NEUROPSYCHOLOGICAL AND
COGNITIVE DEFICITS IN CHILDREN
WITH DISRUPTIVE BEHAVIOUR
DISORDERS

by

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DECLARATION

I, Bob Thomas Shikwambana, declare that the dissertation hereby submitted to the University of Limpopo as partial fulfilment for the degree of Master of Arts in Clinical Psychology, has not been previously been submitted by me for a degree at any other university, that it is my own work in design and execution, and that all the material contained therein has been duly acknowledged.

Signature   ………………………………………..

Date   ………………………………………...

ACKNOWLEDGMENTS

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ABSTRACT

Background

Disruptive Behaviour Disorders are often associated with high rates of school drop outs, academic skills deficits and low achievement, drug abuse, low self esteem, depression, delinquency and incarceration. The main aim of the study was to establish whether children with DBDs are cognitively and/or neurologically impaired. The study investigated (1) whether children with DBDs show deficiencies in cognitive and motor functions and (2) whether gender and subtype influence cognitive and motor functions.

Method:

The sample of 137 children with DBDs (ADHD, ODD and CD) and those without a diagnosis of DBDs was drawn from children aged between 8 and 15 years. They were assessed using instruments that were selected to be measures of Executive Functions, cognitive functioning, and motor functions. The scores obtained from the administration of these measures were compared for significant differences between the DBD subtypes and a non-DBD control group as a possible function of gender.

Results:

The findings indicate that children with symptoms of DBDs performed poorer than the control group on all tests with the exception of the Digits backward. EF and motor impairments are associated with ADHD-C and ADHD-PI, and not with ADHD-HI, ODD and CD. Although among the DBDs, neuropsychological and cognitive impairments have been found to be severe in children with ADHD-PI and ADHD-C, the ADHD-C subtype showed qualitatively larger differences with the normal control group on most measures. There were no differences found between the genders in the performance on all tests that were administered.

Conclusion

Children of the ADHD-C and ADHD-PI subtypes are significantly more impaired on measures of Executive, cognitive and motor functions than those with ADHD-HI, ODD and CD and those without externalising disorders. However, the ADHD-C subtype found to be more severely impaired when compared with the ADHD-PI subtype.

INTRODUCTION

1.1 General introduction

Children with Disruptive Behaviour Disorders, Attention Deficit/Hyperactivity Disorder (ADHD), Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) are at risk for a range of abnormalities in personality development including delinquency, antisocial behaviour, personality disorders, substance abuse, sexual promiscuity and subsequent risk at for HIV, psychiatric problems such as anxiety and depression, accident proneness and traffic offences, and academic underachievement (Barkley, 2004; Barkley, Fischer, Smallish, & Fletcher, 2004; Carroll, Riffenburgh, Roberts, & Myhre, 2002; Kahn, Kaplowitz, Goodman, & Emans, 2002; Lam, 2002; Molina, Bukstein, & Lynch, 2002; Roberts & Tanner, Jr., 2000; Tercyak, Lerman, & Audrain, 2002).

Children who exhibit certain behavioural problems such as high levels of hyperactivity are found to be significantly more likely to report major and minor injuries affecting the head region. High scores for Conduct Disorder and emotional symptoms are significant risk factors for injuries, while high scores for hyperactivity and Conduct Disorders were significantly related to major and minor head injuries (Lalloo, Sheiham, & Nazroo, 2003).

Antisocial behaviour, youth violence and student safety are increasingly becoming a primary concern in schools and the larger society (Sprague & Walker, 2000). Preschool behavioural problems have been found to be a strong indicator of risk for future disruptive disorders, and aggressive behaviours of children at eight years were good predictors of aggression during adolescence (Hendren & Mullen, 2003). Research studies that correlate

correlation adult sex offences and childhood behavioural disorders, ADHD, ODD, and CD, found a positive correlation of childhood ADHD and CD with rape and violent sexual assault against minors. The comorbidity of ADHD and CD showed a high significant correlation to violent sexual delinquency. Children with ADHD seem to have a higher risk for involvement in violent sexual delinquency in adulthood if they also show symptoms of CD (Castellanos, 1997). ODD has been linked to deficits in self regulatory abilities which are necessary to prevent reactive acting out of negative emotions (Baving, Rellum, Laucht, & Schmidt, 2005). Research based practices, tools, and approaches in interventions are needed to help alleviate the problem (Sprague & Walker, 2000). This therefore brings about the importance and necessity for early identification of youth exhibiting antisocial and violent behaviours and thereafter developing and implementing strategic plans for early identification and intervention.

A number of causal factors have been identified in the development of Disruptive Behaviour Disorders (DBDs) including neurological, genetic, environmental and psychosocial factors. Some researchers believe that watching TV for many hours in a day by children may stifle the creative processes of children, and this lack of creative expression may lead into hyperactivity and inattention as children are presented with ready made images. They argue that the development of the right brain needs to be encouraged in children before the age of 12, and therefore children should be allowed to explore and be creative. Parents are further advised to cut down on refined sugar from their children's diet such as cakes, biscuits and pastries because they cause dramatic highs and lows in blood sugar level. However, the National Institute of Mental Health (2002) argues against these beliefs, it says that ADHD is not caused by too much TV, food allergies, excess sugar, poor home life and poor schooling. Sadock and Sadock (2003) also believe that there is no scientific evidence to support the claims that these factors are among the causal factors of ADHD.

Most research studies point to heredity as an important factor that contributes to ADHD and antisocial behaviour. Antisocial behaviour in parents has been found to increase the risk of antisocial behaviour in children (Maughan, Taylor, Caspi, & Moffitt, 2004). Concordance is greater among monozygotic twins compared with dizygotic twins. Adoption studies also indicate that conduct disorder and criminality in offspring are more likely when the biological relative has shown this behaviour (Mandel, 1997; Meyer, 1999; Sagvolden & Sergeant, 1998).

However, it is unlikely that a sole causal factor will be identified. A variety of causal factors have been identified including neurological, genetic, environmental and psychosocial factors (Brown, 2000). Since about three decades ago, clinical neuroscience and neuropsychopharmacology, a revolution of psychiatric perspective, were launched to challenge the empirically unverified psychoanalytical theories; this enabled diagnosis that relies on specific observable criteria as the basis for making reliable diagnoses (Castellanos, 1997).

Progress has been made in documenting age and sex differences. Some symptoms can be more serious, more atypical to the child's sex, or more age atypical. Progress has also been made in terms of methods of assessment of DBDs although some critical issues such as combined information from different informants still remain to be addressed (Loeber, Burke, Lahey, Winters, & Zera, 2000).

1.2 Purpose of the study

The main goal of this study is to establish whether children with Disruptive Behaviour Disorders are cognitively and neurologically impaired. Measurement of cognitive and neurological impairment will be achieved through systematic and experimental testing.

1.3 Operational definitions

Attention Deficit Hyperactivity Disorder (ADHD) is a persistent and severe impairment of psychological development resulting from a high level of impulsive, overactive and inattentive behaviour. The symptoms are recognised as a disorder when these behaviours are severe, developmentally inappropriate, and impair functioning at home and at school. The onset is before age seven. (Swanson, Sergeant, Taylor, Sonuga-Barke, Jensen, & Cantwell, 1998b).

Oppositional Defiant Disorder (ODD) is a pattern of negativistic, defiant, disobedient and hostile behaviour toward authority figures as evident in temper tantrums, argumentativeness, refusing to comply with request and deliberately annoying others (Lahey, Miller, Gordon, & Riley, 1999).

Conduct Disorder (CD) is a persistent pattern of behaviour in which the rights of others or important age appropriate societal norms or rules are violated. The problem behaviours are organised into four areas of functioning; Aggression towards people or animals, destruction of property, deceitfulness or theft, and serious rule violations (American Psychiatric Association, 2000).

Executive functioning (EF) is the combination of abilities that permit the individual to function in a constantly changing environment (Rock, Fessler, & Church, 1997). It has two primary components: an awareness of which skills, strategies and resources are needed to perform a task effectively, and the ability to use self-regulatory mechanisms to ensure successful completion of a task. Specific skills include maintaining an appropriate problem solving set of procedures to attain a future goal. Inhibiting or deferring a response, formulating a sequential strategic plan of action, and encoding relevant information in memory for future use. According to Pennington & Ozonoff, (1996) EF's are necessary for organising information, planning short and long term strategies, future oriented

behaviour, set maintenance, self monitoring and self regulation, selective attention, vigilance of attention, inhibiting irrelevant behaviour, and switching strategies when old ones are no longer rewarding.

For the purposes of this study children with ADHD, ODD and CD will be defined on the basis of the results of their scoring on the Disruptive Behaviour Disorders (DBD) scale which will be completed by teachers and parents.

1.4. Background and significance of the study

The high crime rate in South Africa is one of the major concerns of the nation. Antisocial patterns that appear early in a child's life, and characterised by high frequency, intensity and severity predict a number of outcomes later on including victimization of others, drug and alcohol abuse, violence, school failure and drop out. The review of research literature indicates that preschool behavioural problems are a strong indicator of risk for future disruptive disorders, and that DBDs tends to be followed by a wide range of emotional, social, and relationship problems (Hendren & Mullen, 2003).

The project is part of the comprehensive study which aims at objective, non-verbal neuropsychological diagnostic methods, which can be used to diagnose DBD in the different populations of South Africa. Children and youth who are likely to encounter serious negative outcomes in their lives need support and intervention services early on within schools and community setting to reduce, buffer and offset early risk.

1.5 Delineation of the study

In chapter two a general introduction of DBDs is discussed including operational definition of concepts, diagnostic criteria, symptoms, prevalence and gender differences, aetiologies, secondary deficits, comorbidity of the disorders, developmental course of the disorders, and treatment options.

The neurobiological basis of DBDs is discussed in chapter three.

Chapter 4 is a presentation of diagnostic and assessment processes, the different screening and assessment methods that are often used in the diagnosis and treatment of patients with DBDs.

Chapter 5 provides a discussion of problem statement and research hypothesis. The methodology of the study is discussed in chapter 6, while chapter 7 gives a presentation of research results of the study.

The results of the study, description of the limitations, and outlines of possible areas of future research are discussed in chapter 8.

THE DISRUPTIVE BEHAVIOUR DISORDERS

2.1. General introduction

The cost and burden of Disruptive Behaviour Disorders (DBD) on the individual, his/her family and society are high (Harpin, 2005). According to Walker & Reid (1997) the consequences of DBDs include higher rates of high school drop outs, drug abuse, delinquency, and incarceration (Richards, Symons, Greene, & Szuszkiewicz, 1995). DBDs are often associated with academic skill deficits and low achievement, also frequently associated with low self-esteem and depression (Klassen, Miller, & Fine, 2004).

Meyer (1999) believes that children with DBD are at risk for a range of abnormalities in personality development including delinquency, antisocial behaviour, personality disorders, substance abuse, sexual promiscuity and subsequent risk for HIV, psychiatric problems such as anxiety and depression, accident proneness and traffic offences, and academic underachievement. These children display a pattern of externalising behaviour problems including hyperactivity, attention problems and more off-task behaviours.

Kim-Cohen, Caspi, Moffitt, Harrington, Milne, & Poulton (2003) see ODD as a significant risk factor for a wide range of concurrent and future psychiatric disorders including, conduct, and anxiety and mood disorders even in adult age.

Hunter (2002) maintains that DBDs which include Attention Deficit/Hyperactive Disorder (ADHD), Oppositional Defiant Disorder (ODD), and Conduct Disorder (CD) are among the most prevalent and stable child psychiatric disorders. DBDs are serious psychiatric disorders and when left untreated can adversely affect the lives of children. Even though the base rates of adolescent violence are high, the majority of violent acts are

perpetrated by a minority of persons. Over 50 % of violent behaviours are committed by only 6 % of the population (Dodge & Pettit, 2003).

Antisocial behaviour, youth violence, and student safety have become primary concerns in schools and larger society. Research based practices, tools, and approaches in interventions are needed to help alleviate the problems (Sprague & Walker, 2000).

In their investigation of the progression of DBDs from ODD to CD among children with ADHD, Biederman, Faraone, Spencer, Wilens, Mick, & Lapey (1994) found that nearly all those children with CD had comorbid ODD, with ODD preceding CD by several years. DBD children display poor cognitive and social functioning as a result of deficits in knowledge and mature thinking which is shown in their understanding of emotions and their interpersonal consequences of aggressive acts, as well as their reasoning about others' feelings and perspectives on moral dilemmas. Children with ODD or with CD are at high risk for both criminality and antisocial personality disorders in adulthood (Van Goosen, Matthys, Cohen-Kettenis, Westenberg, & Van Engeland, 1999).

Children who are hyperactive and aggressive have been found to have encoding problems and interpretation difficulties which may be contributory to social adjustment problems. Research studies report that the inattentive, disruptive, off-task, immature, provocative aggressive, and non compliant behaviours of ADHD children quickly elicit a pattern of controlling and directive behaviour from their peers when they must work together (Richards, Symonds, Greene, & Szuskiewicz, 1995). DBDs are unlikely to appear suddenly without developmental antecedents.

Tremblay, Loeber, Gagnon, Charlebois, Larivëe, & LeBlanc (1991) indicate that there is evidence that oppositional and aggressive elementary school children generally had behaviour problems during their preschool years. According to Meyer (1999) and Meyer and Aase, (2003) the comorbidity of ADHD with ODD and CD should not necessarily be

seen as differential diagnosis but as a complication. Cairns and Cairns (1991) indicate that most models of the development of DBDs specify a temperament component as one of the primary influences on the development of these disorders.

Most adolescents with DBDs and antisocial adults had problems during childhood. ADHD is characterised by symptoms of inattention, motor hyperactivity, and impulsivity, while ODD and CD are characterised by antisocial behaviour (Hunter, 2002).

According to Swanson et al. (1998b) ADHD is a persistent and a severe impairment of psychological development resulting in high levels of impulsive, hyperactive and inattentive behaviour that impairs functioning at home and at school. The onset of ADHD is in early childhood and it often persists into adolescent and adult life (Taylor, 1998). Children with both ADHD and CD often display a mixture of both the cognitive, attention, and inhibitory deficits as well as greater likelihood of factors associated with social adversity, family psychiatric problems, and family conflict (Quay, 1999). Sergeant (2000) believes that deficiency in executive functions is not specific to ADHD, but also applies to children with the associated disorders of ODD, and CD.

2.2 The comorbidity of ADHD, ODD and CD

Barkley (2006) sees ADHD as a developmental disorder of behaviour disinhibition associated with neuro-maturational immaturity. In supporting this view the symptoms of ADHD, ODD, and CD appear to be age related, symptoms of ODD are within the physical and mental capacity of a four year old to perform, while symptoms of CD that include school truancy, use of weapons, stealing with confrontation, forced sexual activity, breaking and entering can be done by older children. Studies have found that 54 – 67 % of children with ADHD will meet a full diagnostic criteria of ODD. According to Mangus, Bergman, Zieger, and Coleman (2004), 65% of children with ADHD have at least one comorbid disorder such as a learning disorder or abnormal intelligence, while one quarter

to one third of these children qualify for the additional diagnosis of ODD, CD or anxiety disorder. ADHD children are more likely to have developmental delays and cognitive deficits than those with conduct disorders (Quay 1999). According to Holmes, Slaughter, and Kashani, (2001) children presenting with ADD without hyperactivity are much less likely to present with ODD or CD than their ADHD peers. Children with ADHD occurring comorbid with CD tend to be far more likely to sustain their antisocial tendencies into adulthood than children with uncomplicated CD.

In their study Rey, Sawyer, and Prior (2005) found that aggressive behaviour is associated with the impulsive-hyperactive subtype of ADHD, and that comorbidity between ADHD and antisocial behaviour decreased when the children were older, with the exception of the inattentive subtype in which comorbidity was found to be higher among adolescents.

The substantial comorbidity existing between ADHD, CD and ODD has led some investigators to suggest that these are not independent diagnostic entities, but components of a unitary or heterogenous syndrome. In their study, Moss and Lynch (2001) found that ADHD could not be combined into a unitary construct, however, they could not confirm that ADHD and ODD/CD can be best viewed as two distinct entities. The high degree of comorbidity among the DBDs raises fundamental questions about these disorders, whether they are discrete and independent disorders or whether they represent variants of a single underlying disposition (Moss & Lynch, 2001). Although ODD and CD are highly interrelated in school-age, the two disorders have different symptoms, predictors, age profiles and patterns of comorbidity (Baving et al., 2005). Even though there is evidence that support the distinction between the symptoms of ODD and many symptoms of CD, there is controversy whether aggressive symptoms should be considered to be part of ODD or CD (Loeber et al., 2000).

Research investigating the behavioural precursors of DBD in early childhood has tended to focus on specific problematic behaviours such as non-compliant and aggressive behaviours, usually assessed by using rating scales. Because very few children receive a formal diagnosis of ODD and CD, most researchers have adopted a dimensional rather than categorical or diagnostic approach. Campbell and Pierce (1996) found that children with more extreme and pervasive problems with disruptive, high intensity, non-compliant and irritable behaviours showed the most disorganised and non-compliant behaviour in middle childhood.

The DSM-IV distinguishes two types of CD: Child onset Conduct Disorder and Adolescent onset Conduct disorder (American Psychiatric Association, 2000). In child onset Conduct Disorder the dysfunction is evident in early childhood beginning with ADHD or ODD. The symptoms progress to CD even though most still retain the symptoms of the ADHD and/or ODD diagnosis (Kazdin, 1995). Adolescent onset CD is more common than the childhood onset type, and adolescents tend to exhibit many of their behaviour problems in the company of peers (Mandel, 1997).

Faraone, Biederman, and Monuteaux (2002) point out that there are frequent reports of comorbidity between ADHD and CD which has been consistently found in clinical samples in studies of children with ADHD and those with CD, also in follow up studies of ADHD children. The DSM IV-TR as used by the American Psychiatric Association, (2000) sees them as two disorders co-occurring while the ICD-10, used by the World Health Organization, (1993) recognises it as a separate category of the Hyperkinetic Conduct Disorder

Gaub and Carlson (1997) found that girls with ADHD tend to have lower rates of CD. Some children with ADHD were found to have ODD, and that some ODD children progress to CD while others do not. Although girls are less overtly aggressive, than boys

they indulge in more relational aggression including damaging the reputation of others through gossip and rumours, and although only a few seriously delinquent girls became violent criminals, the majority of them cohabitated with violent, abusive men, and most who gave birth to children from these couplings were unable to care for them (Hendren & Mullen, 2003).

Research also reports that while a proportion of children with ODD later develop CD, a proportion of those with CD later meets the criteria for antisocial personality disorder (Loeber et al., 2000).

2.3 Diagnostic criteria and primary symptoms for DBDs

Currently there are two manuals that are used in diagnosis of mental disorders, the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) (American Psychiatric Association, 2000) and the International Classification of Diseases (ICD-10) (World Health Organization, 1993). For the diagnostic criteria used in this study reference is used from specifically the DSM-IV. Assessment instruments that are used to diagnose DBDs include clinical interviews, behaviour rating scales and behaviour observation (Brown, 2000).

2.3.1 Diagnostic criteria for ADHD

ADHD is a persistent and severe impairment of psychological development resulting from a high level of impulsive, overactive and inattentive behaviour. The combination of symptoms of inattention, impulsiveness and hyperactivity in children is recognised as a disorder when these behaviours are severe, developmentally inappropriate, and impair function at home and at school (Swanson et al., 1998b). The onset of ADHD must be in early childhood before the age of seven (Taylor, 1998).

Inattentiveness, overactivity and impulsiveness are regarded as the main clinical symptoms of ADHD. However, it is argued that the ADHD Inattentive type may have

heterogenous origins and is qualitatively different from ADHD Hyperactive/Impulsive subtype (Johansen, Aase, Meyer, & Sagvolden, 2002; Sagvolden, Johansen, Aase, & Russell, 2005).

The DSM-IV-TR criteria for ADHD are as follows (American Psychiatric Association, 2000):

A. Either (1) or (2)

Inattention

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

    (a) often fails to give close attention to details or makes careless mistakes in work, school work or other activities

    (b) often has difficulty sustaining attention in tasks or play activities

    (c) often does not seem to listen when spoken to directly

    (d) often does not follow through on instructions and fails to finish schoolwork, chores or duties in the workplace (not due to oppositional or failure to understand instructions)

    (e) often has difficulty organising tasks and activities

    (f) often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)

    (g) often loses things necessary for tasks or activities (e.g. toys, school assignments, pencils, books or tools)

    (h) is often distracted by extraneous stimuli

(i) is often forgetful in daily activities.

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

Hyperactivity

(a) often fidgets with hands or feet or squirms in seat

(b) often leaves seat in classroom or in other situations in which remaining seated is expected

(c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)

(d)   often has difficulty playing or engaging in leisure activities quietly

(e) is often "on the go" or often acts as if "driven by a motor"

(f) often talks excessively

Impulsivity

(g) often blurts out answers before questions have been completed

(h) often has difficulty awaiting turn

(i) often interrupts or intrudes on others (e.g. butts into conversations or games)

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

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

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

E. The symptoms do not occur exclusively during the course of pervasive developmental disorder, schizophrenia or other psychotic disorder and are not better accounted for by another mental disorder (e.g. mood disorder, anxiety disorder, dissociative disorder, or a personality disorder.

Diagnosis based on type:

Attention-Deficit/Hyperactivity Disorder, combined type (ADHD-C): if both criteria A1 and A2 are met for the first six months

Attention-Deficit/Hyperactivity Disorder, predominantly inattentive type (ADHD-PI): if criterion A1 is met but criterion A2 is not met in the past six months

Attention-Deficit/Hyperactivity Disorder, predominantly hyperactive-impulsive type (ADHD-HI): criterion A2 is met but criterion A1 is not met for the past six months (American Psychiatric Association, 2000).

2.3.2 Diagnostic criteria for ODD

Oppositional Defiant Disorder is characterised by enduring patterns of negativistic, disobedient, and hostile behaviour toward authority figures as well as inability to take responsibility for mistakes, leading to placing blame on others. Children with Oppositional Defiant Disorder frequently argue with adults and become annoyed with others. A child's temper outbursts, active refusal to comply with rules, and annoying behaviours exceed expectations for these behaviours for children of their age. These enduring patterns of negativistic, hostile and defiant behaviours occur in the absence of serious violations of social norms or of the rights of others (Sadock & Sadock, 2003).

ODD is associated with compromised social relations with parents and peers, and impaired school and academic performance (Baving, 2005). Greene, Biederman, Zerwas, Monuteaux, Goring, and Faraone, (2002) state that children with ADHD and comorbid

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with ODD present with a greater number of ADHD symptoms, which are in turn associated with increased severity of the disorder and a poorer prognosis.

In the International Statistical Classification of Diseases and Related Health Problems (ICD 10) (World Health Organization, 1993) ODD is seen as a less severe variant of Conduct Disorder rather than a distinct type (Sadock & Sadock, 2003).

The following are the DSM-IV-TR criteria (American Psychiatric Association, 2000) for Oppositional Defiant Disorder:

A. A pattern of negativistic, hostile and defiant behaviour lasting at least six months, during which (four or more) of the following are present:

(1) often loses temper

(2) often argues with adults

(3) often actively defies or refuses to comply with adults' requests or rules

(4) often deliberately annoys people

(5) often blames others for his or her mistakes or misbehaviour

(6) is often touchy or easily annoyed by others

(7) is often angry and resentful

(8) is often spiteful or vindictive

A criterion is met only if the behaviour occurs more frequently than is typically observed in individuals of comparable age and developmental level.

A. The disturbance in behaviour causes clinically significant impairment in social, academic, or occupational functioning.

B. The behaviour does not occur exclusively during the course of a psychotic or mood disorder.

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C. Criteria are not met for Conduct Disorder, and if the individual is 18 years or older, criteria are not met for antisocial personality disorder (American Psychiatric Association, 2000).

2.3.3 Diagnostic criteria for CD

Conduct Disorder is considered as one of the most serious, incapacitating chronic conditions encountered in child psychiatry. Children with conduct disorders compared with those having other diagnoses are likely to have more serious emotional disturbance, and require longer and more outpatient treatment and have higher rates of psychiatric hospitalization. These children report the highest number of problems and are significantly more withdrawn, have more somatic problems, more anxiety, more depression and more thought problems. Using the Child Behaviour Checklist, they are found to have significantly higher pathological externalising scores. These children also have more serious internalizing symptoms. The diagnostic category of Conduct Disorder is very inclusive and covers a multitude of biopsychosocial vulnerabilities. The category is so broad that it encompasses many different kinds of behaviours that it requires more sophistication to avoid the diagnosis than to make it. Conduct Disorder represents an array of child, parent, family, and contextual conditions (Hendren & Mullen, 2003).

In the ICD-10 (World Health Organization, 1993) Conduct Disorders include disorders confined to the family context, unsocialised Conduct Disorder, socialised Conduct Disorder, Oppositional Defiant behaviour, other Conduct Disorders and Conduct Disorder not specified. Conduct Disorder is characterised as repetitive and persistent pattern of dissocial, aggressive or defiant conduct (Sadock and Sadock, 2003).

Diagnostic criteria for conduct disorder as contained in the DSM-IV-TR are as follows (American Psychiatric Association, 2000):

A. A repetitive and persistent pattern of behaviour in which the basic rights of others or major age-appropriate societal norms and rules are violated, as manifested by the presence of three (or more) of the following criteria in the past twelve (12) months, with at least one criterion present in the past 6 months.

Aggression to people and animals

(1) often bullies, threatens and intimidates others

(2) often initiates physical fights

(3) has used a weapon that can cause serious physical harm to others (e.g. a bat, brick, broken bottle, knife, gun)

(4) has been physically cruel to people

(5) has been physically cruel to animals

(6) has stolen while confronting a victim (e.g. mugging, purse snatching, extortion, armed robbery

(7) has forced someone into sexual activity

Destruction of property

(8) Has deliberately engaged in fire setting with the intention of causing serious damage

(9) Has deliberately destroyed others' property (other than by fire setting)

Deceitfulness and theft

(10) has broken into someone's house, building, or car

(11) often lies to obtain goods or favours or to avoid obligations (i.e. 'cons' others)

(12) has stolen items of nontrivial value without confronting a victim (e.g. shoplifting, but without breaking and entering; forgery)

(13) often stays out at night despite parental prohibitions, beginning before 13 years

(14) has run away from home overnight at least twice while living in parental or parental surrogate home (or once without returning for a lengthy period)

(15) is often truant from school, beginning before age 13 years

B. The disturbance in behaviour causes clinically significant impairment in social, academic, or occupational functioning.

C. If the individual is age 18years or older, criteria are not meant for antisocial personality disorder.

Diagnosis based on age at onset

Conduct Disorder, childhood onset type: onset of at least one criterion characteristic of conduct disorder prior to age 10 years.

Conduct Disorder, adolescent onset type: absence of any criteria characteristic of conduct disorder prior to age 10 years.

Conduct Disorder, unspecified onset: age at onset is not known.

Severity:

Mild: few if any conduct problems in excess of those required to make the diagnosis, and conduct problems cause only minor harm to others

Moderate: number of conduct problems and effect on others intermediate between "mild' and "severe"

Severe: many conduct problems in excess of those required to make the diagnosis or conduct problems cause considerable harm to others

2.4 Differential diagnosis for DBDs

ODD and CD are very common in ADHD, which according to Meyer (1999) should not be necessarily seen as differential diagnosis but as a complication. ADHD frequently coexists with problems of anxiety and depression. Children with ADHD are likely to show neurodevelopmental delays such as delayed language milestones, impaired sensory motor coordination, delayed reading ability. Language delays may include expressive problems, limited vocabulary, use of simple, short utterances, and a relatively weak appreciation of what other people in conversation already know and understand. Other problems include poor handwriting and reading errors, while sensory problems may include erratic performance in tests requiring dexterity, and poor performance of rapid alternative movements.

