COMORBID DISORDERS IN PRIMARY SCHOOL CHILDREN WITH ATTENTION DEFICIT/HYPERACTIVITY DISORDER

by

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DECLARATION

I, Busisiwe Siwelani, declare that the dissertation hereby submitted to the University of Limpopo as partial fulfilment for the degree of Master of Arts in 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 ……...

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ABSTRACT

Background: Although ADHD does occur as a single disorder in a minority of diagnosed individuals, it is generally comorbid with other behavioural and emotional disorders. The most frequent co-occurring psychiatric disorders are Oppositional Defiant Disorder, Conduct Disorder, Anxiety Disorder and Mood Disorder. The aim of the study was to establish a relationship between the core symptoms of ADHD (Inattention and Hyperactivity/Impulsiveness) and the externalising disorders, Oppositional Defiant Disorder and Conduct Disorder, and the internalising disorders anxiety and depression and to establish differences in comorbid symptoms (ODD, CD, anxiety, and depression) between children with ADHD and a non-ADHD control group as a function of gender and subtype.

Method: A total of 100 Tsonga speaking primary school children (50 with ADHD symptoms and 50 non-ADHD controls) participated in the study. The ODD and CD scale of the Disruptive Behaviour Disorders rating scale was used to establish the symptoms of ODD and CD, while the Anxiety and Depression scales of the "Terry" Picture Questionnaire was used to establish the symptoms of Anxiety and Depression.

Results: Findings of the study showed that the relationship between the hyperactive/impulsive component of ADHD and the externalizing disorders (ODD and CD) was moderate to strong, while there was no significant relationship between hyperactivity/impulsiveness and the internalizing disorders (anxiety and depression). The inattentive component of ADHD showed a strong relationship with ODD, but no significant relationship with CD was observed. The relationship of inattentive symptoms with the internalizing disorders (anxiety and depression) was weak to moderate.

The comparison study showed that boys with the predominant hyperactive/impulsive subtype of ADHD had significantly more symptoms of CD than

their non-ADHD counterparts, but not significantly more ODD symptoms, while girls of the predominantly hyperactive/impulsive subtype had significantly more symptoms of both ODD and CD than the non-ADHD girls. A comparison of the predominantly inattentive subtype of ADHD showed that the boys had more symptoms of ODD than their non-ADHD counterparts, while there were no significant extra symptoms for CD. The predominantly inattentive girls did not show more symptoms of both externalizing disorders than the non-ADHD controls. The ADHD-combined subtype had more symptoms of both disorders but no gender differences were observed. When the ADHD subtypes were compared with non-ADHD controls for internalizing disorders, only the predominantly inattentive subtype showed significantly more symptoms of both anxiety and depression. This was the case for both genders.

Conclusion: The study showed that there is a relationship between the symptoms of ADHD (hyperactivity/impulsiveness and inattention) and the externalizing disorders, ODD and CD. Only the Inattentive component of ADHD showed a relationship with internalizing disorders (anxiety and depression).

The comparison study showed that the predominantly hyperactive/impulsive and the combined subtypes displayed most symptoms of externalizing disorders, while the predominantly inattentive subtype had significantly more symptoms of internalizing disorders. Gender differences were observed in the symptoms of externalizing disorders, but not in the symptomatology of internalizing disorders.

Chapter 1

GENERAL INTRODUCTION

1.1 Introduction

Attention Deficit/Hyperactivity Disorder (ADHD) is a chronic, debilitating disorder which may impact upon many aspects of an individual's life, including academic difficulties (Seidman, Biederman, Monuteaux, Doyle, & Faraone 2001), social skills problems (Bagwell, Molina, Pelham, & Hoza, 2001), and strained parent-child relationships (Johnston & Mash, 2001). The disorder consists of a persistent pattern of inattention, hyperactivity and impulsiveness that is inconsistent with the child's developmental level (American Psychiatric Association, 2000). Whereas it was previously thought that children eventually outgrow ADHD, recent studies suggest that 30-60% of affected individuals continue to show significant symptoms of the disorder into adulthood (Weiss & Hechtman, 1993). Its onset is in early childhood, by definition before the age of 7, nearly always before the age of 5 and frequently before the age of 2 years (Barkley, 2006). About half of the children diagnosed as having ADHD grow up to have psychiatric disorders later on in life (American Psychiatric Association, 2000). The consequences of the disorder are therefore extremely costly, both to the affected individuals, their families and to the society (Harpin, 2005).

