, 1997) and smoking (Landgren, Kjellman, & Gillberg, 1998).

Epilepsy: Links have been made between epilepsy and AD/HD. It has been reported that AD/HD symptoms are more highly exhibited in children with seizure disorders. However, it is not known whether this is caused by a common underlying cause for both epilepsy and AD/HD, or brain damage as a result of the seizures (Seager & O'Brien, 2003).

IQ: It was reported by Dekker and Koot (2003) that in a earlier study undertaken by Halperin and Gittelman (1982) hyperactive boys were found to have significantly lower IQ's than their non-hyperactive siblings. From this it was suggested that a direct link might exist between AD/HD and lowered intelligence. However, to substantiate this finding, it is reported that more research into the direct relationship between IQ levels and AD/HD is needed.

Socio-environmental risk factors: Socio environmental risk factors associated with children with ID include low economic status, parental divorce,

childhood abuse/neglect, and loss and separation (Cantwell, 1996). Experience of low status, failure and rejection is common in children with ID, and due to poor problem solving skills and deficiencies in conceptual thinking and communication skills, children with ID are less able to develop constructive, compensatory strategies that would be protective against the development of mental health problems (Szymanski, & King, 1999).

Further factors associated with AD/HD and ID includes low levels of social support, reduced social network, and poor social skills (Dekker & Koot, 2001). Experiencing more life events within the family environment, and poor social intelligence, are also problematic and can be contributory factors to the onset of AD/HD (Dekker & Koot, 2001; Shaffer et al., 2000).

Although the specific aetiological relevance, and association, of these factors with AD/HD in children with ID is not clear, the presence of ID is reported to increase the presence of general psychopathology in this population (Dekker & Koot, 2003). ID within a family appears to increase the potency of influential 'risk factors' such as diet, family discord, poor peer relationships, parenting and parental psychological difficulties. However, in order to gain a greater understanding of the association and aetiological relevance of these factors within the ID population much more research is needed.

Co-existing and associated features of AD/HD in children without ID

AD/HD is associated with a number of long-term psychosocial difficulties; however it remains unclear as to whether these are as a result of AD/HD per se, or due its co-existing disorders (Biederman, Mick, Faraone, & Burback, 2001; Kadesjo, 2000; Rasmussen & Gilberg, 2000). The significance of co-existing disorders in AD/HD has

only recently been identified as an important aspect of AD/HD (Brown, 2000; Jensen et al., 2001; Jensen et al., 1999).

Depending on the sample, AD/HD is associated with another DSM diagnosis in between 60%-100% of school age children diagnosed (Gillberg & Melander, 1997; Jensen, Martin, & Cantwell, 1997; Kadesjö, 2000; Spencer et al., 1999). In a community based study undertaken by Kadesjö and Gillberg (2001), 87% of seven year olds met criteria for at least one other DSM disorder, and it was found that two thirds of the general population diagnosed with AD/HD also had two additional DSM diagnoses. Co-existing disorders identified included oppositional defiant disorder (ODD), conduct disorder (CD), depression (including bipolar disorder), anxiety, tic disorders, obsessive compulsive disorder (OCD), and autistic spectrum disorder (ASD) (Gillberg et al., 2004).

Autism is rarely found to be co-morbid with AD/HD in children without ID (Gillberg et al., 2004). The DSM-IV discourages clinicians from making a double diagnosis of AD/HD and ASD; in fact one of the DSM-IV's exclusion criteria is the presence of a pervasive developmental disorder such as autism. Nevertheless, in very young children it can be difficult to determine whether presenting behaviours are due to severe combined subtype of AD/HD or autistic disorder (Gillberg et al., 2004). A number of studies have found a similar symptom signature often associated with ASD in children with AD/HD, such as difficulties with social interaction, peer relationships and empathy (Barkley, 1998; Kadesjö, 2000). It has been found that between 65% and 80% of children with AD/HD attending clinics also have several DSM-IV autistic spectrum symptoms (Clark, Feehan, Tinline, & Vostanis, 1999). This is compared with less than 10% of the general population (Constantino & Todd, 2003).

CD and ODD are reported to be the most commonly associated disorders with AD/HD, with up to 60% of children diagnosed with AD/HD meeting DSM IV criteria for ODD/CD (Cunningham & Boyle, 2002; Kadesjö & Gillberg, 2001; Kadesjö, Kadesjö, Hagglof, & Gillberg, 2001; Lalonde, Turgay, & Hudson, 1998).

Whilst few studies have investigated the co-existence of AD/HD with OCD (Gillberg et al., 2004), AD/HD is reported to be present in between 6-15% of cases (Gillberg et al., 2004). However, in clinical samples it has been reported that up to 30% of adolescents with OCD also fulfil DSM criteria for AD/HD, with the onset of OCD occurring at a later stage (Banaschewski, Sinatchkin, Uebel, & Rothenberger, 2003; Geller et al., 1996; Hanna, 1995).

Busch et al., (2002) reported that 57% of children in primary care settings with AD/HD were found to have a co-existing mood disorder such as depression or anxiety. However, in the recent British Child Mental Health Survey (Ford, Goodman, & Meltzer, 2003) it was found that the link between anxiety and AD/HD was nonexistent when the adjustment was made for the presence of a third DSM disorder. In an Italian study of children between 7-18 years attending bi-polar disorder clinics, 24% of them were also identified as having AD/HD (Masi et al., 2003). Furthermore, Masi et al., (2003) stated that AD/HD might be a precursor of a child-onset subtype of bi-polar disorder.

In addition to the associations found with other psychiatric disorders, AD/HD is associated with a wide range of secondary problems including marked educational and social impairment, and is recognised as a major risk factor for the development of further problems in adolescence and adulthood including: educational failure, criminality, and personality disorder (Taylor et al., 1996).

Children with AD/HD find it hard to conform to parental, educational, and societal rules and expectations, which in turn results in chronic conflicting relationships within their environment (Mellor et al., 1996). A child with AD/HD is frequently the focus of family and classroom difficulties, often resulting in additional problems such as low self-esteem and self worth (Mellor et al., 1996). Green and Chee (1994) reported a number of difficulties commonly associated with AD/HD including insatiability, social clumsiness, poor co-ordination, disorganisation, variability of mood, and specific learning difficulties such as dyslexia and language problems.

Upon school entry, children with AD/HD are likely to be behind their non-AD/HD counterparts in the following areas: basic maths, pre-reading skills, and fine motor abilities (Lahey, Pelham, & Stein, 1998; Mariani & Barkley, 1997; Shelton, Barkley, & Crosswait, 1998). They are more likely to exhibit increased problem behaviours (in both the internalising and externalising domains) and show a reduction in expected social functioning (DuPaul et al., 2001).

Co-existing and associated features of AD/HD in children with ID

Although a variety of behavioural and emotional difficulties have been identified to co-occur with AD/HD in children without ID, less is known regarding the concomitant behavioural and emotional problems in children with ID (Pearson et al., 2000). However, it would appear that anxiety (Perrins & Last, 1996; Rucklidge & Tannock, 2001) and aggressive behaviour (Shelton et al., 1998) are amongst the most reported co-existing difficulties associated with AD/HD in children with ID. Aggressive behaviour is reported, by parents, to be one of the most difficult behaviours to cope with in their children with ID and AD/HD (Shelton et al., 1998) with violent encounters reported to be a common occurrence (Diamond and

Siqueland, 2001). In 2000 Pearson and colleagues conducted a study to investigate the concomitant behavioural and emotional difficulties experienced by children with AD/HD and ID. The behavioural adjustment of 48 children with ID and AD/HD was compared to that of 47 children with ID without AD/HD. It was found that the AD/HD group of children displayed significantly more symptoms of depression, family conflict, non-compliance, anxiety, hyperactivity, inadequate social skills, and more academic difficulties. The findings of this study suggest that children with ID and AD/HD experience significant behavioural and emotional problems that mirror the pattern associated with AD/HD and children of average intellect (Pearson et al., 2000).

In addition, children with ID and AD/HD appear to be more socially isolated (Lufi & Parish-Plass, 1995) and to experience increased difficulties both initiating and maintaining peer relationships (McCormick, 2000). This results in increased feelings of low self-esteem, which are reported to remain well into adulthood (Trenting & Himshaw, 2001; Mannuzza & Klein, 2001).

Intervention for AD/HD experienced by children and adolescents without ID

Interventions for AD/HD in children without ID can be categorised into two main groups: pharmacological (psycho-stimulant medications) and psychosocial interventions. Psycho-stimulant medications, such as methylphenidate, dexedrine and pemoline, have been found to be clinical efficacious in between 70-90% of school aged children with AD/HD upon initial prescription (Handen et al., 1999; Reid, Hakendorf, & Prosser, 2002; 2002; MTA 1999; Goldman, Genel, Bezman, Slanetz, 1998). Psycho-stimulant medication directly affects the neurotransmitter functioning in the area of the brain responsible for inhibition and attention (Fewell, & Deutscher, 2002). Psycho-stimulant drugs release and inhibit the reuptake of catecholamines,

mainly dopamine, in the central nervous system (Swanson et al., 1998). By controlling these levels, motivation, concentration, and attention abilities in children with AD/HD appear to increase (Pelham et al., 1999).

Psychosocial interventions mainly focus on behavioural interventions. Hibbs (2001) undertook a review evaluating empirically based psychotherapy for children. For the treatment of AD/HD, behavioural parent training (Anastopoulos, 1993; Gitteleman et al., 1980; Horn et al., 1990) and behavioural intervention in the classroom setting (Gitteleman et al., 1980; Pelham, Wheeler, & Chronis, 1998) were found to be the only two that were clinically efficacious. Cognitive treatments alone did not gain empirical support, however could potentially achieve clinical efficacy if combined with behavioural therapy (Hinshaw & Erhard, 1991). Other psychosocial interventions such as play therapy, and diet management were not found to be clinically effective (Pelham, Wheeler, & Chronis, 1998).

In 1999 the Multimodal Treatment Study for children with AD/HD (MTA), was conducted by the National Institute of Mental Health (NIMH). The aim of the study was to examine both the short and long-term effects of systematic treatment on children's AD/HD symptoms, co-morbid conditions, and general impairment. Comparisons were made between 4 main treatment groups, medication alone, psychosocial treatment alone and combination of medication and psychosocial intervention. The fourth comparison group aimed to compare intensive manual-based treatments to routine community care. The MTA is viewed as one of the most influential intervention studies and results have dominated intervention practices for the treatment of AD/HD since (Jensen et al., 1999).

The results of the MTA found that the core symptoms of AD/HD were better controlled by medication alone than by psychosocial intervention, either alone or

combined with medication, or by routine community care (Jensen et al., 1999). In addition it was found that higher and more frequent stimulant dosing produced greater reductions in AD/HD symptoms than lower and less frequent doses (Jensen et al., 1999). These findings influenced subsequent recommendations made by the National Institute for Clinical Excellence (NICE) on interventions for AD/HD, stating that stimulant medication should be the initial treatment for the management of AD/HD, followed by additional psychosocial intervention if necessary (NICE, 2000). Similar findings regarding levels and frequency of medication were reported by Rapport and colleagues, with the important caveat that each child's response will be individual (Rapport, Denney, DuPaul, & Gardner, 1994).

Subsequently, Swanson et al., (2001) conducted further analysis of the MTA data and found that in the long-term, the multi-model intervention package of medication and psychosocial interventions *together* was clinically more efficacious. Further comment has been made regarding the MTA study results by Greene and Ablon (2001). They highlighted the fact that medication regimes given to each child in the study had been carefully tailored to each child, whereas the children in the psychosocial intervention group received an intervention that was much less tailored to their individual needs, and therefore was likely to be less effective (Greene, & Ablon, 2001). In addition, no theoretical rationale for the content of the behavioural component of the psychosocial intervention was ever published (Morell, & Murray, 2003), and due to the multitude of individual sessions for the child including additional classroom support, summer camps and individual sessions for the parents, no clinical replication was possible (Greene, & Ablon, 2001). These issues therefore impact upon the relevance of the MTA's findings on clinical practice.

Although in the short-term at least, stimulant intervention appears to be more effective (MTA, 1999) there is good evidence to suggest that when provided together value can be added to both treatment approaches. Evidence suggests that whilst stimulant medication reduces the levels of core AD/HD symptoms, psychosocial interventions have been found to be especially effective in reducing the further impact of comorbid difficulties such as oppositional, defiant behaviour (Sonuga-Barke et al., 2003).

More recently, it has been suggested that psychosocial interventions are most effective when delivered during the early stages of AD/HD (during pre-school years; Sonuga-Barke et al., 2001). Sonuga-Barke et al., (2001) found that parent training intervention produced significantly reduced AD/HD symptoms that were maintained for at least 15 weeks post intervention. The effect sizes were reported to be comparable with those reported for stimulants (Sonuga-Barke et al., 2003). Bor, Sanders and Dadd (2002) found similar results when they compared standard and enhanced family behaviour therapy with waiting list controls. Post-intervention results found that both groups had significantly lower levels of AD/HD symptoms as well as greater parental competence. These findings were maintained 1 year post-intervention (Bor, Sanders, & Dadd, 2002).

From these findings it would appear that psychosocial interventions are a valuable non-pharmacological alternative to stimulant medication, especially when delivered in a tailored, timely fashion. It is likely that psychosocial interventions are most successful early on in the development of AD/HD due to associated, compounding factors such as school failure, peer rejection and the deterioration of adult-child relationships not yet being entrenched.

Intervention for AD/HD experienced by children and adolescents with ID

In terms of treatment approaches for AD/HD in children with ID, the dominant intervention is the use of stimulant medication. Recent studies indicate that although, overall, stimulants appear to be successful in the treatment of AD/HD in children with ID, there is a less consistent response pattern than that seen in the general, non ID population. For example, Pearson et al., (2003, 2004) recently reported that higher, more frequent doses of methylphenidate (MPH) were required in this population in order to maintain efficacy in the reduction of cognitive and behavioural problems associated with AD/HD, compared to their non-ID counterparts. However, they found that in general children with mild ID responded as well to stimulant medication as their non ID counterparts. It was also reported that MPH efficacy was greater among those with IQ's greater than 45.

Atypical antipsychotics, such as resperidone, have been found to significantly decrease problematic behaviours including hyperactivity in children with ID (Aman, Buican, & Sillick, 2002; Synder et al., 2002). Improvements continue to be reported for at least 48 weeks, with the greatest improvements occurring in the first 4 weeks (Findling et al., 2004; Turgay et al., 2002).

Due to few studies specifically investigating the use of medications on the management of AD/HD symptoms in children with moderate ID, Filho et al. (2005) conducted a study whereby the use of risperidone (anti-psychotic) and MPH (psycho-stimulant) in this population was examined. They found that both types of medication reduced AD/HD syptomatology.

With regard longer term outcomes, Aman et al. (2002) undertook a study whereby 20 children with AD/HD were followed up 4.5 years post-intervention. It was found that approximately half of the sample continued to exhibit AD/HD

symptoms and the children appeared to have a variety of co-morbid conditions not commonly reported in typically-developing children with AD/HD, such as tics and anxiety disorders.

From the heterogeneity of responses to pharmacological interventions Aman et al. (2002) suggested that there are likely to be qualitative differences that distinguish children with AD/HD and ID from their typically developing counterparts, suggesting that AD/HD may have a different 'signature' for children with ID. Due to the fact that few studies have been concerned with the specific presentation of AD/HD in children with ID, Aman et al. (2002) concluded that more research is needed regarding the specificity of AD/HD symptomatology in children with ID.

There are a number of ethical issues that need to be considered when using stimulant medication for the treatment of AD/HD in children with ID. Due to the paucity of literature regarding the long-term side effects of medication use in the treatment of AD/HD, there continues to be a lack of convincing evidence for the long-term benefits of pharmacological interventions for the treatment of AD/HD in children with and without ID (Pelham, Wheeler, & Chronis, 1998). In terms of short-term side effects of treating childhood AD/HD with pharmacological interventions, the literature suggests that there appears to be an increase in parental reports of sadness, nightmares, dysphoria, appetite suppression, and lower levels of interest and interaction with peers (Firestone et al., 1998). Evidence suggests that these reported side-effects appear to be exaggerated in children with ID and AD/HD (Ghuman et al., 2001; Handen et al., 1999). With regard to long-term side effects of chronic administration of stimulant medications, there are no clinical studies that investigate this issue directly. Through investigations with animal models, however, concerns have been raised in terms of a child's physical and neurological development (Moll et

al., 2001). As a result questions remain regarding whether although a child' s behaviour may become easier to manage, they may be less able to learn naturally from their environment, their actions and those around them, reducing their ability to reach their developmental potential. For example, stimulants and the behavioural effects they may create are likely to alter both parental and child experiences of typical, but challenging emotional, social, and behavioural episodes in their lives, reducing the ability for both parties to learn from these experiences. This could in turn result in parental disengagement from their AD/HD child and the development of less skilled and competent parenting (Sonuga-Barke et al., 2003). It remains the case that it is currently the increased ease of management of a child's behaviour that determines whether or not treatment has been successful. No research appears to take into account, or attempt to evaluate, whether or not a child's quality of life or ability to reach their full developmental, emotional, and social development has been optimised.