CD is a common expression of numerous different conditions. At some point in the evolution of neuropsychiatric conditions ranging from schizophrenia to encephalitis, antisocial or aggressive behaviour may occur. Because children and adolescents are limited in their abilities both to conceptualise and to convey words on how they are feeling, what they are thinking, and why they are acting as they are, any condition that diminishes impulse control, jeopardises reality testing, increases suspiciousness, and impairs judgement is likely to result in a conduct disorder. Various disorders, ADHD, Learning disabilities, mood disorders, dissociative disorders, seizures and other kinds of central nervous system (CNS) dysfunction, and schizophreniform may present as behaviour disorder. Conduct Disorder is often a transitional designation when underlying causes for abnormal behaviours have not yet been identified. Through careful evaluation a multiplicity of neuropsychiatric and psychosocial vulnerabilities will be revealed each of which will need to be identified and addressed (Hendren & Mullen, 2003).

Inattention, overactivity, impulsiveness and poor judgement are characteristics not only of ADHD and CD but also of numerous other neuropsychiatric conditions including,

bipolar disorders and dissociative disorders all of which must be part of differential dignosis (Hendren & Mullen 2003).

A comprehensive diagnostic assessment should always be made with a view not only of determining the presence or absence of ADHD, ODD or CD, but also of the differential diagnosis from other childhood disorders (Meyer, 1999).

2.5 Prevalence of Disruptive Behaviour Disorders

The prevalence rates of ADHD, ODD and CD in South Africa is similar to those found in Western countries. Taken together, ADHD, ODD, and CD account for at least 75% of all psychopathological disorders of childhood and adolescence (Meyer & Sagvolden, 2001).

ADHD has long been recognised as a disorder of poor impulse and distractibility with considerable higher rates among disadvantaged sectors such as child welfare, mental health, and juvenile system. In childhood and adolescence it is associated with a variety of self management deficits and impairments in cognitive, social, school and family functioning. ADHD persists into adulthood in about 65% of those diagnosed with the childhood syndrome. In adulthood it is associated with social, intellectual and neuropsychological deficits, impulsivity, marital dissolution, and criminality (Pomerleau, Downey, Snedecor, Mehringer, Marks, & Pomerleau, 2003).

ADHD is a common neurobehavioral problem afflicting 5 - 10% of the children and adolescents, and persisting into adulthood in 30-50% or more of cases. Family twin and adoption studies suggest that genetic factors contribute to ADHD and symptoms of inattention, impulsivity and hypertension (Smalley, Bailey, Palmer, Cantwell, McGough, Del'Homme, et al., 1998).

Prevalence rates for ODD range from 2 -16%, it can begin from as early as 3 years though it is formally diagnosed at 8 years of age before adolescence. Before puberty ODD

is more prevalent in boys than in girls, but the sex ratio becomes equal after puberty. Many parents of CD children are themselves overly concerned with issues of power, control and autonomy (Sadock & Sadock, 2003).

Prevalence of CD among the general population is estimated to range from 1% to 10%. CD is more common among boys than girls and the ratio of boys to girls range from 4 to 1 and goes up to 12 to 1. CD is more prevalent in children of parents with antisocial personality disorder and alcohol dependence than in the general population. Children from poor socioeconomic background are at a higher risk for the development of CD than those who grow up in an urban environment (Sadock & Sadock, 2003).

2.6 Behaviour symptoms of children with DBDs

2.6.1 Behaviour symptoms of children with ADHD

Restlessness, inattentiveness, and impulsiveness are the manifestation of behavioural symptoms of ADHD. These children exhibit excessively or developmentally inappropriate levels of motor or vocal activity, they are more active, restless and fidgety than normal children (Barkley, 2006).

Abnormalities in response inhibition are a central component in the description and explanation of child psychopathological disorders, and in particular ADHD. The inattentive, hyperactive and impulsive behaviour that characterise children with ADHD is explained in terms of failure to suppress inappropriate responding. Children with ADHD have problems responding to rules and instructions, particularly when instructions are not repeated (Barkley, 2006)

Some children with CD have a low plasma dopamine β-hydroxylase, an enzyme that converts dopamine to norepinephrine suggesting decreased noradrenergic functioning in CD. High serotonin levels in the blood of some conduct juvenile offenders indicates that blood serotonin levels correlate inversely with levels of the serotonin metabolite 5-

hydroxyindoleacetic acid (5-HIAA) in the cerebrospinal fluid(CSF), and that 5-HIAA levels in the CSF correlates with aggression and violence (Sadock & Sadock, 2003). This will further be discussed in chapter three.

Research also indicates that Disruptive Behaviour Disorders i.e. ODD and CD have also been linked to a deficit in response inhibition. ADHD children are impaired in their ability to inhibit inappropriate responding, and impairments in response inhibition are not uniquely related to ADHD as DBD children showed shallower inhibition functions than normal controls. Besides the deficits in response inhibition and response reengagement, ADHD children demonstrate impairments in the response execution process suggesting a more general deficit in cognitive performance rather than a deficit confined to response inhibition. Children with ADHD do not expend the necessary effort to achieve and maintain optimal performance. There is a greater variability of task performance in these children, the standard deviation of multitask performance has been found to be considerably larger, with variability in homework and test grades and number of items completed varying from moment to moment (Barkley, 1998; Oosterlaan & Sergeant, 1998).

Research evidence indicate that hyperactive children could perform at normal or near normal levels of sustained attention under condition of continuous and immediate reinforcement but deteriorate dramatically when partial reinforcement is used. Performance of ADHD children rely more strongly on the presence of contingencies than normal children. The performance deficit in ADHD children may reflect a general impairment in executive functioning, which in turn may be attributable to a frontal lobe dysfunction, while poor achievement of ADHD children may reflect a non-optimal energy state. The non-optimal energy state causes impaired motor processing which becomes evident in slow

responding, high variability in the speed of responding and inaccurate responding (Oosterlaan & Sergeant, 1998).

2.6.2 Behaviour symptoms of children with ODD

Children with ODD often argue with adults, lose temper, and they are angry, resentful, and easily annoyed by others. They frequently defy adults' requests or rules, and deliberately annoy others. They tend to blame others for their mistakes and misbehaviours. Chronic ODD always interferes with interpersonal relationships and school performance. ODD children are often without friends and perceive human relationships as unsatisfactory. Despite the fact that they may have adequate intelligence, they do poorly or fail in school; they withhold participation, resist external demands, and insist on solving problems without others' help. These children suffer from low self esteem, poor frustration tolerance, depressed mood, and temper outbursts. Adolescents may abuse alcohol and illegal substances (Sadock & Sadock, 2003).

2.6.3 Behaviour symptoms of children with CD

According to the DSM-IV-TR American Psychiatric Association, 2000), the criteria for diagnosis of CD is divided into four categories of behaviours: (1) aggression to people and animals, (2) destruction of property, (3) deceitfulness and theft, and (4) serious violations of rules. Children with CD express their overt behaviours in various forms. It may take the form of bullying, physical aggression, and cruel behaviours towards peers. They may be hostile, verbally abusive, impudent, defiant, and negativistic towards adults. Persistent lying, frequent truancy, and vandalism are common. Destructiveness, stealing, and physical violence often occur in severe cases. Sexual behaviours and regular use of tobacco, liquor or non-prescribed psychoactive substances usually begin early in such children. Suicidal thoughts, gestures, and acts are frequent. Some children with aggressive behavioural patterns have difficulties with social relationships; they lack skills to communicate in socially acceptable ways, and appear to have little regard for feelings,

wishes, and welfare of others. Many children with conduct problems have poor self esteem, even though they may project an image of toughness. They are typically uncooperative, hostile and provocative (Sadock & Sadock, 2003).

2.7 Cognitive symptoms and cognitive vulnerabilities

This is manifested as a decrease in the flexibility in problem solving, an increase in perseveration of inappropriate responses, and overfocus on limited aspects of available information, and an inability to think divergently and creatively (Quay, 1999).

Children with DBD are more likely to exhibit the following characteristics; failure to finish tasks, daydreaming, failure to concentrate and shifting from one activity to another. They have problems with arousal, alertness, selectivity, distractibility, span of apprehension, and sustained attention. They also fail to consider potentially negative, destructive, or dangerous consequences that may be associated with particular situations and behaviours and a pattern of rapid inaccurate responding to tasks (Barkley, 2006).

Children and adolescents with CD have a clinical picture of a multiplicity of neuropsychiatric and cognitive vulnerabilities. While some studies are consistent with the idea that both ADHD and ODD/CD are associated with EF deficits, other studies have found that ADHD, not ODD/CD is associated with deficits in executive functions and that planning deficits in children with ADHD combined type (not the inattentive type) were independent of ODD and CD (Banaschewski, Hollis, Oosterlaan, Roeyers, Rubia, Willcutt, & Taylor, 2005). ADHD, like CD seems to be an impairment of self-regulation (Castellanos, 1999) and like children with conduct disorders, children with ADHD have problems with executive function as well as with other aspects of cognitive functioning.

These frontal lobe dysfunctions include foresight, judgement and control of impulses. In adolescents with CD, learning disabilities are important manifestations of cognitive dysfunction. Most adolescents with CD as opposed to a few that are severely

retarded have low normal scores on standard intelligence tests which place them on the border of diagnosis with potential treatment implication. Because many delinquent youngsters come from minority and socioeconomic backgrounds, clinicians tend to dismiss low scores as merely evidence of cultural deprivation rather than as an indication for remediation, suggesting that these cognitive problems seem to reflect either intrinsic limitations or environmental adversity. Poor judgement, impaired abstract reasoning, and difficulty planning ahead and anticipating consequences all contribute to behavioural problems. These children often have language and reading problems that impair their ability to put their thoughts, feelings, attitudes into words rather than actions (Hendren & Mullen, 2003).

2.8 Psychiatric vulnerabilities

The behavioural characteristics of CD can be manifestations of different kinds of neuropsychiatric conditions. ADHD is probably the most common child psychiatric disorder, or comorbid diagnosis of delinquent children. It is characterised by impulsiveness, short attention span, disinhibition, overactivity, socially inappropriate behaviours, poor judgement and school difficulties, the very same characteristics as those observed in children with CD (Barkley, 1997b). Irritability and rage that often accompany adolescent depression, especially when aggravated by alcohol or drug abuse, can masquerade as aggressive conduct disorder (Zoccolillo, 1992). Alcohol and drug abuse are frequent concomitants of serious delinquency and violence in adolescence. Mania in adolescence can mimic ADHD, ODD, and CD. Manic youngsters with their grandiosity and boastfulness, and their heedlessness of consequences of their acts and their apparent lack of empathy can be mistakenly dismissed as simply being narscistc and sociopathic (Hendren & Mullen, 2003).

Studies of adult psychiatric patients and those of psychopathology in violent delinquent adolescents indicate that violence and psychopathology go hand in hand. The most common psychiatric symptoms associated with violent behaviour are delusions of thought control, thought insertion, and persecution. Another most common symptom associated with recurrent violence is paranoid ideation. Researchers have found that the more violent and bizarre the adolescent behaviours the more likely the existence of underlying psychotic symptoms (Hendren & Mullen, 2003).

Dissociative disorders play a role in many violent acts of children, adolescents and adults, and consequently dissociative phenomena might explain some of the symptoms and bizarre behaviour of some violent adolescents. During dissociative episodes children may scream obscenities, take belongings of others, set fires and even attack others, and later deny their acts because they do not recall their actions. Their erratic school performances, distractibility, episodic aggression and denial of destructive acts that others may have actually witnessed cause many such children to be diagnosed with ADHD and CD. Episodic aggression, inappropriate sexual behaviours, leaving school or home for hours to days, and having in their possession the property of others but denying having taken it constitute some of the most common externalising or antisocial behaviours of children and adolescents to be misdiagnosed as having CD (Hendren & Mullen, 2003).

2.9 Social effects of Disruptive Behaviour Disorders

ADHD has a significant effect on children's relationships with peers, family and adults. They are often disruptive, interfering, intrusive, abrupt, impatient, and both verbally and physically aggressive. They are more socially busy, intrusive, critical and overbearing, and less attuned to the social agenda of peers. These children are inattentive to subtle social cues and procedures of entering a peer group, and they have a tendency to attribute hostile tendencies to others. As a result of these characteristics, they experience a high level of

rejection by their parents and teachers, and their parents respond to them in a controlling and negative fashion (Quay, 1999).

Treatment with stimulants results in a substantial and immediate improvement in the quality of the children's social interactions with their peers, parents and teachers. It reduces the frequency of negative verbalisations such as teasing and swearing, and of conduct problems such as physical aggression, lying, stealing and destruction of property. They become less dominating and annoying and initiate a fewer negative social interaction with their peers. They also become more cooperative with their parents, peers and teachers, and they are less disruptive in the classroom (Barkley, 1999). Treatment of DBDs will be dealt with in subsequent chapters.

There is an extensive overproduction of dopamine receptors in the striatum and accumbens during prepubertal development, and an extensive pruning of dopamine receptors in striatum after puberty, which is consistent with the observation that ADHD often recedes or diminishes in severity in males (Andersen & Teicher, 2000).

Clinical studies with Magnetic Resonance Imaging show that children with ADHD have enhanced asymmetry of the caudate nucleus with the left side larger than the right. Decreased size of the corpus callosum has also been reported in children with ADHD (Andersen & Teicher, 2000).

2.10 Gender and age differences in the manifestation of DBDs

Enduring differences in D1 receptor density in the nucleus accumbens may have some bearing on the greater incidence of substance abuse in males given the putative role of accumbens D1 system in addictive behaviours (Andersen & Teicher, 2000)

ADHD is more often diagnosed in males than females by 2-9 fold, however females seek more treatment at a higher rate than males. This suggests that gender

differences exist in the severity and course of the illness, and females may be more severely affected than males. Research found that girls with ADHD were found to have lowest glucose metabolism than boys with ADHD and normal girls, and girls with ADHD tend to have a higher genetic loading for the disorder (Smalley et al., 1998).

The incidence of DBDs is generally lower for girls than for boys (Gaub & Carlson, 1997). In childhood ADHD is more common in boys than in girls while more boys than girls will mostly continue to exhibit the disorder and consequently more boys are likely to meet the criteria for CD as CD is diagnosed in older children. According to Carlson, Tamm, and Gaub (1997) the difference in male to female ratios could be as a result of the fact that males show more aggression and antisocial behaviours than females, which is more attributed to societal expectations that girls should be more well behaved than boys. Ohan and Johnston (2005) suggest that, because the DSM-IV symptom criteria for ADHD, ODD, and CD were developed and validated using samples composed primarily of school aged boys, questions have arisen regarding the appropriateness of using these criteria for diagnosing girls. The basis of this argument is that the symptom criteria in the DSM inadequately represent how girls manifest the core pathology of these disorders as the expression of these disorders may differ between boys and girls despite the presence of the underlying pathology. As an example the expression of defiance which is more central to the conceptualisation of ODD it is possible that girls are more likely to defy passively by ignoring and neglecting to do what has been asked in contrast to boys who may be more likely to defy actively by arguing vociferously which is represented in the DSM symptoms (Ohan & Johnston, 2005).

High rates of psychopathology have been reported in female delinquent population, with CD, substance abuse, depression and anxiety particularly frequent. There is, however, emerging evidence that the characteristics of female offenders are distinctly different from

male offenders. Research evidence suggests that female juvenile offenders are particularly susceptible to trauma exposure and trauma related symptomatology, and that trauma is more strongly associated with involvement in serious delinquent activity in girls than in boys. It is widely accepted that juvenile delinquency is the results of complex interactions between numerous risk factors over time and environment. Studies based predominantly on male delinquent samples have consistently related juvenile offending behaviour to factors such as history of physical and sexual abuse, neglect, poverty, poor academic achievement, and family dysfunction, including parental criminality and substance abuse (Dixon et al, 2004).

Predominantly gender segregated peer relationships during early elementary school lead to systematically different social experiences for boys and girls. Boys' relative to girls' peer ecologies are characterised by higher levels of competition, aggression, rough tumble play and domination, and by lower levels of cooperation, supportive verbal exchange, mutual accommodation and conflict avoidance. Boys on average experience higher levels of impulsivity than girls, and these early gender differences in self regulation, along with the quality of peer relationships, have been reported to account for 50-65% of the variance in gender differences in conduct problems and antisocial behaviour (Snyder, Prichard, Schrepferman, Patrick, & Stoolmiller, 2004). According to Moffitt (2003) deficits in self regulation and poor peer relations (along with temperamental traits of under-control and weak constraint) predict antisocial behaviour in both sexes, but males are more likely to experience them.

Cultural effects have more to do with whether important institutions of enculturation are consistent or inconsistent with the demands made and standards set for children behaviour and development. According to Ross and Ross (1982) the cultural view will determine the threshold for defiance that will be tolerated in children and exaggerate a

predisposition to ADHD and DBD in some children. Consistent cultures will therefore have fewer children diagnosed because they minimise individual differences among children and provide clear and consistent expectations and consequences for behaviour that conform to the expected norms, while on the other hand inconsistent cultures will have more children diagnosed with ADHD and consequently DBD and produce ambiguous expectations and consequences to children regarding inappropriate conduct. Highly consistent and highly conforming cultures may have more children who are unable to conform to societal expectations, while inconsistent and low conforming cultures may tolerate deviant behaviours to a greater degree.

Cairns, Cairns, Neckerman, Ferguson, and Gariepy, (1989) state that symptoms of DBD are expressed differently for boys and girls, boys' physical aggression develops out of rough play whereas indirect or relational aggression which takes the form of alienation, ostracism, character defamation, and collusion directed at relational bonds is reported to be more common among girls. According to Lalloo et al. (2003) boys score significantly higher for antisocial behaviour, hyperactivity, Conduct Disorder and peer problems. They found that girls scored higher for emotional symptoms. Younger children were found to experience more hyperactive and conduct disorder behaviours, while older children have more emotional symptoms and peer problems.

Symptoms that are more serious, more atypical for the child's sex, or more age atypical, appear to be prognostic of serious dysfunction (Loeber et al., 2000).

Available assessment instruments are much more tuned to male type emotional behaviour problems. Zoccolillo (1992) argues that in the DSM-IV the diagnostic criteria for CD have not been validated with females, so researchers may not accurately assess the nature of CD in girls.

Another factor contributing to the gender differences is related to production and density of dopamine D1 and D2 in the ventral striatum, nucleus accumbens or dorsal striatum. According to Sagvolden, (1999) males overproduce D1 in the nucleus accumbens and a fivefold overproduction of D1 and D2 receptors in the dorsal striatum of males has been reported as compared to females.

2.11 Conclusion

The diagnosis of CD has a grim prognosis, a minority of children and adolescents with CD go on to commit aggressive antisocial acts in adulthood. The overall adjustment of seriously, behaviourally disordered adolescents is often poor, and is reflected in unstable marriages, unsatisfactory job histories, and other symptoms of maladaptation (Lewis, 2004).

Research has shown that a proportion of children with ADHD later develop ODD, and a proportion of those with ODD later develop CD, while a proportion of those with CD will develop antisocial personality disorder (Loeber et al., 2000). The high comorbidity of DBDs has led to some researchers to question whether these disorders are discrete and independent or whether they represent variants of a single underlying disposition. Comorbidity of DBDs should not necessarily be seen as differential diagnosis but as a complication and that ADHD appears to be the main cause of early onset of conduct disorder (Meyer, 1999).

The development of DBDs through ADHD, ODD to CD and Antisocial Personality Disorder necessitates timely diagnosis, intervention and treatment to offset the risk of DBDs in children and adolescents. Since DBDs are associated with compromised social relations with parents and peers, and are impaired in school and academic performance, treatment should be applied in those different settings. Strategies should

focus on early prevention of behavioural problems in preschool children than late

treatment strategies.

AETIOLOGY OF DISRUPTIVE BEHAVIOUR DISORDERS

3.1 Introduction

Over the last decade, scientists came up with many possible theories to explain the causes of DBDs and Learning Disabilities. Some believed that ADHD and Learning Disabilities were caused by minor brain injuries or undetectable brain damage, and early infections or complications at birth. Refined sugar and food additives were believed to make children hyperactive and inattentive. These theories were rejected because they could only explain a small number of cases (Barkley, 1997a).

The birth of clinical neuroscience and neuropharmacology, has loosened the grip of empirically unverified psychoanalytic theories that dominated psychiatry for half a century, and has led toward a syndrome based diagnostic system that relies on specific, observable criteria as the basis for a reliable diagnosis. The development of neuroimaging techniques has made it possible to propose theoretical models of how the brain works in individuals who have identifiable disorders. These developments have enabled more scientists to test more theories about the causes of DBDs (Castellanos, 1999).

The advances in neuroimaging techniques including Computerised Tomography (CT), Magnetic Resonance Imaging (MRI), and Positron Emission Tomography (PET) scans have helped to confirm the involvement of the frontal lobes, and to identify other structures involved in DBDs. Some research findings from imaging studies report smaller right prefrontal cortical regions, and show the involvement of the prefrontal cortex, the striatum (especially the caudate nucleus) and the basal ganglia in children with ADHD (Rapoport, Castellanos, Gogate, Janson, Kohler, & Nelson, 2001).

The combinations of neuropsychiatric and cognitive vulnerabilities with an upbringing in a violent, abusive household have been found to be highly correlated with violent criminality (Hendren & Mullen, 2003).

Factors that have been identified in the development of DBDs include neurological, genetic (hereditary), environmental factors and psychosocial factors (Brown, 2000).

Neuroanatomical and neuropsychological studies suggest that ADHD may be a frontal lobe disorder with deficits predominantly in the prefrontal lobe, mediating self-regulating and executive functions. A dysfunction in inhibitory control is seen as the main cause of the major deficits observed in hyperactive children. These children are unable to withhold a planned response, to interrupt a response that has been started, and to protect an ongoing activity from interfering activities. Failure to sustain attention may be due to failure to inhibit interfering activities, and distractibility may be caused by not inhibiting attention to irrelevant information. Response inhibition and self control are essential for behaviour and cognition, and play a substantial role in social adaptation (Rubia, Oosterlaan, Sergeant, Brandeis, & van Leeuwen, 1998).

The neurotransmitters, norepinephrine (NE), dopamine (DA), and serotonin (5-HT) are involved in the regulation of several behavioural systems that play an important role in the interaction of the organism with its external environment (Van Goosen et al., 1999). According to Gray (1997) DA appears to be involved in the expression of behavioural patterns, including aggression and sexual behaviour whereas behavioural inhibition seems to be regulated by NE and 5-HT. Research studies have reported associations between low cerebrospinal fluid levels of 5-hydroxyindoleacetic acid (a serotonin metabolite), and impulsiveness and aggression. Others researchers reported increased aggression in men depleted of tryptophan (a serotonin precursor), increased

prolactin responsivity to fenfluramine, and increased blood serotonin levels (Hendren & Mullen, 2003).

3.2 Neurobiology of DBDs

3.2. 1 Neuroanatomy of DBDs

Findings from magnetic resonance imaging (MRI) studies have identified two brain regions, the frontal lobes and the basal ganglia that were found to be smaller in ADHD children. Children with ADHD were found to have smaller brain volumes in the anterior superior region (posterior prefrontal and midanterior) cingulated and anterior inferior region (anterior basal ganglia), abnormalities that implicate neuroanatomical networks of executive control and alerting (Swanson, Castellanos, Murias, LaHoste, & Kennedy, 1998a). Major functions regulated by the prefrontal lobes include inhibition, regulation of arousal, emotional expression and behaviour, planning, distractibility, and judgement (Bradley & Golden, 2001). An insult to the frontal lobes can cause disruption in many areas of behaviour regulation such as behavioural inhibition, regulation of emotional impulses and motivation, planning behaviour, and using external feedback in organising behaviour across time. Symptoms of lesions to the frontal lobes and prefrontal cortex include deficits in sustained attention, inhibition, self-regulation of emotion, and motivation, and capacity to organise behaviour over time. There is a decreased cerebral blood flow to prefrontal regions and pathways connecting the regions to the limbic system via striatum specifically the caudate nucleus (Barkley, 2006).

Electroencephalography (EEG) and galvanic skin response studies have reported diminished central nervous system arousal in ADHD children, which is evidence of dysfunction in the prefrontal region, the reticular activating system, and/or their interconnective fibres. Although ADHD children have been shown to have increased beta wave activity in the left hemisphere, which is an indication of overarousal, other studies of

evoked responses during tests of motor functioning, have consistently found ADHD children to have slower amplitudes during the end phase of their response. Qualitative EEG studies have also identified abnormalities reflecting slowing of brain waves in ADHD children suggesting changes in arousal and attentional processes. It can therefore be concluded that under tasks where stimulation is provided, ADHD children will demonstrate under-arousal during later stages of their responses (Bradley & Golden, 2001).

The right anterior frontal, caudate and globus pallidus regions were found to be smaller in an ADHD group compared to normal children. Structural studies have found reduced volumes in the right frontal brain region, caudate, corpus callosum, cerebellum, and in parietal, temporal, and occipital brain regions (Castellanos, Giedd, Marsh, Hamburger, Vaituzis, Dickstein et al., 1996; Mostofsky, Cooper, Kates, Denckla, & Kaufmann, 2002). Using positron emission tomography (PET) scan, a general decreased metabolic activity was found in adults with ADHD even though it could not be replicated in adolescents. However a significant correlation between ADHD symptom severity and cerebral glucose metabolism in the left frontal lobe in adolescents was reported (Bradley & Golden, 2001).

Hypoperfusion (below normal levels of cerebral blood flow) bilaterally in the frontal lobes was found to be common in children suffering from ADHD (Lou, 1990), and methylphenidate which significantly reduces symptoms, also increases blood flow to the frontal regions (Bradley & Golden, 2001).

Functional imaging studies using single positron emission computerised tomography (SPECT) and functional magnetic resonance imaging (fMRI) have found abnormal brain activation patterns during attention tasks and response inhibition in the frontal, caudate and parietal lobes. Consistent reduced caudate activation and over- and under-activated prefrontal lobes have been reported. Recent ERP studies show

abnormalities in prefrontal lobe activation in children with pure CD. Electrophysiological activity was also found to differ between comorbid ADHD+ODD/CD and pure ADHD or pure ODD/CD groups (Banaschewski et al., 2005).

3.2.2 Neurochemistry of DBDs

Research on emotionally and behaviourally disturbed boys suggests that both orbitofrontal and amygdaloid dysfunction may underlie diminished emotional responsiveness to changed stimuli. Psychomotor seizures (complex partial seizures) are more common in violent delinquent individuals than in the general population. Psychomotor symptoms such as impaired memory for non-violent and violent behaviour, olfactory hallucinations, and recurrent episodes of deja vu are fairly common in the aggressive delinquent population (Lewis, 2004).