Taylor, Dopfner, Sergeant, Asherson, Banaschewski, and Buitelaar (2004) postulated that Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) are very common in ADHD. They should be seen, not necessarily as a differential diagnosis but as a complication. It seems to be the main cause of early onset of Conduct Disorder and twin studies suggest that hyperactive behaviour may be the main genetic pathway into the development of Conduct Disorder (Taylor et al., 2004). Cantwell (1996) maintains that

internalising problems, such as anxiety and mood disorders may be underreported by parents or teachers, because externalising forms of behaviour are more observable. Children with attention deficits without hyperactivity have higher rates of anxiety disorders than those with hyperactivity. Therefore, ADHD with comorbid anxiety disorder may imply substantially worse outcomes in children with both disorders (Spencer, Biederman, & Wilens, 1999).

ADHD with comorbid affective illness may have a distinct clinical presentation, aetiology, course, and outcome. Therefore, understanding the characteristics associated with comorbidity in children with ADHD is a critical step in designing appropriate diagnostic (Jensen, Martin, & Cantwell, 1997), prevention and treatment options (Jensen, Hinshaw, Kraemer, Lenora, Newcorn, & Abikoff et al., 2001).

The combination of ADHD and a depressive disorder could represent a subtype of ADHD, with both shared and specific features related to aetiology, outcome, and clinical presentation (Jensen, 2003; Keiley, Lofthouse, Bates, Dodge, & Pettit, 2003). Angold, Costello and Erkanli, (1999) suggest, that the association between depression and ADHD may be epiphenomenal, that is, attributed to the relationship that both disorders have with anxiety or conduct disorders.

1.2 Purpose of the study

The main goal of the research is to show a relationship between the primary disorder (ADHD), and the comorbid disorders, the Externalizing Disorders, Oppositional Defiant Disorder (ODD), and Conduct Disorder (CD) and the Internalizing disorders, Anxiety Disorders and Mood Disorders and to compare children with ADHD with a non-ADHD control group for the symptoms of those comorbid disorders.

The aim of the investigation is therefore twofold:

1. To establish a relationship between the symptoms of the various comorbid disorders (internalising as well as externalising) and the symptoms of ADHD (Inattention and Hyperactivity/Impulsiveness).
2. To establish differences in the symptoms of comorbid disorders (internalising and externalising) between a group of children classified as having ADHD and a non-ADHD control group as a function of gender and subtype.

1.3 Significance of the study

ADHD is a common problem in children; it is diagnosed in about 30-50% of patients who are referred to child psychiatric clinics (Biederman, 2005). The prevalence of ADHD in the community has been reported to be from 1.7 to 16% (American Psychiatric Association, 2000; Barkley, 2006; Taylor, Dopfner, Sergeant, Asherson, Banaschewski, & Buitelaar et al., 2004).

More than 50% of patients with Attention Deficit/Hyperactivity Disorder have comorbid disorders (Biederman, Faraone, & Lapey, 1992; Biederman, Faraone, Keenan, & Tsuang, 1991; Pliszka, 2000). About 30-40% of children with ADHD have Oppositional Defiant Disorder (August, Realmuto, MacDonald, Nugent, & Crosby, 1996), 30-50% have Conduct Disorder (Biederman et al., 1992), 38% have Depressive Disorders (Pliszka, 1998), and 25% have Anxiety Disorders (Jensen, Hinshaw, Kraemer, Lenora, Newcorn, & Abikoff et al., 2001).