In terms of psychosocial interventions, although there is no data on the efficacy of parent behaviour management with children with ID, there is no reason to assume that similar intervention methods would not be as effective with this population as it is in the non-ID population (Sonuga-Barke et al., 2001). If developmental delay is taken into account, parent behaviour management could be just as efficacious when delivered to children with ID at a later age. Psychosocial interventions, such as parent behaviour management, have additional advantages for use with children with ID; many children with ID and AD/HD are likely to be on medications for an array of associated medical conditions and are therefore at increased risk of experiencing an adverse response to stimulants. And given the greater heterogeneity in stimulant response in children with ID compared to their non-

ID counterparts (Aman, Buican, & Arnold, 2003), further emphasis on alternative, non-pharmacological interventions is greatly needed within the research fraternity.

Conclusion

Although there is a vast amount of knowledge and understanding regarding AD/HD in typically developing children, there continues to be a paucity of research investigating AD/HD in individuals with ID. From what we know, the prevalence of AD/HD is considerably greater in children with ID (Dekker & Koot, 2003), and given the methodological difficulties associated with assessment and diagnosis of psychopathology in individuals with ID, many co-existing disorders, including AD/HD, remain undiagnosed (Enfield & Tonge, 1996a; Emerson, 2003). The interaction between biological and environmental 'risk factors' known to increase the development of AD/HD in individuals without ID (Larrson, Larrson, & Litchenstein, 2004), are likely to be accentuated by difficulties associated with ID (Dekker & Koot, 2001). In terms of intervention, although a number of studies report the short-term efficacy of psycho-stimulant treatments for use with children with ID (e.g. Filho et al., 2005), Aman et al. (2002) found that approximately half of the sample in their study continued to exhibit AD/HD symptoms 4.5 years post-intervention. Recent outcome data on parent behaviour management interventions for pre-school children with AD/HD indicate that if delivered early enough, such interventions can be clinically efficacious (Bor, Sanders, & Makie-Dadds, 2002; Sonuga-Barke et al., 2001). Given developmental delay, similar intervention methods might be just as effective with parents of children with ID. Due to many questions remaining unanswered, and the fact that AD/HD is becoming an increasingly recognised disorder in children with ID, further research is needed, particularly in terms of understanding the specific phenomenology of AD/HD in individuals with ID.

Implications for further research arising from this review.

From this review there appear to be a number of areas that would benefit from further research. Firstly, although from the limited literature currently available it would appear that AD/HD is more prevalent in children with ID, compared to other disorders and populations (Dekker & Koot, 2003; Linna et al., 1999; Stromme & Diseth, 2000) there continues to be a lack of studies that investigate the prevalence of AD/HD in children with ID. In addition there are however, no studies that have attempted to investigate why this should be the case.

Secondly, in order to be able to accurately diagnose the disorder in children with ID, a greater understanding of the presentation of AD/HD in this population is needed. This will enable the development and valid adaptation of appropriate and accurate assessment measures. In addition, further research needs to take place to evaluate the accuracy of parental reports, for example whether parental reports are indicative of an underlying disorder such as AD/HD or whether they are reporting behaviours more associated with a child's ID.

Implications for clinical practice arising from this review.

In terms of clinical practice there are a number of clinical implications arising from this review. Primarily there is the issue of diagnostic overshadowing. It would appear that the prevalence of AD/HD in children with ID is as common, if not more common, than in their non ID counterparts; however it continues to remain largely undiagnosed in the ID population, with many behavioural difficulties being attributed to the child's ID rather than to an underlying disorder (Dekker & Koot, 2003). It may be found that if disorders such as AD/HD were assessed for and addressed as early as possible, the number of referrals for other behavioural difficulties associated with children with ID such as challenging behaviour and stereotypy may be reduced. It

may be possible that AD/HD may be significantly contributing to the development of more difficult and entrenched behaviours. In addition, given that the majority of research states that early intervention yields the best results, the need to be able to recognise and accurately assess and diagnose AD/HD in this population is of extreme importance.

References

Aman, M., Armstrong, S., Buichan, B. & Sillick, T. (2002) Four year follow-up of children with low intelligence with ADHD: A replication. *Research and Developmental Disabilities, 23,*119-134.

Aman, M., Buchanan, B., & Arnold, L. (2003). Methylphenidate treatment in children with borderline IQ and mental retardation. Analysis of 3 aggregated studies. *Journal of Child and Adolescent Psychopharmacology, 13,* 29-40.

Anastropoulos, A., Shelton, T., DuPaul, G., & Guevremont, D. (1993). Parent training for attention deficit-hyperactivity disorder. Impact on parent functioning. *Journal of Abnormal Child Psychology, 21,* 581-596.

Anastropoulos, A., Barkley, R., & Shelton, T. (1996). Family based treatment: Psychosocial intervention for children and adolescents with attention deficit hyperactivity disorder. In E. Hibbs and P. Jensen (eds.), *Psychosocial treatments for Child and Adolescent Disorders: Empirically Based Strategies for Clinical Practice* (pp. 267-284). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders 4th Edition (DSM-IV).* Washington DC: American Psychiatric Association.

Aronson, M., Hagberg, B., & Gillberg, C. (1997). Attention deficits and autistic spectrum problems in children exposed to alcohol during gestation: a follow-up study. *Developmental Medicine and Child Neurology, 39,* 583-587.

Ashgari, V., Sanyal, S., Buchwaldt, S., Paterson, A., Jovanovic, V., & van Tol, H. (1995). Modulation of intracellular cyclic AMP levels by different human dopamine D4 receptor variants. *Journal of Neurochemistry, 65,* 1157-1165.

Banaschewski, T., Siniatchkin, M., Uebel, H. & Rothenberger, A. (2003). Zwangsphanomene bei Kindern mit Tic-Storung bzw. Aufmerksamkeitsdefizit/Hyperaktivitatsstorung. *Zeitschrift fur kinder- und Jugendpsychiatrie und Psychotherapie, 31,* 203-211.

Barkley, R. (1990). *Attention Deficit Hyperactivity Disorder: A handbook for diagnosis and treatment.* New York: Guildford.

Barkley, R. (1998). *Attention Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment,* 2nd ed. New York: Guildford.

Batemen, B., Warner, J., Hutchinson, E., Dean, T., Rowlandson, P., Gant, C., Grundy, J., Fitzgerald, C., & Stevenson, J. (2004). The effects of a double-blind, placebo controlled, artificial food colouring and benzoate preservative challenge on hyperactivity in a general population sample of preschool children. *Archives of Diseases in Childhood, 89,* 506-511.

Benjamin, J., Patterson, C., Greenberg, B., Murphy, D., & Hamer, D. (1996). Population and familial association between the D4 dopamine receptor gene and measures of Novelty Seeking. *Nature Genetics, 12,* 81-84.

Berney, T., Ireland, M., & Burn, J. (1999). Behavioural phentype of Cornelia de Lange syndrome. *Archives of Disease in Childhood, 81,* 333-336.

Biederman, J., Mick, E., Faraone, S., & Burback, M. (2001). Patterns of remission and symptom decline in conduct disorder: a four-year prospective study of an ADHD sample. *Journal of the American Academy of Child And Adolescent Psychiatry, 40,* 290-298.

Bor, W., Sanders, M. & Markie-Dadds, C. (2002). The effect of Triple P-Positive Parenting Programme on preschool children with co-occurring disruptive behavior and attentional/hyperactive difficulties. *Journal of Abnormal Child Psychology, 30,* 571-578.

Brown, T. (2000). *Attention-Deficit Disorders and Comorbidities in Children, Adolescents, and Adults*. Washington DC: American Psychiatric Press, Inc.

Busch, B., Biederman, J., Cohen, L., Sayer, J., Monuteaux, M., Mick, E., Zallen, B., & Faraone, S. (2002). Correlates of ADHD among children in pediatric and psychiatric clinics. *Psychiatric Services, 53,* 1103-1111.

Cantwell, D. (1996). Attention deficit disorder: A review of the past 10 years. *Journal of the American Academy of Child and Adolescent Psychiatry, 35,* 978-987.

Carlson, C., Papolos, D., & Pandita, R. (1997). Molecular analysis of velo-cardio-facial syndrome patients with psychiatric disorders. *American Journal of Human Genetics, 60,* 851-859.

Clark, T., Feehan, C., Tinline, C., & Vostanis, P. (1999). Autistic symptoms in children with AD/HD. *European Child and Adolescent Psychiatry, 8(1),* 50-55.

Constantino, R., & Todd, R. (2003). Autistic traits in the general population - A twin study. *Archives of General Psychiatry, 60 (5),* 524-530

Cook, E., Stein, M., Krasowski, M., Cox, N., Olkon, D., Kieffer, J. & Leventhal, B. (1995). Association of attention-deficit disorder and the dopamine transporter gene. *American Journal of Human Genetics, 56,* 993-998.

Cunningham, C., & Boyle, M. (2002). Preschoolers at risk for attention deficit hyperactivity disorder and oppositional defiant disorder: family, parenting and behavioural correlates. *Journal of abnormal child psychology, 30,* 555-569.

Daley, D. (In press). Attention Deficit Hyperactivity Disorder: A review of the essential facts.

Dekker, M., & Koot, H. (2001). Prediction of DSM-IV disorders in children and adolescents with intellectual disability. *Proceedings of the 34th Annual Gatlinburg Conference.*

Dekker, M., & Koot, H. (2003). DSM-IV disorders in children with borderline to moderate intellectual disability. I: Prevalence and impact. *Journal of the American Academy of Child and Adolescent Psychiatry, 42(8),* 915-922.

Diamond, G., & Siqueland, L. (2001). Current status of family intervention science. *Child and Adolescent Psychiatry Clinical North America, 10,* 641-661.

DuPaul, G., McGoey, K., Eckert, T., & VanBrackle, J. (2001). Preschool Children With Attention-Deficit/Hyperactivity Disorder: Impairments in Behavioral, Social, and School Functioning. *Journal of the American Academy of Child and Adolescent Psychiatry, 40(5),* 508-515.

Durston, S. (2003). A review of the biological bases of ADHD: What we have learned from imaging studies? *Mental Retardation and Developmental Disabilities Research Reviews, 9(3),* 184-195.

Ebstein, R., Novick, O., Umansky, R., Priel, B., Osher, Y., Blaine, D., Bennett, E., Nemanov, L., Katz, M., & Belmaker, R. (1996). Dopamine D4 receptor (D4DR) exon III polymorphism associated with the human personality traits of Novelty Seeking. *Nature Genetics, 12,* 78-80.

Eigenmann, P., & Haenggeli, C. (2004). Food colouring and preservatives-allergy and hyperactivity. *Lancet, 364,* 780-788.

Emerson, E. (2003). Prevalence of psychiatric disorders in children and adolescents with and without intellectual disability. *Journal of Intellectual Disability Research, 47,* 51-58.

Enfield, S., & Tonge, B, (1996a). Population prevalence of psychopathology in children and adolescents with intellectual disability: I rationale and methods. *Journal of Intellectual Disability Research, 40(2),* 91-98.

Enfield, S., & Tonge, B, (1996b). Population prevalence of psychopathology in children and adolescents with intellectual disability: II epidemiological findings. *Journal of Intellectual Disability Research, 40(2),* 99-109.

Faraoné, S., Sergeant, J., Gillberg, C., & Biederman, J. (2003). The worldwide prevalence of ADHD: is it an American Condition? *World Psychiatry, 2,* 104-113.

Fewell, R., & Deutscher, B. (2002). Attention deficit hyperactivity disorder in very young children: Early signs and interventions. *Infants and Young Children, 14(3),* 24-32.

Filho, A., Bodanese, R., Silva, T., Alvares, J., Aman, M. & Rohd, L. (2005). A comparison of Resperidone and Methylphenidate for reducing ADHD symptoms in children and adolescents with moderate mental retardation. *Journal of the American Academy of Child and Adolescent Psychiatry, 44 (8),* 748-755.

Findling, R., Aman, M., De Smedt, G., Derivan, A., Lyons, B. & the Risperidone Disruptive Behaviour Study Group. (2004). A long-term open-label study of risperidone in children with severe disruptive behaviors and subaverage IQ's. *American Journal of Psychiatry, 161,* 677-684.

Firestone, P., Musten, L., Pisterman, S., Mercer, J., & Bennett, S. (1998). Short-term side effects of stimulant medication are increased in preschool children with attention deficit hyperactivity disorder: a double-blind placebo controlled study. *Journal of Child and Adolescent Psychopharmacology, 8(13),* 13-25.

Ford, T., Goodman, R., & Meltzer, H. (2003). The British Child and Adolescent Mental Health Survey 1999: the prevalence of DSM-IV disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 42,* 1203-1211.

Geller, D., Biederman, J., Griffin, S., Jones, J., & Lefkowitz, T. (1996). Comorbidity of juvenile obsessive-compulsive disorder with disruptive behavior disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 35,* 1637-1646.

Gillberg, C., Gillberg, I., Rasmussen, P., Kadesjö, B., Söderström, H., Råstam, M., Johnson, M., Rothenberger, A., & Niklasson, L. (2004). Co-existing disorders in ADHD-implications for diagnosis and intervention. *European Child and Adolescent Psychiatry, 13(1),* 80-92.

Gillberg, C., Melander, H., von Knorring, A., Janols, L., Thernlund, G., Hägglöf, B., Eidevall-Wallin, L., Gustafsson, P., & Kopp, S. (1997). Long-term stimulant treatment of children with attention deficit hyperactivity disoreder symptoms. A randomised, double-blind, placebo-controlled trial. *Archives of General Psychiatry, 54,* 857-864.

Gitteleman, R, Abikoff, H., Pollack, E., Klein, D., Katz, S. & Matts, J. (1980) A controlled trial of behaviour modification and methylphenidate in hyperactive children. In Whalen C. & Henker, B. (Eds) Hyperactive Children: The Social Ecology of Identification and Treatment (pp. 221 -243). New York Academic.

Goldman, L., Genel, M., Bezman, R., & Slanetz, P. (1998). Diagnosis and treatment of attention deficit-hyperactivity disorder in children and adolescents. *Journal of the Americam Medical Association, 279,* 1100-1107.

Green, G., & Chee, K. (1994). Management of attention deficit disorder: A personal perspective. *Modern Medicine,* 37, 38-53.

Greene, R. & Ablone, J. (2001). What does the MTA Study tell us about effective psycho-social treatments for ADHD? *Journal of Clinical & Child Psychology, 30 (1),* 114-121.

Hanna, G. (1995). Demographic and Clinical features of obsessive-compulsive disorder in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 34,* 19-27.

Hallowell, E., & Ratey, J. (1994). *Driven to Distraction.* New York: Pantheon Books.

Halperin, J., & Gittelman, R. (1982). Do hyperactive children and their sibling differ in IQ and academic achievement? *Psychiatry Research, 6,* 253-258.

Handen, B., Feldman, H., Lurier, A. & Huszar-Murray, P. (1999). Efficacy of methylphenidate among preschool children with developmental disabilities and ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 38,* 805- 812.

Hastings, R., Beck, A., Daley, D., & Hill, C. (2005). Symptoms of ADHD and their correlates in Children with Intellectual Disabilities. *Research in Developmental Disabilities, 26,* 456-468.

Hibbs, E. (2001). Evaluating empirically based psychotherapy research for children and adolescents. *European Child & Adolescent Psychiatry, 10(1),* 1-11.

Hinshaw, S., & Erhard, D. (1991). Attention Deficit Hyperactive Disorder. In Kendall, P. (Ed) *Child and Adolescent Therapy: Cognitive Behavioural Procedures.* (pp. 98-128). New York Guilford.

Hinshaw, S. (1994). *Attention deficits and hyperactivity in children.* Thousand Oaks, CA: Sage.

Horn, W., Ialongon, N., Greenberg, G., Packhard, T. & Smith-Winberry, C. (1990). Addictive effects of parental behaviour training and self- control therapy with ADHD children. *Journal of Clinical Child Psychology.* 19, 98-110.

Jensen, P., Martin, D., & Cantwell, D. (1997). Comorbidity in ADHD: implications for research, practice and DSM-V. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 1065-1079.

Jensen, P., Rubio-Stipec, M., Canino, G., Bird, H., Dulcan, M., Schwab-Stone, M. & Lahey, B. (1999). Parent and child contributions to diagnosis of mental disorder: are both informants always necessary? *Journal of the American Academy of Child and Adolescent Psychiatry, 38,* 1569-1579.

Jensen, P., Hinshaw, S., Kraemer, H., Lenora, N., Newcorn, J., Abikoff, H., March, J., Arnold, L., Cantwell, L., Connors, C., Elliot, G., Greenhill, L., Hechtman, L., Hoza, B., Pelham, W., Severe, J., Swanson, J., Wells, K., Wigal, T., & Vitiello, B. (2001). ADHD comorbidity findings from the MTA study: comparing comorbid subgroups. *Journal of the American Academy of Child and Adolescent Psychiatry, 40,* 147-158.

Kadesjö, B. (2000). Neuropsychiatric and neurodevelopmental disorders in young school-age population. Epidemiology and comorbidity in a school health

paerspective. *Department of Child and Adolescent Psychiatry, Institute for the health od women and children.* Goteberg: Goteberg University.

Kadesjö, B., & Gillberg, C. (2001). The comorbidity of ADHD in the geneal population of Swedish school-age children. *Journal of Child Psychology and Psychiatry, 42,* 487-492.

Kadesjö, C., Kadesjö, B., Hägglöf, B., & Gillberg, C. (2001). ADHD in Swedish 3-7 year old children. *Journal of the American Academy of Child and Adolescent Psychiatry, 40,* 1021-1028.