Research has established a relationship between diminished central serotonergic (5-HT) function and aggressive behaviour in animals and human adults. However, discrepant findings in children suggest that age or the presence of ADHD may influence this relationship. According to research findings, there is no clear relationship between central 5-HT function and aggression in disruptive boys (Schulz, Newcorn, McKay, Himelstein, Koda, Siever, Sharma, & Halperin, 2001). However, a positive association between CSF 5-HIAA and ratings of aggression in a sample of boys selected for the presence of ADHD was found. The presence of ADHD may influence the relationship between central 5-HT function and aggression in children. While ADHD is generally associated with catecholaminergic dysfunction, perinatal insults to central dopaminergic systems have been shown to produce morphological and functional changes in 5-HT neurons. Consequently, perturbations in dopamine activity associated with the presence of ADHD could alter central 5-HT function in children thereby modifying its relationship with aggression. The hypothesis that 5-HT varies with the presence or the absence of ADHD, could help

resolve the apparent inconsistencies between child and adult literature. Symptoms of ADHD decrease with age in many individuals, and these longitudinal changes in symptomatology may be mediated in part by developmental changes in central 5-HT function (Schulz et al., 2001).

Van Goosen et al. (1999) see 5-HT activity as stabilizing information flow which results in controlled behavioural, affective, and cognitive output, whereas deviations of 5-HT activity result in altered neural information processing tendencies.

The general psychobiological model, Gray's model (Gray, 1982) of relevance helps to provide an explanation for the occurrence of DBD's. The model has to do with the dopaminergic and serotonergic functioning.

According to Gray's model of relevance (Pliszka, 1999), aggressive behaviour can result from enhanced or diminished functioning in the following three systems:

a) The behaviour inhibition system which is represented in the brain primarily by noradrenergic (NE) projections from the locus coeruleus and serotonergic (5HT) projections from the brain stem raphe nuclei to diverse areas of the lower brain and higher cortical centres. It reduces the frequency of response when reward is unlikely and when punishment is likely. A hyposerotonergic state can lead to inhibited impulsive behaviour.

b) The reward system which is primarily linked to dopaminergic (DA) projections, controls behaviour that is motivated by the prospect of reward and the escape from or avoidance of punishment. A hyperdopaminergic state can lead to enhanced levels of aggression.

c) The Fight/Flight system which is primarily activated during times of perceived threat to the organism resulting in defensive actions or flight

behaviour. Supersensitive F/F can lead to exaggerated defensive reaction to minimal provocation.

Gray's psychobiological theory of learning and emotion has also been used to explain the symptoms of ADHD. The three collaborative brain systems i.e. behaviour activation system (BAS); behaviour inhibition system (BIS), and the non specific arousal system (NAS) work together to modulate behaviour. The NAS is activated by both the BAS and the BIS and acts to increase the intensity (speed/force) of behaviour. In normal children the BAS and BIS cooperate with one another to meet situational demands such that when response inhibition is required, the BIS is activated and temporarily predominates over the BAS. Children with ADHD have difficulty inhibiting ongoing and anticipated motor behaviour because of under active BIS (Luman, Oosterlaan, & Sergeant, 2005).

However, the functioning of neurotransmitter systems though they may be genetically determined can be induced by environmental factors like drugs of abuse or environmental pollutants (Sagvolden, 1999).

Attention may be seen as a set of neural networks that carries different functions, including orientation to sensory stimuli, exercise of executive control, and maintenance of the alert state. The attentional network is like an aggregate in which a posterior system is responsible for the orientation towards new stimuli, and an anterior executive system coordinates the frontal lobe functions necessary for the analysis, selection, and initiation of responses. A dysregulated central norepinephrine system in persons with ADHD may not efficiently prime the posterior attention system to external stimuli (Schachar & Ickowicz, 1999).

The anterior system which is responsible for executive functions (inhibitory control and working memory) is to a great extent dopaminergic. DA plays a major role in

regulating the excitability of the cortical circuitry on which the memory function of the prefrontal cortex depends. Inadequate dopaminergic activity at the prefrontal synapse may lead to dysfunction in inhibitory control (Barkley, 1997b). Dopamine is involved in the mesolimbic circuitry mediating sensitivity to rewards and in cerebellar activity. The cerebellum has been classically associated with motor incoordination, and was also found to play a role in higher cognitive functions, including memory, learning and attention. The central and the peripheral epinenephrine systems are involved in a person's response to information through their effects in the locus ceruleous, which is an essential part of the preparatory mechanisms for attention (Pliszka, 1999).

Central epinephrine inhibits the locus ceruleous directly, and the peripheral effects of epinephrine (increased heart rate and blood pressure) may reset the locus ceruleous at a lower level of activity (Pliszka, McCracken, & Maas, 1996).

ADHD is not merely regarded as a deficit of attention, an excess of locomotor activity, or their simple conjunction, but the unifying abstraction that best encompasses the faculties principally affected in ADHD have been termed Executive Functions (EF), whose consistent deficits can be measured with tests of vigilance. Central DA activity was found to be playing a mediating role in stimulant drug efficacy and in motor activity and not in vigilance. Of the three monoamines, DA is the most developmentally dynamic, its metabolic levels in cerebrospinal fluid (CSF) peak in infancy and decline rapidly over the next 12 years. Research found that in a four year longitudinal study of 106 children with ADHD, the symptoms of hyperactivity and impulsiveness, but not inattention declined linearly with increasing age further supporting the hypothesis that central DA levels are correlated with symptoms of hyperactivity-impulsivity in ADHD. An NE posterior parietal network was found to be associated with vigilance and DA prefrontal-striatal-thalamo-cortical circuits associated with executive functions (Pliszka, 1999).

3.2.3 Neuropharmacology of DBDs

Castellanos (1997) proposed that ADHD may result from different abnormalities in two dopamine regions, underactivity in a cortical region (anterior cingulate) which results in cognitive deficits, and overactivity in a subcortial region (dorsolateral prefrontal), which results in primary memory deficits, and overactivity in a subcortical region (locus coerulus) which results in overarousal.

Activity in networks of these regions is modulated by subcortical input from midbrain neurons that have receptors strategically located in specialized neuroanatomical networks. The primary site of action of stimulants is through the dopamine system. The baseline level of a primary dopamine metabolite (HVA) was positively correlated with behavioural response to multiple stimulant medications, but the baseline levels of norepinephrine or serotonin metabolites i.e 3-methoxy-4-hydroxyphenylglycol (MHPG) and 5-hydroxyindoleacetic acid (5-HIAA) were not significantly related to drug response. Studies in molecular biology support the notion that both dopamine deficiency and excesses contribute to ADHD depending on the domain receptor type affected. Research studies have been successful in identifying the site of action of methylphenidate, which blocks the dopamine transporter and thus increasing the temporal and spatial presence of dopamine at the synapse (Swanson et al., 1998a).

Although the treatment of DBDs will be discussed in the subsequent chapter of diagnosis and treatment the following will suffice to be included in this section. Treatment with stimulants results in a substantial and immediate improvement in the quality of the children's social interactions with their peers, parents, and teachers. Treatment leads to reduced frequency of negative verbalizations, such as teasing and swearing, and of conduct problems. They become less dominating and annoying, and initiating a fewer negative social interactions with their peers resulting in their improved standing in the eyes of their peers, they become more cooperative with their teachers and peers, and less disruptive in

the classroom. Their parents and siblings become more positive and less critical of them. Despite the beneficial effects discussed above, medication has been shown to have minimal effects on the processesing of social information, including their typical attribution of hostile intentions. Even with medication these may not cease their aggressive response when provoked (Schachar & Ickowicz, 1999).

3.2.4 Neuropsychology of ADHD

A number of studies indicate that ADHD is associated with deficits on variety of neuropsychological measures. ADHD children have been shown to be impaired in various executive function domains. Other abnormalities of impairment include altered motivational processes (Sonuga-Barke, 2002) or insufficient ability to regulate the state of activation (Sergeant, Geurts, & Oosterlaan, 2002), measures of domains with less of an executive component, such as processing speed, rapid naming, fine and gross motor skills, timing functions and early and automatic processing stages (Banaschewski et al., 2005).

Research also reports that a series of minor injuries such as mild concussion can have cumulative adverse effects. Neuropsychological impairments have been found to be associated with frontal lobe functions, and therefore causality of ADHD is conceptualised to include, frontal lobe dysfunctions, delayed frontal maturation, and dysfunctional subcortical frontal motor subsystems (Johnson, Morrow, Accornero, Xue, Anthony, & Bandstra, 2002). Damage to the frontal lobes (regions of the brain that are involved with judgement, foresight, impulse control, and recognition of interpersonal cues) can contribute to behaviour problems and violence, while frontal lobe dysfunction has been shown to be characteristic of adult offenders. Clinical evaluations of delinquent adolescents have shown that the more aggressive teenagers tend to have more neurological signs indicative of frontal lobe dysfunctions (Lewis, 2004).

Several studies have reported that ADHD in children are associated with slower and inaccurate performance as demands for more effortful processing of information increase (Johnson et al, 2002). These children were also found to be unable to inhibit responding on neuropsychological tests (Oosterlaan, Logan, & Sergeant, 1998). Although most studies found that only ADHD and not ODD or CD is associated with deficits in EF, Oosterlaan et al. (1998) have concluded that ADHD+ODD/CD, and CD are associated with inhibitory dysfunctions. The fact that CD in other studies such as that of Aronowitz, Liebowitz, Hollander, Fazzini, Durlach-Misteli, Frenkel et al., (1994) was found to be associated with poor cognitive flexibility, suggests that ADHD and ODD/CD may differ in terms of their profile of EF deficits.

The performance of adults with ADHDS on the Wechsler memory scales (Wechsler, 1997) as a measure of intelligence showed deficits in both immediate and later recall of information, supporting the notion that they have difficulties encoding information. However, with non verbal memory test of visual reproduction deficit ADHD adults similar to normal group when they were asked to reproduce immediately after viewing it, but less accurately after a 30 minute delay (Johnson et al., 2002).

Research studies have shown that ADHD children perform more poorly than non-ADHD children on neuropsychological tasks measuring sustained attention, executive functioning, motor inhibition, and verbal learning and memory which are acknowledged as neuropsychological deficits in children with ADHD. Neuropsychological processes that permit or assist a person with self regulation are defined as executive functions. Domains of executive functions that fall in the field on neuropsychology include motor coordination and sequencing, working memory and mental computation, planning and anticipation, verbal fluency and confrontational communication, effort allocation, application of organisational strategies, internalisation of directed speech, adherence to restrictive

instructions, and self regulation of arousal and are considered to be mediated by the frontal cortex, particularly the prefrontal lobes (Barkley, 1997; Johnson et al, 2002). Although ADHD has always been conceptualised as a childhood disorder, adolescents also demonstrated impaired performance on neuropsychological tests that assess attention, executive functioning, impulse control, and verbal learning. Approximately 30-50% of ADHD patients continue to meet the diagnostic criteria in adulthood (Johnson et al, 2002). Sergeant (2000) found that ADHD and CD children had slower Stop Signal Reaction Time (SSRT) which does not support the notion that response inhibition deficits are specifically related to ADHD, and that measures of inhibition do not differentiate ADHD children from CD children.

ADHD children have inefficient response styles that are slow and inaccurate rather than an impulsive response style which is fast and inaccurate. Three theories have been proposed for attentional deficits, the first one being Barkley's theory (Barkley, 1997a), which is based on theories of language and frontal lobe function and it suggests that behaviour inhibition is the core of ADHD and attention are secondary and are a consequence of behaviour inhibition. The cognitive energetic theory suggests that the core deficit in ADHD is a state deficit rather than a process deficit that selectively affects output stages rather than input stages of information processing (Sergeant, 2000). The third theory is based on the neuroanatomical network theory of attention. It asserts that the core deficits of ADHD are a combination of alerting and executive control deficits suggesting that state and behavioural inhibition would be considered as attentional deficits (Swanson & Castellanos, 1998).

Attention and executive functions are a variety of functions of the prefrontal cortex.. Neuroanatomical regions that emphasise cognitive capacity of the computational modeling approach give consideration to the prefrontal cortex involved in working

memory. The clinical neurology approach, which emphasise cognitive processes, but excludes emotional or motivational processes considers the dorsolateral prefrontal cortex as playing an important role, but excludes the orbital and medial frontal regions. The cognitive model that emphasizes conflict resolution considers the anterior cingulated brain region of importance (Swanson, Posner, Cantwell, Wigal, Crinella, Filipek et al., 1998). However recent neuropsychological evidence which demonstrates temporal processing deficits and non executive memory deficits suggests the involvement of wider brain areas implying that the common perception of ADHD as a cortico-striato-thalamo-cortical disorder may be too limited (Banaschewski et al., 2005)

3.3 Genetic factors

Genetic factors account for moderate amounts of variance in children's externalising problems, substance abuse, and in self-reports of adolescent delinquent behaviours. The genetic base for most problem behaviours likely reflects combinations of genes that are expressed in different ways at different points in life. Such polygenetic factors may operate additively of interactively to increase the probability of specific disorders, and may render certain children ill-equiped to manage ordinary tasks of social life placing them at risk for conduct disorders. Because of genes and in-utero experiences, some children are born with a hyperpersistent behaviour facilitation system (Gray, 1997), an under-active behavioural inhibition system, autonomic nervous system hyperarctivity, cognitive problems in sustaining attention to cues, low cerebrospinal fluid concentration of serotonin metabolites (5HIAA).

In families of children with ADHD, 10-35% of family members have been found to have the disorder with concordance estimated at 15-20% in mothers, 25-30% of fathers, and 32% in siblings (Bradley & Golden, 2001). ADHD is a developmental disorder with both genetic and environmental underpinnings. It is a familial disorder, children of parents

or siblings with ADHD are at increased risk of receiving a childhood diagnosis of ADHD. The heritability estimates of twin studies range from 75-91% (Auerbach, tzaba-Poria, Berger, & Landau, 2004). Adoption studies suggest specific genetic and environmental risk factors rather than a general addictive tendency or pattern of deviant behaviour (Hicks, Krueger, Iacono, McGue, & Patrick, 2004)

Molecular genetic studies have identified risk alleles that are widely distributed in the population each accounting for small increase of risk of ADHD (Banaschewski et al., 2005).

Molecular genetic research of ADHD has focused on examination of RNA (responsible for encoding of dopamine) receptors within alleles of neurons. The research focus is on the RNA responsible for the encoding of dopamine receptors because the cerebral structures heavily enervated by dopamine have been implicated in the development of ADHD. Studies have shown that D4 receptors play a greater role in cognitive and emotional functions as opposed to motor functions. ADHD children have demonstrated D4 gene polymorphism as they differed significantly in the distribution of DRD4 alleles when compared to control subjects, a finding which supports the notion that that a genetically inherited defect in the encoding of the D4 receptor may contribute to ADHD (Bradley & Golden, 2001).

However, despite this findings Bradley and Golden (2001) showed that there remain a large number of ADHD children who did not have the defect and a small percentage of normal children who had the defect did not show any symptoms of ADHD. Swanson, Oosterlaan, Murias, Schuck, Flodman, Spence, et al., (2000), also suggests that the presence of the D4 7 repeat allele is associated with increased risk for ADHD, and that this is neither a necessary condition as about half of the ADHD children do not have a 7 repeat allele, nor a sufficient condition since about 20% of unaffected controls have a 7

repeat allele. ADHD children with 7 repeat allele showed normal response and speed variability in neuropsychological tests designed to probe attention networks in neuroanatonical foci in D4-rich brain region, while those without a D4 7 repeat allele showed abnormality of slow and variable response. The D4 7repeat allele is therefore believed to be associated with novelty seeking and perseverance, while the 10 repeat allele of the DAT1 gene is believed to be associated with increased reuptake of dopamine (Ding, Chi, Grady, Morishima, Kidd et al., 2002; Swanson et al., 2000).

Sagvolden & Sergeant, (1998), believe that ADHD has a genetic component associated with genes coding for receptors in the dopamine D2 family and membrane dopamine transporter (DAT) proteins.

The density of the dopamine transporter DAT was found to be higher in patients with ADHD. The elevated DAT density could be uniquely related to ADHD and provides an intriguing lead into the neurobiology of ADHD. Elevated dopamine transporter density may represent "hypertrophy" of dendritic trees or dopaminergic neurons due to inadequate pruning during neurodevelopment (Madras, Miller, & Fischman, 2002).

Too slow and too fast pruning has been hypothesized to be associated with psychopathology. Densities of dopamine receptors of D1 and D2 families may be expressed differently according to gender, a five fold overproduction and elimination of the D1 and D2 receptors was found in infant and adolescent male rats as compared to females, but settling on the same densities as adults. Males overproduced D1 but not D2 in the nucleus accumbens and retained the elevated densities in adulthood (Sagvolden & Sergeant, 1998).

The elevated dopamine transporter expression may reflect a state of ADHD which arise from neuroadaptive processes that compensate for increased or decreased dopamine neurotransmitter. Enhanced dopamine transporter may also arise from excess dopamine

production and release, diminished vesicular storage of dopamine, overactivity of D_1 –D_5 dopamine receptor subtypes or abnormal receptor-effector coupling. An association between the DRD 4 7 repeat allele and ADHD comorbid with CD (Holmes, Payton, Barrett, Harrington, McGuffin, Owen et al., 2002).

Behavioural genetic studies suggest that there is a genetic contribution to Conduct Disorder and adolescent antisocial behaviour. Most parents of children with ODD are themselves overly concerned with issues of power, control, and autonomy. CD is more common in children of parents with antisocial personality disorder and alcohol dependence than in the general population (Hicks et al., 2004).

Several studies have demonstrated higher concordance across MZ in comparison to DZ twin pairs. Concordance rates of ADHD have been found to range between 50 and 80% for MZ pairs and between 0 and 33% for DZ twins with heritability accounting for 30-40% of the variance in the symptom presentation (Bradley & Golden, 2001). More monozygotic twins showed higher levels of antisocial behaviour than dizygotic twins indicating that antisocial traits are heritable (Holmes et al., 2001). In twin studies concordance rate of antisocial behaviour was found to be more than twice as great among monozygotic twins as compared with dizygotic twins. Adoption studies also indicate that conduct disorder and criminality in offspring are more likely when the biological relative has shown these behaviours (Kazdin, 1995).

Sadock and Sadock, (2003) have reported that biological families of ADHD children have high rates of alcoholism, mood disorders, and antisocial personality disorders. Lahey et al. (1999) have also reported a higher incidence of antisocial disorders, depression, and substance abuse among relatives of ADHD+CD compared to ADHD only.

Relatives of the ADHD, ADHD+ODD, and ADHD+CD proband subgroups were found to be at greater risk for ADHD compared with relatives of a control group, and relatives of the ADHD subgroup were found to be significantly at greater risk for ODD compared to control subjects. No significant differences in the rates of ODD were found between relatives of ADHD+CD and ADHD+ODD subgroups and relatives of control. Relatives of CD probands were found to have increased rates of CD and the combined category of CD and or Antisocial Personality Disorder (ASPD). The difference in the rates of CD/ASPD between the ADD+ODD was not found to be significant. Furthermore relatives of ADHD+ODD and ADHD only probands did not differ in rates of ADHD, ODD, CD or ASPD. In contrast relatives of ADHD+CD probands were found to have higher rates of CD and CD/ASPD compared with ADHD only subgroup (Faraone, Biederman, & Monuteaux, 2000).

3.4 Pre-, peri- and postnatal factors

Conception at early maternal age, birth complications, use of forceps and brain damage are associated with greater incidence of ADHD (Taylor, Dopfner, Sergeant, Asherson, Banaschewski, Buitelaar, et al., 2004).

Brain development during the foetal period can be altered by chemical agents such as alcohol, nicotine and drugs, by mother's behaviour and health, and by the environmental effects on the mother (Quay, 1999). Exposure of a foetus to a toxic or diseased prenatal environment such as opiates, methadone, alcohol, marijuana, and cigarette by-products during pregnancy may lead to conduct problems. Both before and after birth lead poisoning can also lead to long term conduct problems in adolescence (Dodge & Pettit, 2003).

Chronic intake of dopamine agonists like cocaine, crack, and amphetamines will produce down regulation of dopamine synthesis (Holene, Nafstad, Skaare, & Sagvolden,

1998). Maternal smoking during pregnancy may lead to the development of behavioural and cognitive impairments in children. Nicotine is believed to cause foetal brain damage secondary to prolonged hypoxia, as well as through nicotine's hypothesised role in modulating the dopaminergic activity of the brain. Nicotine directly leads to cerebral pathology (via hypoxia), which manifests through symptoms of ADHD. Maternal smoking during pregnancy raises the risk of having a child with ADHD (Bradley & Golden, 2001).

Maternal smoking has been found to be significantly higher in ADHD children than normals, which supports the notion that maternal smoking during pregnancy raises the risk of having a child who develops ADHD. Nicotine is believed to cause foetal brain damage secondary to prolonged hypoxia, as well as nicotine hypothesized role in modulating dopaminergic activity of the brain leading to the development of behavioural and cognitive impairments in children that manifest through symptoms of ADHD (Milberger, Biederman, Faraone, & Jones, 1998).

Low birth weight has also been linked elevated rates of ADHD in children. Rates of ADHD in children of low birth weight was found to be significantly higher than in children of normal birth weight (Bhutta, Cleves, Casey, Cradock, & Anand, 2002; Breslau, Chilcoat, Johnson, Andreski, & Lucia, 2000; Saigal, Stoskopf, Streiner, & Burrows, 2001). Among children who were born with low birth weight, those that later developed ADHD were those who showed most extensive signs of hypoxia and ischaemia (Whitaker, Van Rossem, Feldman, Schonfeld, Pinto-Martin, Tore, et al.,1997). This has led to the hypothesis that early hypoxic-ischemic episodes through the high susceptibility of the spiny neurons of the striatum to hypoxic injury, and that this damage caused to the striatum leads to de-autoregulation of behaviour (Bradley & Golden, 2000).

Prenatal factors may affect ODD and CD, foetal exposure to alcohol and other drugs have been found to be correlated with the development of ADHD, CD and ODD.

Other factors that contribute to the development of CD include malnutrition, low birth weight, premature birth and potential exposure to toxins such as lead ingestion (Holmes et al., 2001).

3.5. Biopsychosocial factors

3.5.1 The Biopsyshosocial model

According to the biopsychosocial model of development of adolescent conduct problems as proposed by Dodge and Petit, (2003), the biological predisposition and sociocultural contexts place certain children at risk, and that life experience with parents, peers, and social institutions increase and mediate the risk. The emergence of chronic antisocial behaviour across time can best be explained by a transactional model. Reciprocal influences among the dispositions, contexts, and life experiences exacerbate or diminish antisocial development (Dodge, & Pettit, 2003). The model proposes that the predispositions are related to conduct-problems outcomes, but that the path from predisposition to outcomes may be indirect.

A biopsychosocial model of development posits that biological dispositions and sociocultural contexts place certain children at risk for chronic conduct problems in early life, and that life experiences with parents, peers, and social institution increment and mediate the risk. The emergence of chronic antisocial behaviour across time can be described by a transactional model. According to this model a non-linear relation exist between the individual and his or her interpersonal context which are correlated with each other and mediate each other to lead to conduct outcomes. Reciprocal influences among dispositions, contexts, and life experiences lead to recursive iterations across time that exacerbate or diminish antisocial development. Cognitive and emotional processes within the child including acquisition of knowledge and social information processing patterns mediate the relation between life experiences and conduct problems outcomes. The model

proposes that dispositions, contexts, and life experiences lead children to develop idiosyncratic social knowledge about their world which is represented in memory offering a link between past life experiences and future behavioural tendencies. Upon presentation of social stimulus, the child uses social knowledge to guide the processing of social information, and the child's pattern of processing social information leads directly to specific social or antisocial behaviours and mediates the effects of early life experiences on later chronic conduct problems (Dodge & Pettit, 2003).

3.5.2 Family factors

A relationship exists between the sociocultural environment in which a child is raised and the development of behaviour problems. Violence is more common in socioeconomically disadvantaged neighbourhoods than in suburban and rural settings. Environmental factors contributing to disruptive behaviours include impoverished households, high crime neighbourhoods, broken homes, household violence, and parental psychopathology (Lewis, 2004)

Family studies have consistently shown that the presence of substance dependence or antisocial behaviour disorder in first degree relatives greatly increases a person's risk of developing the disorder (Hicks et al., 2004). Family problems in the form of parental psychopathology, marital stress and other stressful life events are associated with ADHD. Elevated levels of parenting stress are associated with disruptions to the parent-child relationship and parenting practices, and disruptions in parent psychological functioning. Child externalising behaviour was found to be the major contributor to child domain parenting stress, while parents' locus of control and depressive symptomatology predicted parent domain parenting stress in mothers. Greater depressive symptomatology was associated with parental locus of control characterised by beliefs of reduced efficacy,

increased control of child over parents' lives, and decreased parental control over the child's life (Tripp, 2005).

The biopsychosocial model of development of conduct disorder of Dodge and Petit, (2003) proposes that early context of disadvantage place a child at probabilistic risk for later conduct problems and that as with predispositions the path is likely to be indirect. Neighbourhood level scores for the proportions of families of characterised by poverty, unemployment, marital divorce, low education, single parent households, high residential mobility, and low income represent a significant risk factors for individual conduct problems (Dodge & Pettit, 2003).

Bradley and Corwyn (2002) found that at family level, socioeconomic status at birth indexed by income, occupation, and education of parents, is one of the strongest and most consistent of all risk factors for later conduct problems, throughout childhood and adolescent years. Early life experiences that involve harsh treatment and inconsistent discipline, rejection of the self and failure place a child at risk for conduct problems. Other risk factors include inadequate parenting, the physical harshness of discipline, lack of warmth between parent and child, the amount of exposure the child has to aggressive peers in day care and preschool, social rejection by peers, exposure to violence on television is predictive of later child aggressive behaviour.

Comorbid association of ADHD with ODD and CD is linked to greater degrees of parenting stress, parental psychopathology, marital discord and divorce. Dixon, Howie, and Starling, (2004) found that the most significant factor associated with female juvenile offender status was the number of psychological diagnosis, with the presence of two or more disorders posing the highest risk. Exposure to two or three traumatic events also increased the likelihood of being an offender. Other factors that were found include that the offenders tend to be more likely to have been homeless, living independently, in a

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residential or foster care setting or with extended family. DBD in children is associated with parental substance abuse. According to McGee and Williams (1999) family climate and child interaction with parents was found to be predictive of both CD and ODD and may operate as a general risk for disruptive and antisocial behaviour.

Childhood aggression, lack of social skills, and social withdrawal can contribute to peer rejection which when combined with academic failure and poor parental monitoring can lead to deviant behaviour (Holmes et al., 2001).

3.5.3 Environmental factors and toxins

Some environmental pollutants may also alter the functioning of neurotransmiters, as may certain drugs. Although environmental factors have been shown to influence the development of DBDs, the main influence of the environment is on the biology of the individual. It is therefore the environmental influence on the biological elements of a person's functioning, and not the factors in themselves that contributes to the development of DBDs, environmental factors can thus be seen as secondary causative factors (Barkley, 2006).

Environmental pollutants that may also cause dopamine hypofunctioning include polychlorinated biphenyls (PCBs) which is a group of halogenated aromatic hydrocarbons with a variety of industrial uses such as in paints, lubricants and in dielectric and heat-exchange fluids in transformers and heat exchangers. Because PCBs are lipophilic, they make the brain vulnerable. Intake of these pollutants will cause developmental abnormalities in humans including low birth weight, disruptive behaviour and overactivity. Studies of effects of PCB exposure on behaviour and brain chemistry showed that male rats exposed to sub-toxic doses of the PCB congener 153 through mother's milk when pups were hyperactive and impulsive when they grow up (Holene et al., 1998).