According to Hechtman, (2000) comorbid disorders affect the manifestation, severity of symptoms, prognosis and treatment response in patients with ADHD. For example, when CD is comorbid with ADHD, it makes ADHD symptoms more severe and patients exhibit more aggression, anxiety and poor relationships (Barkley,

Anastopoulos, Guevremont, & Fletcher, 1992). According to Biederman, Faraone, Mick, Wozniak, Chen, Quellette et al. (1996), comorbid disorders such as Conduct Disorders, Affective Disorders and Anxiety Disorders are predictive factors that affect the chronic course of ADHD. The presence of anxiety disorders is associated with a poor response of ADHD symptoms to stimulant medication (DuPaul, Barkely, & McMurray, 1994; Pliszka, 1998). It is therefore important that comorbid disorders of ADHD should be thoroughly investigated as they may influence diagnosis, treatment and outcome of the disorder.

1.4 Delineation of the study

The study represents an attempt to answer comorbid disorders and behavioural issues in ADHD. Chapter 2 provides the general background of ADHD as a childhood disorder. Chapter 3 focused on ADHD: Mimicking disorders and comorbidity. Chapter 4 discussed in detail, the Externalising Disorders: Oppositional Defiant Disorder and Conduct Disorder. Chapter 5 discusses in detail, the Internalising Disorders: Anxiety and Mood Disorders. Chapter 6 outlines the problem statement as well as formulates the research hypotheses. Chapter 7 describes the methodology. Chapter 8 gives a representation of the results of the study. Chapter 9 presents the discussion of results and possible areas for future research, limitations of the study and clinical implications.

Chapter 2

ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

2.1 Introduction

ADHD is one of the most common childhood psychiatric disorders and affects between 3% and 5% of children world-wide (American Academy of Pediatrics, 2000; Meyer, Eilertsen, Sundet, Tshifularo, & Sagvolden, 2004; Taylor et al., 2004). ADHD is a condition characterized by different levels of inattention, hyperactivity, and impulsiveness and gives rise to significant academic, social, and emotional problems at home and at school (Taylor et al., 2004). Academically, children with ADHD often underachieve or fail in school. Socially, they have poor relationships with peers, teachers, and parents. Emotionally, they often have poor self-esteem and are at considerably increased risk for depression, anxiety and delinquent behaviour (Barkley, 2006).

2.2 Historical Background

Attention Deficit/Hyperactivity Disorder was first described 100 years ago by Still (1902) as a childhood disorder found mainly in boys. These children were often aggressive, defiant, resistant to discipline, and excessively emotional or "passionate" and showed little "inhibitory volition." Still believed that this behaviour was not the result of poor parenting or moral weakness, but had a biological cause, either inherited or caused by parental injury. Still and Tredgold became instrumental in determining characteristics that are still valid today (Still, 1902; Tredgold, 1908).

An outbreak of an encephalitis epidemic in 1917-1918 led to interest in the precursors of Attention Deficit/Hyperactivity Disorder in North America. The disease was also known as "Postencephalitic Behaviour Disorder." Many children survived the epidemic, but were left with residual behavioural and cognitive sequelae (Cates, 2002; De Arnas, 2001). Such children were described as being impaired in attention, regulation of

activity, and impulsiveness as well as other cognitive abilities, including memory, and were often noted to be socially disruptive.

The association of brain disease with behavioural pathology apparently led early investigators to study other potential causes of brain injury in children and their behavioural manifestations for example, birth trauma; other infections such as measles, lead toxicity, epilepsy, and head injury were studied in children and were found to be associated with numerous cognitive and behavioural impairments, including the triad of ADHD symptoms (Barkley, 2006).

In the 1950s -1960s these children were classified as having "hyperkinetic impulse disorder" (Barkley, 2006). Hyperactivity was accepted as a brain damage syndrome. Chess (1960) was the first to describe hyperactivity. She used the term "hyperkinetic child syndrome." She emphasized activity as the defining feature and the concept of syndrome of hyperactivity was separated from that of brain damage syndrome. The association of hyperactivity with brain damage led to the conclusion that children who were hyperactive necessarily also had brain damage. The term minimal brain dysfunction became a widely used general term to describe these children (Cates, 2002; Gilles-Thomas, 1989; Rafalovich, 2001).