Koskentausta, T., Iivanainen, M., & Almqvist, F. (2002). Psychiatric disorders in children with intellectual disability. *Nordic Journal of Psychiatry, 56,* 126-131.

Kuntsi, J., Oosterlaan, J., & Stevenson, J. (2001). Psychological mechanisms in hyperactivity, 1: response inhibition deficit, working memory impairment, delay aversion, or something else? *Journal of Childhood Psychology and Psychiatry, 42,* 199-210.

Lahey, B., Pelham, W., & Stein, M. (1998). Validity of DSM-IV attention deficit/hyperactivity disorder for younger children. *Journal of the American Academy of Child and Adolescent Psychiatry, 37,* 695-702.

LaHoste, G., Swanson, J., Wigal., S., Glabe, C., Wigal, T., King, N., & Kennedy, J. (1996). Dopamine D4 receptor gene polymorphism is associated with attention deficit hyperactivity disorder. *Molecular Genetics, 1,* 121-124.

Lalonde, J., Turgay, A., & Hudson, J. (1998). Attention deficit hyperactivity disorder subtypes and comorbid disruptive behaviour disorders in a child and adolescent mental health clinic. *Canadian Journal of Psychiatry, 43,* 623-628.

Landgren, M., Kjellman, B., & Gillberg, C. (1998). Attention deficit disorder with developmental co-ordination disorders. *Archives of Disease in Childhood, 79,* 207-212.

Larsson, J., Larsson, H., & Lichtenstein, P. (2004). Genetic and environmental contributions to stability and change of ADHD symptoms between 8 and 13 years of age: a longitudinal twin study. *Journal of the American Academy of Child and Adolescent Psychiatry, 43,* 1267-1275.

Levy, F., Hay, D., McStephen, M., Wood, C., & Waldman, I. (1997). Attention deficit-hyperactivity disorder: A category or a continuum? Genetic analysis of a large-scale twin study. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 737-744.

Linna, S., Moilanen, I., Ebeling, H., Piha, J., Kumpulainen, K., Tamminen, T., 7 Almqvist, F. (1999). Psychiatric symptoms in children with intellectual disability, *European Child and Adolescent Psychiatry, 8,* 77-82.

Lou, H. (1996). Etiology and pathogenesis of attention-deficit hyperactivity disorder (ADHD): significance of prematurity and perinatalhypoxic-hemodynamic encephalopathy. *Acta Paediatrica, 85,* 1266-1271.

Lufti, D., & Parrish-Plass, J. (1995). Personality assessment of children with ADHD. *Journal of Clinical Psychology, 51,* 94-99.

Mannuzza, S., & Klein, R. (2000). Long-term prognosis in ADHD. *Child and Adolescent Psychiatry Clinical North America, 9,* 711-726.

Mariani, M., & Barkley, R. (1997). Neuropsychological and academic functioning in preschool boys with attention deficit hyperactivity disorder. *Developmental Neuropsychology, 13,* 111-129.

Masi, G., Toni, C., Perugi, G., Travierso, M., Millepiedi, S., Mucci., M., & Akiskal, H. (2003). Externalising disorders in consecutively referred children and adolescents with bipolar disorder. *Comprehensive Psychiatry, 44,* 184-189.

McCormick, L. (2000). Improving social adjustment of children with ADHD. *Archives of Family Medicine, 9,* 94-99.

Mellor, D., Storer, S., & Brown, J. (1996). Attention deficit hyperactivity disorder: Perceptions, practice and politics. *Journal of Paediatric Child Health,* 32, 218-222.

Meltzer, H., Gatward, R,. Goodman, R., & Ford, F. (2000). *Mental health of children and adolescents in Great Britain.* London: HMSO.

Morrell, J. & Murray, L. (2003). Parenting and the development of Conduct Disorder and hyperactive symptoms in childhood. A prospective longitudinal study from 2 months to 8 years. *Journal of Child Psychology & Psychiatry, 44,* 489-508.

MTA Cooperative Group. (1999). 14-month randomized clinical trial of treatment strategies for attention deficit-hyperactivity disorder. *Archives of General Psychiatry, 56,* 1073-1086.

Neef, N., Bicard, D., & Endo, S. (2001). Assessment of impulsivity and the development of self-control in students with attention deficit hyperactivity disorder. *Journal of Applied Behavior Analysis, 34,* 397-408.

National Institute of Clinical Excellence (NICE). Technology appraisal guidance No. 13. Guidance on the use of methylphenidate (Ritalin, Equasym) for ADHD in childhood, October 2000.

Paule, M., Rowland, S., Fergusson, S., Chelonis, J., Tannock, R., Swanson, J., & Castellanos, F. (2000). Attention deficit/hyperactivity disorder: characteristics, interventions, and models. *Neurotoxicology and Teratology, 22(5),* 631-651.

Pearson, D., Lacher, D., Loveland, K., Santos, C., Faria, L., Azzam, P., Hentges, B., 7 Cleveland, L. (2000). Patterns of behavioural adjustment and

maladjustment in mental retardation: Comparison of children with and without ADHD. *American Journal of Mental Retardation, 105 (4),* 236-251.

Pearson, D., Santos, C., Roche, J., et al. (2003). Treatment effects of methyphenidate on behavioural adjustment in children with mental retardation and ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry,42,* 209-216.

Pearson, D., Lane, D., Santos, C. et al. (2004). Effects of methylphenidate treatment in children with mental retardation and ADHD: Individual variation in medication response. . *Journal of the American Academy of Child and Adolescent Psychiatry 43,* 686-698.

Pelham, W., Wheeler, T., & Chronis, A. (1998). Empirically supported psychosocial treatments for ADHD. *Journal of Clinical Child Psychology,* 27 (2), 190-205.

Pelham, W., Aronoff, H., Midlam, J., Shapiro, C., Gnagy, E., & Chronis, A. (1999). A comparison of Ritalin and Adderall: Efficacy and time-course in children with attention deficit-hyperactivity disorder: Just say yes to drugs alone? *Pediatrics Electronic Pages, 103.*

Pennington, B., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Childhood Psychology and Psychiatry, 37,* 51-87.

Perrins, S., Last, C. (1996). Relationship between ADHD and anxiety in boys: results from a family study. *Journal of the American Academy of Child and Adolescent Psychiatry, 35,* 988-996.

Rapport, M., Denney, C., DuPaul, G. &Gardner, M. (1994). ADHD and methyl-phenidate: Normalisation rates, clinical effectiveness, and response prediction in 76 children. *Journal of American Academy of Child and Adolescent Psychiatry, 33,* pp 882-893.

Rasmussen, P., & Gillberg, C. (2000). Natural outcome of ADHD with Developmental Coordination Disorder at age 22 years: a controlled, longitudinal, community-based study. *Journal of the American Academy of Child and Adolescent Psychiatry, 39,* 1424-1431.

Reid, R., Hakendorf, P., & Prosser, B. (2002). Use of psycho-stimulant medication for ADHD in South Australia. *Journal of American Academy of Child & Adolescent Academy, 41 (8),* 906-913.

Rucklidge, J., & Tannock, R. (2001). Psychiatric, psychosocial and cognitive functioning of female adolescents with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 40,* 530-540.

Sagvolden, T., Aase, H., Zeiner, P., & Berger, D. (1998). Altered reinforcement mechanisms in attention-deficit/hyperactivity disorder. *Behavior Brain Research, 94,* 61-71.

Scahill, L., Schwab-Stone, M., Merikangas, K., Leckman, J., Zhang, H., & Kasl, S. (1999). Psychosocial and Clinical Correlates of ADHD in a Community Sample of School-Age Children. *Journal of the American Academy of Child and Adolescent Psychiatry, 38(8),* 976-984.

Schachar, R., Mota, V., Logan, D., Tannock, R., & Klim, P. (2000). Confirmation of an inhibitory control deficit in attention-deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology, 28,* 227-235.

Schnoll, R., Burshteyn, D., & Cea-Aravena, J. (2003). Nutrition in the treatment of Attention Deficit Hyperactivity Disorder; a neglected but important aspect. *Applied Psychophysiology and Biofeedback, 28,* 63-75.

Schweitzer, J., & Sulzer-Azaroff, B. (1995). Self-control in boys with attention deficit hyperactivity disorder: Effects of added stimulation and time. *Journal of Childhood Psychology and Psychiatry, 36,* 671-686.

Seager, M., & O'Brien, G. (2003). Attention deficit hyperactivity disorder: review of ADHD in learning disability: the Diagnostic Criteria for Psychiatric Disorders for Use with Adults with Learning Disabilities/Mental Retardation [DC-LD] criteria for diagnosis. *Journal of Intellectual Disability Research, 47(1),* 26-31.

Sergeant, J., Abikoff, H., & Sonuga-Barke, E. (2002). How specific is a deficit of executive functioning for attention deficit hyperactivity disorder? *Behavior Brain Research, 130,* 3-28.

Shaffer, D., Fisher, P., Lucas, C., Dulcan, M., & Schwab-Stone, M. (2000). NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV): Description, differences from previous versions, and reliability of some common diagnoses, *Journal of the Academy of Child and Adolescent Psychiatry, 39,* 28-38.

Shelton,T., Barkley, R., & Crosswait, C. (1998). Psychiatric and psychological morbidity as a function of adaptive disability in preschool children with aggressive and hyperactive-impulsive-inattentive behaviour. *Journal of abnormal child psychology, 26,* 475-494.

Smalley, S., McGough, J., Del'Homme, M., NewDelman, J., Gordon, E., Kim, T., Liu, A., & McCracken, J. (2000). Familial clustering of symptoms and disruptive behaviours in multiplex families with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 39,* 1135-1143.

Solanto, M., Abikoff, H., Sonuga-Barke, E., Schachar, R., Logan, G., Wigal, T., Hechtman, L., Hinshaw, S., & Turkel, E. (2001). The Ecological Validity of Dealy Aversion and Response Inhibition as Measures of Impulsivity in ADHD: A Supplement to the NIMH Multimodal Treatment Study of ADHD. *Journal of Abnormal Child Psychology,* 29(3), 215-228.

Sonuga-Barke, E. (1994). On dysfunction and function in psychological accounts of childhood disorder. *Journal of Childhood Psychology and Psychiatry, 35,* 801-815.

Sonuga-Barke, E. (2002). Psychological heterogeneity in AD/HD: a dual pathway of cognition and performance. *Behavior Brain Research, 130,* 29-36.

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

Sonuga-Barke, E., Daley, D., Thompson, M., Weeks, A., & Laver-Bradbury, C. (2001). Parent based therapies for preschool attention-deficit/hyoperactivity disorder: a randomised control trial with a community sample. *Journal of the American Academy of Child and Adolescent Psychiatry,* 40, 402-408.

Sonuga-Barke, Dalen, L., & Remington, B. (2003). Do Executive Deficits and Delay Aversion Make Independent Contributions to Preschool Attention Deficit Hyperactivity Disorder Symptoms? *Journal of the American Academy of Child and Adolescent Psychiatry,* 42(11), 1335-1342.

Spencer, T., Biederman, M., Coffey, B., Geller, D., Wilens, T., & Faraone, S. (1999). The 4-year course of tic disorders in boys with attention deficit hyperactivity disorder. *Archive of General Psychiatry, 56,* 842-847.

Stevenson, J., Asherson, P., Hay, D., Levy, F., Swanson, J., Thapar, A., & Willcut, E. (2005). Charaterising the ADHD phenotype for genetic studies. *Developmental Studies, 8,* 115-121.

Stromme, P., & Diseth, T. (2000). Prevalence of psychiatric diagnoses in children with mental retardation: Data from a population-based study. *Developmental Medicine and Child Neurology, 42,* 266-270.

Swanson, J., Kraemer, H., Hinshaw, S. et al. (2001). Clinical relevance of the primary findings of the MTA: Success rates based on severity of ADHD and ODD symptoms at the end of treatment. *Journal of the American Academy of Child & Adolescent Psychiatry, 40,* 168-179.

Snyder, R., Turgay, A., Aman, M., Binder, C., Fisman, S., Carroll, A., & The Risperidone Conduct Study Group. (2002). Effects of risperidone on conduct and disruptive behavior disorders in children with sub-average IQ's. *Journal of the American Academy of Child and Adolescent Psychiatry, 41*, 1026-1036.

Szymanski, L., & King, B. (1999). Psychiatric diagnostic evaluation of mentally retarded individuals. *Journal of the American Academy of Child and Adolescent Psychiatry, 38,* 1606-1610.

Tannock, R. (1998). Attention deficit hyperactivity disorder: Advances in cognitine, neurobiological and genetic research. *Journal of Child Psychology and Psychiatry,* 39, 65-100.

Taylor, R. (1994). Syndromes of attention deficit and overactivity. In M. Rutter, E. Taylor, and L. Hersov (eds.), *Child and Adolescent Psychiatry: Modern Approaches* (3rd ed., pp. 258-307. Oxford: Blackwell.

Taylor, E. (1999). Developmental neuropsychopathology of attention deficit and impulsiveness. *Development and Psychopathology, 11,* 607-628.

Taylor, E., Chadwick, O., Hepinstall, E., & Danckaerts, M. (1996). Hyperactivity and conduct problems as risk factors for adolescent development. *Journal of the American Academy of Child and Adolescent Psychiatry, 35,* 1213-1226.

Thapar, A. (1995). Childhood hyperactivity scores are highly heritable and show sibling competition effects: Twin study evidence. *Behavior Genetics, 25,* 537-543.

Thapar, A., Holmes, J., Poulton, K., & Harrington, R. (1999). Genetic basis of attention deficit and hyperactivity. *British Journal of Psychiatry, 174,* 105-111.

Trenting, J., & Hinshaw, S. (2001). Depression and self-esteem in boys woth ADHD: associations with comorbid aggression and explanatory attributional mechanisms. *Journal of Abnormal Child Psychology, 29,* 23-39.

Tripp, G., & Alsop, B. (2001). Sensitivity to reward delay in children with attention deficit hyperactivity disorder (AD/HD). *Journal of Childhood Psychology and Psychiatry, 42,* 691-698.

Turgay, A., Binder, C., Snyder, R., & Fisman, S. (2002). Long-term safety and efficacy of risperidone for the treatment of disruptive behavior disorders in children with sub-average IQ's. Pediatrics, 110, 16-34.

Udwin, O., & Dennis, J. (1995). Psychological and behavioural phenotypes in genetically determined syndromes: a review of research findings. G. O'Brien & W. Yule (Eds.). In: *Behavioural Phenotypes' Clinics in Developmental Medicine, pp. 94-95.* Cambridge University Press: Cambridge.

Voeller, K. (2004). Attention-deficit hyperactivity disorder (ADHD). *Journal of Child Neurology, 19 (10),* 798-814.

Wallander, J., Brown, D., & Stankovic, S. (2002). *Mental health problems in African American adolescents in EMR Special Education reported by multiple sources.* Manuscript submitted for publication, University of Alabama at Birmingham.

Wallander, J., Dekker, K., & Koot, H. (2003). Psychopathology in children and adolescents with intellectual disability: Measurement, prevalence, course, and risk. *International Review of Research in Mental Retardation, 26,* 93-133.

WHO. *The ICD-10classification of mental and behavioural disorders: clinical descriptions and diagnostic guidelines 1992; diagnostic guidelines for research 1993.* Geneva: WHO.

Whitaker, A., Van Rossem, R., Feldman, J., Schonfeld, I., Pinto-Martin, J., Tore, C., Shaffer, D., & Paneth, N. (1997). Psychiatric outcomes in low birth weight children at age 6 years: relation to neonatal cranial ultrasound abnormalities. *Archives of General Psychiatry, 54,* 847-856.

SECTION 4

RESEARCH PAPER

The Presentation of Attention Deficit Hyperactivity Disorder (AD/HD) in Children with Intellectual Disabilities (ID)

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Research paper written in the style appropriate for the Journal of Intellectual and Developmental Disability. For author's instruction, please see attached material.

Abstract

Background: Attention deficit hyperactivity disorder is one of the most common childhood disorders, and is thought to affect more children with intellectual disabilities than those without. However, there has been little exploration into why this population appear to be at increased risk.

Aims: The study aimed to examine whether parental reports of hyperactivity were measuring the presence of an underlying liability to AD/HD in their children with intellectual disability, or whether they were being influenced by other factors, such as levels of adaptive behaviour or the presence of other behavioural problems.

Method: Parental reports of hyperactivity on a screening questionnaire were compared with semi-structured clinical interviews and an objective measure of AD/HD.

Results: Results found that the majority of children (n = 14), who were reported by parents as being hyperactive on the screening measure, were also found to exhibit AD/HD symptoms on subsequent measures.

Conclusions: These findings support those from previous findings that AD/HD is commonly exhibited by children with intellectual disability. However, they also highlight a need for more research to be undertaken on the specificity of AD/HD in children with intellectual disability. This would allow advancements to be made in the domains of assessment, diagnosis and intervention for children with intellectual disability and AD/HD.