3.6 Cultural factors

Cultural factors include the school, teachers' educational approach, parenting styles, family structure, and societal expectation regarding productivity and leisure. Some literature suggests that cultural factors may modulate the clinical manifestation of DBDs (Reid, Riccio, Kessler, DuPaul, Power, Anastopoulos, et al., 2000; Reid, DuPaul, Power, Anastopoulos, Rogers-Adkinson, et al., 1998). Cultural factors influence the aetiology, expression, course, outcome and epidemiology of childhood mental disorders. In adult psychiatry, cross cultural comparisons of major psychiatric disorders have indicated differences in symptom presentation (Sartorius, 1986). Consequently differences in the presentation of symptoms of DBDs across different cultures can be expected.

Meyer et al. (Meyer, Eilertsen, Sundet, Tshifularo, &Sagvolden, 2004; Meyer, 2005; Meyer & Sagvolden, (2006a) state that the influence of culture on behaviour disturbance has a long history in psychiatry, and the question is, to what extent is culture contributing to behaviour disturbance? Cultural norms and rules will modify how the disorder is manifested, making it essential that ethnic, cultural, and language factors be taken into account in considering the manifestation, diagnosis and treatment of childhood disorders. Although prevalence and sex ratios of ADHD in Limpopo, South Africa were found to be similar to those reported in Western countries (Meyer et al., 2004) suggesting that ADHD is caused by the same fundamental neurobiological processes, which may be caused by genetic factors independently of cultural differences, cultural differences, however were found to affect performance on neuropsychological measures. The reason may be that cultural factors are important determinants of child rearing practices which affect the brain's organisation of cognition (Meyer, 2005a). Taylor (1998) reports that culturally related differences may be explicable in terms of one or more of the children's behaviour, adult perception, referral practices, and diagnostic criteria used.

According to Barkley (2006) variations in the prevalence of DBD occur across different socioeconomic status and that in early and middle childhood, the care giving environment is the more powerful predictor of behaviour than early biological factors.

3.7 Conclusion

A number of theories about the aetiology of DBDs have been developed. Factors that have been identified in the development of DBDs include neurological, genetic (hereditary), environmental factors and psychosocial factors (Brown, 2000).

The combinations of neuropsychiatric and cognitive vulnerabilities with an upbringing in a violent, abusive household have been found to be highly correlated with violent criminality (Hendren & Mullen, 2003). Ongoing research is needed to refine these theories, because an improved understanding of the aetiology of DBDs will contribute to more improved approaches in the quest for treatment of DBDs.

C h a p t e r   4

DIAGNOSIS AND TREATMENT

4.1 Introduction

Assessment of DBDs must be accurate to provide for effective treatment. It should span multiple domains, and multiple informants should contribute to the process. Categorically and/or dimensionally culturally validated assessment tools should be used. The possibility of sensory receptive disorders such as hearing and visual impairment, underlying physical illnesses, developmental delays, and substance abuse should be investigated. It is also crucial to determine whether there are comorbid conditions or complicating health conditions for referral to relevant specialists (Kutcher, Aman, Brooks, Buitelaar, van Daalen, Fegert et al., 2004). The diagnosis of DBDs is difficult. The category of Conduct Disorder is so broad that it includes different kinds of behaviours. Other psychiatric conditions of childhood disorders can manifest themselves in disruptive and aggressive behaviours.

Like with other psychiatric disorders, there is no single diagnostic test for Disruptive Behaviour Disorders. The diagnosis is based on careful elicitation and integration of reports by parents and teachers of past and current development and behaviour, along with systematic exclusion of other conditions that may present with other features, such as learning disorders, anxiety, depression, and post traumatic stress disorder (Solanto, 1998).

The diagnosis of DBDs is based on clinical history, and not based on psychological nor biological tests that are presently recommended for clinical use. However psychological

and biological tests are used by researchers to investigate the links between symptoms and underlying cognitive processes (Swanson et al., 1998b).

Assessment can be seen as a basis from which the diagnostic and treatment procedures can be organised. Inaccurate assessments will have an impact on diagnosis and treatment planning. A thorough clinical interview, medical tests and behaviour rating scales are imperative in the diagnostic process. Diagnosis of DBDs requires both medical and psychosocial expertise (Meyer & Aase, 2003).

Symptom descriptions may be obtained from parents and teachers in open ended clinical interviews or structured interviews such as the Comprehensive Diagnostic Interview Schedule for Children and Adolescent (DISC) (Shaffer, Schwab-Stone, Fisher, Cohen, Piacentini, Davies, et al., 1993) and age-and gender-normed questionnaires such the as Conners Parent and Teacher Rating Scales (Conners, 1998) and the Child Behaviour Checklist (Achenbach, 1991). The rating scale used in this study is the Disruptive Behaviour Disorder Scale developed by Pelham (Pelham, Jr., Gnagy, Greenslade, & Milich, 1992; Pillow, Pelham, Jr., Hoza, Molina, & Stultz, 1998) and translated and normed for the populations of the Limpopo Province by Meyer (Meyer et al., 2004). It contains 42 items of Disruptive behaviour disorders from the DSM-IV-TR.

4.2 Cultural influences on behaviour and assessment

Culturally different individuals are more likely to be exposed to different prenatal risk factors, psychosocial stressors, and socio-economic status which in turn will affect educational and behavioural outcomes. Commonly used available assessment instruments could be misleading and invalid when used with culturally different populations (Reid et al., 1998).

Cultural influences in the diagnosis of hyperactivity and consequently DBDs appear to be very strong. Some of the geographic variations in the diagnosis of DBDs may

derive from different practices and different criteria in case identification. The standards of the recognition of the disorder in different populations can be different. In some cultures adults may have an intolerance of relatively minor degrees of disruptive behaviour and as a result may be more ready to identify children with DBDs. Deviance is socially constructed, that which is acceptable or diagnosed in one culture may not be so in another culture (Taylor, 1998).

The multidimensional nature of DBDs, makes the process of assessment even more complicated than in most other psychiatric disorders. Multiple aetiologies, differences in clinical presentations and the many comorbid conditions can impact on results gained from assessment.

4.3 Culturally sensitive assessment

The need to serve children from diverse cultures calls for the use of standardised procedures that have proven applicability to multiple cultures (Crijnen, Achenbach, & Verhulst, 1999).

Most assessment instruments used in clinical psychiatry have been developed, tested and applied in Western countries. Rating scales are therefore culturally biased and most of them, to be applicable for use outside the Western countries have to be translated into languages of the local populations for which they are intended. Research is ongoing to investigate the applicability of assessment methods and interventions in other parts of the world especially in developing countries (Meyer & Aase, 2003).

Assessment instruments used in one culture could be misleading or invalid when used with children from different cultures (Reid et al., 1998). The validity and reliability of assessment instruments can be influenced by a variety of factors including culture. Assessment instruments should therefore in all cases be valid and reliable in the cultural context within which they are being administered. Although ADHD (like other DBDs) is

expected to be present all over the world, cultural norms and rules will modify how the disorder is manifested. It is therefore essential that the ethnic, cultural and language factors be taken into account in considering the development, manifestation, diagnosis and treatment (Meyer & Aase, 2003). In evaluating children with DBDs it may be necessary to draw on measures that are normed for the child's ethnic background if such instruments are available so as to preclude cultural bias (Barkley, 1997a)

Assessment and diagnosis should not only be aimed at the determination and of the presence or absence of psychiatric disorders, such as ADHD, ODD, or CD but also differential diagnosis from other childhood psychiatric disorders (Meyer, 1999).

Parental checklists and accounts from teachers are valuable in detecting the symptoms. Assessment should comprise the following; a clinical interview with the parents, and separately with the child, obtaining school information, IQ testing, behavioural observations and physical evaluation. A physical examination should be done to make sure that there is no underlying illness (Meyer, 1999).

4.4 The clinical interview

Although parent and child interviews are often controversial because of reliability issues, they are recommended as they produce a wealth of information. As the diagnosis of DBDs is based on the historical information of the child, the parent is the person who knows the child's history better than anyone (Barkley, 2006).

Clinical interviews may provide a rich source of information, but may not systematically cover every area necessary for diagnosis of a given psychiatric disorder. Clinical information may be increased by making use of semi structured or structured interviews (Sadock & Sadock, 2003).

Standardised interviews for the assessment of child and adolescent psychopathology include the Diagnostic Interview Schedule for Children which has two versions, DISC-P the parent version, and DISC-C the child version. The Diagnostic Interview Schedule for Children is based on the DSM-IV and as a result permits the clinician to make accurate diagnoses of ADHD, ODD and CD (Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000).

4.4.1 Interview with the parent

The clinical interview with the parent should clarify presenting problems, and make a systematic evaluation of psychopathological symptoms. It must include developmental history and previous medical and professional reports, an adequate account of affected family members, pregnancy and birth history, and early developmental history. Parent rating scales are useful as a supplement to the interview, not as a replacement. Although these scales are only opinions, and subject to oversights, prejudices, and limitations on reliability, they provide a means of quantifying the opinions of parents and often along qualitative dimensions and of comparing the scores to norms collected on large groups of children (Barkley & Murphy, 2006).

4.4.2 Interview with the child

The duration of the interview with the child depends on the child's age, intellectual level, and language abilities. Self report rating scales may be useful as a supplement to interview as children are often not reliable in their reports of their own behaviour which may be due to diminished self awareness and underreporting of the seriousness of their behaviour (Hinshaw, 1994)

According to the diagnostic criteria for ADHD specified in the ICD-10 (World Health Organization, 1993) and DSM-IV-TR (American Psychiatric Association, 2000), the clinician must document specific and impairing symptoms of the disorder in at least two

settings. The sources of information are typically parent and teachers, who provide information about the child's functioning at school and home setting.

4.5 Medical examination

A general examination including assessment of physical health is needed. The possibility of sensory receptive disorders such as hearing and visual impairments, underlying physical illnesses and developmental delays should be investigated (Kutcher et al., 2004). The physical examination should also look for any evidence of neurodevelopmental immaturity. Although results from a physical examination may not be used in isolation to diagnose DBDs, a thorough clinical examination may provide a useful detailed clinical picture. Medical interview is aimed at differentiating between the diagnosis of DBDs from other medical conditions, a thorough evaluation of any coexisting conditions that may require medical management, and to determine whether physical conditions that may be present are contra-indications for treatment with medication (Barkley, 2006).

4.6 Behaviour rating scales

A rating scale is any instrument that provides rapid assessment of behaviour or psychological dimension, yielding a numerical score that is easily interpreted to complement clinical care. Rating scales provide systematic coverage and quantification of behaviours for comparison of youths with self and peers over time, setting, and context (Myers & Collett, 2004).

One of the primary and most efficient ways to collect information from parents and teachers is with behaviour rating scales (Swanson, Lerner, March, & Gresham, 1999). Parent-teacher discrepancies sometimes occur. Some of the reasons for discrepancies that the clinician should consider can be a true difference in the manifestation of symptoms at home and at school (situational specificity of behaviour), different standards for behaviour

at home and school (situational tolerance of certain behaviours), and different task demands at home and school (situational requirement for academic work. The clinician should also be aware of some common biases in parent and teacher rating, such as "halo" effects (the presence of a salient behaviour may impact ratings of other behaviours) and measurement errors (Swanson et al., 1999).

Available rating scales with excellent psychometric properties for the assessment of DBDs include the Disruptive Behaviour Disorder (DBD) rating scale (Pelham, Jr. et al., 1992); Child Behaviour Checklist (CBCL) (Achenbach, 1991); the Conners Rating scales (Conners, 1998). The following are types of behaviour rating scales:

4.6.1 Parent, Teacher, and Self reports

Multiple informants (parents, teachers and children themselves) can contribute towards the identification and understanding of children's behavioural and emotional adjustment (Kutcher et al., 2004) . For younger and developmentally delayed children who may lack the cognitive and linguistic skills to verbalise their feelings and thoughts, or those who lack reading skills and persistence to complete a rating scale, teacher and parent reports are of vital importance. Self report is not suitable for younger children, but for older children and adolescents especially when concerns are about feelings and behaviours that are difficult to observe directly they are a good source of information (Wingenfield & Peabody, 2002).

Parents rating scales should not be seen as a substitute for, but as a supplement to the interview. It must however be remembered that the scales are opinions and are subject to oversights, prejudices, and limitations on reliability and validity (Taylor et al., 2004). Multiple domains (school, home and community) and multiple informants should be part of the diagnostic procedure (Kutcher et al., 2004). Parents are knowledgeable about the child's development, relationships with family and peers, and behaviours in a range of

diverse settings, while teachers spend a considerable amount of time of the day with children, observing them in both structured (highly structured , task oriented classroom setting) and unstructured situations during recess and play with peers. However teacher reports at secondary school may be less accurate as a result of reduced contact with the children, and youth self reports may be the most appropriate indicator of the learner's feelings and behaviours (Wingenfield & Peabody, 2002).

Information across informants may be different due to among others the following reasons:

- Differences in child's behaviour across settings given the differences in expectations, structure, and parent/teacher skill in behaviour management at home and at school.

- Parental reports on children behaviour may underestimate problem behaviour when children are gaining independence and likely spending significant amount of time away from home (Gelhorn, Stallings, Young, Corley, Rhee, & Hewitt, 2005).

- The differences in scales used in assessment, the scales may vary in terms of item construction, and in the accuracy and description of the target behaviour. Diagnostic criteria are often based on symptom count and arbitrary cut-offs that may vary as new editions of the diagnostic criteria are developed (Gelhorn et al., 2005).

- Differences in respondents' perceptions, parents and teachers may rate behaviour as more problematic than parents, fathers and mothers may rate behaviour differently. Though not supported by research, overworked mothers and teachers may have more negative views of students, and teacher experience with behaviour management or familiarity with the student influence rating. Response sets and

response styles, lack of motivation or compliance may lead to an inconsistent and random response style, and informants may not want to admit to their mental health status or may exaggerate the true nature of their problem (Wingenfield & Peabody, 2002). Parents and teachers from different cultures may have thresholds for reporting particular kinds of problems (Crijnen et al., 1999).

4.6.2 Unidimentional and multidimensional rating scales

Unidimensional scales are disorder-specific and have been developed for a wide range of areas. Measures of these scales may vary in their conceptual bases, breadth, specificity to the problem, and psychometric properties (Barkley, 2006). Parent and teacher versions are only available for externalizing behaviours, while those scales for assessing internalizing problems such as depression or anxiety rely on self report by the child or adolescent (Wingenfield & Peabody, 2002).

Multidimensional scales may contain either specific or broad areas of adjustment. They allow screening for the presence or absence of specific problem and of comorbid conditions (Wingenfield & Peabody, 2002).

4.7 The use of Psychometric tests

Although psychometric tests may have not been standardised for individual diagnosis, they can be helpful in giving clues to the nature of the problem in an individual case.

IQ tests and tests of reading and other academic achievement must be considered when there is any problem related to classroom adjustment or progress. Speech and language tests may be indicated when there is evidence of difficulty in communication (Meyer, 1999; Taylor et al., 2004). Tests used may be biased if they are used for populations to which it has not been standardised. Most tests that have been developed in Western countries need to be translated into African languages for use in African countries. Non-

verbal tests with a minimum of instructions are more appropriate to counter the bias related to cultural differences and problems associated with the use of foreign language. Symptoms of ADHD include deficient sustained attention, motor and cognitive impulsiveness. Research evidence indicates that cognitive difficulties are also observed in ODD and CD children (Sergeant, 2000). Tests that are used in this study, the Tower of London (Krikorian, Bartok, & Gay, 1994), Memory for Digits (SSAIS-R) (Wechsler, 1991; Van Eeden, 1997), Maze Coordination Task (Matthews & Kløve, 1964), and Blocks Design (Wechsler, 1991; Van Eeden, 1997) may load on symptoms of DBDs (Badenhorst, 2003; Barkley, 2006; Lezak, Howieson, & Loring, 2004) and do not rely much on verbal or linguistic abilities. These tests will require a minimum of instructions and their choice will minimise effects of linguistic abilities and cultural bias.

The diagnostic process consists of an integration of data from various sources when reliance on one source is considered insufficient. Although none of the psychometric instruments are indicated as a basis on which to make a diagnosis, they can be used to differentiate between possible co-morbid disorders, or to indicate the level of impairment (Barkley, 2006). Psychometric instruments that have been used in the assessment of children with DBDs (executive functions, memory deficits, and inattention and motor problems) include intelligence tests, general neuropsychological batteries, individual neuropsychological instruments and projective tests (Barkley, 2006; Brown, 2000).

4.8 Treatment of DBDs

4.8.1 Introduction

The high incidence of, and significant psychiatric comorbidity calls for the opportunity of intervention to decrease psychological distress. Comprehensive assessment and treatment of all symptoms should be a critical consideration to intervention efforts (Dodge & Pettit, 2003). Comorbid conditions complicate the diagnostic process, influence

the type of treatment to be offered, and have a significant impact on the efficacy of treatment. Comorbidity needs to be recognised and addressed to improve the identification and management of DBDs and to improve outcome (Kutcher et al., 2004).

Given that cultural norms and rules will modify how DBDs are manifested, it is essential that ethnic, cultural and language factors be taken into account in considering the manifestation, diagnosis and treatment of these disorders (Meyer et al., 2004). Since DBDs are associated with compromised social relations with parents and peers, and impairment in school and academic performance, treatment should be applied in those different settings. Children who experience neurochemical abnormalities, such as decreased levels of central dopamine and serotonin may present with hyperactivity, aggression, depression and inattention (Holmes et al., 2001). The fact that ADHD usually occurs in early years of primary school children and may in later years develop into ODD and CD implies that treatment strategies should focus rather on early prevention of behavioural problems in preschool children than on later treatment strategies. Early recognition and intervention are imperative, ADHD and childhood emotional disorders such as depression often respond to psychotherapy and pharmacological intervention (Holmes et al., 2001). The focus of treatment for DBDs is not all symptoms, but target symptoms. Target symptoms for ADHD are the core symptoms of hyperactivity, impulsiveness, and inattention, while CD symptoms will include various forms of physical aggression, destruction of property, rule violations and socialisation. Pharmacological treatment of pure ODD should not be considered except where aggression is a significant and persistent problem. In comorbid cases of DBDs the target symptoms will include hyperactivity, impulsivity, inattention, socialisation and aggression (Kutcher et al., 2004).

4.8.2 Pharmacological agents

The concern that treatment of ADHD with medication could lead to drug addiction in later life is not supported by research. There are also conflicting research results stating that stimulating treatment suppress growth of children, implying that clinicians should monitor the growth of hyperactive children receiving stimulants, and to consider dose reductions in individual cases should evidence of growth suppression occurs (Harpin, 2005).

Psychostimulants in terms of efficacy outrank all other classes of medication in the treatment of ADHD. Psychostimulants have been shown to improve the core symptoms of ADHD (inattention, hyperactivity, and impulsivity), and to improve oppositional behaviour, impulsive aggression and social interactions (Kutcher, et al., 2004). Psychostimulant drugs such as D-amphetamine (Dexedrine®), Methylphenidate (Ritalin®, Concerta®) and D-,L-ampetamine (Adderral®) have been shown to significantly ameliorate symptoms of ADHD that is, inattentiveness, hyperactivity, and impulsivity. Antipsychotics have reportedly helped children control aggressive and assaultive behaviours. To this effect newer antipsychotic drugs such as risperidone (Risperda®l), and olanzapine (Zypresxa®) have replaced haloperidol because they carry a lower risk of extrapyrimidal symptoms including acute dystonic reaction and parkinsonism (Kutcher et al., 2004; Sadock & Sadock, 2003). Other drugs that have been found to be useful in the treatment of aggressive behaviour include Lithium (Eskalith®), carbamazepine (Tegretol®) and clonodine (Catapres®). Selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine (Prozac®), setraline (Zoloft®), paroxetine (Paxil®), and citalopram (Celexa®) have been used to attempt to diminish impulsivity, irritability, and lability of mood which often occur with Conduct Disorder (Sadock & Sadock, 2003). While chlorpromazine causes significantly problematic sedation even at low doses, Lithium is associated with adverse side effects including thyroid stimulated hormone elevations, hypothyroidism,

polydisia and polyuria, and may be lethal in overdose. Pemoline is not recommended due to its association with hepatotoxicity. Common side effects of stimulants include loss of appetite, insomnia, headache and stomach ache, appetite suppression, and motor tics. Abrupt cessation of psychostimulant medication may lead to withdrawal reactions including increased activity, excitability, irritability and insomnia. Depression and fatigue may occur at a later stage. To alleviate the significant depressive symptoms, addition of antidepressants may be helpful. To minimise the adverse effects of medication and if medication is to be discontinued, doses should be reduced gradually at approximately 25% per week (Kutcher et al, 2004).

Atomoxetine (Strattera®), a non-stimulation agent is a highly selective presynaptic inhibitor of the norepinephrine transporter with virtually no affinity for other transporters or neurotransmitter receptor sites. It increases dopamine and norepinephrine in the prefrontal cortex, similarly to methylphenidate although unlike the latter it does not increase dopamine in the striatum or the nucleus accumbens, and it therefore carries a minimal risk for substance abuse. Atomoxetine was found to produce a statistically significant and clinically meaningful improvement in ADHD and ODD symptoms among children and adolescents (Newcorn, Spencer, Biederman, Milton, & Michelson, 2005).

The most beneficial effect of stimulant therapy is the rapid improvement in the behavioural manifestation such as restlessness, inattentiveness and impulsiveness. The behavioural effects of stimulants are limited to 1 – 4 hr of pharmacological activity; there is no evidence of any carryover effect to periods in which the medication is not pharmacologically active. Methylphenidate (MPH) is rapidly absorbed and easily crosses the blood-brain barrier. Tolerance to the effect of MPH may develop in some children over months and years of continuous therapy (Schachar & Ickowicz, 1999).

Despite considerable debate about the possible impact of stimulants on cognition, such as a decrease in the flexibility of problem solving, an increase in perseveration of inappropriate responses, an overfocus on limited aspects of available information, and an inability to think divergently and creatively, MPH generally has a beneficial effect on many of cognitive processes that are deficient in children with ADHD. MPH decreases reaction time, decreases response variability, improves accuracy of performance, facilitates detection and correction of errors, improves the ability to focus on the most relevant aspects of information, and decreases impulsive responding (Schachar & Ickowicz, 1999).

A greater proportion of medicated children exhibit clinically significant improvement in classroom behaviour and attention to tasks and academic efficiency (Rapport, Denney, DuPaul, & Gardner, 1994)

Children with DBDs are often disruptive, interfering, intrusive, abrupt, impatient, and both verbally and physically aggressive. They are more socially busy, critical and overbearing, less attuned to the social agenda of their peers, less attentive to subtle social cues and procedures for entering a peer group, and they have a tendency to attribute hostile motives to others. Treatment with stimulants results in a substantial and immediate improvement in the quality of the children's social interactions with their peers, parents and teachers. Treatment reduces the frequency of negative verbalisations, teasing and swearing and of conduct problems such as physical aggression, lying, stealing and destruction of property (Lewis, 2004).

4.8.3 Psychosocial interventions

Medical treatment alone is often not enough to satisfy the comprehensive therapeutic needs of children. The most appropriate form of treatment should be selected for each case individually following through diagnostic assessment and taking into account the capacities of parents, educational system, and culture of the child (Mandel, 1997).

Parents' educational programs designed to emphasise the necessity of parental support, supervision and involvement could help alter the child's psychosocial environment (Holmes, et al., 2001; Meyer, 2005b). Social coaching and advice giving appear to be especially important in young children's initial social encounters with peers and forms of guidance in ways of handling socially challenging situations especially in families with older children and adolescents (Dodge & Pettit, 2003). Parents' proactive teaching of social skills in the early childhood years predicts lower levels of behavioural problems in middle childhood and early adolescent. A heightened tendency among parents is to engage in effective monitoring and supervision in the later adolescent years (Dodge & Pettit, 2003).

Social skills groups training for parents of children with DBDs, and behavioural interventions at school and at home have also been found to be effective especially when used with medication. In parent management training, parents are taught ways of changing dysfunctional parent child interaction and social learning principles to implement (Mandel, 1997; Meyer, 2005b). Special tutoring and social skills training including anger management training may alleviate academic underachievement and peer rejection that could lead to permanent deviance (Holmes et al., 2001). Parent management training teaches the parents techniques for interacting with their offspring to encourage appropriate behaviour and diminish oppositional, aggressive behaviours (Meyer, 2005b). Therapists teach parents methods for positive reinforcement of children's desirable behaviours, the use of mild non-physical punishment, and communication techniques for negotiation with their offspring. This form of treatment may have its limitation in that mostly behaviourally disturbed children come from the most chaotic households in which parents will have the great difficulty cooperating with treatment (Lewis, 2004)

Social skills training focuses on getting the child or the adolescent to think about how to approach any potentially problematic situation. They are taught specific steps in

constructive problem solving and are provided structured situations in which to practice these principles. Skills targeted in social skills training include carrying on constructive conversations, receiving and sending complaints in a non-destructive manner, dealing with anger more effectively, and dealing productively with authority figures (Kazdin, 1995).

Cognitive problem-solving skills training is based on the premise that children with behaviour problems do not know how to make use of their potential cognitive abilities to identify problems, anticipate consequences, and consider alternative ways of understanding and coping with difficult situations (Lewis, 2004). Cognitive skills training is designed to get the child to examine his or her thought processes, Children are taught principles and techniques of behavioural analysis including self monitoring, self evaluation and self reinforcement (Mandel, 1977). Cognitive problem skills training have been less successful with children who have other neuropsychiatric disorders in addition to CD (Lewis, 2004). Cognitive behaviour therapy (which aims to alter negative and inefficient self instruction and to improve problem-solving skills), and multisystemic therapy (a family based therapy integrating components of parent training, marital and cognitive therapy) were found to have some efficacy in treating ODD and CD (Kutcher et al., 2004).

According to Barkley (1997a) the goals of parent and teacher programs include (1) to improve parental (and teacher) management skills and competence in dealing with child behaviour problems, (2) to increase parent and teacher knowledge of the cause of childhood defiant behaviours, (3) to improve the child's compliance with commands, directives and rules given by parents and teachers, and (4) to increase family harmony through improvement of parental and teacher use of positive attention,   the provision of clear guidelines and rules, the application of swift, fair, and just discipline for inappropriate child's behaviour and general reliance on principle guided behaviour.

Non-pharmaceutical therapies such as individual therapy, behaviour modification techniques, parenting classes, parent support groups, school involvement, and education about DBDs are recommended in addition to the use of medications. In several behaviour modification programs, reinforcement has proven to be highly effective in the treatment of ADHD. Reinforcement contingencies are found to normalise behaviour that characterise ADHD in school, sports, home settings and to improve academic functioning (Luman et al., 2005).

Children who are prescribed medications should be told the purpose of the medication and be given the opportunity to say how they feel about it in order to dispel misconceptions about medication use. Parents should be helped to recognise that in spite of their child's difficulties, all children face the normal tasks of maturation, including significant building of self-esteem when a sense of mastery is developed, which therefore implies that these children should not be exempted from the requirements, expectations, and planning applicable to normal children. Group therapy is aimed at refining social skills and increasing self-esteem, a sense of success may be useful for children who have great difficulty functioning in group setting. An environmental structure that provides support, along with consistent rules and expected consequences can help control a variety of problem behaviours. Parents should be alerted to the hazard of parental substance abuse, child neglect, and violence may help alter the child's environment (Holmes et al., 2001).