In 1937 hyperactive children were first treated with the stimulant dextroamphetamine known as Benzedrine (Bradley, 1937). Later studies would also confirm such a positive drug response in half or more of hyperactive hospitalized children (Laufer, Denhoff, & Solomons, 1957). As a result, by the 1970s stimulant medications had become the treatment of choice for the characteristics of Attention-Deficit/Hyperactivity Disorder (Barkley, 2006).

In the 1950s, researchers began a number of investigations into the neurological mechanisms underlying these behaviour symptoms (Laufer et al., 1957). These writers referred to ADHD children as having Hyperkinetic Impulsive Disorder and reasoned that

the central nervous system (CNS) deficit occurred in the thalamic area. Their study was based on a study of the effects of the photometrozol method in which the drug metrozol is administered while flashes of lights are presented to the child. These findings suggested that hyperactive children had a lower threshold for stimulation in the thalamic area

In the 1970s there was an explosion of research interest into hyperactivity (Barkley, 2006). Virginia Douglas was the first to focus on "cognitive impulsiveness," then described as daydreaming and lack of attention. Her model culminated in the view that four major deficits could account for symptoms of ADHD: (1) The investment, organization, and maintenance of attention and effort (2) The inhibition of impulsive responding (3) problems regulating arousal levels (4) An unusually strong inclination to seek immediate reinforcement. She argued that deficits in sustained attention and impulsive control were more likely to account for the difficulties seen in these children than hyperactivity (Douglas, 1972).

Douglas (1972) was credited as being influential in the fact that the American Psychiatric Association created a new diagnostic category, Attention-Deficit/Hyperactivity Disorder, with or without hyperactivity in the eighties (DSM-III) (American Psychiatric Association, 1980). In this revised official taxonomy, deficits in sustained attention and impulse control were formally recognized as of greater significance in the diagnosis than hyperactivity. Hyperactivity was induced in the Diagnostic and Statistical Manual of Mental Disorders, for the first time in the second edition (American Psychiatric Association, 1968).

The third edition of the DSM was published as the decade of the 1980s opened. It contained a radical reconceptualisation of hyperactivity, changing from hyperkinetic reaction in children in DSM-II (American Psychiatric Association, 1968), to Attention Deficit Disorder, in DSM-III (American Psychiatric Association, 1972) when the DSM-III was revised in 1987, Attention-Deficit Disorder became Attention-Deficit/Hyperactivity Disorder (American Psychiatric Association, 1987).

In DSM-IV the essential features of ADHD are a pattern of inattention and/or hyperactivity-impulsiveness, and the diagnosis is currently made on the basis of developmentally inappropriate symptoms of inattention, impulsiveness and motor restlessness (American Psychiatric Association, 1994).

2.3 Primary symptoms

Three core symptoms, which are displayed by children suffering from ADHD, are inattention, impulsiveness and hyperactivity. These characteristics of ADHD are believed to be displayed in the early stages of development and more than is expected (Barkley, 2006). The combination of inattention, hyperactive, and impulsive behaviour are severe, developmentally and impair function at home and school (Swanson, Sergeant, Taylor, Sonuga-Barke, Jensen, & Cantwell, 1998).

2.3.1 Impulsiveness or behavioural inhibition

Impulsiveness is seen as the symptom of greatest significance in children with ADHD (Sagvolden & Sergeant, 1998). Impulsiveness is reflected in an inability to withhold inappropriate responses, such as premature responding, over-rapid responsiveness, excessive attraction to immediate reward, acting without reflecting, reckless and impetuous behaviour (Johansen, Aase, Meyer, & Sagvolden, 2002). These are difficulties frequently identified in children with ADHD (Sagvolden, Johansen, Aase, & Russell 2005). The concept of impulsiveness has both a motor and a cognitive component. Motor impulsiveness is presently defined as bursts of responses with short inter-response times, and this behaviour has been shown to emerge in ADHD children (Johansen et al., 2002; Sagvolden et al., 2005).