Introduction

Attention deficit hyperactivity disorder (AD/HD; American Psychiatric Association, 1994) and Hyperkinesis (a form of AD/HD based on alternative diagnostic criteria; ICD-10, WHO 1992), are commonly recognised as one of the most prevalent disorders in children with and without intellectual disabilities (Dekker & Koot, 2003; Faraone, Sergeant, Gillberg, & Biederman, 2003; Mellor, Storer, & Brown, 1996). AD/HD is associated with marked social and educational impairment, and is recognised as a major risk factor for the development of problems throughout adolescence and adulthood such as conduct disorder, educational failure and personality disorder (Taylor, Chadwick, Heptinstall, & Danckaerts, 1996).

Although much research has attempted to investigate the aetiology of AD/HD (e.g. Castellanos & Acosta, 2002; Rutter, 2000; Stevenson et al, 2005), the precise cause of the disorder continues to remain unclear (Durston, 2003). Whilst genetic factors (Stevenson, 2005) and environmental factors (Dekker & Koot, 2003) both go some way in explaining why some children are at greater risk for the development of ADHD, neither can fully account for the development of the disorder (Daley, in press). However, there are currently two predominant, theoretical models that seek to separately explain the development, and maintenance, of AD/HD. They have both independently focussed on two single, neuropsychological deficits; firstly, executive dysfunction caused by deficient inhibitory control mechanisms (Barkley, 1997) and secondly, disturbances in motivational processes (Sagvolden, Aase, Zeiner, & Berger, 1998), and an aversion to delay (Sonuga-Barke, 1994). The first theoretical model, inhibitory dysfunction states that a child's AD/HD behaviour results from insufficient forethought, planning and control due to cognitive dysregulation (Schachar et al., 2000). This model is supported by evidence from a wealth of studies that demonstrate

poorer performance in the executive function of children with AD/HD when compared to non-AD/HD matched controls (Pennington & Ozonoff, 1996, Sergeant, Abikoff, & Sonuga-Barke, 2002).

The second theoretical model describes AD/HD, not as a disorder of cognitive dysregulation, but as a motivational style and therefore a *functional* response by the child (Sonuga-Barke, Houlberg, & Hall, 1994). This model was developed following investigations by Sonuga-Barke et al. (1994) into the findings of previous studies that reported AD/HD as a disorder of cognitive dysfunction; Sonuga-Barke et al., (1994) found that these results were consistently confounded by delay. The delay aversion hypothesis characterised the influence of delay on behaviour as dependent upon whether the child has control over their environment or not. For example, when a child with AD/HD is in control of their environment they can choose to minimise delay by acting impulsively, for example skipping the queue at the end of the slide! When the same child is not in control of their environment, or at least where they are expected to behave in a certain way or face sanctions, they will choose to distract themselves from the passing of time by either daydreaming (inattention) or fidgeting (hyperactivity).

Estimates regarding the prevalence of AD/HD continue to vary and unfortunately few studies have specifically investigated the prevalence of AD/HD in children with ID (Dekker & Koot, 2003). However, from the few that have AD/HD has been found to be more prevalent in children with ID than those without (Dekker & Koot, 2003; Linna et al., 1999; Rutter, Tizzard, & Whitmore, 1970; Strømme & Diseth, 2000). AD/HD is thought to affect between 2% and 5% of typically developing school-aged children and up to 2% of preschool children (Scahill et al., 1999). One study investigating prevalence of AD/HD in children with ID found a

prevalence rate of 15% (Dekker and Koot, 2003), and similarly, a population-based study undertaken by Strømme and Diseth (2000) found that 16% of their total sample with ID were experiencing symptoms of Hyperkinesis (ICD-10 criteria). High rates of AD/HD have also been found in children with ID when smaller group comparisons of children with and without ID have been undertaken (Epstein, Cullinan, & Gadow, 1986; Fee, Matson, & Benavidez, 1991). For example, Epstein and colleagues investigated the prevalence of AD/HD in children with special educational needs and found that between 14% and 21% of children with mild-moderate ID scored above the level of clinical concern for AD/HD symptoms on the Connors' Abbreviated Symptom Questionnaire (ASQ; Sprague & Sleator, 1973). More recently, Hastings, Beck, Daley, & Hill (2005) reported that 60% of their ID sample scored above the level of clinical concern on a questionnaire of AD/HD/hyperactivity, compared to only 2.7% of non-ID siblings. More importantly, the significantly increased prevalence of AD/HD remained, once chronological and mental age differences had been controlled.

Although it is generally agreed that AD/HD is one of the most prevalent childhood disorders (Dekker & Koot, 2003; Faraone, Sergeant, Gillberg, & Biederman, 2003; Mellor, Storer, & Brown, 1996) accurate assessment leading to the diagnosis of the disorder continues to be fraught with difficulties. The main problem faced by researchers and clinicians is a continued lack of understanding regarding the *specificity* of ADHD in contrast to other childhood disorders. This issue is especially pertinent in children with ID. It may be that higher reports of AD/HD symptoms in children with ID result from behaviours associated with their ID rather than as a result of underlying AD/HD symptomatology (Hastings et al., 2005). Due to most research having compared differences between typically developing children with and without

AD/HD, much less is known regarding its presentation in children with ID (Banaschewski et al., 2005). Another problem remains the issue of co-morbidity, and until more is known about how co-existent conditions and their associations differ from one another, this problem will remain (Gillberg et al., 2004); this too is particularly pertinent within the field of ID. Although research findings to date have identified many associated correlates, uncertainties remain regarding their specificity in contrast to other disorders and other populations (Hastings et al., 2005). Banaschewski et al., (2005) undertook a selective review of research that focussed upon the specificity of associations of AD/HD. They examined specificity between AD/HD and ODD/CD at a neuropsychological, structural, and pathophysiological level. The review revealed that to date no specific pathophysiological pathways for AD/HD have been identified. However, based on findings from a study by Solanto et al. (2001), Sonuga-Barke (2002) proposed a dual-pathway model of AD/HD, proposing two specific, independent pathways between biology and behaviour. One pathway mediated by deficits in executive function and the other, inhibition dysfunction and delay aversion. However, the specificity of these pathways in relation to other disorders has not yet been fully investigated.

It would appear that the specificity of AD/HD continues to be the key issue if researchers are to fully understand findings of elevated rates of AD/HD in children with ID. One study that has attempted to look at the specificity of AD/HD symptoms was conducted by Halperin and Gittelman (1992). They compared children with AD/HD with a non-AD/HD clinical control group (children diagnosed with a psychiatric disorder) and normal controls. They found that both patient groups displayed higher levels of inattention compared to controls, although both groups remained indistinguishable from each-other, and the AD/HD group was more active

compared to the other groups. The authors proposed that AD/HD may be uniquely characterised by hyperactivity.

In terms of researching the prevalence and presentation of AD/HD in children with ID there continue to be a number of methodological difficulties. Firstly, many studies largely gather information from behaviour-rating scales (as generally used with their non-ID counterparts). Information gathered in this way results in difficulties when it comes to accurately mapping symptom profiles of children with ID onto criteria used in current psychiatric diagnostic systems (Hastings et al., 2005). Clinical interviews have been found to elicit a greater degree of accuracy and detail regarding symptomatology needed within this client group for an accurate diagnosis (Dekker and Koot 2003).

Secondly, both sets of diagnostic criteria (DSM IV & ICD 10) specify that only behaviours inappropriate for the child's age should be regarded as symptoms, however no guidelines are given for to how to account for mental age, or differences in developmental course, when assessing children with ID (Wallander, Dekker, & Koot, 2003). Children with ID will, by definition, be developmentally delayed and will be experiencing a different developmental course to their non ID peers. In addition, research within the developmental tradition has implied that once children have been matched for mental age there is no strong evidence for the presence of attention deficits, thus concluding that the apparent increase in AD/HD in children with ID could be due to their developmental delay rather than an additional disorder (Burack, Evans, Klaiman, Iaroci, 2001). Given this research on attention, and current diagnostic criteria, it could be concluded that the apparent increased risk of AD/HD in children with ID could be simply due to their developmental delay.

There are however, at least two problems with this conclusion. Firstly, research has only taken into account the impact of developmental delay on the differential prevalence of inattention; impulsivity and hyperactivity, key aspects of the disorder appear to have been neglected (Hastings et al., 2005). Secondly, if developmental delay is correlated with AD/HD symptoms, one would expect to see increased AD/HD symptomatology in children with greater intellectual deficits. There is currently no data to support expectation (Dekker & Koot, 2003; Pearson & Aman, 1994).

Given these methodological difficulties there continue to be difficulties with being able to draw any conclusions regarding causes for the apparent increase in prevalence of ADHD in children with ID. For example, it remains unclear whether the increased prevalence arises from parental rating biases or a greater vulnerability to ADHD. Since few studies have addressed the specificity of AD/HD in children with ID, this study aimed to examine whether parental reports of hyperactive behaviour remained consistent with more in depth information obtained by semi-structured clinical interviews, and an additional objective measure of impulsivity. If increased ADHD symptoms were only evident in questionnaire measures, then rating biases might be the most likely explanation, however if increased ADHD symptoms remained evident in structured clinical interviews and objective measures of impulsivity, then a this might suggest a greater vulnerability to ADHD for these children with ID.

Methodology

Participants

In stage 1 (screening phase) a total of 58 parents completed a hyperactivity screening measure, thus giving an overall response rate of 28%. From the initial 58 parents that took part in the screening phase of the study, 31 parents agreed to continue participation in phase 2, thus yielding a response rate of 53% in stage 2.

The clinical and demographic characteristics of children in each stage of the study are illustrated in table 1. In both stages age was distributed between 6 and 12 years of age (mean age = 9.9 years), 75% of children were male, and they all attended schools for children with special needs in North Wales. Children included in both stages of the study were known to have additional diagnoses including autism, cerebral palsy, and Down syndrome, and other sensory impairments such as eyesight and hearing difficulties. Between 10-25% of children included in the study were experiencing additional difficulties such as epilepsy, mobility impairments, and other health problems such as bowel disorders. No exclusion criteria were applied. Disorders such as autism and cerebral palsy tend to be among the most common co-morbid diagnoses associated with children with ID, as are a number of medical difficulties, such as epilepsy, sensory impairments, and mobility difficulties (Chadwick et al., 2005). For example, in a recent population study undertaken by Chadwick et al. (2005) a similar percentage of their sample (4-22%) had mobility difficulties, epileptic seizures, and bowel problems.

{insert table 1 here}

Measures

Clinical and demographic information

Clinical and demographic information was requested from each parent/guardian about them and their child with an intellectual disability. Information requested about the parent/guardian included: sex, age, marital status, number of adults and children in household, educational qualifications, employment, post code, and their relationship to the child with a disability. Information about the child included: age, sex, diagnoses/conditions, sensory impairment, epilepsy, mobility difficulties, and other health problems.

Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997, 1998, 1999)

The SDQ is a brief informant-rated measure of 25 behavioural attributes, some positive and some negative, for use with children aged between 3-16 years with and without ID. The 25 items are divided into 5 subscales which assess emotional symptoms (e.g., 'often seems worried'), conduct problems (e.g., 'often has temper tantrums'), hyperactivity/inattention (e.g., 'is easily distracted'), peer relationships (e.g., 'has at least one good friend'), and prosocial behaviour (e.g., is kind to younger children'). Scores obtained from the subscales, excluding the prosocial behaviour subscale, are used to generate a total difficulties score.

The SDQ is a well validated tool, and has been found to be as effective in identifying clinically significant levels of behavioural disturbance in children as both the Child Behaviour Checklist (Achenbach, 1991), and the Rutter Scales (Elander & Rutter, 1996; Goodman, 1997; Goodman & Scott, 1999). The form is quick to administer, taking only approximately 5 minutes to complete and has been found to have good test-retest reliability (mean: .62) and internal consistency (mean Cronbach [alpha]: .73; Goodman, 2001).

Parent Account of Childhood symptoms (PACS – a structured clinical interview; Taylor et al., 1991).

The PACS is a structured clinical interview used to assess the core symptoms of AD/HD. Parents describe the severity and frequency of these symptoms across a range of situations (e.g., in the home, with friends, in public etc.) over the previous six months. These descriptions are then rated by trained interviewers using criteria validated according to clinical practice (Taylor et al., 1991). It is designed for use with children aged between 6 and 11 years and takes approximately 15 minutes to administer. The PACS has high inter-rater reliability and good construct validity and has been well validated against clinical judgement.

Vineland Adaptive Behaviour Scale – Survey Form (VABS; Sparrow, Balla, & Cicchetti, 1984)

The VABS is a semi-structured interview measure that contains 297 items in total. Not all 297 items are used during its administration, rather enough specific questions are asked to allow the interviewer to make an accurate rating of adaptive behaviour. The VABS is sub-divided into 4 domains which each give an adaptive behaviour score, and when added together yield a total adaptive behaviour composite score. The 4 domains are: socialisation, daily living skills, communication, and motor skills. The latter is only applicable to children with a developmental age of five years and under, and is not needed to derive the total adaptive behaviour composite score; therefore this domain was not administered. Administration of the VABS took approximately 30 minutes and was administered at the same time as the PACS. Inter-rater reliability for the VABS in this study was .94.

Cookie Delay Task (C-DT; delay of gratification; Campbell et al., 1982).

The Cookie-Delay Task involves an experimenter placing a desired item e.g. a chocolate button under a transparent cup in placed front of the child. They are

informed/shown that they can only have the treat after the experimenter has clapped her hands. Three practise trials are run to ensure the child has understood the rule. During each trial the child is reminded not to move before hearing the sound of the clap. If during these practise trials the child is unable to inhibit their behaviour, they are asked to place their hands on the table at the start of each trial. This enables the experimenter to identify a purposeful move over a twitch.

Up to eight trials are run with time delays varying randomly between 5 seconds and 30 seconds between first placing the desired item under the cup and giving the signal that the item could be taken (clap). During the midpoint of each trial the experimenter raises her hands as if intending to clap, but not doing so (e.g. if total time delay was 30 seconds, the experimenter would raise hands as if about to clap after 15 seconds). The child is rated on the level of inhibition shown at this time. The scoring of inhibition is as follows: 0 = fully inhibited (there was no movement toward the cup); 1 = partially inhibited (any movement towards or touching of the cup); and 2 = not inhibited (the cup was lifted and/or the sweet was taken).

The range of possible scores over 8 trials varies between 0-16, with higher scores indicating greater impulsivity. The C-DT has been used successfully with typically developing children with AD/HD in previous research (see Sonuga-Barke, 2002; Sonuga-Barke, Dalen & Remington, 2003). A study undertaken by Sonuga-Barke et al. (2003) incorporated all the tests described for the purpose of this study (SDQ; PACS; & C-DT) and found that test-re-test reliability was acceptable for all the tests ($r > .66$).

Procedure

All schools in North Wales for children with special needs were invited to participate in the research (1 school per county; 7 schools). In total 6 schools stated

that they would be willing to participate. Following this, questionnaire packs (stage 1) were issued to parents/guardians of children aged between 6-12 years by classroom teachers across all participating schools. Each pack contained information about the research, 2 consent forms (one for them to retain and one to return), a Strengths and Difficulties Questionnaire, and a demographics information sheet. A total of 255 questionnaire packs were distributed, and 58 completed questionnaires were returned (22.7%). Parents/guardians were also asked to state whether they would be willing to be contacted for participation in the next stage of the study. Out of the total sample that took part in the stage 1, 94% gave their consent to be contacted for participation in stage 2.

Information outlining stage 2, and consent forms were issued to all parents/guardians who had stated that they would be willing to continue their participation. A total of 31 consent forms were returned (53%). These parents were subsequently contacted and convenient dates and times were scheduled in order to complete the telephone interview. Telephone interviews were carried out whereby parental reports of their child's AD/HD symptoms (PACS) and levels of adaptive behaviour (VABS) were obtained. Each telephone interview lasted for approximately 45 minutes and was tape recorded for inter-rater reliability purposes.

Finally, the Cookie Delay Task (CDT) was conducted in the school environment with each child whose parent/guardian had been interviewed.

Results

Data Preparation

Outliers that were likely to affect the analysis of the data were identified and adapted scores relative to the mean of the remaining scores were applied. Missing data was identified and acknowledged by the application of missing data values. The parametric state of the data was examined using the Kolmogorov-Smirnov test. Results confirmed that the scores on the PACS ($Z = .832$; $p = .493$), cookie delay task ($Z = 1.038$; $p = .231$), and VABS ($Z = .947$; $p = .331$) all had good approximations to normal distributions. However, the results of the Kolmogorov-Smirnov test on SDQ scores, in all five subscales, indicated that this data was non-parametric ($Z \geq 3.279$; $p \leq .000$). Thus, the Mann-Whitney non-parametric test was used in order to determine differences on SDQ scores. SDQ hyperactivity scores were used as a grouping variable to create two groups for all subsequent analysis, a low hyperactivity group (based on normal and borderline SDQ hyperactivity scores), and a high hyperactivity group (based on children in the clinical range for hyperactivity on the SDQ). 68% of children that were reported by parents to be exhibiting high levels of hyperactive behaviour according to the SDQ also achieved ratings of clinical levels of hyperactivity according to the PACS. All demographic differences were explored prior to subsequent analysis. Gender, epilepsy, other health problems, and additional diagnoses (including autism) were found to have no influence on SDQ total difficulties scores or hyperactivity scores, as reported by parents on the SDQ and PACS. However, mobility was found to have a significant effect on parental ratings of hyperactivity on the SDQ, $Z = .483$; $p = <0.05$, indicating that children with greater mobility difficulties were reported by parents to exhibit higher levels of hyperactive behaviour. Further, mobility was found to have a marginally significant influence on

parental perceptions of hyperactivity as reported during the PACS, Z = 1.722; p = >.05. Parents of children with mobility impairments were found to report higher levels of hyperactivity.