Malnutrition predisposes to neurocognitive deficits, which in turn predispose to persistent externalising behaviour problems throughout childhood and adolescence. Malnutrition is believed to negatively affect brain growth and development and that brain impairments predispose to antisocial and violent behaviour by affecting cognitive functioning. Reducing early malnutrition will therefore help reduce later antisocial and aggressive behaviour (Liu, Raine, Venables, & Mednick, 2004). In some cases the structure

can be applied to family life, so that parents become aware of behavioural techniques and grow proficient at using them to foster appropriate behaviours. Individual psychotherapy oriented toward improving problem-solving skills can be useful, as children with Conduct Disorder may have a longstanding pattern of maladaptive responses to daily situations (Sadock & Sadock, 2003).

4.9 Prognosis

Symptoms of ADHD may persist into adolescence or adult life, or remit at puberty. Symptoms persist into adulthood in 15 to 20 percent of cases and may cause disruptions to both personal and professional life of the patient (Harpin, 2005). Those children whose symptoms persist into adolescence are at risk of developing CD, while those that have both ADHD and CD are at risk of developing a substance related disorder. Adults with ADHD are more likely to experience interpersonal difficulties with employers and colleagues. At family level relationship difficulties and break-ups are more common. The outcome of ADHD in childhood is related to the amount of persistent comorbid psychopathology especially CD, social disability and chaotic family factors (Harpin, 2005; Sadock & Sadock, 2003).

In children with ADHD the presence of comorbid CD predicts poor social adjustment in later development, while on the other hand the presence and severity of hyperactivity in children with CD predicts higher levels of antisocial outcomes in adolescence. For a child with ADHD to develop into an adolescent with comorbid CD appears to be modulated by environmental factors such as high levels of expressed criticism from family members and rejection by peers. If children with ADHD can be identified early, early interventions may preclude the development of CD and minimise the developmental risk conferred by ADHD (Kutcher et al., 2004).

About a quarter of all children who receive the diagnosis of ODD do not continue to meet the diagnostic criteria over the next several years. Persistence of ODD symptoms depends largely on the severity of symptoms and leads to the development of CD and substance use disorders. The prognosis of ODD children depends to some extent on family functioning and the development of comorbid psychopathology. Pharmacological treatment is not recommended for cases of pure ODD (without comorbidity), but psychosocial intervention may be necessary unless severe aggression or destructive behaviour persists despite psychological intervention (Kutcher et al., 2004; Sadock & Sadock, 2003).

Treatment programs have been more successful in decreasing overt symptoms of CD than the covert symptoms. Prognosis for children with CD is poor on those who have symptoms at a young age, exhibit the greatest number of symptoms, and express them more frequently. Given that most adolescents with severe Conduct Disorder have a multitude of other kinds of neuropsychiatric and cognitive vulnerabilities, the comorbid disorders have a better prognosis than Conduct Disorder (Lewis, 2004). A good prognosis may be expected for mild CD in the absence of coexisting psychopathology, and the presence of normal intellectual functioning.

4.10 Conclusion

The prevalence rate of DBDs is found to be comparable across different cultures and countries, the neuropsychological factors that result from studies of DBD children and the aetiology of DBDs suggest that treatment of DBDs needs to begin early in the life of the primary school child. Treatment could be both pharmacological and psychosocial. Comorbid conditions complicate the diagnostic process, influence types of treatment offered, and have significant effect on efficacy of that treatment (Kutcher et al, 2004). It is

clear from the discussion that prognosis in the treatment of DBDs depends on the severity

and the presence of other comorbid psychiatric conditions.

PROBLEM STATEMENT

5.1 Introduction

Problems in preschool children such as excesses in aggression/restlessness and motor and cognitive deficits strongly predict persistent delinquent behaviour (Bor & Sanders, 2004). Children with ADHD exhibit deficits in their performance on learning, colour, and position discrimination, and short term memory tasks (Paule, Rowland, Ferguson, Chelonis, Tannock, Swanson, & Castellanos, 2000). Deficits in response inhibition were found in CD and ODD indicating that they are not unique to ADHD (Paule et al., 2000).

ADHD is associated with deficits on a variety of neuropsychological measures (Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003). Impulse control dysfunction and the presence of hyperactivity and inattention are the most highly related predisposing factors for the presentation of antisocial behaviour. ADHD can contribute greatly to problematic behaviour and antisocial acts, as these children have difficulty analysing and anticipating consequences and learning from past behaviour (Holmes et al., 2001).

Although there is evidence for the hypothesis that EF deficits play a role in the aetiology of ADHD (Willcutt, DeFries, Pennington, Olson, & Cardon, 2003), many deficits are shared with other disorders, and that some differences with other disorders may be quantitative rather than qualitative (Banaschewski et al., 2005). According to research evidence, deficiency in executive functioning is not specific to ADHD, but also applies to children with both ADHD and CD (Sergeant, 2000). Children with both ADHD and CD often display a mixture of cognitive, attention, and inhibitory deficits (Quay, 1999).

Executive Functions (EF) encompass meta-cognitive processes that enable efficient planning, execution, verification and regulation of goal directed behaviour. Evidence of EF impairment is considered stronger for ADHD than for ODD/CD, and ODD was found to be associated with superior performance on the Tower of Hanoi (ToH) from which the Tower of London (ToL) is adapted. The performance of ODD/CD and ADHD+ODD/CD was found to fall midway between performance of children with ADHD and normal control children suggesting that impairment in planning in terms of hasty decision making appears to be associated with ADHD, but not with ODD/CD nor with comorbid ADHD+ODD/CD (Oosterlaan, Scheres, & Sergeant, 2005).

Deficits in components of executive functions particularly those associated with control of motor responses (planning, preparation, execution, inhibition) and working memory have been found. Motor planning deficits are observed by consistent poor performance on neuropsychological tasks such as the Tower of Hanoi, and Porteus Mazes or WISC-mazes (Paule et al., 2000).

The construct validity of the various psychological processes implicated in ADHD (attention, response inhibition, working memory, executive function and timing) and the validity of tasks used to measure these processes needs to be investigated. Distinct neuropsychological constructs may partly rely on the same interconnected neuronal circuits, and similar neuropsychological processes may rely on different neuronal structures (Banaschewski et al., 2005).

Working memory deficits are reflected by poor performance on tests such as the backward digit span (Paule et al., 2000). Working memory is critical to conscious thought; it permits internal representation of information or rules to guide decision making and overt behaviour or responses during activity so that behaviour is not dominated by the

sensory cues in the environment. Working memory functions are believed to be highly dependent on frontostriatal brain regions. Working memory processes were found to be impaired in children with ADHD especially with spatial storage as opposed to verbal storage of information. Poor academic achievement of ADHD children may be the result of working memory deficiencies rather than a direct consequence of behavioural symptoms of inattention and or hyperactivity-impulsivity (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005).

5.2 The focus of the current research study

The focus of this research is to test whether there are differences in performance on cognitive and neuropsychological tests between children with DBDs and a non-DBD comparison groups as a function of gender and DBD subtype.

5.3 Major research questions and hypotheses

5.3.1 Research questions
1. Do children with DBDs show deficiencies in executive, cognitive and motor functions?
2. Do gender and subtype influence executive, cognitive and motor functioning in children with DBDs ?

5.3.2 Hypotheses

Research hypothesis:

Children with DBDs will have lower scores on tests that measure executive and cognitive impairments and motor functions than non-DBD children. There will be differences in performance between the genders and DBD subtypes.

Null hypothesis:

The will be no differences in the performance of children with DBDs and non-DBD children in tests that measure executive, cognitive and motor impairments, there will be no gender or subtype differences.

Specific null hypotheses:

Null hypothesis 1:

Children with ADHD will not have lower scores than non-DBD children on the Block Design Test of the SSAIRS-R. There will be no differences in the scores between the genders and DBD-subtypes.

Null hypothesis 2:

Children with ADHD will not have lower scores than non-DBD children on the Tower of London (ToL). There will be no differences in the scores between the genders and DBD-subtypes.

Null hypothesis 3:

Children with ADHD will not have lower scores than non-DBD children on the Memory for Digits test of the SSAIRS-R. There will be no differences in the scores between the genders and DBD-subtypes.

Null hypothesis 4:

Children with ADHD will not perform poorer than non-DBD children on the Maze Coordination Test. There will be no differences in the scores between the genders and DBD-subtypes.

A description of the statistical tests employed to accept or reject the hypotheses formulated here will be supplied in the next chapter.

C h a p t e r   6

RESEARCH METHODOLOGY

6.1 Introduction

The aim of this research is to establish whether children with Disruptive Behaviour Disorders (i.e. ADHD, ODD and CD) are cognitively or neurologically impaired. Children with DBDs have problems with arousal, alertness, selectivity, distractibility, span of apprehension and sustained attention. They are more likely to exhibit the following characteristics: failure to finish tasks, daydreaming, failure to concentrate (Barkley, 2006). Children with ADHD, ODD and CD appear to have an impairment of self regulation, and problems with executive functions and other aspects of cognitive functioning (Castellanos, 1999). ADHD children are more likely to have developmental delays and cognitive deficits than those with ODD and CD (Quay & Hogan, 1999). It is also associated with a variety of self management deficits and impairments in cognitive, social, school and family functioning. The social, intellectual and neuropsychological deficits and impulsivity often persist into adulthood (Pomerleau et al., 2003). Neuropsychological impairments have been found to be associated with frontal lobe functions (Johnson et al., 2002).

6.2 Method

6.2.1 Research design

This is a quantitative study and a quasi-experimental research design was employed as the subjects could not be randomly assigned to the conditions of the independent variable because they already exhibit the variable. It is a comparative study where the performance in tests that measures cognitive and neuropsychological impairments will be compared between the DBD groups. Boys and girls will be compared separately. Children

with ADHD, ODD, CD and non-DBD children will be compared on areas of executive functioning (cognitive impulsiveness) and motor skills.

6. 2.2 Sample

The schools recruited for the selection of the groups were from Limpopo Province of South Africa. Participants were selected from schools in rural and semi-rural areas of the Ritavi area falling within the Mopani Municipal District. Participation was voluntary and parental consent was sought before testing was done. The schools were selected on the basis of proximity and accessibility, and were schools that include the teaching of Xitsonga as the first language, and therefore Xitsonga speaking. The Disruptive Behaviour Disorders (DBD) rating scale (Pelham, Jr. et al., 1992; Pillow et al., 1998) was used to screen the children to form four groups: ADHD, ODD, CD and non-DBD children. The DBD scale was designed to measure externalising disorders; it contains scales that are composed of the DSM-IV-TR items for ADHD-PI, ADHD-HI, ADHD-C, ODD, and CD. The DBD questionnaire has been translated, standardised with established norms for all the language groups in the Limpopo Province (Meyer et al., 2004). The DBD comprise 42 items that assess the presence and degree of ADHD, ODD and CD as formulated in the DSM IV-TR. Respondents are required to rate behaviour on a four point scale comprising the following options: not at all (0); just a little (1); pretty much (2); and very much (3). The higher the score on the DBD the more impaired the child is. School teachers from the selected schools were required to complete the DBD rating scale. The sample of 137 children with DBDs (i.e ADHD, ODD and CD) and those without DBDs (i.e. the normal control, non-DBD children) was drawn from children aged between 8 and 15 years who had been screened for ADHD, ODD and CD as this is the age group when the DBDs will manifest themselves (American Psychiatric Association, 2000). The Disruptive Behaviour Disorders Rating Scale (DBD) was used in the screening process (Pelham, Jr. et al., 1992;

Pillow et al., 1998). The experimental group was matched for age and sex with normal children, who formed the control group. (See Fig. 6.1)

Figure 6.1

Sample: DBD Subtypes
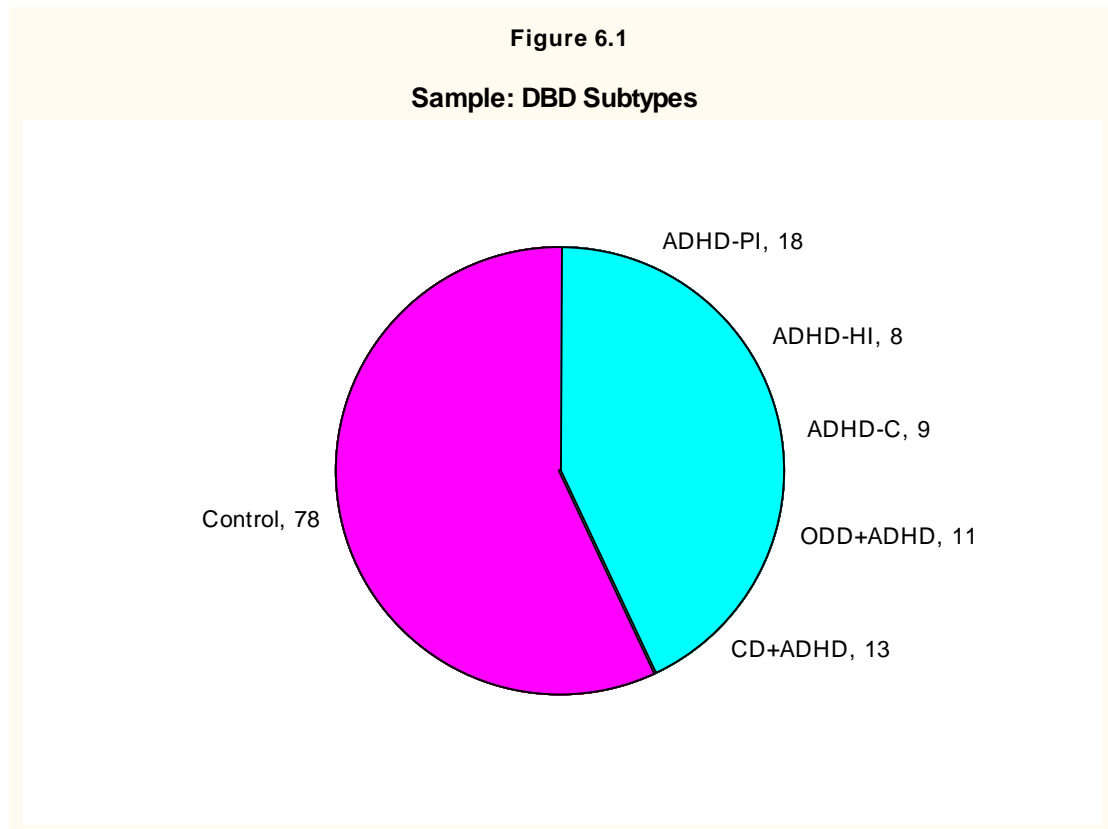
The average age of the DBD subtypes is depicted in Table 6.1. There was no statistical difference in age between the DBD-groups and the non-DBD control group (p = 0.15)

Table 6.1 Age of Sample

DBD subtype	N	Mean age (months)
ADHD-PI	18	139.00 ± 30.70
ADHD-HI	8	158.50 ± 22. 14
ADHD-C	9	136.89 ± 28.40
ODD+ADHD	11	151.11± 23.21
CD+ADHD	13	156.85 ± 21.76
Control	78	152.24 ± 20.94
All groups	137	150.18 ± 23.78

Table 6.2 shows the DBD scores for the sample.

Table 6.2: DBD Scores for the groups

DBD Subtype	N	Inattention Mean	Hyp/Imp Mean	ODD Mean	CD Mean
ADHD-PI	18	20.11± 1.13	9.83± 3.09	8.17 ± 2.94	6.11 ± 4.14
ADHD-HI	8	8.13 ± 2.64	18.75 ± 1.69	9.75 ± 4.33	6.86 ± 3.04
ADHD-C	9	19. 89 ± 0.93	17.89 ± 1.05	6.56 ± 3.94	5.00 ± 2.50
ODD+ADHD	11	12.78 ± 6.48	18.89 ± 6.35	20.00 ± 2.06	8.67 ± 4.44
CD+ADHD	13	17.31± 6.06	19.86 ± 4.88	17.92 ± 6.21	22.77 ± 6.08
Control	78	7.217 ± 3.97	5.37 ± 3.10	5.58 ± 3.81	4.31 ± 3.75
All Groups	137	11.204	9.93	8.51	6.79

6.2.3 Measuring instruments
SSAIS-R

The Senior South African Individual Scale-Revised (SSAIS-R) is an instrument that is used to measure general intelligence. It consists of verbal and non-verbal (performance) sections. The test has been standardised for South African children between 7 years 0 months and 16 years 11 months.

Two subtests from Senior South African Individual Scale (Revised) were used to measure cognitive impairment i.e. Block Designs, and Memory for Digits (Van Eeden, 1997).

Block designs

Patterns have to be built with plastic cubes from a design example. The test consists of 15 items. Four cubes are used for the first seven designs and nine for the rest. All the test items are timed, items 1-3 should be completed in 90 seconds, items 4-7 in 120 seconds, while items 8-15 should be completed in 150 seconds. The test measures non-verbal intelligence and problem solving skills. The testee has to solve problems in spatial relationships through logical reasoning and planning (Van Eeden, 1997). The test is discontinued after four consecutive failures. It measures non-verbal intelligence and non-verbal problem solving skills. It also measures non-verbal concept formation, including perceptual organisation, spatial visualisation and orientation and abstract conceptualisation. Concentration and visual motor coordination are important for the successful reproduction of the designs.

The test is based on the assumption that the ability to analyse, synthesise, and copy an abstract two dimensional geometric pattern is a valid criterion for general intelligence. It requires a certain amount of perseverance and dedication, as the test becomes progressively more difficult.

Instructions

The blocks are spilled on the table in front of the testee. The tester takes two blocks and say;

"Look at these blocks. Both have different colours on different sides. (Show simultaneously the red sides, red and white sides, etc. of both blocks.) *All the blocks look exactly like these two".*

After the five out nine blocks are removed out of sight the tester says:

"I am now going to put four blocks together in such a way that they make a picture or a pattern. Watch carefully".

The tester builds design 1 in front of the testee. The testee is then given the four blocks and the tester says:

"Now try to make one exactly like this one" (pointing to the model)

If the testee fails to complete the design successfully within the time limit, the tester will demonstrate again. The testee is allowed to redo items one to item three, after which, he/she will not be assisted and only one trial is permitted when using the nine blocks from item 4-15 (Van Eeden, 1997).

Memory for digits

The test consists of strings of digits that are read to the test taker, and the subject repeats them after they have been read to them at a steady rate. Some items are repeated forwards while some are repeated backwards. The second series of each string of eight digits is administered only if the testee repeats the first series incorrectly. The test requires concentration for the testee to be able to encode and remember the digits. Each of the memory for digits subtest i.e. digits forward and digits backwards is discontinued after two consecutive items or four consecutive series (Van Eeden, 1997).

Instructions for Memory for digits subtest were as follows:
Digits forward:

The tester says:

>"*I am going to say some numbers. Listen carefully and repeat them after me*".

Digits backwards:

>The tester starts with practice examples and says
>
>"*Now I am going to say some numbers which you have to repeat. This time start with the last number I say and say the numbers backwards*".
>
>The tester may only assist the testee with practice example 1.
>
>The digits are read at the rate of one a second. If the testee repeats the first series of an item correctly, the first series of the next item is read. If the testee repeats the first series incorrectly, the tester reads out the second series of the same item.

The maze-co-ordination test

>The test measures fine motor coordination. The testee is required to move the stylus through the maze without touching the sides. The number of touches and the time the touches last are recorded electronically (Matthews & Kløve, 1964). The child has to follow the trail (using both the dominant and the non-dominant hand) without touching the sides using a stylus.

>The instructions for the Maze coordination test are as follows:

>>"*Take this stylus and move it through the maze all the way to here* (point). *Try to avoid touching the sides* (show). *Do this with about this speed.* (Show by moving stylus through about ¼ of the maze). *You do not have to rush, if you move too quickly you will make more errors. Try to be accurate. Start with your (dominant) hand. Do not rest your arm against anything.*"

Tower of London

The Tower of London (ToL) test (Krikorian et al., 1994) is used to measure deficit attention responses, ability to plan and poor behavioural organisation. According to (Barkley, 2006), the test places heavy emphasis on working memory. It requires the subject to construct a design using coloured disks of different sizes and three upright pegs, employing the least moves possible with several restraints. The test involves substantial mental planning that must occur before and while undertaking the actual motor execution of arrangement. It requires forethought and planning and the individual must be able to mentally represent and test out different ways of removing and replacing disks on a set of pegs or spindles to match the design presented by the examiner.

The materials include three wooden peg of different lengths mounted on a strip of wood and three balls of red, yellow and blue colour that are manipulated by rearranging them on the pegs (the longest peg can hold three balls, the middle can hold two balls while the third ball can hold one ball) to reproduce a pictured end state of each of the 12 problems that have a graded difficulty. The ToL consists of four problems that require at least two or three steps to be solved, four problems requiring at least four steps, and four problems requiring at least five steps. The demand for planning is manipulated by presenting problems that differ in the minimum number of moves that are required for a solution (Scheres, Oosterlaan, Swanson, Morein-Zamir, Meiran, Schut, et al., 2003).

The Tower of London instructions are as follows:

The board is placed in front of the child with the highest stick to the left of the child. The child may explore and try the material.

"As you can see for yourself the sticks have different heights: The first one here (point) *can take (1) ball. The next one* (point) *can take 1 or 2 balls, and this one has the space for 1, 2 or (3) balls.*

The balls are placed in the right starting position.

"We will start every time in this position; so this is the starting position for every trial."

The example picture card is shown.

"Now I want you to place the balls on the sticks like they are positioned on this picture."

If necessary, the child may be helped to finish the task so that it becomes right.

When this is completed:

"I am now going to show you more pictures and I will ask you to copy these pictures by moving the balls on the sticks. I am also going to ask you to do it in a certain number of moves: it means I am going to ask you to copy this drawing by using 2, 3, 4 or 5 moves. With one move I am taking the ball from one stick and moving it to another stick.

You cannot take up one ball and hold it as you are moving another ball. You can also not move two balls at the same time."

The balls are placed back to starting position.

Trial number one is presented, and then followed by the rest of the drawings.

6.2.4 Procedure

A sample of 137 children with DBDs (i.e. ADHD, ODD and CD) aged 8-15 years and those without DBDs (i.e. normal control non-DBD children), matched for age and gender, was recruited from schools around the Ritavi district in the Limpopo province. The DBD screening test was used for the selection process. Permission was sought and

obtained from the University Ethics Committee, provincial and district Departments of Education, parents and principals of the participating schools from which the sample was drawn. Parents of all children participating in the study signed an informed consent form to allow their children to participate. Participation was voluntary and teachers were given the DBD rating scale to complete. Children with a history of neurological problems such as epilepsy and head injuries or those using psychostimulant medications at time of testing were excluded. The children meeting the criteria for inclusion into the clinical group were selected for further testing. These children were matched for gender and age with non-DBD children who formed the control group.

The other tests i.e. SSAIS-R (Blocks designs and Memory for digits), ToL, and Maze Coordination tests were given following the screening test by using the DBD rating scale. The children were tested in their own language. Test instructions were also be given in their mother tongue. The assessment was done at the schools during school hours. The tests were given in the following order; SSAIR-R block patterns, SSAIR-R memory for digits, The Tower of London, and the Maze Coordination Task.

6.3. Methods of data analysis

Computer programme STATISTICA version 6.1 (StatSoft, 2003) was used. MANOVA models and other inferential statistical methods were used to investigate possible differences in raw scores between the groups.

RESULTS

7.1. Introduction

The aim of this study was to establish whether children with DBDs (i.e. ADHD, ODD and CD) are cognitively and/or neuropsychologically impaired. This chapter will present the results of the performances on cognitive and neuropsychological tests between children with DBDs and a non-BDB comparison groups.

A battery of neuropsychological tests used in this assessment include two subtests from the SSAIR-S i.e. the Block Designs subtest and Memory for Digits (digits forward and digits backward) subtest (Van Eeden, 1997), a test to measure Executive Functions (attention and planning), the Tower of London (ToL) (Krikorian et al., 1994), and a test to measure motor functions, the Maze Coordination Task (Matthews & Kløve, 1964). The scores obtained from the administration of these measures are compared for significant differences between the DBD subtypes and a non-DBD control group as a possible function of gender. Test results of each neuropsychological test are presented in the following format: a table of descriptive statistics, a graphical representation to be followed by Analysis of Variance (ANOVA), to establish between-group differences, and a post-hoc (Bonferroni correction) test to establish within-group differences.

7.2 Results of the study

7.2.1. Block designs

Descriptive statistics

Table 7.1 illustrates the descriptive statistics for the results of the Block Designs subtest of the SSAIS-R according to DBD subtypes.

Table 7.1: Descriptive Statistics of Block designs (DBD subtypes)

	N	Mean
ADHD-PI	18	4.556 ± 4.805
ADHD-HI	8	11.625 ± 9.561
ADHD-C	9	3.111 ± 4.256
ODD	11	8.000 ± 7.629
CD	13	10.692 ± 5.588
Control	78	11.077 ± 6.544

Figure 7.1 illustrates the results of the Block Designs subtest of the SSAIS-R

Fig. 7.1 Graphical illustration of the results obtained on the Block designs subtest of the SSAIS-R
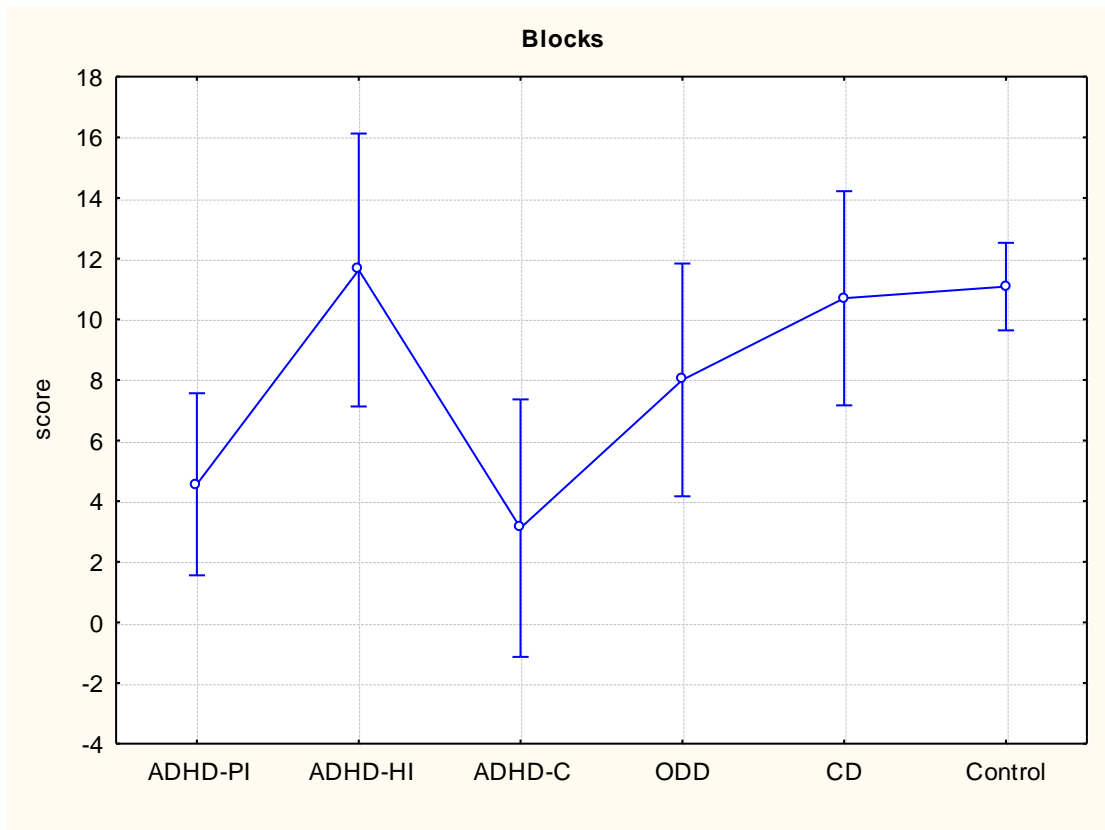
Analysis of variance (ANOVA)

Table 7.2 depicts the results of the ANOVA for the Block Designs

Table 7.2: ANOVA (Block designs)

	DF	F	p
Gender	1, 125	0.52216	0.471
Subtype	5, 125	4.59622	0.001*
Gender x Subtype	5, 125	0.29334	0.9156

*$p < 0.05$

There was no effect of gender, neither main nor interacting, therefore the subtypes were not analysed separately according to gender groups.

The differences in performances between the DBD subtypes and the non-DBD control groups on the Block designs subtest of the SSAIS-R were significant ($p < 0.05$).

Post-hoc tests (Bonferroni) were used to establish where the differences in performances between the DBD subtypes and non-DBD control group occurred on the Block Designs.

Table 7.3 illustrates the post-hoc (Bonferroni) test on the results of the Block Designs for the DBD groups according to subtypes.

Table 7.3: Results of the post-hoc (Boferroni) test: Blocks design (DBD subtypes)

Subtypel	ADHD-PI	ADHD-HI	ADHD-C	ODD	CD	Control
ADHD-PI	-	n/s	n/s	n/s	n/s	0.003*
ADHD-HI	n/s	-	n/s	n/s	n/s	n/s
ADHD-C	n/s	n/s	-	n/s	n/s	0.009*
ODD	n/s	n/s	n/s	-	n/s	n/s
CD	n/s	n/s	n/s	n/s	-	n/s
Control	0.003*	n/s	0.009*	n/s	n/s	-

*p < 0.05

The difference in performance between the ADHD-PI and the ADHD-C subtypes and the non-DBD control group was statistically significant ($p = 0.003$ and $p = 0.009$ respectively). When compared with the non-DBD control group, none of the other subtypes (ADHD-HI, ODD and CD) did show statistically significant differences in performance on the Block Designs test. Although the performance of either of the ADHD-PI and the ADHD-C subtypes was poorer than those of the other DBD subtypes, the difference was not statistically significant.

7.2.2 Digits Forward and Backward

Table 7.4 represents the descriptive statistics for the Memory for Digits test (Digits Forward and Digits Backward) of the SSAIS-R according to subtypes.

Table 7.4 Descriptive Statistics (Digits forward and Digits backward)

Subtype	N	Digits F Mean	Digits B Mean
ADHD-PI	18	5.833 ± 1.295	2.611 ± 2.033
ADHD-HI	8	8.125 ± 2.295	4.125 ± 1.456
ADHD-C	9	4.667 ± 1.871	2.111 ± 2.421
ODD	11	7.818 ± 3.628	3.273 ± 1.421
CD	13	6.769 ± 2.803	3.385 ± 1.758
Control	78	8.026 ± 2.168	3.923 ± 1.771

Figure 7.2 is a graphical representation of the results on the Memory for Digits subtests i.e. Digits Forward and Digits Backward according to DBD subtypes.

Figure 7.2 Graphical representation of the results obtained on the Memory for Digits subtest of the SSAIS-R
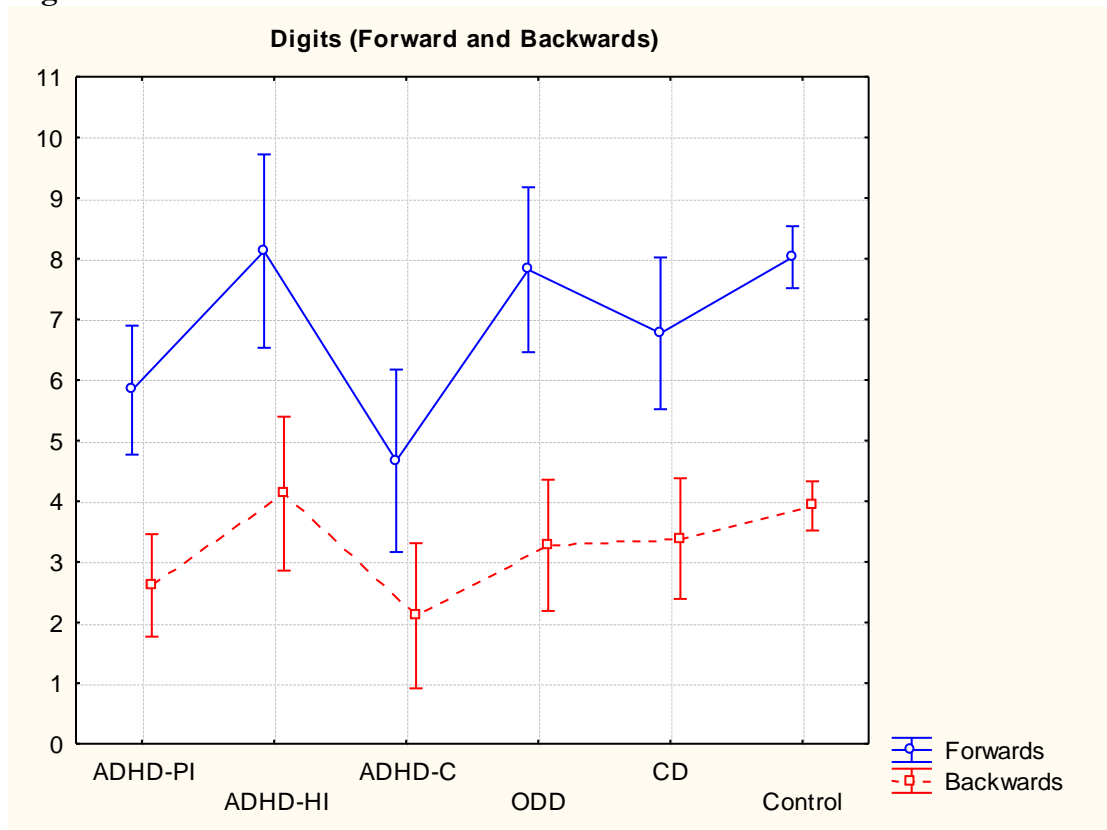
Table 7.5 depicts the analysis of variance (ANOVA) on the Digits Forward and Digits Backward subtests from the SSAIS-R Memory for Digits test according to DBD subtypes.

Table 7.5 ANOVA Digits Forward and Digits Backward (repeated measures)

	DF	F	P
Gender	1, 125	0.0193	0.889
Subtype	5, 125	6.4998	0.000**
Gender x Subtype	5, 125	1.1409	0.342
Digits (F & B)	1, 125	147.4325	0.000**
Digits (F & B) x Gender	1, 125	0.3280	0.568
Digits (F & B) x Subtype	5, 125	0.6387	0.671
Digits (F & B) x Gender x Subtype	5, 125	1.7566	0.127

$**p < 0.001$

There was no effect of gender, neither main nor interacting, therefore the subtypes were not analysed separately according to gender groups.

The difference in performance on Digits Forward and Digits Backward tests between the DBD subtypes and non-DBD control children is significant (p=0.001).

Post-hoc (Bonferroni) tests were used to establish where the differences occurred on the Digits Forward and Digits Backwards subtest, when the groups were compared

Tables 7.6 and 7.7 depict the results of the post-hoc tests on the Digits Forward and the Digits Backward according to the DBD subtypes.

Results of the post-hoc (Bonferroni) tests:

Table 7.6 Digits forward

	ADHD-PI	ADHD-HI	ADHD-C	ODD	CD	Control
ADHD-PI	-	n/s	n/s	n/s	n/s	0.005*
ADHD-HI	n/s	-	0.033*	n/s	n/s	n/s
ADHD-C	n/s	0.033*	-	0.038*	n/s	0.001*
ODD	n/s	n/s	0.038*	-	n/s	n/s
CD	n/s	n/s	n/s	n/s	-	n/s
Control	0.005*	n/s	0.001*	n/s	n/s	-

*p<0.05

Table 7.7 Digits Backward:

	ADHD-PI	ADHD-HI	ADHD-C	ODD	CD	Control
ADHD-PI	-	n/s	n/s	n/s	n/s	n/s
ADHD-HI	n/s	-	n/s	n/s	n/s	n/s
ADHD-C	n/s	n/s	-	n/s	n/s	0.078
ODD	n/s	n/s	n/s		n/s	n/s
CD	n/s	n/s	n/s	n/s	-	n/s
Control	n/s	n/s	0.078	n/s	n/s	-

Digits forward:

The difference in performance was statistically significant between ADHD-PI and the non-DBD control group (p = 0.05) with the ADHD-PI performing significantly poorer on the Digits Forward test. The difference in performance was not statistically significant between ADHD-PI group and all the other DBD subtypes.

The ADHD-C group also showed a significant poorer performance on this test when compared with the non-DBD control group (p = 0.001).

When the ADHD-PI's performance was compared with those of the other DBD groups (ADHD-HI, ADHD-C, ODD, and CD), no statistically significant difference in performance were shown. However, a comparison of the ADHD-C group's performance with the other DBD types did show a significant poorer performance than the ADHD-HI (p = 0.033) and the ODD (p = 0.038) groups. There was no difference in performance when compared with the ADHD-PI and CD groups.

Digits backward

When compared to the non-DBD control group, none of the DBD groups showed statistically significant differences in performance on the Digits Backward subtest.

The difference in performance between ADHD-C and the non-DBD control group was not statistically significant, but showed a tendency towards statistical significance (p=0.078).

No significant differences in performance were revealed when the DBD groups were compared with one another.

7.2.3 The Tower of London (ToL)

Descriptive statistics:

Table 7.8 illustrates the descriptive statistics of the results obtained on the tower of London according to DBD subtypes.

Table 7.8 Descriptive Statistics of the results on the Tower of London (ToL)

Subtype	N	Mean
ADHD-PI	18	18.000 ± 7.993
ADHD-HI	8	22.625 ± 5.236
ADHD-C	9	14.222 ± 7.032
ODD	11	22.727 ± 9.166
CD	13	22.615 ± 4.770
Control	78	24.192 ± 4.931

Figure 7.3 is a graphical illustration of the results on the Tower of London for the DBD subtypes and the non-DBD control group.

Figure 7.3 Graphical illustration of the results obtained on the Tower of London
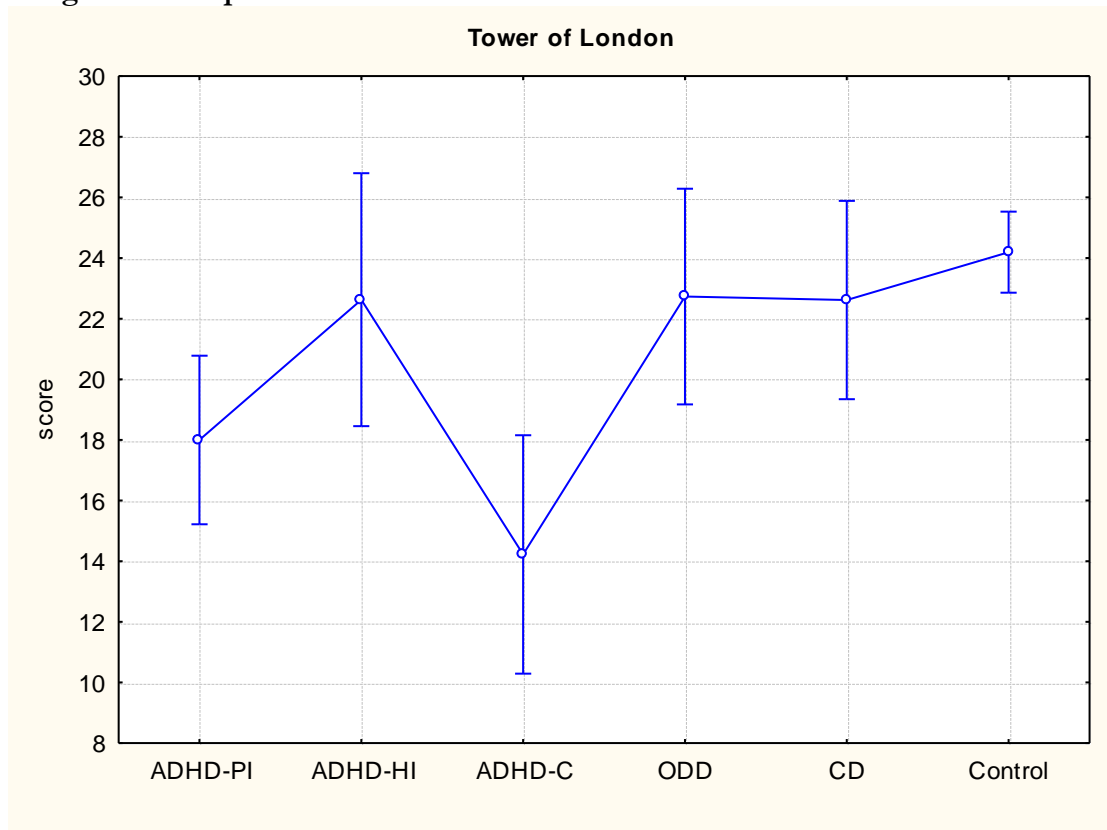
Analysis of Variance on the results of the ToL

Table 7.9 depicts the results of analysis of variance (ANOVA) on the Tower of London.

Table 7.9 ANOVA: Tower of London

	DF	F	P
Gender	1, 125	0.2010	0.655
Subtype	5, 125	5.0666	0.000*
Gender x Subtype	5, 125	0.7917	0.558

* $p < 0.001$

There was no effect of gender, neither main nor interacting, therefore the subtypes were not analysed separately according to gender groups.

The differences in the performance between the DBD subtypes and the non-DBD control group on the Tower of London test was statistically significant (p=0.000).

Post-hoc (Bonferroni) tests

Post-hoc (Bonferroni) tests were used to establish where the differences in performance occurred on the Tower of London.

Table 7.10 depicts the results of the post-hoc (Bonferroni) test on the Tower of London.

Table 7.10 Post-hoc (Bonferroni) tests: Tower of London (DBD subtypes)

	ADHD-PI	ADHD-HI	ADHD-C	ODD	CD	Control
ADHD-PI	-	n/s	n/s	n/s	n/s	0.002*
ADHD-HI	n/s	-	0.066	n/s	n/s	n/s
ADHD-C	n/s	0.066	-	0.028*	0.022*	0.000**
ODD	n/s	n/s	0.028*	-	n/s	n/s
CD	n/s	n/s	0.022*	n/s	-	n/s
Control	0.002*	n/s	0.000**	n/s	n/s	-

*p < 0.05;
**p< 0.001

When compared with the Non-DBD control group, only the ADHD-PI (p = 0.002) and ADHD-C (p = 0.000) subtypes showed a significantly poorer performance on the Tower of London. The difference in performance between the other DBD subtypes and the non-DBD control group was not statistically significant.

When the DBD subtypes were compared with each other, the ADHD-C subtype performed significantly poorer than the ODD (p = 0.028) and CD (p = 0.022) groups, while there was a tendency towards significance (p = 0.066) when compared with the ADHD-HI subtype. There were no statistically significant differences in performance when the ADHD-C subtype was compared with the ADHD-PI subtype.

7.2.4 The Maze Coordination Task

Descriptive statistics:

Table 7.11 represents the results obtained on the Maze coordination Test

Table 7.11 Descriptive Statistics: Results of the Maze Coordination Test

Subtype	N	Dom. hand Mean	Non-dom. hand Mean
ADHD-PI	18	80.889 ±20.588	91.0556±13.959
ADHD-HI	8	51.875 ±20.364	54.750 ± 19.934
ADHD-C	9	89.667 ±7.071	92.000 ± 7.298
ODD	11	61.000 ±13.229	69.364 ±12.372
CD	13	57.000 ±18.601	68.308 ±18.848
Control	78	56.718 ±21.259	64.089 ± 18.717

Figure 7.4 illustrates the graphical representation of the means for the number of touches on the Maze Coordination Test according to DBD subtypes

Figure 7.4 Graphical illustration of the performance on the Maze Coordination Task (no. of touches).

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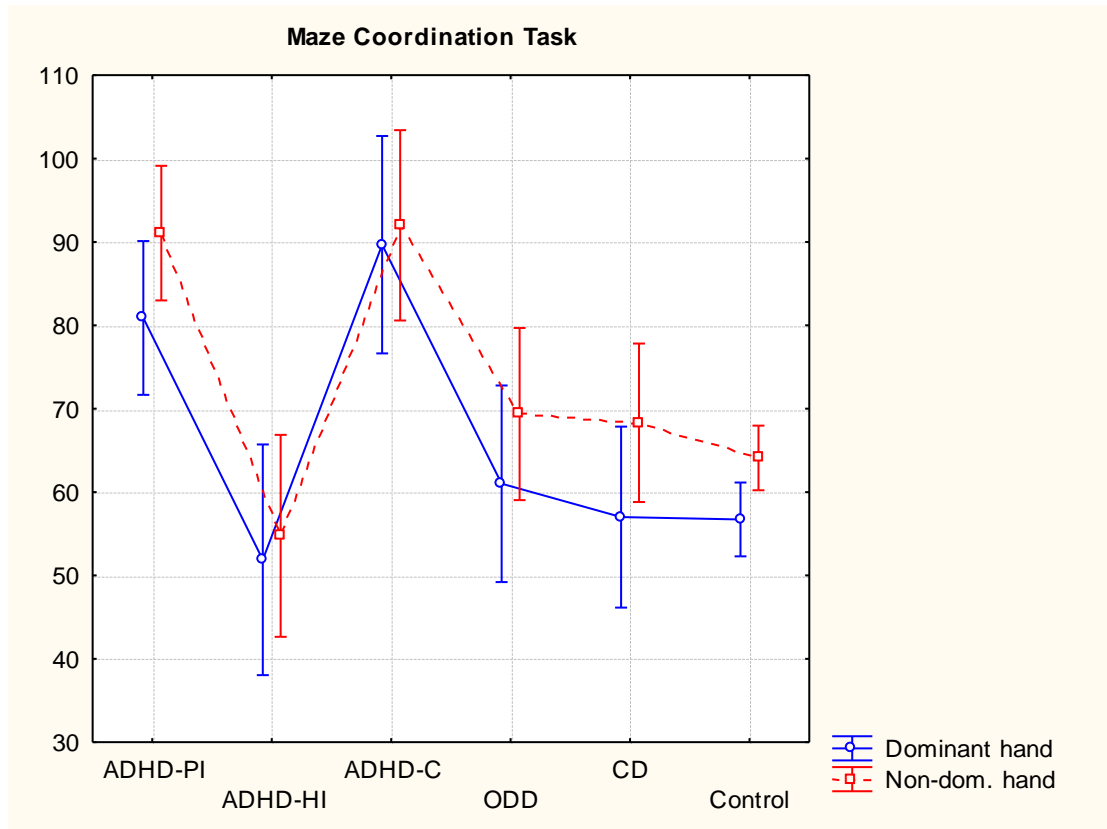
Table 7.12 depicts the results of the analysis of variance (ANOVA) for the number of touches on the Maze Coordination Test for the DBD groups according to subtypes.

Table 7.12 Results of the ANOVA (repeated measures) on the results of the Maze Coordination Task

	DF	F	P
Gender	1, 125	0.0433	0.836
Subtype	5, 125	8.8577	0.000**
Gender x Subtype	5, 125	0.2468	0.941
Hand dominance (Dom. & Non-dom)	1, 125	13.5622	0.000**
Hand dominance x Gender	1, 125	0.0651	0.799
Hand dominance x Subtype	5, 125	0.9413	0.457
Hand dominance x Gender x Subtype	5, 125	0.4305	0.827

*p < 0.001

There was no effect of gender, neither main nor interacting, therefore the subtypes were not analysed separately according to gender groups.

There were statistically significant differences in performance when the DBD groups were compared (p = 0.000).

Post-hoc (Bonferroni) test.

Tables 7.13 and 7.14 illustrate the results of the post-hoc (Bonferroni) test performed on the Maze Coordination Test for the number of touches for each (Dominant and non-Dominant) hand respectively according to DBD subtypes.

Table 7.13 Post-hoc (Bonferroni) test results for the Dominant hand

Subtype	ADHD-PI	ADHD-HI	ADHD-C	ODD	CD	Control
ADHD-PI	-	0.011*	n/s	n/s	0.018*	0.000**
ADHD-HI	0.011*	-	0.002*	n/s	n/s	n/s
ADHD-C	n/s	0.002*		0.024*	0.003*	0.000**
ODD	n/s	n/s	0.024*		n/s	n/s
CD	0.018*	n/s	0.003*	n/s		n/s
Control	0.000**	n/s	0.000**	n/s	n/s	

*p < 0.05
**p < 0.001

Table 7.14 Results of post-hoc (Bonferroni) test for the Non-Dominant hand

Subtype	ADHD-PI	ADHD-HI	ADHD-C	ODD	CD	Control
ADHD-PI	-	0.000**	n/s	0.020*	0.007*	0.000**
ADHD-HI	0.000**	-	0.000**	n/s	n/s	n/s
ADHD-C	n/s	0.000**	-	0.064	0.029*	0.000**
ODD	0.020*	n/s	0.064	-	n/s	n/s
CD	0.007*	n/s	0.029*	n/s	-	n/s
Control	0.000**	n/s	0.000**	n/s	n/s	-

*p < 0.05
**p < 0.001

Dominant hand:

When compared to the Non-DBD Control group, the ADHD-PI (p = 0.000) and

ADHD-C (p = 0.000) had statistically significant more touches in the Maze Coordination

Task. The ADHD-PI subtype also had a poorer performance than the ADHD-HI (p =

0.000), the ODD (p = 0.020) and the CD (p = 0.007) subtypes. They did not differ significantly from the ADHD-C group.

The ADHD-C subtype performed significantly poorer on the Maze Coordination Task than the Non-DBD controls. The also performed significantly poorer than the other DBD groups, except for the ADHD-PI subtype, when on comparison, no statistically significant difference was found.

Non-Dominant Hand:

When compared to the Non-DBD Control group, the ADHD-PI (p = 0.000) and ADHD-C (p = 0.000) had statistically significant more touches in the Maze Coordination Task.

When the ADHD-PI group was compared to the other DBD types on the results of the non-dominant hand, they also performed poorer than the other groups, except for the ADHD-C group, where no significant difference was found.

When the ADHD-C subtype was compared with the Non-DBD controls, they also showed a statistically poorer performance (p = 0.000).

The ADHD-C subtype also had a statistically significant poorer performance with the non-dominant hand than the ADHD-HI (p = 0.000) and CD (p = 0.029) groups, while there was a tendency towards significance when compared with the ODD group (p = 0.064).

7.3 Hypotheses testing

Based on the research results, the following conclusions about the hypotheses can be made:

Null hypothesis 1:

The null hypothesis 1 which states that there will be no differences in the performance of children with DBDs and non-DBD children on the Blocks design Test, and that there will be no gender or subtype differences must be partially rejected.

No gender differences in performance were observed. Significant statistical differences were found only between the ADHD-PI subtype and the non-DBD control group, and between the ADHD-C subtype and the Control group. The other groups (ADHD-HI, ODD, and CD) did not differ significantly in their performance on the Block Design test from the controls. There were no statistically significant differences observed when the DBD subtypes were compared with each other.

Null hypothesis 2:

The null hypothesis 2 must be partially rejected; there are differences in the performance on the digits forward between the DBD subtypes and the non-DBD control group with the latter performing better than the DBD experimental group. No differences were found between the genders. Statistical significant differences were found between ADHD-C subtype and each of the ADHD-HI, ODD and CD subtypes and the non-DBD control group where in each case the ADHD-C performed more poorly. A significant statistical difference in the performance was also observed between ADHD-PI subtype and the non-DBD control group, with the ADHD-PI subtype performing poorer.

A comparison between the ADHD subtypes shows that the performance by the ADHD-HI was better than either of ADHD-PI and ADHD-C subtypes.

There was no statistically significant difference in the performance on the digits backwards between all the DBD subtypes and the non-DBD control group, and between DBD subtypes themselves.

Null hypothesis 3:

The null hypothesis 3 must be partially rejected as there are statistical significant differences in performance on the Tower of London (ToL) between the DBD and the non-DBD control group. No significant difference in performance was found between the genders. Statistically significant differences in performance were observed between the DBD subtypes. The performance of the ADHD-C subtypes was significantly poorer when compared with that of each of the ODD, and CD subtypes, and that of the non-DBD control group. The ADHD-PI subtype also showed a significantly poorer performance when compared with the non-DBD control group. When the ADHD subtypes were compared among themselves, no statistical differences in performance between the ADHD-HI and either of the ADHD-PI and the ADHD-C subtypes were observed.

Null hypothesis 4:

The null hypothesis 4 must be partially accepted, as there are differences in performance on the Maze Coordination Test between the DBD children and non-DBD control children, but no differences in performance between the genders.

When the dominant hand was used the difference in performance on the Maze Coordination Test was found to be statistically significant when the ADHD-PI group was compared with non-DBD control group, and the ADHD-HI and CD subtypes, with ADHD-PI performing poorer (making significantly more touches on the sides of the Maze trail) in each case. Similar results were found to occur when the non-Dominant hand was used with ADHD-PI performing poorer than the non-DBD control group, ADHD-HI, CD subtypes, but in this case including ODD subtype.

A significant statistical difference in performance with the dominant hand can also be observed between ADHD-C and the non-DBD control group, ADHD-HI, ODD and CD subtypes, where the former performed more poorly in each case. A similar pattern of

results emerges when the non-dominant hand is used, but with the ODD subtypes also performing better than the ADHD-C subtype.

The results further indicate that there was no significant difference for the dominant hand between the performance on the Maze Coordination Test of the ADHD-PI and the ADHD-C.

A discussion of the results will follow in the next chapter.

DISCUSSION OF RESULTS

8.1 Introduction

Research has found that inhibitory control deficits are not a unique marker for ADHD, but have also been found in ODD and CD (Oosterlaan et al., 1998). Klorman, Hazel-Fernandez, Shaywitz, Fletcher, Marchione, Holahan, et al. (1999) found evidence for planning deficits in children with ADHD combined type that was independent of ODD and CD. The tests used in the current study were selected as measures of EF and motor functions. Major neurological underpinnings for EF is the frontal cortex and its subcortical connections (Eslinger, 1996), and therefore only measures that have been shown to rely heavily on frontal cortex functioning should be used to study EF (Sergeant, Geurts, & Oosterlaan, 2002). EF is a summary of psychological processes involved in the organisation and planning of behaviour (Tannock, 1998), and include working memory, motivation and state of arousal and reconstitution (Barkley, 1997a). EF deficits include impairments in the ability to plan, execute, verify and regulate own behaviour (Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003). Motor deficits are associated with execution of gross and fine motor tasks (Meyer & Sagvolden, 2006b).

Research evidence indicates that hyperactive children could perform at normal or near normal levels of sustained attention under conditions of continuous reinforcement and immediate reinforcement, but deteriorate when partial reinforcement is used (Oosterlaan & Sergeant, 1998). Children with ADHD are not always cognitively impulsive as they temporarily do manage to plan ahead, organise their behaviour and remember important things if their behaviour is maintained by potent and frequent reinforcers (Johansen et al., 2002).

The aim of the study was to establish whether children with DBDs are cognitively and/or neuropsychologically impaired. Neuropsychological testing of children screened for DBDs were assessed on measures of neuropsychological and cognitive deficits. Neuropsychological tests that were used, the Blocks Design, Digits Forward and Digits Backward from the Senior South African Intelligence Scale (Van Eeden, 1997), The Tower of London, a test for cognitive impulsiveness and planning ahead (Krikorian et al., 1994), and the Maze Coordination Test which measures fine motor skills (Matthews & Kløve, 1964).