Differences between participants in stage 1 and stage 2 of the study

A Mann-Whitney *U*-test was employed to investigate differences between the 58 children in the screening phase of the study and the 31 who participated in the AD/HD and adaptive behaviour measures (stage 2). No significant differences in SDQ subscale scores were found between those who took part in the screening phase and those who agreed to participate in stage 2.

Difficulties identified by the SDQ

The percentage of difficulties reported by parents in both stage 1 and 2 on the SDQ were identified. It can be seen from table 2 that parents in both samples reported peer problems and hyperactive behaviours to be the most clinically problematic behaviours exhibited by their children with ID. Emotional problems were reported to be the least problematic, with over 54% of both samples being within the normal range. These findings are similar to normative data available on the SDQ generated from the ONS study (see Meltzer et al., 2000). As with the findings of this study, hyperactive behaviour was the most clinically problematic behaviour reported by parents, and the least problematic (excluding prosociality) was emotional problems. However, according to the normative data conduct problems were reported to be more problematic than peer problems; this was not the case in the present study.

{insert table 2 here}

Other behaviour problems

Mann Whitney *U*-tests were employed to examine differences between the hyperactive and non-hyperactive groups on other behaviour problems on the SDQ. The results of this analysis are displayed in table 3 and show that the groups differed significantly on conduct problems and pro-social behaviour. Children with ID and high hyperactivity were rated by parents, as displaying significantly *more* conduct problems and significantly *less* pro-sociality than their non-hyperactive ID peers.

(insert table 3 here}

Differences between the groups on measures of ADHD, adaptive behaviour and impulsivity.

Subsequent analysis was carried out to investigate the influence of hyperactivity on AD/HD and adaptive behaviour scores. Both PACS and the VABS scores were entered into the analysis of variance (ANOVA) as the dependent variables, with high and low SDQ hyperactivity scores as the fixed factor. Results of the ANOVA are shown in table 4. Children who were rated by their parents as displaying high hyperactivity were also reported to exhibit significantly higher ADHD symptoms in a structured clinical interview (PACS), and significantly more impulsive responses in the cookie delay task. These statistically significant differences also yielded large effect sizes. In addition, children with high hyperactivity were also reported by parents to display significantly lower levels of adaptive behaviour.

{insert table 4 here}

Differences between the groups on measures of ADHD, controlling for other differences.

Previous analysis indicated significant differences between the high and low hyperactivity group for prosociality, conduct problems, and levels of adaptive behaviour. Correlational analysis also indicated significant associations between the high and low hyperactivity group and prosociality, $r = .521$; $p < .05$, conduct problems, $r = .401$; $p < .05$, and adaptive behaviour, $r = 0471$; $p < .05$. Due to limitations with power in this analysis, before controlling for prosocial behaviour, conduct problems, and adaptive behaviour, a new composite score for AD/HD was created. PACS typical scores and cookie delay scores were standardised and then aggregated to yield a new score for overall AD/HD behaviour. This new composite score was entered into an analysis of covariance (ANCOVA) as the dependent variable, with the high and low SDQ hyperactivity score entered as the fixed factor. SDQ conduct and prosocial scores, along with the total adaptive behaviour scores were entered as covariates. Results indicated that even when all other differences were controlled, high scoring SDQ hyperactivity children demonstrated significantly more AD/HD behaviour, $F(1,29) = 9.90$; $p<0.05$.

Discussion

The results from this study highlight a number of findings. Firstly, hyperactivity was rated by parents on the SDQ, as the most problematic behaviour exhibited by their child with ID, after peer problems. Furthermore, the majority of parental reports of high hyperactivity on the SDQ remained consistent with additional measures of AD/HD symptomatology, namely the PACS and CDT. This finding signifies that parental reports of high hyperactive behaviour are likely to be indicative of an underlying liability of AD/HD; and not due to the presence of intellectual disability. These results appear to be consistent with findings from previous research that suggest AD/HD is as common, if not more common, in children with ID as it is in those without (Dekker & Koot, 2003; Linna et al., 1999; Rutter et al., 1970; Strømme & Diseth, 2000). In addition due to the fact that hyperactivity was confirmed by additional, objective measures of AD/HD, we can also conclude that reported AD/HD symptoms were not as a result of rating bias; a difficulty frequently experienced in the past, before information was sought from a number of individuals in a number of settings (Mellor, Storer, & Brown, 1996).

From these findings it would appear that the increased prevalence of AD/HD in children with ID (Dekker & Koot, 2003; Linna et al., 1999; Rutter et al., 1970; Strømme & Diseth, 2000) is more accurately explained by a greater vulnerability to AD/HD in this population, rather than by behaviours associated with ID per se, or rating biases. This conclusion appears to be consistent with that of Halperin and Gittelman (1992) who stated that AD/HD appears to be uniquely characterised by hyperactivity. However, much more research is needed into the specificity of AD/HD before this can be known for sure.

The high rate of reported peer difficulties could be explained by a number of factors. Primarily, all of the children involved in this study attended special schools for children with ID that cater for children living in a wide geographical area. Parents reported to most commonly observe their child interacting with their non-ID siblings at home; having had very little experience of their child's interactions with contemporaries at school. Unfortunately no questionnaire measures were completed by school staff to investigate differences in reports of peer relationships in different environments. On the other hand, it is reported that children with ID and AD/HD often find themselves socially isolated (Lufi & Parish-Plass, 1995; Mino et al., 1990) and experiencing difficulties both initiating and maintaining peer relationships (McCormick, 2000). Therefore, high levels of reported peer relationship difficulties reported by parents of children in this sample could have been due to increased AD/HD symptoms impacting upon their ability to make and maintain friendships.

The second main finding was that children, who were rated as most hyperactive by parents in the study, also appeared to display the lowest levels of adaptive behaviour. This finding could lead us to presume that AD/HD symptoms are correlated with severity of intellectual disability (more pronounced with more developmental delay). However, there appears to have been no research that has specifically investigated the relationship between severity of ID and vulnerability to AD/HD (Hasting et al., 2005; Dekker & Koot, 2003; Pearson & Aman, 1994). In addition to low adaptive behaviour, children with high hyperactivity were also reported to display significantly more conduct problems, and significantly less prosociality than children with low hyperactivity. In order to investigate whether adaptive behaviour, conduct problems and prosociality were influencing parental reports of hyperactivity, analyses were conducted that controlled for the influence of

these factors. Results showed that increased AD/HD symptoms in semi-structured clinical interviews and an objective measure of impulsivity remained evident. However, more research investigating the influence of factors, such as degree of ID, on the development of AD/HD would be extremely interesting and useful.

Clinical implications

There are a number of clinical implications resulting from the findings of this study. Primarily, none of the children, who were reported by parents to be displaying high levels of hyperactivity, were either undergoing assessment for, or had previously received a diagnosis of, AD/HD. It would appear that much of the problematic behaviour displayed by the children with high hyperactivity continues to be attributed to their ID rather than an underlying disorder, such as AD/HD. Given what researchers and clinicians alike know regarding the impact of undiagnosed AD/HD in children without ID, the need for early assessment and diagnosis of the disorder cannot be emphasised enough. However, due to the lack of understanding regarding the specificity of AD/HD in contrast to other childhood disorders, especially in children with ID, accurate assessment and subsequent diagnosis of AD/HD continues to be the main problem faced by researchers and clinicians alike. More research is needed into the specific signs of AD/HD symptoms in children with ID. It may be possible that further studies will also find that hyperactivity, as reported by parents, is strongly indicative of underlying AD/HD (Glascoe, Altemeier, & MacLean, 1989). If this is found to be the case, parental reports of hyperactivity could lead to earlier assessment, diagnosis and treatment of AD/HD for these children before associated difficulties such as school failure, peer rejection and the deterioration of adult-child relationships has become entrenched (Reid, 1993).

In terms of intervention, recent research indicates that early psycho-social intervention for AD/HD yields the best results (Sonuga-Barke et al., 2001). Recent outcome data on parent behaviour management interventions for pre-school, non-ID, children with AD/HD has shown that it was clinically efficacious (Bor, Sanders, & Makie-Dadds, 2002; Sonuga-Barke et al., 2001). Although there is no data on the efficacy of parent behaviour management with children with ID, there is no reason to assume that similar intervention methods would not be as effective with this population. If developmental delay is taken into account, parent behaviour management could be just as efficacious when delivered to children with ID at a later age, as it is in the non-ID population.

Psychosocial interventions, such as parent behaviour management, have additional advantages for use with children with ID; many children with ID and AD/HD are likely to be on medications for an array of associated medical conditions and are therefore at increased risk of experiencing an adverse response to stimulants (Handen et al., 1991). In addition, it is reported that there is a greater heterogeneity in stimulant response in children with ID compared to their non-ID counterparts (Aman, 1996; Aman, Buican, & Arnold, 2003), further emphasising the need for increased research into alternative, non-pharmacological interventions.

Limitations of the study

The primary limitation of the study was a lack of power, and although good effect sizes were found, it would have been interesting to have been able to make comparisons between other possible impacting variables. For example, although the presence of autism was not found to significantly influence parental reports of hyperactivity, a purer sample, gained by excluding children with additional diagnoses such as autism, would have been preferred. In addition, reports of AD/HD symptoms

on the SDQ and in the semi-structured interviews were mainly maternal. It would have been more insightful to have gained additional paternal and teacher reports of behaviour. This would have allowed for comparisons to have been made between different accounts of observable behaviour, further strengthening the identification of AD/HD symptomatology. Clinically, in order for a child to obtain a diagnosis of AD/HD, symptoms have to be observed and reported in different settings e.g. at home and at school (APA, 1994).

A further limitation of the study was the inclusion of only two groups (high and low scorers on parental reports of hyperactivity). Initially the study aimed to include non-ID children with and without AD/HD in addition to the two groups included. This would have allowed for greater comparisons to be made between groups, allowing further insight into the specificity of AD/HD in children with ID. The study originally aimed to include two further neuropsychological tests; the Stop-Signal Task (SST), and the Choice-Delay Task (CH-DT). Both these tasks have been reported to differentiate children with and without AD/HD (Solanto et al., 2002). The inclusion of these tests would have firstly given further confirmation of AD/HD symptoms, and secondly allowed greater insight into the utility of the Cookie Delay Test with children with ID and AD/HD. However, as with the CDT, neither the SST nor CH-DT has been validated for use with children with ID. A number of children who scored highly for hyperactivity on the SDQ did not score highly on the CDT. One explanation for this may be that good parenting is a moderator of AD/HD symptoms (Maniadaki, Sonuga-Barke, & Kakouro, 2005). Parents may have been correctly rating their child's AD/HD symptoms, but due to good parenting skills the child has learnt how to manage their behaviour in an appropriate manner, thus full AD/HD symptomatology does not develop. More research needs to be undertaken on

both the specificity of AD/HD in children with ID, and the diagnostic utility of clinical measures of AD/HD for use with individuals with ID (Hastings et al., 2005).

Many questions are left unanswered in relation to AD/HD in children with intellectual disabilities. As well as a need for greater understanding into the specificity of AD/HD in children with ID, more research needs to investigate why AD/HD develops in some children and not in others, and what diagnostic measures can be reliably used for the assessment of the disorder in this client group. Although, there is a lot of research that has identified copious risk factors associated with the development of AD/HD (Larsson, Larsson, & Lichtenstein, 2004; Tannock, 1998), both genetic (Stevenson et al., 2005) and environmental (Hinshaw, 1994; Taylor, 1994; Anastropoulos, Barkley, & Shelton, 1996), it remains unclear as to why AD/HD develops in some children and not others who are equally exposed to them (Durston, 2003). Future studies investigating the influence of parenting styles on the development of the disorder would be extremely beneficial. Research of this kind would help inform clinicians and researchers alike as to what interventions would be most appropriate. This is crucial if children are to avoid the long-term, associated features of AD/HD. Given that recent clinically and statistically significant outcome data has been obtained from research into the efficacy of parent management interventions for pre-school children (Bor, Sanders, & Markie-Dadds, 2002; Sonuga-Barke, et a.l, 2001), it would appear that there is much potential in a coherent effort being directed at psycho-social interventions. The presence of AD/HD in children with intellectual disabilities is being increasingly recognised, so it is important that developments in research, theory, and treatment for children with ID and AD/HD continue.

References

Achenbach, T. (1991). *Manual for the Child Behaviour Checklist.* Department of Psychiatry: University of Vermont.

Aman, M. (1996). Stimulant drugs in the development disabilities revisited. *Journal of Developmental and Physical Disabilities, 8,* 347-365.

Aman, M., Buican, B., & Arnold, L. (2003). Methylphenidate treatment in children with borderline IQ and mental retardation: analysis of three aggregated studies. *Journal of Child and Adolescent Psychopharacology, 13,* 29-40.

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders 4th Edition (DSM-IV).* Washington DC: American Psychiatric Association.

Anastropoulos, A., Barkley, R., & Shelton, T. (1996). Family based treatment: Psychosocial intervention for children and adolescents with attention deficit hyperactivity disorder. In E. Hibbs and P. Jensen (eds.), *Psychosocial treatments for Child and Adolescent Disorders: Empirically Based Strategies for Clinical Practice* (pp. 267-284). Washington, DC: American Psychiatric Association.

Banaschewski, T., Hollis, C., Oosterlaan, J., Roeyers, H., Rubia, K., & Taylor, E. (2005). Towards an understanding of unique and shared pathways in the psychopathophysiology of ADHD. *Developmental Science, 8(2),* 132-140.

Barkley, R. (1997). Behavioural inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin, 121,* 65-94.

Biederman, J., Faraone, S., & Keenan, K et al. (1992). Further evidence for family-genetic risk factors in attention deficit hyperactivity disorder: patterns of comorbidity in probands and relatives psychiatrically and pediatrically referred samples. *Archives of General Psychiatry,* 49: 728-738.

Bor, W., Sanders, M. & Markie-Dadds, C. (2002). The effect of Triple P-Positive Parenting Programme on preschool children with co-occurring disruptive behavior and attentional/hyperactive difficulties. *Journal of Abnormal Child Psychology, 30,* 571-578.

Burack, J., Evans, D., Klaiman, C., & Iarocci, G. (2001). The mysterious myth of attention deficits and other defect stories: Contemporary issues in the developmental approach to mental retardation. *International Review of Research in Mental Retardation, 24,* 299-320.

Campbell, S., Szumowski, E., Ewing, L., Gluck, D., & Breaux, A. (1982). A multi-dimensional assessment of parent-identified behaviour problem toddlers. *Journal of Abnormal Child Psychology, 10,* 569-592.

Castellanos, F. & Acosta, M. (2002). Syndrome od attention deficit with hyperactivity as the expression of an organic functional disorder. *Revists De Neurologia, 35,* 1-11.

Chadwick, O., Kusel, Y., Cuddy, M., & Taylor, E. (2005). Psychiatric diagnoses and behaviour problems from childhood to early adolescence in young people with severe intellectual disabilities. *Psychological Medicine, 35,* 751-760.

Daley, D. (In press). Attention Deficit Hyperactivity Disorder: A review of the essential facts.

Dekker, M., & Koot, H. (2003). DSM-IV disorders in children with borderline to moderate intellectual disability. I: Prevalence and impact. *Journal of the American Academy of Child and Adolescent Psychiatry, 42(8),* 915-922.

Durston, S. (2003). A review of the biological bases of ADHD: What we have learned from imaging studies? *Mental Retardation and Developmental Disabilities Research Reviews, 9(3),* 184-195.

Elander, J., & Rutter, M. (1996). Use and development of the Rutter parents' and teachers' scales. *International Journal of Methods in Psychiatric Research, 6,* 63-78.

Epstein, M., Cullinan, D., & Gadow, K. (1986). Teacher ratings of hyperactivity in learning disabled, emotionally disturbed, and mentally retarded children. *Journal of Special Education, 20,* 219-229.

Fee, V., Matson, J., Moore, L., & Benavidez, D. (1993). The differential validity of hyperactivity/attention deficits and conduct problems among mentally retarded children. *Journal of Abnormal Child Psychology, 21,* 1-11.

Faraone, S., Sergeant, J., Gillberg, C., & Biederman, J. (2003). The worldwide prevalence of ADHD: is it an American Condition? *World Psychiatry, 2,* 104-113.

Gillberg, C., Gillberg, I., Rasmussen, P., Kadesjö, B., Söderström, H., Råstam, M., Johnson, M., Rothenberger, A., & Niklasson, L. (2004). Co-existing disorders in ADHD-implications for diagnosis and intervention. *European Child and Adolescent Psychiatry, 13(1),* 80-92.

Gillis, J., Gilger, J., Pennington, B., & DeFries, J. (1992). Attention deficit disorder in reading-disabled twins: Evidence for a genetic etiology. *Journal of Abnormal Child Psychiatry, 20,* 303-315.

Glascoe, F., Altemeier, W., & MacLean, W. (1989). The importance of parents' concerns about their child's development. *American Journal of Disabilities and Childhood, 143,* 955-958.

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

Goodman, R, Meltzer, H., & Bailey, V. (1998). The Strengths and Difficulties Questionnaire: A pilot study on the validity of the self-report version. *European Child and Adolescent Psychiatry, 7,* 125-130.