The main objective of the study was to investigate the differences in measures of executive and motor functions, EF refers to a variety of behaviours and abilities related to planning and strategy use, as well as maintenance of behaviour in the pursuit of some goal (Culbertson & Zillmer, 1998; Riccio, Wolfe, Romine, Davis, & Sullivan, 2004), it is a system of interconnected behaviours or processes to facilitate goal oriented behaviour and the ability to form mental representation of a task (Fuster, 1997; Stuss & Benson, 1986); on the other hand motor deficits include, problems with gross and fine motor skills, problems with planning movement and execution, motor clumsiness, ability to adapt task performance to environmental requirements (parameter setting) (Meyer & Sagvolden, 2006b). The ToL and memory for digits i.e. digits forward and digits backwards were used in the assessment of EF (Riccio et al., 2004). The Tower tasks are presumed to tap Executive Function and problem-solving (Shallice, Marzocchi, Coser, Del Savio, Meuter, & Rumiati, 2002), and are viewed as planning tasks because of the presumption that the individual will engage in more efficient problem-solving if he/she plans the course of action before starting to move the beads (Riccio et al., 2004). According to Paule et al. (2000) working memory deficits are reflected by poor performance on tests such as the backward digit span, while motor planning deficits are observed by consistent poor performance on tower and maze tasks. Working memory refers to an individual's ability to

hold relevant information in mind for the purposes of completing a task, a functional storage that provides for temporary storage and manipulation of information (Baddeley, 1992). The Blocks Design & Maze Coordination tasks are used as measures of motor functioning, attention, and visuo-spatial perception. While Blocks are more sensitive to gross motor functioning, the Maze measures complex coordination, goal directed fine motor movements, accuracy and stability of movement (Meyer & Sagvolden, 2006b). The current research also seeks to investigate if there are gender differences in the manifestation of EF and neuropsychological impairments.

The Blocks Design is also a measure of cognitive functioning, it requires of a testee to solve problems in spatial relations by using logical reasoning. In the Maze Coordination tasks, poor performance will show eye-hand coordination deficits, and a measure of control of task by means of pre structured motor plans (Schoemaker, Ketelaars, van, Minderaa, & Mulder, 2005) as it requires planning ahead (Meyer & Sagvolden, 2006b).

8.2 Summary of results

Table 8.1 illustrates the summary of all significant results for all the tests administered.

Table 8.1.Summary of significant results

Test	Significant results
Blocks	*ADHD-PI vs Control
	ADHD-C vs Control
Memory for Digits	
Digits Forward	ADHD-PI vs Control
	ADHD-C vs ADHD-HI
	ADHD-C vs ODD
	ADHD-C vs Control
Digits backward	
	None
Tower of London	
	ADHD-PI vs Control
	ADHD-C vs Control
	ADHD-C vs ODD
	ADHD-C vs CD
Maze Coordination Task	
Dominant hand	ADHD-PI vs Control
	ADHD-PI vs ADHD-HI
	ADHD-PI vs CD
	ADHD-C vs Control
	ADHD-C vs ADHD-HI
	ADHD-C vs ODD
	ADHD-C vs CD
Non-dominant hand	ADHD-PI vs Control
	ADHD-PI vs ADHD-HI
	ADHD-PI vs ODD
	ADHD-PI vs CD
	ADHD-C vs Control
	ADHD-C vs ADHD-HI
	ADHD-C vs CD

*In each case the subtype with the poorest performance is mentioned first

The results of the tests were analysed on the basis of the differences between children with DBDs and non-DBD children as a function of gender and subtype. A number of neuropsychological tests have been used over the years by researchers to assess

neuropsychological deficits in children with DBDs. The results of the current study show that the performance of non-DBD children is overall better than those of the DBD children in the Blocks, ToL, Maze, and Memory for digits tests with the ADHD-C performing worse in all measures used in the current research. A more detailed discussion of the results will follow in the subsequent sections of this chapter.

Gender differences

Sex differences in cognitive functions have been well documented with males performing better than females on spatial tasks, and females performing better than males on tests of verbal memory (Kimure, 1992).

However no differences have been found between the genders in the performance on all the tests used in the current study i.e. Blocks, Digits forward & backward, ToL and Maze Coordination Task. Although Carlson et al. (1997) found no differences in motor skills between genders, Meyer and Sagvolden, (2006b), found that girls performed significantly poorer than boys in the Maze Coordination Task when the dominant hand was used. Yang, Jong, Chung, and Chen (2004) found that ADHD boys performed better than girls on the Blocks Design test.

Other studies (Culbertson & Zillmer, 1998; Krikorian et al., 1994; Welsh, Pennington, & Groisser, 1991) also did not find any significant differences between girls and boys on ToL performance. From the results of this study, it can be suggested that cognitive and motor impairments are expressed the same in both boys and girls.

The tests administered were therefore able to successfully differentiate neuropsychologically between children with DBD's and those without DBD symptoms, especially isolating the ADHD-PI and ADHD-C subtypes as the poorer performers.

The data results can be summarized as follows:

The performance by ADHD-C and ADHD-PI children on the ToL, Block Designs and Digits forward and the Maze Coordination Task was found to be poorer than that of the non-DBD control group, with the ADHD-C performing the worst. These results are consistent with current research studies that found that the impairment in cognitive and motor functioning was most severe for the subtype with symptoms of ADHD-C and less severe for the subtypes with symptoms of ADHD-PI, while HI, ODD and CD subtypes appeared to be not significantly affected (Aase, Meyer, & Sagvolden, 2006; Meyer & Sagvolden, 2006a; Schmitz, Cadore, Paczko, Kipper, Chaves, Rohde, Moura, & Knijnik, 2002).

Schmitz et al. (2002) have found that subjects with ADHD-PI or ADHD-C subtypes showed poorer neuropsychological performance than did control subjects, while subjects with ADHD-HI did not show significant differences when compared with a control group. The results in this study support the notion that evidence of cognitive and motor impairment is considered stronger for ADHD subtypes particularly the ADHD-PI and ADHD-C than for ODD and CD.

The performance of the ODD and CD subtypes on the Tower of London (ToL) was found to fall midway between performance of children with ADHD and normal control children suggesting that impairment in planning appears to be associated with ADHD, but not necessarily with ODD and CD nor with comorbid ADHD+ODD/CD, a finding which supports the findings by Oosterlaan et al. (2005).

Findings from the current study confirm other studies that have consistently found that ADHD-PI and ADHD-C subtypes show deficits in components of executive functions particularly those associated with control of motor responses (planning, preparation, execution, and inhibition) and working memory. Motor planning deficits are

observed by consistent poor performance on neuropsychological tasks such as the Tower of London, and Mazes (Paule et al., 2000).

It is interesting to note the relationship of the results of the performance of the ADHD-PI subtype and that of the ADHD-C subtype. In all the tests used in the current study there were no significant differences between the results of scores of the ADHD-PI subtype and ADHD-C subtype. What is common in the diagnosis of both these subtypes is the inattention component as found in the diagnostic criteria in the DSM-IV-TR (American Psychiatric Association, 2000). This could suggest that the results of the performance could be due to the inattentive component in both the ADHD-PI and ADHD-C disorders, and that inattention could be a risk factor for the development of cognitive and motor problems. The presence of both the inattention symptoms and externalising behaviour in ADHD combined subtype could be a complicating factor which places these children in more increasing risk for cognitive and motor problems (Carlson et al., 1997; Meyer & Sagvolden, 2006b).

The similar performance between ADHD-C and ADHD-PI subtypes, and the difference in performance between either of the subtypes with that of ADHD-HI could suggest that ADHD-HI subtype is different from the former subtypes on measures of neuropsychological and cognitive functions, supporting the conclusion made by Schmitz et al. (2002) who asserted that there are neuropsychological differences among ADHD subtypes (Barkley, 1997a; Rosenthal, Riccio, Gsanger, & Jarratt, 2006). Another significant finding from the results is that the performance by each of the DBD subtypes (ADHD-HI, ADHD-PI, ADHD-C, ODD and CD) was consistent on all measures suggesting that the tests used were able to discriminate well between the groups.

Blocks Design

This test requires a certain amount of perseverance, and dedication due to the fact that it becomes progressively more difficult. The test involves a spatial component in perception at the conceptual level and in motor execution. Blocks design load highly on perceptual organisation. Correct reconstruction reflects the ability to perceive errors and willingness to correct them (Lezak et al., 2004). The performance of the non-DBD control group is not different to the performance by ADHD-HI, ODD and CD but differed from the ADHD-PI and ADHD-C subtypes whose performance was found to be significantly poorer when compared the non-DBD control group. Of the ADHD subtypes, only the ADHD-HI performed as well as the non-DBD control group, a finding consistent with that of Garcia-Sanchez, Estevez-Gonzalez, Suarez-Romero, and Junque (1997), also confirming their assertion that Blocks Design is a visuo-spatial tasks with the highest discriminant to differentiate between subjects with ADHD-PI and ADHD-HI.

The poor performance by the ADHD-PI and the ADHD-C subtypes suggests that their lower performance could be due the inattentive component of the ADHD spectrum. Poor performance on the Block Design shows that the ADHD-C subtype has deficiencies in their ability to perceive errors and in their willingness to make corrections if they notice them (Lezak et al., 2004). In contrast with ADHD-HI children, subjects with ADHD-PI have been found to perform poorly on the Block Design, a finding that is consistent with the right hemisphere dysfunction which suggests that inattention, defective response inhibition and impersistence are more commonly seen in right hemisphere lesions (Garcia-Sanchez et al., 1997). Other brain areas of functioning implicated with deficit cognitive functioning include the areas supplied by the meso-cortical dopamine branch (Johansen et al., 2002). Because the performance on the Block Designs provides information whether testees are able to order and plan ahead, it follows that these two subgroups, the ADHD-C and the ADHD-PI subtypes are more affected than the ADHD-HI subtype and the other

externalising disorders, the ODD and CD subtypes. Of the ADHD group the ADHD-HI subtypes appears not to be as dysfunctional in terms of cognitive and motor functions as the ADHD-C and the ADHD-PI subtypes supporting the suggestion by other researchers that the ADHD-PI (and most likely ADHD-C) could have different aetiology from that of ADHD-HI (Johansen et al., 2002; Sagvolden, Johansen, Aase, & Russell, 2005).

Therefore, a diagnosis of ADHD-PI and ADHD-C predisposes an individual to be more severely affected by cognitive deficits than the ADHD-HI, ODD and CD subtypes. They are incapable of planning and foresight, they also fail to sustain goal directed behaviour. It could further be suggested that these subtypes adopt a concrete attitude of the surroundings, taking everything at most obvious face value. Nigg, Blaskey, Huang-Pollock, and Rappley (2002) have also suggested that the ADHD-C and ADHD-PI are related disorders that share deficits in vigilance and effort functions and on other measures, with them differing only in severity.

Although the graphical illustrations of the results for all the tests used in this study shows that the performance of the ADHD-C was poorer than that of ADHD-PI, the difference in performance is not significant. These findings support those found by Geurts, Verte, Oosterlaan, Roeyers, and Sergeant, (2005) who concluded that ADHD-C and ADHD-PI subtypes did not differ from one another in measures of Executive Functioning, but differed from the control group.

Memory for digits

Poor performance on the digit test shows a deficiency in working memory (Paule et al., 2000).

Memory for digits measures how fast attentional systems operate and how much it can process at once (Lezak et al., 2004). As a measure of attentional capacity, the test exposes the subject to increasingly larger amounts of information.

Working memory deficits are reflected by poor performance on tests such as digit span (Hale, Hoeppner, & Fiorello, 2002; Paule, et al., 2000). Working memory is critical to conscious thought; it permits internal representation of information or rules to guide decision making and overt behaviour or responses during activity so that behaviour is not dominated by the sensory cues in the environment. Working memory processes were found to be impaired in children with ADHD especially with spatial storage as opposed to verbal storage of information. Poor academic achievement of ADHD children may be the result of working memory deficiencies rather than a direct consequence of behavioural symptoms of inattention and or hyperactivity-impulsivity (Martinussen et al., 2005).

The overall performance of the non-DBD control group was significantly better than that of the DBD groups in the digits forward test, while there were no significant differences observed between the DBD groups (ADHD-HI, ADHD-PI, ADHD-C, ODD and CD) and the non-DBD control group on the digits backwards task. Results show significantly poor performances by ADHD-PI and ADHD-C subtypes on the digits forward test when compared with the control group, with the performance by the ADHD-C subtype also differing significantly from those of ADHD-HI and ODD subtypes.

Digits forward test

Digits forward is a task of short-term auditory memory, sequencing and simple verbal expression (Hale et al., 2002). Studies have shown that ADHD children were found to have impairment in working memory (Barkley, 1997a; Karatekin & Asarnow, 1998; Martinussen et al., 2005; Paule et al., 2000). Consistent with the findings by Rosenthal et al. (2006) the current study did not find any significant differences between ADHD-PI and ADHD-C subtypes on the digits forwards tasks. In the present study the ADHD-C subtype showed a significant poorer performance on the digits forward test than the non-DBD control group, also a significant poorer performance than the ADHD-HI and the

ODD groups emerged. The ADHD-PI subtype also showed a significant poorer performance when compared with the non-DBD control group. Consistent with these findings Schmitz et al. (2002) reported poor performance on digit span by adolescents with ADHD-PI and much poorer performance by the combined subtype. As in the current research, they also reported that the ADHD-HI subtype did not differ significantly from the control subjects, and showed better performance than did those with ADHD-C subtype. The current results support the findings by Schmitz et al. (2002) that ADHD-HI does not seem to be linked to cognitive problems. The poor performance by the ADHD-C and ADHD-PI subtypes suggests that these children have more problems with immediate memory, attention and concentration, a deficiency in the processes pertaining to storage and processing of information, and are more likely to forget information than ADHD-HI, and ODD and CD subtypes. The poor performance of the ADHD-PI and ADHD-C subtypes on this test also suggests that these subtypes have a very limited capacity to retain information and to utilize it for adaptive purposes. Although both the ADHD-PI and ADHD-C groups shows a significant poorer performance when compared with the non-DBD control group, however unlike as it is the case with ADHD-C, there were no significant differences between ADHD-PI and CD, and between ADHD-PI and ODD. This could be an indication that ADHD-C children are more severely affected in measures of cognitive and motor functions than ADHD-PI children.

Digits backward test

Digits backward is a simple test of mental tracking. It tests how many bits of information a person can attend to at once and repeat in reverse order (Lezak et al., 2004). It requires the testee to hold information while performing a mental operation. This subtest differs from Digits Forward in that more effortful activity is required as data bits are briefly stored while simultaneously juggling them around mentally. It involves both memory and reversing operation simultaneously. Factor-analytic studies have found that

the memory processes involved in the forward recall of digits are distinctively different from those involved in backward recall (Reynolds, 1997).

There were no differences in performance between the non-DBD control group and the DBD subtypes and between the subtypes themselves, but a tendency towards a statistical significant difference was observed between the ADHD-C and the non-DBD control group (p=0,078). Digits backward is a more challenging task than digits forward in that it requires more complex skills than the later. Literature trends report an association between impulsiveness and slower or poorer performance on complex speeded tasks (Dougherty, Bjork, Harper, Marsh, Moeller, Mathias, & Swann, 2003). Keilp, Sackeim and Mann (2005) maintain that when information-processing demand and response complexity are increased, requiring subjects to respond as quickly as possible, impulsive individuals appear to be slower.

Although there were no significant differences in performance between the DBD and non-DBD subtypes, the ADHD-C group performed qualitatively worse, an indication that the ADHD-C subtype appears to have a difficulty in both the ability to store information and to process information. It could be that compared to ADHD-PI, ADHD-HI, ODD and CD subtypes, the ADHD-C group has more problems with attention; they have a deficiency in the capacity or processes that are related to aspects of how they can become receptive to stimuli and to process incoming excitation. Compared with other subgroups it may be that even if some measure of information could be retained the ADHD-C are more likely to quickly lose the information or experience diminished access to retained information. Rosenthal et al., (2006) found significant differences on DB performance of children with ADHD-C and those with ADHD-PI with ADHD-C performing poorer. The significant differences between the combined and the

predominantly inattentive subtypes lend support to Barkley's contention that the subtypes have different underlying deficits (Barkley, 1997).

The Tower of London

The results of the ANOVA did show a significant difference in performance between the DBD and the non-DBD control group (p=0.000) on this test. The ADHD-C and ADHD-PI subtypes showed poorer performance when compared to the non-DBD control group. The performance of the ADHD-C subtype was also found to be poorer when compared to the performance of the ADHD-HI, ODD and CD subtypes. This is consistent with research findings by Culbertson & Zillmer (1998), Meyer (2005a), Meyer and Sagvolden, (2006a), Nigg, Butler, Huang-Pollock, and Henderson (2002), and Sarkies, Sarkies, Marshall, and Archer (2005) who found that ADHD children performed poorly in the ToL tasks. The ToL has a large planning component; it measures the ability to plan ahead and to some extent other factors including working memory, response inhibition and visuo-spatial memory (Lezak et al., 2004).

Children with ADHD predominantly inattentive and those with ADHD combined type differed from the control group on the ToL tasks. These results are consistent with those of other researchers (Klorman et al., 1999; Nigg et al., 2002), which further suggest that the ADHD-C subtypes are more likely to show planning deficits. Some studies have been more specific to suggest that the ADHD combined subtype differed significantly in perserveration and response inhibition (Houghton, Douglas, West, Whiting, Wall, Langsford, et al., 1999).

This study seems to have successfully demonstrated that it is particularly the ADHD-C subtype of ADHD that is associated with such deficits. ADHD-PI is also associated with such deficits, but the ADHD-C group had qualitatively larger differences when compared with the normal control group suggesting that it has a more serious deficit,

a finding which is consistent with those of other researchers (Klorman et al., 1999; Meyer & Sagvolden, 2006a; Nigg et al., 2002). The performance by the ADHD-HI subtype was not found to be different compared to ODD, and CD subtypes, and also when compared with the non-DBD control group. The result of the current study suggests that the presence of HI is not a necessary risk for cognitive and motor deficiencies. The comparable performance of the ADHD-HI subtype with those of ODD and CD subtypes shows that there is a strong relationship between the subtypes on measures of cognitive and motor functioning. It is therefore more likely that the ADHD-HI could develop into ODD or/and CD if left untreated. The presence of both the inattention and hyperactivity symptoms in an individual appears to be a risk factor in the development of motor and cognitive problems.

Maze Coordination Test

Children with ADHD showed deficits in motor coordination particularly the ADHD-C and ADHD-PI subtypes who showed a much poorer performance (shown by the number of touches on the maze alley sides) when compared to the non-DBD control group. The difference was statistically significant for both the dominant hand ($p=0.00$) and non-dominant hand ($p=0.00$). The results of the current study are in accordance with research findings that also found that children with ADHD-C and ADHD-PI performed significantly poorer than the non-ADHD control group on the Maze Coordination test (Meyer & Sagvolden, 2006b; Piek, Pitcher, & Hay, 1999; Schoemaker et al., 2005). Consistent with these findings, research studies have demonstrated that poor fine motor skills are associated with ADHD-PI while ADHD-C was found to be also associated with poor gross motor skills, further noting that inattention is predictive of severe difficulties in motor coordination (Dewey, Kaplan, Crawford, & Wilson, 2002; Pitcher, Piek, & Hay, 2003). The poor performance by the ADHD-PI and ADHD-C subtypes suggests that children with ADHD-C and ADHD-PI have serious deficiencies in planning and foresight

or in sustaining goal directed behaviour that are deficiencies associated with frontal lobe dysfunctions particularly those that control psychomotor functioning (Barkley, 1997). Poor motor functioning is associated with the cortical areas supplied by the nigro-striatal dopamine branch (Johansen et al., 2002). The poor performance of the ADHD-PI and ADHD-C subtypes further suggests that children with these subtypes have problems with eye-hand coordination and control of tasks through pre-structured motor plans, deficits that are more pronounced in ADHD-C children due to the qualitatively poor performance when compared to ADHD-PI children (Meyer & Sagvolden, 2006b).

Other studies consistent with these findings and asserting that deficits in components of executive functions particularly those associated with control of motor responses (planning, preparation, execution, and inhibition) and working memory have been found, and that motor planning deficits can be observed by consistent poor performance on neuropsychological tasks including the mazes (Barkley, 1997a; Karatekin & Asarnow, 1998; Paule et al., 2000b)

8.3 Integration of results and Findings

The findings in the current study indicate that, although both ADHD-PI and ADHD-C children show neuropsychological deficits, the ADHD-C children are more severely affected in their cognitive and psychomotor functioning, while the ADHD-HI children were less affected; they performed as well as ODD, CD and non-DBD control groups in all the tests used in this study. In a study by Nigg et al. (2002) the ADHD-C subtype was found to have a lower IQ than the control group, while ADHD-C (there were no significant differences between the combined and the inattentive subgroups) and ADHD-PI were not found to have different IQ's. They further noted that the diagnosis of ADHD-PI and ADHD-C were associated with weaker academic achievement, a finding supported by several other researchers (Faraone, Biederman, Weber, & Russell, 1998;

Gaub & Carlson, 1997). In their study Klorman et al. (1999) found that the ADHD-C children encountered deficits in planning and cognitive flexibility. Other researchers have also shown that the ADHD-HI subtype does not seem to be linked to cognitive problems (Schmitz et al., 2002).

Since the DSM-IV-TR diagnostic criteria were used in this study, which requires that if the DBDs are comorbid only the more serious disorder is diagnosed (American Psychiatric Association, 2000) implying that most ODD and CD diagnoses may have comorbid ADHD-PI, ADHD-C or ADHD-HI, while a diagnosis of one of the ADHD subtypes do not have comorbid ODD and CD. Therefore, while ODD and CD may have comorbid ADHD, the two ADHD subtypes i.e. ADHD-PI and ADHD-C subtypes do not have comorbid ODD, or CD according to the DSM-IV criteria (American Psychiatric Association, 2000). This shows that the impairments in executive function are clearly located in ADHD particularly in ADHD combined subtype. However in the current study it was not established how much comorbidity of ADHD-PI and ADHD-HI was contained in the ODD and CD subgroups. Future research should establish whether such comorbidity could influence the results differently.

Other researchers argue that ADHD-PI is associated with more specific deficits of selective attention (Barkley, 2006; Schmitz et al., 2002) while ADHD-C is associated with broader deficits of executive functioning (Schmitz et al., 2002). Barkley (1997) further reports that individuals with the inattentive subtype experience deficits in focussed and selective attention and speed of information processing, whereas the ADHD-HI subtype has deficits primarily in sustained attention, behavioural inhibition, and affect regulation. Children with ADHD-C, but not those with ADHD-HI are significantly more impaired on measures of executive functions and motor deficits (Tervo, Azuma, Fogas, & Fiechtner, 2002). Barkely (1997) posits that Executive Function deficits are central to the conceptions

of ADHD-C. In their study Nigg et al. (2002) have noted that ADHD-C is associated with low IQ when compared with the control group.

The present study has successfully identified that it is the ADHD-C and the ADHD-PI subtypes of ADHD that clearly exhibit the deficit in cognitive and executive functions.

With the exception of Digits Backwards, a common pattern was observed in all the tests used in the present study, the ADHD-C and the ADHD-PI performed more poorly than ADHD-HI, ODD and CD subgroups. Of the ADHD subtypes the ADHD-HI performed as well as the non-DBD control groups and other DBD subtypes i.e. ODD and CD. ODD and CD are not associated with deficits in planning and working memory as found in ADHD-PI and ADHD-C in the current study, but instead with enhanced performance on these measures (Oosterlaan et al., 2005). Most research studies of ADHD, ODD and CD focus on ADHD as a unitary group and not according to ADHD subtypes resulting in obscured effects of the interrelationships between them that affect their conclusions. Hyperactivity and impulsiveness appears to be less impairing than inattention in terms of cognitive function, as it is mainly children with a ADHD-PI and ADHD-C diagnosis have been found to be most severely impaired. This supports the notion that ADHD-C may have different aetiology from ADHD-HI (Johansen et al., 2002) and therefore also different from ODD and CD subtypes whose performance is at about the same level as the ADHD-HI subtype.

Individuals with ADHD-PI are often more socially withdrawn and experience greater academic problems and develop comorbid anxiety or other mood problems, while those with early hyperactive-impulsive behaviour are associated with externalising problems like aggression, oppositional behaviour, adolescent delinquency and substance abuse (Johansen et al., 2002). Hyperactivity and impulsiveness, but not inattention

predicted criminal activity in children (Wechsler, 1991). The attentional problems of children with ADHD-PI subtype are general and non-specific, related to poor focused or selective attention and less accurate information processing and is more associated with reduced levels of IQ (Johansen et al., 2002), which is also more evident in the ADHD-C subtype (Nigg et al., 2002), and may manifest in cognitive deficiencies and academic underachievement. Nigg and his co-workers (2002) have also suggested that the ADHD-C subtype and the ADHD-PI subtype are related disorders that share deficits in vigilance and effort functions and on other measures, with them differing only in severity. That ADHD-C and ADHD-PI did not differ from one another in measures of cognitive and neuropsychological function is consistent with findings by Geurts et al. (2005).

The ADHD-PI subtype and particularly the ADHD-C subtype appear to have more deficits in both the cognitive and motor control areas. An association was reported between inattentive symptomatology and poor motor skills, particularly fine motor skills, and that the ADHD-C subtype is also associated with poor gross motor skills (Piek et al., 1999; Pitcher et al., 2003). The low performance in tests of Executive Functions and motor functions suggest that these children fail to plan ahead before they engage in some action (Lezak et al, 2004), and is also evidence of poor gross and fine motor skills (as can be observed in the performance on Blocks Design and Maze Coordination tasks) . In the current study, the ADHD-C and ADHD-PI subgroups accordingly appear to have more problems in generating and following plans, organising behaviour, and forgetfulness than those diagnosed with ADHD-HI, ODD and CD.

Executive functions are a summary of psychological processes involved in organisation and planning of behaviour (Hale & Fiorello, 2001; Johansen et al., 2002). The reason for the difference in the results of the ADHD-C and ADHD-PI subtypes and that of the ADHD-HI subtype could be due to the fact that tests and measures of aspects of

neuropsychological functions tapped for ADHD-HI and those for ADHD-PI or ADHD-C are different. Researchers have argued that overactivity in children with ADHD might not always be present as it happens to be absent in novel situations (Sagvolden et al., 2005b) and this could explain the findings that impairment in ADHD-HI was not as seriously affected as in the ADHD-PI subtype, particularly the ADHD-C subtype. It appears therefore that in situations when the ADHD-HI subtype could perform optimally, the ADHD-C and ADHD-PI subtypes will show poorer performances.

Impaired timing, poor motor control, longer reaction times, poor response timing, as seen in children with ADHD-PI and ADHD-C are associated with dysfunctioning in nigro-striatal dopamine branch (Johansen et al., 2002). Research has found that dopamine dysfunctioning in the neostriatum is associated with impaired motor control (Sagvolden & Sergeant, 1998). Dysfunction in the frontal striatum has been implicated in disinhibition, self-regulation deficits, and impaired attention which are the characteristics exhibited by ADHD-C (Teeter & Semrud-Clikeman, 1995), whereas disruption in the brain stem and posterior region including the parietal lobes have been considered to play a causative role in ADHD-PI (Goodyear & Hynd, 1992; Posner, 1992).

Poor performance of children with ADHD-C and ADHD-PI could be a reflection of a motivational deficit, which could be a failure to achieve and maintain optimal performance (Oosterlaan & Sergeant, 1998). Given that the performance of ADHD-HI is the same as that of the non-DBD control group, if the results of the current study were analysed with ADHD as a unitary group and not separated according to subtypes, the of significance of the difference in performance could be different. The present findings suggest that the ADHD-C and ADHD-PI subtypes show more deficits in motor and executive functions than the ADHD-HI subtype.

Another explanation of the poor performance of the ADHD-PI and ADHD-C subtypes could be due to developmental delay in the brain development of these children. Slow development of the brain and under-developed cognitive structures necessary for optimal functioning may be present in these subtypes when compared with the ADHD-HI and other DBD subtypes. Developmental theories suggest that neuropsychological and cognitive deficits result from a maturational lag of the brain or of permanent neurological impairment (Oosterlaan et al., 2005). EF shows a gradual development from childhood, minor deficits in childhood may cause a cascade of future deficits leading to gross deficits that may translate into severe impairment in the ability to plan, execute, verify and regulate goal directed behaviour (Caspi, Lynam, Moffitt, & Silva, 1993; Lynam, 1998). Raine, Buchsbaum, and LaCasse, 1997; Coccaro, Kavousi, Cooper, and Hauger, (1997) maintain that the individual's risk for Conduct Disorder include ADHD, high impulsiveness, low intelligence and weakness of the executive functioning of the brain which are associated with decreased prefrontal gray matter and glucose hypometabolism in the medial-orbito-frontal region, and decreased level of brain serotonin, low autonomic activation that is, low resting heart rate, low skin conductance, and weak mobilisation of endocrinologic stress responses (van Goozen, Matthys, Cohen-Kettenis, Gispen-de Wied, Wiegant, & van Engeland, 1998). Research has shown that ADHD children are more likely to have developmental delays and cognitive deficits than those with conduct disorders (Quay, 1999). ADHD could be associated with a delay in the maturation of the frontal cortex and its sub-cortical connections of the brain or a permanent impairment suggesting a neurological deficit that remain stable across development (Oosterlaan et al., 2005).

8.4 Limitations of the study

The screening and selection process of the participants in this study was done in accordance with the DSM classification. While the ADHD subtypes in this study were not

comorbid with other DBD subtypes, participants in the clinical group had only two cases each of pure cases of CD and ODD, the rest of the CD and ODD subtypes were comorbid with ADHD. It therefore follows that most CD participants also had comorbid ODD. This is also in accordance with the view that because very few children receive formal diagnosis of ODD and CD most researchers prefer a dimensional rather than a categorical approach (Campbell & Pierce, 1996). The fact that the clinical group was only screened by teachers not properly diagnosed poses another possible limitation.

Although the tests used in this study are simple, inexpensive and easy to use across different cultures, they were all standardized in Western countries (Meyer, 2005a). Cultural factors are important determinants of childrearing practice, and may therefore affect performance on neuropsychological measures.

Other comorbid psychiatric conditions such as anxiety, depression, learning disorders, and pervasive developmental disorders that have not been formally diagnosed before testing, could affect the performance on neuropsychological tests.

Another limitation of this study is the extent to which the results of this study can be generalized to overall population, since the participants were predominantly Tsonga speaking and of low Socio-Economic Status.

Furthermore, the reliability of the results can be enhanced by making use of a larger sample of the DBD subtypes.

8.5 Clinical implications and possibilities for further research

It would be interesting to replicate these findings in a study that will include a much bigger sample of the participants from both high and low socio-economic status, from different ethnic groups and from both rural and urban areas. Further research should strive to investigate and generate knowledge about differences in performance between

cases of "pure" categories of ODD and CD, comorbid conditions and ADHD. Given the results that supports the suggestion that the ADHD-C subtype could have different aetiology than ADHD-HI subtype (Johansen et al., 2002), more research needs to be done to determine whether these disorders could be explained differently, or whether they need to be separated as most research in ADHD does not study the subtypes as separate and findings that are made are more often explained in terms of ADHD inclusive, and not as individually different subtypes isolated into ADHD-HI, ADHD-PI and ADHD-C.

More research should be done to determine whether the effect of comorbid ADHD-PI with ODD or CD, and ADHD-HI with ODD and CD will produce a difference on measures of executive and motor functions. Studies that determine how much of the ADHD-C, ADHD-PI and that of the ADHD-HI develops into ODD and CD will make a valuable contribution to research on ADHD.

It is important to note that children with ADHD are not always cognitively impulsive as they temporarily do manage to plan ahead, organise themselves and remember important things (Johansen et al., 2002). In children with ADHD both motor and cognitive impulsiveness may not occur in some situations such as when reinforcements are used. It may be possible that the performance of ADHD-HI children and that of ADHD-C children could be different in different situations depending on how each subtype reacts given the conditions of the situation.

The results of the current study indicate that among the DBDs, neuropsychological and cognitive functioning is severely impaired in children with ADHD-PI and ADHD-C. ADHD predisposes children to develop ODD and CD. In a study by Burke, Loeber, Lahey, and Rathouz (2005) ADHD predicted ODD, which excessively predicted CD. Of all the DBDs, ADHD-C children are at the greater risk of needing attention to help improve their cognitive functioning. Early diagnosis of children with ADHD and early

intervention will help minimize potential learning problems and offsets risk of development of other problems associated with DBDs which could develop from ADHD. Considering that a number of studies on cognitive and neuropsychological impairments have been about DBD and ADHD without concern of the ADHD subtypes, this research seeks to contribute towards analyzing the results of the DBDs identifying and analysing them according to subtypes of ADHD-HI, ADHD-PI, ADHD-C.

The findings that cognitive impairment is associated with ADHD-C and ADHD-PI, but not with ADHD-HI, ODD nor CD implies that goals and strategies for intervention should be different when learning problems have been diagnosed. The cause of school drop out of the ADHD-C and ADHD-PI subtypes could result from difficulties associated with the cognitive and motor deficits leading to continuous failure and successive repeating of grades, while with ADHD-HI and other DBDs (i.e.ODD and CD) it may be the result of repeated behaviour problems associated with oppositional and conduct problems such as failure to cooperate with authority and aggressive behaviours. Such behaviours may elicit punishment and negative emotional reactions from authorities, parents and peers.

8.6 Concluding remarks

Children with ADHD-C and those with ADHD-PI are significantly more impaired on measures of cognitive and motor functions than those with ADHD-HI, ODD and CD and children without externalizing behaviours. The absence of comorbidity of other DBDs (ODD and CD) in children diagnosed with ADHD-PI and ADHD-C shows that the impairment in cognitive and motor functions are located in ADHD particularly in the inattention component

There could be much truth in the suggestion that poor academic progress in children with ADHD may be the results of Working Memory deficiencies rather than a

direct consequence of their behavioural symptoms of inattention and/or hyperactivity-impulsivity (Rapoport, Giedd, Blumenthal, Hamburger, Jeffries, Fernandez, et al., 1999).

Inattentive and impulsive behaviours invite negative feedback (from peers, parents, and teachers) in children with ADHD-C, and also those with ADHD-PI, putting them at risk for failure leading to dropping out of school.

In the light of these findings, it can be suggested that children with ODD or CD comorbid with ADHD-C could be more likely to experience academic underachievement. However this poses a challenge that needs further investigation. The prevalence of these disorders in school populations seem to be lower than is actually the case because of school drop out as a result of reprimands and alienation by teachers and peers due to behavioural problems or social deficits associated with ADHD and academic deficits displayed by continuous underachievement.

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**University of Limpopo
School of Social Sciences
Psychology**

The Principal
 ………………….. Primary School

Dear Sir / Madam,

Research project: Attention-Deficit/Hyperactivity in the Limpopo Province

Attention-Deficit/Hyperactivity Disorder (ADHD) is a developmental disorder, which affects between 2% and 5% of primary school children. It consists of problems with impulse control, attention span, and activity level. However, it is much more than a matter of being inattentive and overactive. The disorder is an obstacle to benefit from normal education methods and to form acceptable social relations. It is not a temporary state that will be outgrown, for most of the children will still be suffering from the disorder as adolescents and adults.

The child usually is disorganized, has problems with planning his/her activities and may be very forgetful. There are severe problems with sustained attention, especially in the classroom situation. The child has also problems with sitting still, is overactive and fidgety. Problems with gross and fine motor coordination are frequent.

The cause of ADHD is not known yet, but research suggests a genetic origin. Pollutants and poor nutrition may also play a role. It is not caused by failure to discipline or control the child. ADHD children not diagnosed and treated at an early age are at risk for future delinquent behaviour, psychiatric problems, and substance abuse. The financial cost for the society will be considerable. The families of these children experience undue stress and it has severe impact on academic activities at schools.

Diagnosis of ADHD has always caused a problem. Up to now, all instruments, which are used for the diagnosis of ADHD, are rating scales completed by teachers and/or parents and are usually culturally biased and have to be translated into all the official languages. These rating scales are mostly inaccurate because of the subjectivity of the rater. Especially in South Africa, with its many culture and language groups, the rating methods is often invalid.

The Department of Physiology, University of Oslo, Norway has therefore developed a culture-free, non-verbal test sensitive to impulsiveness, inattention and motor activity, the three major symptoms of ADHD. Together with tests for planning deficiencies and fine motor co-ordination, we are hoping to have been implicated to play a role in the disorder. This project is funded by the Norwegian Programme for Development related Research and Education (NUFU).

Postgraduate students from both the University of the Limpopo and University of Oslo also form part of the research team.

Method:

The research team will visit the participating school and will screen the pupils for ADHD. This Disruptive Behaviour Disorder Rating Scale-DBD- (Pelham, Gnagy, Greenslade, and Milich, 1992) will be used. This scale, which is standardized for use with all the population groups of the Province (Meyer, Eilertsen, Sundet, Tshifularo, and Sagvolden; 2004) will be filled in by the child's class teacher. The screened children, who comply with the ADHD criteria, will then undergo further testing. The following will be administered:

- Biographical data questionnaire

- Tests for fine motor co-ordination

- Tests for planning abilities

- Test for overactivity, impulsiveness and impaired sustained attention

The data will be used for statistical analysis only and in no circumstances will the identity of the child and the school be revealed.

Your approval of this very important study will contribute to the establishment of a valid diagnostic method, which will enable professionals to identify children at risk for educational, social and emotional problems.

Yours Sincerely

…………………………….

Prof Anneke Meyer
Project Leader

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Dear Parents,

Research project: Attention-Deficit/Hyperactivity in the Limpopo Province

Attention Deficit/Hyperactivity Disorder or AD/HD is a disorder, which affects between 2% of primary school children. The child has difficulty paying attention, controlling his or her activity and is impulsive. However, it is much more than a matter of being inattentive and overactive. The child has problems in coping with his or her schoolwork and may not be getting along well with other children. They are also unable to complete assigned tasks without supervision and cause disruptions in the family.

The problems may cause that the child is unable to adjust to the normal requirements of ordinary life. They are not likely to be outgrown and could cause future problems with reckless behaviour, possible risk of law-breaking and drug abuse. The disorder is more common in boys than in girls.

The cause of AD/HD is not known yet, but research suggests that it may be an inherited condition. Pollutants and poor nutrition may also play a role. It is not caused by failure to discipline or control the child. The children benefit from medication.

It is extremely important that these children are diagnosed and treated at an early age so that suffering at home and at school can be prevented and the child may not be at risk for future behaviour.

Yours Sincerely

…………………………….

Prof Anneke Meyer
Project Leader

Consent form:

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I, mother, father, guardian of _____ hereby give my consent for my child to be tested by the Psychology team of the University of Limpopo.

Signed: _____

Appendix C

Teacher / Parent DBD Rating Scale

Child's name: _____   Form completed by: _____

Sex: M/F   Age: _____

Grade: _____

Home language: English /Afrikaans/ N-Sotho/ Xitsonga/ Tshivenda/ Setswana/Sesotho iZulu/Other: _____

Check the column that best describes this child. Please put a question mark next to any item for which you do not know the answer.

	Not at All	Just a Little	Pretty Much	Very Much
1. often interrupts or intrudes on others (e.g, butts into conversations or games)				
2. has run away from home overnight at least twice while living in parental or parental surrogate home (or once without returning for a lengthy period)				
3. often argues with adults				
4. often lies to obtain goods or favours to avoid obligations (i.e., "cons others)				
5. often initiates physical fights with other members of his or her household				
6. has been physically cruel to people				
7. often talks excessively				
8. has stolen items of nontrivial value without confronting a victim (e.g. shoplifting, but without breaking and entering; forgery)				
9. is often easily distracted by extraneous stimuli				
10. often truant from school, beginning before age 13 years				
11. often fidgets with hands or feet or squirms in seat				
12. is often spiteful or vindictive				
13. often blames others for his or her mistakes or misbehaviour				
14. has deliberately destroyed others' property (other than by fire setting)				
15. often actively defies or refuses to comply with adults' request or rules				
16. often does not seem to listen when spoken to directly				
17. often blurts out answers before questions have been completed				

	Not at all	Just a little	Pretty much	Very much
18. often initiates physical fights with others who do not live in his or her household (e.g. peers at school or in the neighbourhood)				
19. often has difficulty playing or engaging in leisure activities quietly				
20. often fails to give close attention to details or makes careless mistakes in schoolwork, work or other activities)				
21. is often angry and resentful				
22. often leaves seat in classroom or in other situations in which remaining seated is expected				
23. is often touchy or easily annoyed by others				
24. often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behaviour or failure to understand instructions)				
25. often looses temper				
26. often has difficulty sustaining attention in tasks or play activities				
27. often has difficulty awaiting turn				
28. has forced someone into sexual activity				
29. often bullies, threatens, or intimidate others				
30. is often "on the go" or often acts as "if driven by a motor"				
31. often loses things necessary for tasks or activities (e.g toys, school assignments, pencils, books, or tools)				
32. often runs about or climbs excessively in situations in which it is inappropriate				
33. has been physically cruel to animals				
34. often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)				
35. often stays out at night despite parental prohibitions, beginning before age 13 years				
36. often deliberately annoys people				
37. has stolen while confronting a victim (e.g., mugging, purse snatching, extortion, armed robbery)				
38. has deliberately engaged in fire setting with the intention of causing serious damage				
39. often has difficulty organising tasks and activities				
40. has broken into someone else's house, building, or car				
41. is often forgetful in daily activities				
42. has used a weapon that can cause serious physical harm to others (e.g. a bat, brick, broken bottle, knife, gun.				

APPENDIX D: DBD Rating Scale (Tsonga)

Vito ra N'wana:………………………………………………

Fomo yi tatiwile hi:………………………………………………

Siku Fomo yi nga tatiwa hi rona:………………………………

Rimbewu:………………………………………………………..

Vukhale:…………………………………………………………

Ririmi ra le kaya:……………………………………………….

Ntangha:………………………………………………………

Hlawula ndzhawu eka swiyenge swa mune leyi yi hlamuselaka n'wana wa loyi.
U komberiwa ku tsala AT ethlelweni ra nhlamuselo yi n'wana ni yin'wana leyi u nga tiviku nhlamulo ya yona.

	A swi endleki	Swi endleka ka tsongo	Swi endleka ngopfu	Swi endleka ku lula pimo
1. Minkarhi yo tala u nghenelela van'wana (e.g U nghenelela van'wana loko va vulavula kumbe loko va huha (tlanga).				
2. U balekile ekaya a ya etlela enhoveni ku hundza kan'we loko a ha tshama ekaya na vatswari va yena kumbe ekaya laha a hlayisiwaka kona (kumbe u baleka kan'we laha a hetaka nkarhi wo leha a nga si vuya				
3. Hakanyingi u n'an'isana na vatswatswi				
4. Minkarhi yo tala u vulavula mavun'wa leswaku a ta kuma swilo swo karhi kumbe ku pfuniwa ko karhi.Kumbe ku pfuniwa ko karhi kumbe mintirho yo karhi.(Vutihlamuleri byo karhi)				
5. Hakanyingi u sungula tinyimpi (kulwa) na vanhulava a tshamaka na vona emutini ya vona				
6. U kombile tihanyi hi ku vavisa van'wana emirini ya vona				
7. Hakanyingi u vulavula ku tlula mpimo				
8. U yivile swilo swo ka swi nga ri ni nkonka wa le henhla hi ndlela yaleyo a nga kongomani na n'wini wa swilo xikombiso, ku pambula swiloemavhengeleni handle ka ku tshova a nghena endzeni ka vhengele, kumbe ku endla fojari				
9. Hakanyingi u kokiwa miehleketo hi ku olovahiswilo leswi humelelaka laha a nga kona				
10. Hakanyingi u tinghenisa enghozini yo limala emirini a nga khatali hi leswi swi nga ta n'wi humelela (u endla leswi ku ngari hi ku u nghenelrile mitlangu leyi nga na nghozi), xik., u nghena exitarateni hi kutsutsuma a nga langutanga leswaku u hlayisekile				
11. Hakanyingi u nyenga kumbe u tshama a nga yiexikolweni, mhaka leyi yi sungule a nga si va na malembe ya khumenharhu				
12. Hakanyingi u tlanga hi swandla ni mikondo a				

nyunganyungeka a nga tshamiseki exitulwini				
13. Hakanyingi u ni lunya ni tihanyi				
14. Hakanyingi wa rhukana ni ku tirhisa marito ya hlamba				
15. Hakanyingi u veka nandzu eka vanhu van'wana loko a endlile swihoxo kumbe loko a tikhomile hi ndlela yo biha				
16. U onhile swibye swa vanhu van'wana (ku nga ri hi ku swi hisa hi ndzilo)				
17. Hakanyingi u tsan'wa kumbe ku ala ku landzela (endla) swikombelo kumbe swileriso swa vanhu lavakulu				
18. Hakanyingi u vonaka wonge a nga yingisi loko munhu a vulavula a kongomisa marito eka yena				
19. Hakanyingi u hatlisa ku vula tinhlamulo swivutiso swi nga si vutisiwa swi fika makumu				
20. Hakanyingi u pfuxa tinyimpi ta mavoko na vanhu lava a nga tshamiku na vona endyangwini un'we				
21. Hakanyingi u tshika ntirho wo karhi wu nga si hela a tlulela eka wun'wana				
22. Hakanyingi u tikeriwa hi ku tlanga mitlangu kutani a endla hasahasa				
23. Hakanyingi u tsandzeka ku va ni ku xopaxopa ni vukheta na kona u tala ku endla swihoxo hikwalaho ka vosopfa e ntirhweni wa yena wa xikolo, ntirho wa ku tihanyisa, kumbe mintirho-ntirho yin'wana yo hambana hambana				
24 U tala ku va a kwatile ni ku vilela				
25. Hakanyingi u suka exitulwini xa xikolo kumbe etindzhawini tin'wana laha a faneleke ku va a tshamile ehansi				
26. Hakanyingi u ni xifafa na kona u hatla ku kwatisiwa hi vanhu van'wana				
27. Hakanyingi a nga hetisisi/landzisisi swileriso na swona u tsandzeka ku heta ntirho wa yena wa xikolo, swintirhwa-ntirhwana swa le kaya kumbe vutihlamuleri bya yena bya le ntirhweni (ku ngava hi mhaka ya ku ka a nga swi lavi kumbe ku ka a nga swi twisisi swileriso)				
28. Hakanyingi u kwata hi ku hatlisa				
29. Hakanyingi u tikeriwa hi ku va ni vuxiya-xiya emintirhweni kumbe emitlangwini ya yena				
30. Hakanyingi u hela mbilu a tsandzeka ku yima nkarhi wa yena wu kala wu fika				
31. U tivanile kumbe ku etlelana na un'wana hi ku sindzisa				
32. Hakanyingi u karhata kumbe ku chuhisa van'wana				
33. Hakanyingi u le mikitsikitsimi hi ku ka a nga tshamiseki kumbe ku ka a nga rhuli onge u fambisiwa hi njhini				
34. Hakanyingi u lahla swilavekwa leswi a fanelaka ku swi tirhisa (xik., swo huha hi swona, mintirho leyi va n'wi nyikeke exikolweni, tipenisele, tibuku, na swo tirha hi swona)				

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35. Hakanyingi u tsutsuma-tsutsuma kumbe ku khandziya-khandziya hi laha swi nga fanelangiki (ku fana na loko se munhu a ri jaha/ nhwana kumbe ntswatsi; yi nga ha va mhaka yo munhu wo tano u titwa a nga rhulangi)				
36. U tshama a va ni tihanyi eka swihari hi ku swi vavisa miri				
37. Hakanyingi u tsan'wa, u nyenya, kumbe ku tinonon'whisa eku endleni, mintirho leyi yi lavaka leswaku a chivirika eku tirhiseni miehleketo ya yena				
38. Hakanyingi a nga etleli ekaya hambi loko vatswari va n'wi tshimbisa ku endla mhaka yo tani, mhaka leyi yi sungula loko munhu wo tano a nga si va ni malembe ya khumenharhu				
39. Hakanyingi u nyangatsa vanhu van'wana				
40. U yivile hi ndlela yo kongomana ni muyiveriwa (xik., ku tlimba munhu un'wana u n'wi tekela swa yena, ku vutla xipaci, ku koxa mali hi ku chavisa kumbe ku xungeta, ku tekela van'wana mali kumbe mpahla hi ku tirhisa swolwa).				
41. U hisile swilo hi vomu hi xikongomiso xo endla ku onha ku kulu				
42. Hakanyingi u tikeriwa hi ku lulamisa mintirho ya yena				

Block Design (from SSAIS)

Appendix F
Memory for Digits Test (from SSAIS)

TEST 10: MEMORY FOR DIGITS/TOETS 10: SYFERGEHEUE

DIGITS FORWARD/GEWONE VOLGORDE

DISCONTINUE Digits Forward after 2 consecutive zero scores and continue with Digits Backward.

STAAK Gewone Volgorde na 2 agtereenvolgende nultellings en gaan aan met Omgekeerde Volgorde.

Item	Series 1 Reeks 1	Right/Wrong Reg/Verkeerd + / -	Series 2 Reeks 2	Right/Wrong Reg/Verkeerd + / -	Score Telling
1.	1 - 4		6 - 3		
2.	2 - 8 - 5		1 - 7 - 4		
3.	4 - 5 - 1 - 8		2 - 6 - 4 - 1		
4.	6 - 9 - 2 - 8 - 3		7 - 2 - 9 - 6 - 5		
5.	3 - 8 - 4 - 7 - 1 - 9		5 - 2 - 7 - 8 - 1 - 6		
6.	3 - 6 - 7 - 8 - 2 - 4 - 9		9 - 3 - 2 - 6 - 4 - 7 - 5		
7.	8 - 5 - 6 - 2 - 7 - 9 - 5 - 3		2 - 5 - 3 - 8 - 6 - 7 - 3 - 9		
8.	6 - 4 - 8 - 5 - 1 - 3 - 2 - 9 - 7		4 - 6 - 5 - 7 - 2 - 3 - 8 - 9 - 1		

DIGITS BACKWARD/OMGEKEERDE VOLGORDE

DISCONTINUE Digits Backward after 2 consecutive zero scores.

STAAK Omgekeerde Volgorde na 2 agtereenvolgende nultellings.

Item	Series 1 Reeks 1	Right/Wrong Reg/Verkeerd + / -	Series 2 Reeks 2	Right/Wrong Reg/Verkeerd + / -	Score Telling
1.	3 - 1		5 - 3		
2.	2 - 7 - 5		4 - 9 - 2		
3.	9 - 1 - 8 - 2		7 - 3 - 4 - 8		
4.	3 - 5 - 2 - 6 - 7		8 - 3 - 4 - 6 - 2		
5.	8 - 2 - 9 - 4 - 7 - 5		3 - 1 - 9 - 6 - 8 - 4		
6.	6 - 3 - 8 - 9 - 1 - 4 - 7		6 - 1 - 3 - 7 - 5 - 2 - 8		
7.	4 - 6 - 1 - 5 - 9 - 2 - 7 - 8		7 - 3 - 6 - 9 - 6 - 5 - 1 - 4		

Total score for Memory for Digits Test/Totaaltelling vir Syfergeheuetoets

Comments/Opmerkings: _____

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Tower of London
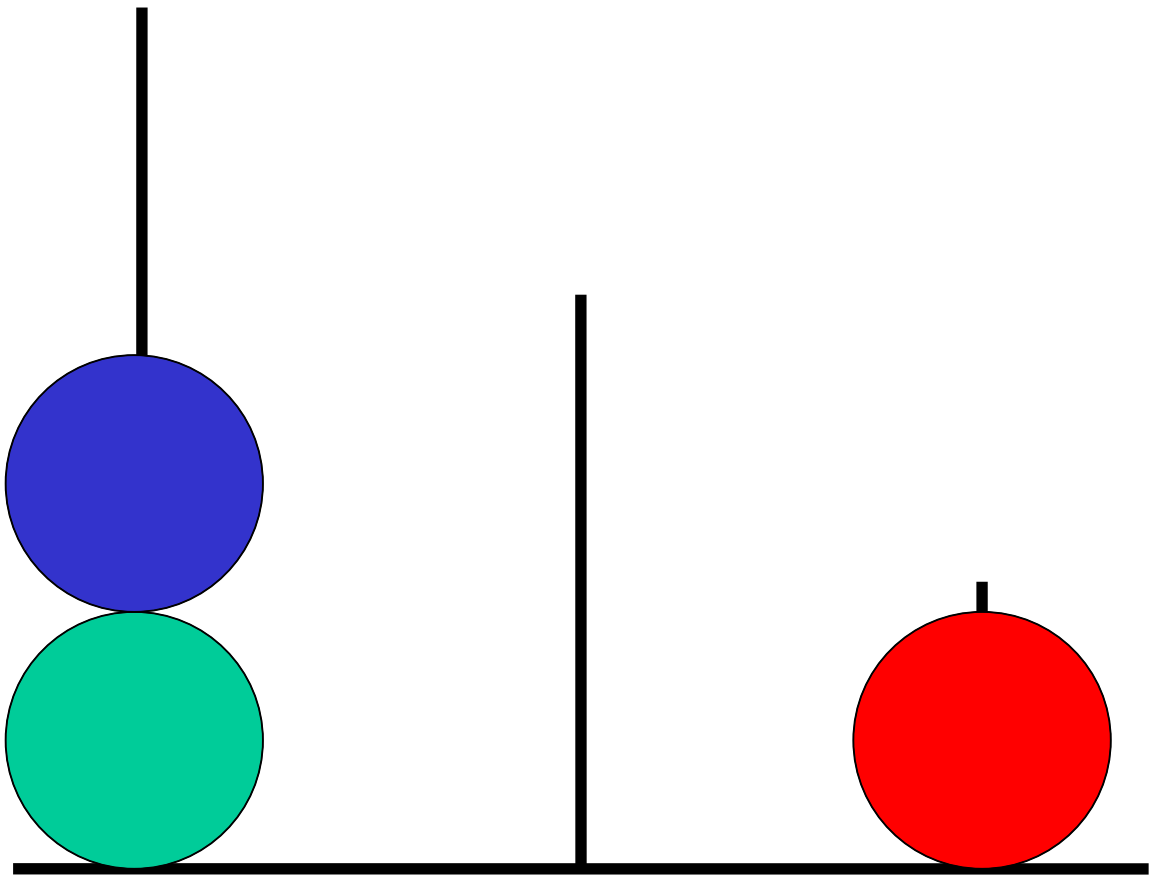
Example: 2 moves

Appendix H

Maze Coordination Task