Goodman, R. (2001). Psychometric properties of the Strength and Difficulties Questionnaire. *Journal of the Academy of Child and Adolescent Psychiatry, 40 (11),* 1337-1345.

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

Halperin, J., & Gittelman, R. (1992). Do hyperactive children and their sibling differ in IQ and academic achievement? *Psychiatry Research, 6,* 253-258.

Handen, B., Feldman, H., Gosling, A., Breaux, A., & McAuliffe, S. (1991). Adverse side-effects of methylphenidate among mentally retarded children with AD/HD. *Journal of the American Academy of Child and Adolescent Psychiatry,30,* 241-245.

Hastings, R., Beck, A., Daley, D., & Hill, C. (2005). Symptoms of ADHD and their correlates in Children with Intellectual Disabilities. *Research in Developmental Disabilities, 26,* 456-468.

Hinshaw, S. (1994). *Attention deficits and hyperactivity in children.* Thousand Oaks, CA: Sage

Larsson, J., Larsson, H., & Lichtenstein, P. (2004). Genetic and environmental contributions to stability and change of ADHD symptoms between 8 and 13 years of age: a longitudinal twin study. *Journal of the American Academy of Child and Adolescent Psychiatry, 43,* 1267-1275.

Levy, F., Hay, D., McStephen, M., Wood, C., & Waldman, I. (1997). Attention deficit-hyperactivity disorder: A category or a continuum? Genetic analysis of a large-scale twin study. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 737-744.

Linna, S., Moilanen, I., Ebeling, H., Piha, J., Kumpulainen, K., Tamminen, T., 7 Almqvist, F. (1999). Psychiatric symptoms in children with intellectual disability, *European Child and Adolescent Psychiatry, 8,* 77-82.

Lufti, D., & Parrish-Plass, J. (1995). Personality assessment of children with ADHD. *Journal of Clinical Psychology, 51,* 94-99.

Maniadaki, K., Sonuga-Barke, E., Kakouros, E. (2005). Parents' causal attributions about attention/hyperactivity disorder: the effect of child and parent sex. *Child, Care, Health, & Development, 31,* 331-340.

McCormick, L. (2000). Improving social adjustment of children with ADHD. *Archives of Family Medicine, 9,* 94-99.

Mellor, D., Storer, S., & Brown, J. (1996). Attention deficit hyperactivity disorder: Perceptions, practice and politics. *Journal of Paediatric Child Health,* 32, 218-222.

Mino, Y., Yasuda, M., Ohara, H., Nagamatsu, K., & Yoshida, T. (1990). Isolation of children with ADD in their classrooms. *Nippon Koshu Eisei Zasshi, 37,* 843-849.

Pearson, D., & Aman, M. (1994). Ratings of hyperactivity and developmental indices: Should clinicians correct for developmental delay? *Journal of Autism and Developmental Disorders, 24,* 395-411.

Pennington, B., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Childhood Psychology and Psychiatry, 37,* 51-87.

Reid, J. (1993). Prevention of conduct disorder before and after school entry: relating interventions to developmental findings. *Development and Psychopathology, 5,* 243-262.

Rutter, M., Tizzard, J., & Whitmore, K. (1970). *Education, health, and behavior.* London: Longwood.

Rutter, M. (2000). Risks and outcomes in developmental psychopathology. *British Journal of Psychiatry, 177,* 569-570.

Sagvolden, T., Aase, H., Zeiner, P., & Berger, D. (1998). Altered reinforcement mechanisms in attention-deficit/hyperactivity disorder. *Behavior Brain Research, 94,* 61-71.

Scahill, L., Schwab-Stone, M., Merikangas, K., Leckman, J., Zhang, H., & Kasl, S. (1999). Psychosocial and Clinical Correlates of ADHD in a Community Sample of School-Age Children. *Journal of the American Academy of Child and Adolescent Psychiatry, 38(8),* 976-984.

Schachar, R., Mota, V., Logan, D., Tannock, R., & Klim, P. (2000). Confirmation of an inhibitory control deficit in attention-deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology, 28,* 227-235.

Seager, M., & O'Brien, G. (2003). Attention deficit hyperactivity disorder: review of ADHD in learning disability: the Diagnostic Criteria for Psychiatric Disorders for Use with Adults with Learning Disabilities/Mental Retardation [DC-LD] criteria for diagnosis. *Journal of Intellectual Disability Research, 47(1),* 26-31.

Sergeant, J., Abikoff, H., & Sonuga-Barke, E. (2002). How specific is a deficit of executive functioning for attention deficit hyperactivity disorder? *Behavior Brain Research, 130,* 3-28.

Smalley, S., McGough, J., Del'Homme, M., NewDelman, J., Gordon, E., Kim, T., Liu, A., & McCracken, J. (2000). Familial clustering of symptoms and disruptive behaviours in multiplex families with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 39,* 1135-1143.

Solanto, M., Abikoff, H., Sonuga-Barke, E., Schachar, R., Logan, G., Wigal, T., Hechtman, L., Hinshaw, S., & Turkel, E. (2001). The Ecological Validity of Dealy Aversion and Response Inhibition as Measures of Impulsivity in ADHD: A Supplement to the NIMH Multimodal Treatment Study of ADHD. *Journal of Abnormal Child Psychology,* 29(3), 215-228.

Sonuga-Barke, E. (1994). On dysfunction and function in psychological accounts of childhood disorder. *Journal of Childhood Psychology and Psychiatry, 35,* 801-815.

Sonuga-Barke, E. (2002). Psychological heterogeneity in AD/HD: a dual pathway of cognition and performance. *Behavior Brain Research, 130,* 29-36.

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

Sonuga-Barke, E., Daley, D., Thompson, M., Weeks, A., & Laver-Bradbury, C. (2001). Parent based therapies for preschool attention-deficit/hyoperactivity disorder: a randomised control trial with a community sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 40(4)*, 402-408.

Sonuga-Barke, Dalen, L., & Remington, B. (2003). Do Executive Deficits and Delay Aversion Make Independent Contributions to Preschool Attention Deficit Hyperactivity Disorder Symptoms? *Journal of the American Academy of Child and Adolescent Psychiatry,* 42(11), 1335-1342.

Sonuga-Barke, E., Thompson, M., Daley, D., & Laver-Bradbury, C. (2004). Parent training for Attention Deficit/Hyperactivity Disorder: Is it as effective when delivered as routine rather than specialist care?. *British journal of Clinical Psychology, 43,* 449-457.

Sparrow, S., Balla, D., & Cicchetti, D. (1984). *The Vineland Adaptive Behaviour Scale (Interview edition survey form).* Circle Time, MN: American Guidance Service.

Sprague, R., & Sleator, E. (1973). Effects of psychopharmacologie agents in learning disorders. *Peadriatric Clinics of North America, 20,* 7191-7735

Stevenson, J., Asherson, P., Hay, D., Levy, F., Swanson, J., Thapar, A., & Willcut, E. (2005). Charaterising the ADHD phenotype for genetic studies. *Developmental Studies, 8,* 115-121.

Strømme, P., & Diseth, T. (2000). Prevalence of psychiatric diagnoses in children with mental retardation: Data from a population-based study. *Developmental Medicine and Child Neurology, 42,* 266-270.

Swanson, J., Sergeant, J., Taylor, E., Sonuga-Barke, E., Jensen, P., & Cantwell, D. (1998). Attention-deficit hyperactivity disorder and hyperkinetic disorder. *The Lancet, 351,* 429-433.

Tannock, R. (1998). Attention deficit hyperactivity disorder: Advances in cognitine, neurobiological and genetic research. *Journal of Child Psychology and Psychiatry,* 39, 65-100.

Taylor, R. (1994). Syndromes of attention deficit and overactivity. In M. Rutter, E. Taylor, and L. Hersov (eds.), *Child and Adolescent Psychiatry: Modern Approaches* (3rd ed., pp. 258-307. Oxford: Blackwell.

Thapar, A., Holmes, J., poulton, K., & Harrington, R. (1999). Genetic basis of attention deficit and hyperactivity. *British Journal of Psychiatry, 174,* 105-111.

Thapar, A. (1995). Childhood hyperactivity scores are highly heritable and show sibling competition effects: Twin study evidence. *Behavior Genetics, 25,* 537-543.

Taylor, E., Chadwick, O., Hepinstall, E., & Danckaerts, M. (1996). Hyperactivity and conduct problems as risk factors for adolescent development.

Journal of the American Academy of Child and Adolescent Psychiatry, 35, 1213-1226.

Taylor, E., Sanberg, S., Thorley, G., & Giles, S. (1991). *The Epidemiology of Childhood Hyperactivity.* Oxford, England: Oxford University Press.

Wallander, J., Dekker, K., & Koot, H. (2003). Psychopathology in children and adolescents with intellectual disability: Measurement, prevalence, course, and risk. *International Review of Research in Mental Retardation, 26,* 93-133.

WHO. *The ICD-10classification of mental and behavioural disorders: clinical descriptions and diagnostic guidelines 1992; diagnostic guidelines for research 1993.* Geneva: WHO.

Table 1.

Demographic variable	% in stage 1	% in stage 2
Child's age in years:		
6	8.80	6.50
7	5.30	6.50
8	14.00	9.70
9	14.00	19.40
10	15.80	22.60
11	19.30	16.10
12	22.90	19.40
Child's gender:		
Male	66.70	74.20
Female	33.30	25.80
Child's diagnosis:		
Autism	32.80	33.30
Cerebral Palsy	3.40	6.70
Down Syndrome	8.60	13.30
Other unspecified	19.00	16.70
None	36.20	30.00
Known impairment:		
Visual impairment	15.80	8.90
Auditory impairment	5.30	4.20
No known impairment	71.90	77.50
Both	7.00	7.50
Epilepsy:		
Yes	10.50	8.40
No	89.50	91.60
Other known health problems:		
Yes	25.0	28.40
No	75.0	71.60
Mobility impairments:		
Yes	15.80	14.90
No	84.20	85.10

Table 2.

SDQ Subscales	n = 58 Normal	Borderline %	Abnormal	n = 31 Normal	Borderline %	Abnormal
Emotional Difficulties	55.20	10.30	34.50	54.80	6.50	38.70
Conduct Problems	22.40	27.60	50.00	9.70	32.30	58.10
Hyperactive Behaviour	25.90	10.30	63.80	22.60	9.70	67.70
Peer Relationships	25.90	5.20	69.00	19.40	3.20	77.40
Prosocial Behaviour	43.10	15.50	41.40	35.50	16.10	48.40

Table 3.

	High hyperactivity scores (n = 21) Mean (SD)	Low hyperactivity scores (n = 10). Mean (SD)	Z	p	d
Emotional difficulties	3.43 (2.44)	4.10 (3.35)	.59	.54	.23
Conduct problems	4.57 (1.50)	3.40 (2.22)	2.52	.01*	.62
Peer problems	5.76 (2.41)	3.90 (1.97)	.76	.45	.85
Prosocial behaviour	3.48 (2.62)	7.00 (1.70)	3.88	.00**	1.44

*** = p<0.05 ** = p<0.01**

Table 4.

	High hyperactivity scores (n = 21) Mean (SD)	Low hyperactivity scores (n = 10). Mean (SD)	F	d
PACS (last week)	16.55 (4.90)	4.60 (4.01)	44.33**	2.66
PACS (typical)	17.05 (4.78)	5.00 (3.97)	46.98**	2.75
VABS	29.53 (6.34)	47.10 (9.61)	37.09**	2.20
CDT	6.94 (5.85)	2.38 (2.55)	4.96*	1.08

*** = p<0.05 ** = p<0.01**

List of table titles

Notes for Contributors

Journal of Intellectual and Developmental Disability

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SECTION 5

CONTRIBUTIONS TO THEORY, CLINICAL PRACTICE AND LEARNING

The Presentation of Attention Deficit/Hyperactivity Disorder in Children with Intellectual Disability.

Contributions to Theory, Clinical Practice and Learning.

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Implications for research and theory development.

The first main finding from the current study was that hyperactivity, as rated by parents, was one of the most problematic behaviours exhibited by their child with intellectual disabilities (ID). Although power in the current study was limited, this finding appears to be consistent with previous studies that report high prevalence rates of AD/HD in children with ID, compared to other disorders and populations (Dekker & Koot, 2003; Linna et al., 1999; Rutter et al., 1970; Strømme & Diseth, 2000). Given these findings and the continued lack of studies that appear to investigate this issue at depth, further research on the prevalence of AD/HD in children with ID would be imperitive. One possible way of achieving this would be to conduct a large, multi-centred, prevalence study of AD/HD in children with ID.

In addition, this study illustrated that by using additional assessment measures in order to confirm parental reports of hyperactivity, such as semi structured interviews, and an objective measure of AD/HD symptomatology, the majority of parental reports of hyperactive behaviour were confirmed.

This finding is important for a number of reasons; firstly it implies that in many cases, parental reports of hyperactivity may be indicative of an underlying liability to an externalising disorder such as AD/HD, and not due to a rating bias or behaviours arising from the presence of ID per se. Secondly, in terms of further research it would appear that there is added value in using a variety of measures in order to be able to generate more accurate rates of prevalence. However, although it cannot be denied that the use of clinical interviews alongside parental questionnaires are likely to generate more accurate results (Reiss, 1990), more research is needed into measures that successfully and appropriately measure AD/HD in children with ID (Hastings et al., 2005). Another main finding from the present study was that

children, who were rated as most hyperactive by parents in the study, also appeared to display the lowest levels of adaptive behaviour. Although, there is no research that has specifically investigated the relationship between severity of ID and vulnerability to AD/HD (Hasting et al., 2005; Dekker & Koot, 2003; Pearson & Aman, 1994), this finding could lead us to presume that AD/HD symptoms are correlated with severity of intellectual disability (more pronounced with more developmental delay), and would benefit from further investigation. In addition, compared to children rated as exhibiting low hyperactivity in the study, children reported as being hyperactive were also reported to display significantly more conduct problems, and significantly less prosociality (resulting in increased peer relationship difficulties). In order to investigate whether these factors were influencing parental reports of hyperactivity, these factors were controlled for in subsequent analyses. Results showed that increased AD/HD symptoms reported by parents during a semi-structured interview, and displayed by children during the objective measure of AD/HD symptomatology remained evident. However, even though low levels of adaptive behaviour, increased conduct problems and low prosociality were not found to significantly influence parental reports of hyperactivity, their presence should not be ignored and further research into the concomitant factors associated with AD/HD symptomatology in children with ID would be extremely beneficial.

Finally, the findings of the current study have implications for further research and theory development in terms of intervention methods used with children experiencing AD/HD and ID. In terms of treatment approaches for AD/HD in children with ID, the use of stimulant medication is currently the most dominant form of management strategy for children with ID and AD/HD. Recent studies indicate that stimulants appear to be successful in the treatment of AD/HD in children with ID,

(Aman et al., 1991, 1993; Handen et al., 1990). For example, Pearson et al., (2003, 2004) found that in general children with mild ID responded as well to stimulant medication as their non-ID counterparts. As well as stimulants, atypical antipsychotics, such as resperidone, have also been found to significantly decrease problematic behaviours including hyperactivity in children with ID (Aman et al., 2002; Synder et al., 2002) with improvements continuing to be reported for at least 48 weeks (Findling et al., 2004; Turgay et al., 2002).

Although pharmacological treatments are more commonly used, very few studies have investigated long-term outcomes. However, one study that did investigate the longer-term outcome of using medication was undertaken by Aman et al. (2002); they undertook a study examining 20 children with AD/HD were they were followed up 4.5 years post-intervention. They found that approximately half of the sample continued to exhibit AD/HD symptoms and appeared to have developed a variety of co-morbid conditions not commonly reported in typically-developing children with AD/HD, such as tics and anxiety disorders.

Due to this finding, Aman et al. (2002) suggested that there are likely to be qualitative differences that distinguish children with AD/HD and ID from their typically developing counterparts, suggesting that AD/HD may have a different 'signature' for children with ID. Due to the fact that few studies have examined the specific presentation of AD/HD in children with ID, Aman et al. (2002) concluded that more research is needed regarding the specificity of AD/HD symptomatology in children with ID. A greater understanding of this issue may be gained by conducting future research that attempts to replicate the findings of Solanto et al. (2001), they found that there were independent associations between AD/HD and executive dysfunction, and AD/HD and inhibitory deficits.

In terms of psychosocial interventions, although there is no data on the efficacy of parent behaviour management with children with ID, there is no reason to assume that similar intervention methods would not as effective with this population, as they are in the non-ID population (Sonuga-Barke et al., 2001). If developmental delay is taken into account, parent behaviour management could be just as efficacious when delivered to children with ID at a later age. Psychosocial interventions, such as parent behaviour management, have additional advantages for use with children with ID; many children with ID and AD/HD are likely to be on medications for an array of associated medical conditions and are therefore at increased risk of experiencing an adverse response to stimulants (Handen, Feldman, Gosling, Breaux, & McAuliffe, 1991). Given the greater heterogeneity in stimulant response in children with ID compared to their non-ID counterparts (Aman, 1996; Aman, Buican, & Arnold, 2003), further research on alternative, non-pharmacological interventions is greatly needed. An interesting idea for future research that would be to replicate Sonuga-Barke et al., (2001) parent training intervention with children with AD/HD and ID at a later age.

To conclude, it would appear that in order to make advances in our understanding of AD/HD in children with ID, a better understanding of whether AD/HD presents itself in a similar way in children with and without ID is needed. Until the specificity of AD/HD in children with ID is better understood, advancements with regards measurement and intervention issues cannot be made successfully.

Implications for clinical practice

Given what researchers and clinicians alike know regarding the impact of undiagnosed AD/HD in children with ID, the need for early assessment and diagnosis of the disorder cannot be emphasised enough. The findings of the current study appear to have a number of clinical implications in terms of how assessment methods and interventions for AD/HD in children with ID could be improved. Firstly, this study found that the majority of parental reports of hyperactivity on the SDQ remained consistent with additional measures of AD/HD symptomatology. This finding implies that parents' early concerns are likely to be indicative of underlying difficulties such as AD/HD, and need to be given greater value. Often parents raise concerns about their child's behaviour at a time when intervention would be extremely beneficial, but are pacified until, in some cases, behaviours have become too difficult to manage. A survey undertaken by Mulhern, Dworkin, & Bernstein (1994) found that 32% of parents waiting for their child's first appointment with child services had been previously concerned about their child's 'hyperactivity'. Although Mulhern et al., (1994) found that parental concerns were not always positively correlated with the specific childhood difficulty later identified by professionals, they did in the majority of cases appear to indicate the presence of an important school-related problem that would benefit from early intervention. In a further study of 100 families seeking professional help and advice from child services, 80% of children who were subsequently diagnosed with a behavioural/developmental difficulty had parents who had previously raised concerns regarding their child's behavioural difficulties (Glascoe, Altemeier, & MacLean, 1989).

In terms of reliable assessment measures, as with the current study, Goodman et al., (2003) found the SDQ to be a reliable early screening tool for a number of

childhood difficulties. Goodman et al., (2003) found that by using a predictive algorithm based on multi-informant SDQ's, they were able to detect children with psychiatric and behavioural disorders with reasonable efficacy. It was also found that the predictive value of the SDQ was greater when both the child's parents and their teacher completed the questionnaire.

However, due to the continued lack of understanding regarding the specificity of AD/HD in contrast to other childhood disorders, especially in children with ID, accurate assessment and subsequent diagnosis of AD/HD continues to be fraught with difficulties. It may be possible that further studies will also find that hyperactivity, as reported by parents, is strongly indicative of underlying AD/HD (Glascoe, Altemeier, & MacLean, 1989). If this is found to be the case, parental reports of hyperactivity, on screening measures such as the SDQ, could lead to earlier assessment, diagnosis and treatment of AD/HD for these children.

This would not only provide a greater understanding of the AD/HD in children with ID, but would enable professionals working within the field to receive training on the unique behavioural precursors to the full development of AD/HD in this population. This would not only identify the presence of the underlying disorder before the associated difficulties such as school failure, peer rejection and the deterioration of adult-child relationships *have become entrenched (Reid, 1993), but* would allow for a wider choice of early intervention options.

As previously discussed, recent research indicates that early psycho-social intervention for AD/HD in typically developing children yields the best results (Sonuga-Barke et al., 2001). Recent outcome data on parent behaviour management interventions for pre-school, non-ID, children with AD/HD has shown that it was

clinically efficacious (Bor, Sanders, & Makie-Dadds, 2002; Sonuga-Barke et al., 2001).

Given the increased risk of experiencing adverse reactions to stimulants (Handen et al., 1991), the greater heterogeneity of stimulant response (Aman, 1996; Aman, Buican, & Arnold, 2003), and the fact that AD/HD is likely to be due to the interaction between biological and environmental 'risk' factors (Larrson, Larrson, & Litchenstein, 2004), by offering psychosocial interventions, such as parent management training, services are likely to have a greater impact upon a greater range of impacting influences than medical regimes. A good therapeutic alliance is known to be one of the most important factors associated with positive outcome (Nixon, 2002), and by working with a family at a systemic level, chances of tackling systemic difficulties such as parental mental health problems are increased. It is likely that parents who have had experienced help and support with coping with their child's challenging behaviour will be more likely to seek help for themselves. Pharmacological interventions on the other hand only seek to reduce AD/HD symptomatology elicited by the child and do little to address systemic factors known to be associated with the development and maintenance of AD/HD (Hinshaw, 1994; Taylor, 1994; Anastropoulos et al., 1996).

Process/personal issues

On the whole undertaking this piece of research was an enjoyable process. However, that's not to say that there weren't any hurdles that had to be overcome along the way. The initial study intended to compare the presentation of 4 clinical groups; children with ID and AD/HD vs. children with ID, but without AD/HD vs. typically developing children with AD/HD vs. typically developing children without AD/HD. In addition, it was originally planned to do two additional

neuropsychological tests with the children involved, namely the Stop-Signal Task and the Choice-Delay Task. The incorporation of these groups and tasks would have enabled a deeper investigation into the differences between the development of AD/HD in children with and without ID and given a greater insight into the specificity of its development in children with intellectual disability. It was frustrating that due to time constraints I was not able to include all that was originally proposed.

In addition, as a result of taking maternity leave at the beginning of my third year I was unable to commence my research until April 2005. This put me under further time pressure, as I had to recruit all my participants and complete the first stage of the research before the summer holidays. Due to only applying for, and obtaining, School of Psychology ethics approval, I was not able to access any children with ID through NHS sources. All schools for children with special educational needs were contacted throughout North Wales, of which all but one agreed to take part. However, this was an extremely time consuming process; I had to arrange a meeting with each Head teacher in order to discuss my research and this meant travelling around a large geographical area. I had to make a number of further visits to the participating schools, both before the summer holidays and afterwards, in order to deliver and collect questionnaire packs (stage 1), and complete the cookie delay task with participating children (stage 2).

In terms of response rate, if I had had more time I probably would have been able to gain more responses by attending school functions such as open evenings and directly approaching parents in order to ask them to participate. The SDQ only takes 5 minutes to complete and by having personal contact with the parents I would have increased the likelihood that they would have responded positively. All parents that

did take part were extremely supportive and interested in the research I was conducting. In addition, I was unable to get hold of many parents that showed an interest in participating in the second stage by telephone. I would often make future appointments with parents at a time that was convenient for them, but upon calling there would often be no reply. Also many parents were working, so were only contactable during the evening. This greatly reduced the number of parents that I could contact. Due to each phone-call lasting between 45minutes – 1 hour, I was only able to manage 1 phone-call per evening, and either them or I were either too busy with family life or too tired to participate. Contacting parents past 9 pm was felt too much of an invasion. In addition, all telephone interviews had to be completed prior to the cookie delay task being performed on participating children. Due to the difficulties contacting parents, I was left little time to visit each school and perform the CDT. In addition, many of the children I needed to test were either taking part in rehearsals for Christmas plays at this time or were out on *their regular school trips* (swimming etc.). This meant that I had to make a number of visits to the same school before I managed to test all the children still involved, and due to the large geographical area covered, this was extremely time consuming. It has to be said though that all the schools involved were extremely patient, supportive and interested in the research that was being undertaken. They always managed to find me a private room, and in most cases provided me with a support worker that would fetch each child involved from their respective classroom, this saved me considerable time hunting for children in schools that I was not familiar with.

On a more personal level, due to maternity leave, I found it difficult not having the direct support of my peer group. From the beginning of my third year, I was six months behind my cohort, so did not have their presence and direct support I

in terms of undertaking the research or in terms of teaching. I did not have anyone else on which to gauge my time management of data collection, and was not able to engage in conversations relating to either the theoretical and/or practical issues relating to our respective studies. I missed out on conversations generated by much of the teaching in my third year, and feel that although the support I have received from the course team has been fantastic, may not have received the peer support that the rest of my cohort would have done, going through the experience together. However, the fact that I have been out of synch with my cohort during at the completion of my LSRP may also have had its benefits. I was not affected by the accumulative stress that was likely to have been experienced by my cohort all doing their studies at the same time, and I may have actually received more support from them individually, than they did from each-other as a result. I have, on the whole, not only thoroughly enjoyed conducting this piece of research, but have learnt a great deal in the process. My only wish is that I could have had more time in which to have included all that was discussed when the idea for this research was first discussed. I can only hope that this study has contributed in a small way to the understanding of the presentation of AD/HD in children with ID.

References

Aman, M. (1996). Stimulant drugs in the development disabilities revisited. *Journal of Developmental and Physical Disabilities, 8,* 347-365.

Aman, M., & Singh, N. (1991). Pharmacological interventions. In J. L. Matson & J. A. Mulick (Eds.), *Handbook of Mental Retardation* (2nd ed., pp. 347-372). New York: Pergamon Press.

Aman, M., Kern, R., McGhee, D., & Arnold, L. (1993). Fenfluramine and methylphenidate in children with mental retardation and attention deficit/hyperactivity disorder: Laboratory effects. *Journal of Autism and Developmental Disorders, 23,* 491-506.

Aman, M., Armstrong, S., Buican, B., & Sillick, T. (2002). Four-year follow-up of children with low intelligence and AD/HD: A replication. *Research in Developmental Disabilities, 23,* 119-134.

Aman, M., Buican, B., & Arnold, L. (2003). Methylphenidate treatment in children with borderline IQ and mental retardation: analysis of three aggregated studies. *Journal of Child and Adolescent Psychopharacology, 13,* 29-40.

Anastropoulos, A., Barkley, R., & Shelton, T. (1996). Family based treatment: Psychosocial intervention for children and adolescents with attention deficit hyperactivity disorder. In E. Hibbs and P. Jensen (eds.), *Psychosocial*

treatments for Child and Adolescent Disorders: Empirically Based Strategies for Clinical Practice (pp. 267-284). Washington, DC: American Psychiatric Association.

Bor, W., Sanders, M. & Markie-Dadds, C. (2002). The effect of Triple P-Positive Parenting Programme on preschool children with co-occurring disruptive behavior and attentional/hyperactive difficulties. *Journal of Abnormal Child Psychology, 30,* 571-578.

Dekker, M., & Koot, H. (2003). DSM-IV disorders in children with borderline to moderate intellectual disability. I: Prevalence and impact. *Journal of the American Academy of Child and Adolescent Psychiatry, 42(8),* 915-922.

Findling, R., Aman, M., De Smedt, G., Derivan, A., Lyons, B. & the Risperidone Disruptive Behaviour Study Group. (2004). A long-term open-label study of risperidone in children with severe disruptive behaviors and subaverage IQ's. *American Journal of Psychiatry, 161,* 677-684.

Glascoe, F., Altemeier, W., & MacLean, W. (1989). The importance of parents' concerns about their child's development. *American Journal of Disabilities and Childhood, 143,* 955-958.

Goodman, R., Ford, T., Simmons, H., Gatward, R., & Meltzer, H. (2003). Using the strengths and difficulties questionnaire (SDQ) to screen for child

psychiatric disorders in a community sample. *International Review of Psychiatry, 15,* 166-172.

Handen, B., Feldman, H., Gosling, A., Breaux, A., & McAuliffe, S. (1991). Adverse side-effects of methylphenidate among mentally retarded children with AD/HD. *Journal of the American Academy of Child and Adolescent Psychiatry,30,* 241-245.

Hastings, R., Beck, A., Daley, D., & Hill, C. (2005). Symptoms of ADHD and their correlates in Children with Intellectual Disabilities. *Research in Developmental Disabilities, 26,* 456-468.

Hinshaw, S. (1994). *Attention deficits and hyperactivity in children.* Thousand Oaks, CA: Sage

Larsson, J., Larsson, H., & Lichtenstein, P. (2004). Genetic and environmental contributions to stability and change of ADHD symptoms between 8 and 13 years of age: a longitudinal twin study. *Journal of the American Academy of Child and Adolescent Psychiatry, 43,* 1267-1275.

Linna, S., Moilanen, I., Ebeling, H., Piha, J., Kumpulainen, K., Tamminen, T., 7 Almqvist, F. (1999). Psychiatric symptoms in children with intellectual disability, *European Child and Adolescent Psychiatry, 8,* 77-82.

Mulhern, S., Dworkin, P., & Bernstein, B. (1994). Do parental concerns predict a diagnosis of attention-deficit hyperactivity disorder? *Developmental and Behavioural Pediatrics, 15(5),* 348-352.

Nixon, R. (2002). Treatment of behaviour problems in pre-schoolers: A review of parent training programs. *Child Psychology Review, 22,* 525-546.

Pearson, D., & Aman, M. (1994). Ratings of hyperactivity and developmental indices: Should clinicians correct for developmental delay? *Journal of Autism and Developmental Disorders, 24,* 395-411.

Reiss, S. Prevalence of dual diagnosis in community-based day programs in the Chicago metropolitan area. (1990). *American Journal of Mental Retardation, 94,* 578-585.

Rutter, M., Tizzard, J., & Whitmore, K. (1970). *Education, health, and behavior.* London: Longwood.

Snyder, R., Turgay, A., Aman, M., Binder, C., Fisman, S., Carroll, A., & The Risperidone Conduct Study Group. (2002). Effects of risperidone on conduct and disruptive behavior disorders in children with sub-average IQ's. *Journal of the American Academy of Child and Adolescent Psychiatry, 41,* 1026-1036.

Solanto, M., Abikoff, H., Sonuga-Barke, E., Schachar, R., Logan, G., Wigal, T., Hechtman, L., Hinshaw, S., & Turkel, E. (2001). The Ecological Validity of Dealy Aversion and Response Inhibition as Measures of Impulsivity in

ADHD: A Supplement to the NIMH Multimodal Treatment Study of ADHD. *Journal of Abnormal Child Psychology,* 29(3), 215-228.

Sonuga-Barke, E., Daley, D., Thompson, M., Weeks, A., & Laver-Bradbury, C. (2001). Parent based therapies for preschool attention-deficit/hyoperactivity disorder: a randomised control trial with a community sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 40(4)*, 402-408.

Strømme, P., & Diseth, T. (2000). Prevalence of psychiatric diagnoses in children with mental retardation: Data from a population-based study. *Developmental Medicine and Child Neurology, 42,* 266-270.

Taylor, R. (1994). Syndromes of attention deficit and overactivity. In M. Rutter, E. Taylor, and L. Hersov (eds.), *Child and Adolescent Psychiatry: Modern Approaches* (3rd ed., pp. 258-307. Oxford: Blackwell.

Turgay, A., Binder, C., Snyder, R., & Fisman, S. (2002). Long-term safety and efficacy of risperidone for the treatment of disruptive behavior disorders in children with sub-average IQ's. *Pediatrics, 110,* 16-34.

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Contributions to Theory, Clinical Practice and Learning.

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Implications for research and theory development.

The first main finding from the current study was that hyperactivity, as rated by parents, was one of the most problematic behaviours exhibited by their child with intellectual disabilities (ID). Although power in the current study was limited, this finding appears to be consistent with previous studies that report high prevalence rates of AD/HD in children with ID, compared to other disorders and populations (Dekker & Koot, 2003; Linna et al., 1999; Rutter et al., 1970; Strømme & Diseth, 2000). Given these findings and the continued lack of studies that appear to investigate this issue at depth, further research on the prevalence of AD/HD in children with ID would be imperitive. One possible way of achieving this would be to conduct a large, multi-centred, prevalence study of AD/HD in children with ID.

In addition, this study illustrated that by using additional assessment measures in order to confirm parental reports of hyperactivity, such as semi structured interviews, and an objective measure of AD/HD symptomatology, the majority of parental reports of hyperactive behaviour were confirmed.

This finding is important for a number of reasons; firstly it implies that in many cases, parental reports of hyperactivity may be indicative of an underlying liability to an externalising disorder such as AD/HD, and not due to a rating bias or behaviours arising from the presence of ID per se. Secondly, in terms of further research it would appear that there is added value in using a variety of measures in order to be able to generate more accurate rates of prevalence. However, although it cannot be denied that the use of clinical interviews alongside parental questionnaires are likely to generate more accurate results (Reiss, 1990), more research is needed into measures that successfully and appropriately measure AD/HD in children with ID (Hastings et al., 2005). Another main finding from the present study was that

children, who were rated as most hyperactive by parents in the study, also appeared to display the lowest levels of adaptive behaviour. Although, there is no research that has specifically investigated the relationship between severity of ID and vulnerability to AD/HD (Hasting et al., 2005; Dekker & Koot, 2003; Pearson & Aman, 1994), this finding could lead us to presume that AD/HD symptoms are correlated with severity of intellectual disability (more pronounced with more developmental delay), and would benefit from further investigation. In addition, compared to children rated as exhibiting low hyperactivity in the study, children reported as being hyperactive were also reported to display significantly more conduct problems, and significantly less prosociality (resulting in increased peer relationship difficulties). In order to investigate whether these factors were influencing parental reports of hyperactivity, these factors were controlled for in subsequent analyses. Results showed that increased AD/HD symptoms reported by parents during a semi-structured interview, and displayed by children during the objective measure of AD/HD symptomatology remained evident. However, even though low levels of adaptive behaviour, increased conduct problems and low prosociality were not found to significantly influence parental reports of hyperactivity, their presence should not be ignored and further research into the concomitant factors associated with AD/HD symptomatology in children with ID would be extremely beneficial.

Finally, the findings of the current study have implications for further research and theory development in terms of intervention methods used with children experiencing AD/HD and ID. In terms of treatment approaches for AD/HD in children with ID, the use of stimulant medication is currently the most dominant form of management strategy for children with ID and AD/HD. Recent studies indicate that stimulants appear to be successful in the treatment of AD/HD in children with ID,

(Aman et al., 1991, 1993; Handen et al., 1990). For example, Pearson et al., (2003, 2004) found that in general children with mild ID responded as well to stimulant medication as their non-ID counterparts. As well as stimulants, atypical antipsychotics, such as resperidone, have also been found to significantly decrease problematic behaviours including hyperactivity in children with ID (Aman et al., 2002; Synder et al., 2002) with improvements continuing to be reported for at least 48 weeks (Findling et al., 2004; Turgay et al., 2002).

Although pharmacological treatments are more commonly used, very few studies have investigated long-term outcomes. However, one study that did investigate the longer-term outcome of using medication was undertaken by Aman et al. (2002); they undertook a study examining 20 children with AD/HD were they were followed up 4.5 years post-intervention. They found that approximately half of the sample continued to exhibit AD/HD symptoms and appeared to have developed a variety of co-morbid conditions not commonly reported in typically-developing children with AD/HD, such as tics and anxiety disorders.

Due to this finding, Aman et al. (2002) suggested that there are likely to be qualitative differences that distinguish children with AD/HD and ID from their typically developing counterparts, suggesting that AD/HD may have a different 'signature' for children with ID. Due to the fact that few studies have examined the specific presentation of AD/HD in children with ID, Aman et al. (2002) concluded that more research is needed regarding the specificity of AD/HD symptomatology in children with ID. A greater understanding of this issue may be gained by conducting future research that attempts to replicate the findings of Solanto et al. (2001), they found that there were independent associations between AD/HD and executive dysfunction, and AD/HD and inhibitory deficits.

In terms of psychosocial interventions, although there is no data on the efficacy of parent behaviour management with children with ID, there is no reason to assume that similar intervention methods would not as effective with this population, as they are in the non-ID population (Sonuga-Barke et al., 2001). If developmental delay is taken into account, parent behaviour management could be just as efficacious when delivered to children with ID at a later age. Psychosocial interventions, such as parent behaviour management, have additional advantages for use with children with ID; many children with ID and AD/HD are likely to be on medications for an array of associated medical conditions and are therefore at increased risk of experiencing an adverse response to stimulants (Handen, Feldman, Gosling, Breaux, & McAuliffe, 1991). Given the greater heterogeneity in stimulant response in children with ID compared to their non-ID counterparts (Aman, 1996; Aman, Buican, & Arnold, 2003), further research on alternative, non-pharmacological interventions is greatly needed. An interesting idea for future research that would be to replicate Sonuga-Barke et al., (2001) parent training intervention with children with AD/HD and ID at a later age.

To conclude, it would appear that in order to make advances in our understanding of AD/HD in children with ID, a better understanding of whether AD/HD presents itself in a similar way in children with and without ID is needed. Until the specificity of AD/HD in children with ID is better understood, advancements with regards measurement and intervention issues cannot be made successfully.

Implications for clinical practice

Given what researchers and clinicians alike know regarding the impact of undiagnosed AD/HD in children with ID, the need for early assessment and diagnosis of the disorder cannot be emphasised enough. The findings of the current study appear to have a number of clinical implications in terms of how assessment methods and interventions for AD/HD in children with ID could be improved. Firstly, this study found that the majority of parental reports of hyperactivity on the SDQ remained consistent with additional measures of AD/HD symptomatology. This finding implies that parents' early concerns are likely to be indicative of underlying difficulties such as AD/HD, and need to be given greater value. Often parents raise concerns about their child's behaviour at a time when intervention would be extremely beneficial, but are pacified until, in some cases, behaviours have become too difficult to manage. A survey undertaken by Mulhern, Dworkin, & Bernstein (1994) found that 32% of parents waiting for their child's first appointment with child services had been previously concerned about their child's 'hyperactivity'. Although Mulhern et al., (1994) found that parental concerns were not always positively correlated with the specific childhood difficulty later identified by professionals, they did in the majority of cases appear to indicate the presence of an important school-related problem that would benefit from early intervention. In a further study of 100 families seeking professional help and advice from child services, 80% of children who were subsequently diagnosed with a behavioural/developmental difficulty had parents who had previously raised concerns regarding their child's behavioural difficulties (Glascoe, Altemeier, & MacLean, 1989).

In terms of reliable assessment measures, as with the current study, Goodman et al., (2003) found the SDQ to be a reliable early screening tool for a number of

childhood difficulties. Goodman et al., (2003) found that by using a predictive algorithm based on multi-informant SDQ's, they were able to detect children with psychiatric and behavioural disorders with reasonable efficacy. It was also found that the predictive value of the SDQ was greater when both the child's parents and their teacher completed the questionnaire.

However, due to the continued lack of understanding regarding the specificity of AD/HD in contrast to other childhood disorders, especially in children with ID, accurate assessment and subsequent diagnosis of AD/HD continues to be fraught with difficulties. It may be possible that further studies will also find that hyperactivity, as reported by parents, is strongly indicative of underlying AD/HD (Glascoe, Altemeier, & MacLean, 1989). If this is found to be the case, parental reports of hyperactivity, on screening measures such as the SDQ, could lead to earlier assessment, diagnosis and treatment of AD/HD for these children.

This would not only provide a greater understanding of the AD/HD in children with ID, but would enable professionals working within the field to receive training on the unique behavioural precursors to the full development of AD/HD in this population. This would not only identify the presence of the underlying disorder before the associated difficulties such as school failure, peer rejection and the deterioration of adult-child relationships have become entrenched (Reid, 1993), but would allow for a wider choice of early intervention options.

As previously discussed, recent research indicates that early psycho-social intervention for AD/HD in typically developing children yields the best results (Sonuga-Barke et al., 2001). Recent outcome data on parent behaviour management interventions for pre-school, non-ID, children with AD/HD has shown that it was

clinically efficacious (Bor, Sanders, & Makie-Dadds, 2002; Sonuga-Barke et al., 2001).

Given the increased risk of experiencing adverse reactions to stimulants (Handen et al., 1991), the greater heterogeneity of stimulant response (Aman, 1996; Aman, Buican, & Arnold, 2003), and the fact that AD/HD is likely to be due to the interaction between biological and environmental 'risk' factors (Larrson, Larrson, & Litchenstein, 2004), by offering psychosocial interventions, such as parent management training, services are likely to have a greater impact upon a greater range of impacting influences than medical regimes. A good therapeutic alliance is known to be one of the most important factors associated with positive outcome (Nixon, 2002), and by working with a family at a systemic level, chances of tackling systemic difficulties such as parental mental health problems are increased. It is likely that parents who have had experienced help and support with coping with their child's challenging behaviour will be more likely to seek help for themselves. Pharmacological interventions on the other hand only seek to reduce AD/HD symptomatology elicited by the child and do little to address systemic factors known to be associated with the development and maintenance of AD/HD (Hinshaw, 1994; Taylor, 1994; Anastropoulos et al., 1996).

Process/personal issues

On the whole undertaking this piece of research was an enjoyable process. However, that's not to say that there weren't any hurdles that had to be overcome along the way. The initial study intended to compare the presentation of 4 clinical groups; children with ID and AD/HD vs. children with ID, but without AD/HD vs. typically developing children with AD/HD vs. typically developing children without AD/HD. In addition, it was originally planned to do two additional

neuropsychological tests with the children involved, namely the Stop-Signal Task and the Choice-Delay Task. The incorporation of these groups and tasks would have enabled a deeper investigation into the differences between the development of AD/HD in children with and without ID and given a greater insight into the specificity of its development in children with intellectual disability. It was frustrating that due to time constraints I was not able to include all that was originally proposed.

In addition, as a result of taking maternity leave at the beginning of my third year I was unable to commence my research until April 2005. This put me under further time pressure, as I had to recruit all my participants and complete the first stage of the research before the summer holidays. Due to only applying for, and obtaining, School of Psychology ethics approval, I was not able to access any children with ID through NHS sources. All schools for children with special educational needs were contacted throughout North Wales, of which all but one agreed to take part. However, this was an extremely time consuming process; I had to arrange a meeting with each Head teacher in order to discuss my research and this meant travelling around a large geographical area. I had to make a number of further visits to the participating schools, both before the summer holidays and afterwards, in order to deliver and collect questionnaire packs (stage 1), and complete the cookie delay task with participating children (stage 2).

In terms of response rate, if I had had more time I probably would have been able to gain more responses by attending school functions such as open evenings and directly approaching parents in order to ask them to participate. The SDQ only takes 5 minutes to complete and by having personal contact with the parents I would have increased the likelihood that they would have responded positively. All parents that

did take part were extremely supportive and interested in the research I was conducting. In addition, I was unable to get hold of many parents that showed an interest in participating in the second stage by telephone. I would often make future appointments with parents at a time that was convenient for them, but upon calling there would often be no reply. Also many parents were working, so were only contactable during the evening. This greatly reduced the number of parents that I could contact. Due to each phone-call lasting between 45minutes – 1 hour, I was only able to manage 1 phone-call per evening, and either them or I were either too busy with family life or too tired to participate. Contacting parents past 9 pm was felt too much of an invasion. In addition, all telephone interviews had to be completed prior to the cookie delay task being performed on participating children. Due to the difficulties contacting parents, I was left little time to visit each school and perform the CDT. In addition, many of the children I needed to test were either taking part in rehearsals for Christmas plays at this time or were out on their regular school trips (swimming etc.). This meant that I had to make a number of visits to the same school before I managed to test all the children still involved, and due to the large geographical area covered, this was extremely time consuming. It has to be said though that all the schools involved were extremely patient, supportive and interested in the research that was being undertaken. They always managed to find me a private room, and in most cases provided me with a support worker that would fetch each child involved from their respective classroom, this saved me considerable time hunting for children in schools that I was not familiar with.

On a more personal level, due to maternity leave, I found it difficult not having the direct support of my peer group. From the beginning of my third year, I was six months behind my cohort, so did not have their presence and direct support I

in terms of undertaking the research or in terms of teaching. I did not have anyone else on which to gauge my time management of data collection, and was not able to engage in conversations relating to either the theoretical and/or practical issues relating to our respective studies. I missed out on conversations generated by much of the teaching in my third year, and feel that although the support I have received from the course team has been fantastic, may not have received the peer support that the rest of my cohort would have done, going through the experience together. However, the fact that I have been out of synch with my cohort during at the completion of my LSRP may also have had its benefits. I was not affected by the accumulative stress that was likely to have been experienced by my cohort all doing their studies at the same time, and I may have actually received more support from them individually, than they did from each-other as a result. I have, on the whole, not only thoroughly enjoyed conducting this piece of research, but have learnt a great deal in the process. My only wish is that I could have had more time in which to have included all that was discussed when the idea for this research was first discussed. I can only hope that this study has contributed in a small way to the understanding of the presentation of AD/HD in children with ID.

References

Aman, M. (1996). Stimulant drugs in the development disabilities revisited. *Journal of Developmental and Physical Disabilities, 8,* 347-365.

Aman, M., & Singh, N. (1991). Pharmacological interventions. In J. L. Matson & J. A. Mulick (Eds.), *Handbook of Mental Retardation* (2nd ed., pp. 347-372). New York: Pergamon Press.

Aman, M., Kern, R., McGhee, D., & Arnold, L. (1993). Fenfluramine and methylphenidate in children with mental retardation and attention deficit/hyperactivity disorder: Laboratory effects. *Journal of Autism and Developmental Disorders, 23,* 491-506.

Aman, M., Armstrong, S., Buican, B., & Sillick, T. (2002). Four-year follow-up of children with low intelligence and AD/HD: A replication. *Research in Developmental Disabilities, 23,* 119-134.

Aman, M., Buican, B., & Arnold, L. (2003). Methylphenidate treatment in children with borderline IQ and mental retardation: analysis of three aggregated studies. *Journal of Child and Adolescent Psychopharacology, 13,* 29-40.

Anastropoulos, A., Barkley, R., & Shelton, T. (1996). Family based treatment: Psychosocial intervention for children and adolescents with attention deficit hyperactivity disorder. In E. Hibbs and P. Jensen (eds.), *Psychosocial*

treatments for Child and Adolescent Disorders: Empirically Based Strategies for Clinical Practice (pp. 267-284). Washington, DC: American Psychiatric Association.

Bor, W., Sanders, M. & Markie-Dadds, C. (2002). The effect of Triple P-Positive Parenting Programme on preschool children with co-occurring disruptive behavior and attentional/hyperactive difficulties. *Journal of Abnormal Child Psychology, 30,* 571-578.

Dekker, M., & Koot, H. (2003). DSM-IV disorders in children with borderline to moderate intellectual disability. I: Prevalence and impact. *Journal of the American Academy of Child and Adolescent Psychiatry, 42(8),* 915-922.

Findling, R., Aman, M., De Smedt, G., Derivan, A., Lyons, B. & the Risperidone Disruptive Behaviour Study Group. (2004). A long-term open-label study of risperidone in children with severe disruptive behaviors and subaverage IQ's. *American Journal of Psychiatry, 161,* 677-684.

Glascoe, F., Altemeier, W., & MacLean, W. (1989). The importance of parents' concerns about their child's development. *American Journal of Disabilities and Childhood, 143,* 955-958.

Goodman, R., Ford, T., Simmons, H., Gatward, R., & Meltzer, H. (2003). Using the strengths and difficulties questionnaire (SDQ) to screen for child

psychiatric disorders in a community sample. *International Review of Psychiatry, 15,* 166-172.

Handen, B., Feldman, H., Gosling, A., Breaux, A., & McAuliffe, S. (1991). Adverse side-effects of methylphenidate among mentally retarded children with AD/HD. *Journal of the American Academy of Child and Adolescent Psychiatry,30,* 241-245.

Hastings, R., Beck, A., Daley, D., & Hill, C. (2005). Symptoms of ADHD and their correlates in Children with Intellectual Disabilities. *Research in Developmental Disabilities, 26,* 456-468.

Hinshaw, S. (1994). *Attention deficits and hyperactivity in children.* Thousand Oaks, CA: Sage

Larsson, J., Larsson, H., & Lichtenstein, P. (2004). Genetic and environmental contributions to stability and change of ADHD symptoms between 8 and 13 years of age: a longitudinal twin study. *Journal of the American Academy of Child and Adolescent Psychiatry, 43,* 1267-1275.

Linna, S., Moilanen, I., Ebeling, H., Piha, J., Kumpulainen, K., Tamminen, T., 7 Almqvist, F. (1999). Psychiatric symptoms in children with intellectual disability, *European Child and Adolescent Psychiatry, 8,* 77-82.

Mulhern, S., Dworkin, P., & Bernstein, B. (1994). Do parental concerns predict a diagnosis of attention-deficit hyperactivity disorder? *Developmental and Behavioural Pediatrics, 15(5),* 348-352.

Nixon, R. (2002). Treatment of behaviour problems in pre-schoolers: A review of parent training programs. *Child Psychology Review, 22,* 525-546.

Pearson, D., & Aman, M. (1994). Ratings of hyperactivity and developmental indices: Should clinicians correct for developmental delay? *Journal of Autism and Developmental Disorders, 24,* 395-411.

Reiss, S. Prevalence of dual diagnosis in community-based day programs in the Chicago metropolitan area. (1990). *American Journal of Mental Retardation, 94,* 578-585.

Rutter, M., Tizzard, J., & Whitmore, K. (1970). *Education, health, and behavior.* London: Longwood.

Snyder, R., Turgay, A., Aman, M., Binder, C., Fisman, S., Carroll, A., & The Risperidone Conduct Study Group. (2002). Effects of risperidone on conduct and disruptive behavior disorders in children with sub-average IQ's. *Journal of the American Academy of Child and Adolescent Psychiatry, 41,* 1026-1036.

Solanto, M., Abikoff, H., Sonuga-Barke, E., Schachar, R., Logan, G., Wigal, T., Hechtman, L., Hinshaw, S., & Turkel, E. (2001). The Ecological Validity of Dealy Aversion and Response Inhibition as Measures of Impulsivity in

ADHD: A Supplement to the NIMH Multimodal Treatment Study of ADHD. *Journal of Abnormal Child Psychology,* 29(3), 215-228.

Sonuga-Barke, E., Daley, D., Thompson, M., Weeks, A., & Laver-Bradbury, C. (2001). Parent based therapies for preschool attention-deficit/hyoperactivity disorder: a randomised control trial with a community sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 40(4)*, 402-408.

Strømme, P., & Diseth, T. (2000). Prevalence of psychiatric diagnoses in children with mental retardation: Data from a population-based study. *Developmental Medicine and Child Neurology, 42,* 266-270.

Taylor, R. (1994). Syndromes of attention deficit and overactivity. In M. Rutter, E. Taylor, and L. Hersov (eds.), *Child and Adolescent Psychiatry: Modern Approaches* (3rd ed., pp. 258-307. Oxford: Blackwell.

Turgay, A., Binder, C., Snyder, R., & Fisman, S. (2002). Long-term safety and efficacy of risperidone for the treatment of disruptive behavior disorders in children with sub-average IQ's. *Pediatrics, 110,* 16-34.

Statement of word count

Thesis Component		Word Count
Thesis abstract		263
Ethics proposal		4494
Literature review		6268
Research paper		5944
Critical review		2970
	Total	**19939**
Appendices and references		
Ethics proposal references		1073
Literature review references		4660
Research paper references and tables		3849
Critical review references		827
Section 2 – Appendices		6554
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