Cognitive impulsiveness implies that private events like thoughts and plans are dealt with for short sequences of time with rapid shifts, resulting in problems generating

and following plans, problems organizing own behaviour, forgetfulness and inefficient use of time (Johansen et al., 2002). Impulsiveness implies that judgment or accuracy is compromised for the sake of speed. These children make careless errors because they often respond quickly before the instructions are completed; they act without reflecting and they fail to plan ahead (Halperin, Wolf, Greenblatt, & Young, 1991).

Children with ADHD seem to engage in unnecessary risk-taking behaviours. Accidental poisoning and injuries are common in these children and they may carelessly damage or destroy other's property more frequently than normal children (Halperin et al., 1991). They are often interested in for tasks that require less work to achieve and rather prefer immediate, smaller rewards. They will take shortcuts in their work performance i.e. they are very impatient to wait. Situations or games which require sharing, cooperation, and restraint with peers can be problematic for impulsive children.

Children with ADHD often blurt out answers to questions prematurely and often interrupt conversations of others (Barkley & Murphy, 2006; Taylor et al., 2004). They also find it difficult to stand in a line or wait for their turn (Barkley et al., 2006). Impulsiveness can also be defined as a pattern of rapid inaccurate responding to tasks. In tasks that require a child to take time before responding, they respond more quickly and often make mistakes (Taylor, 1998).

2.3.2 Inattention

Children with ADHD display difficulties with attention, relative to normal children of the same age and gender. The dimension impaired in ADHD reflects an inability to sustain attention or persistence to tasks, remember and follow through on rules and instructions, and to resist distractions while doing so (Barkley et al., 2006). Inattention may manifest as failure to give close attention to details or making careless mistakes. These children look as if they are not listening or hearing what is said. They tend to shift from one incomplete task to the next and often don't follow through on instructions, failing to

complete school work and chores (American Academy of Pediatrics, 2000; Swanson et al., 1998).

They avoid activities that demand self-application and mental effort. They are easily distracted by outside stimuli and are often forgetful. In social situations, inattention may be expressed as frequent shifts in conversation and not listening to others (American Academy of Pediatrics, 2000). Difficulties with attention can also be seen in situations requiring the child to sustain attention to dull, boring, repetitive tasks such as independent school work, or chore performance (Johansen et al., 2002; Taylor, 1998).

2.3.3 Hyperactivity

Hyperactivity is defined as an excessive or developmentally inappropriate level of activity that is typically seen in ADHD as recklessness, fidgeting, and a general increase in unnecessary gross bodily movements (Taylor, 1998; Teicher, Ito, Glod, & Barber, 1996). Although they move twice as frequently and cover a fourfold wider area, their movement pattern is less complex and more linear (side by side), compared with normal controls (Teicher et al., 1996). Overactivity is seen in some situations like the classroom, but may not be present in others like in play and in novel situations (Johansen et al., 2002; Sagvolden et al., 2005; Taylor, 1998). The ratings of hyperactivity and of impulsiveness involve one element of overstepping implicit or explicit social rules and are judged according to situational appropriateness (Taylor et al., 2004).

2.3.4 Problems with motor coordination

Motor problems often characterised as clumsiness or poor motor coordination, have been associated with ADHD in addition to the main symptom groups of inattention, impulsiveness and overactivity (Meyer & Sagvolden, 2006).

The ADHD child's motor ability is frequently significantly lower than would be expected of his age and level of intellectual functioning (American Psychiatric Association, 2000). The wide range of motor problems include delays in achieving motor milestones,

problems with movement planning and execution (reaction time, movement time, accuracy and variability) (Piek & Dyck, 2004). Children with ADHD who experience motor problems often display deficits in requiring complex co-ordinations of movement, such as handwriting (Barkley, 2006; Schoemaker, Ketelaars, Minderaa, & Mulder, 2005). These problems may interfere with the ADHD child's daily functioning and influence their academic performance (Jongmans, Smits-Engelsman, & Schoemaker, 2003).

2.3.5 Variability of behaviour

A feature of ADHD is a marked moment-to-moment fluctuation in task performance (Aase & Sagvolden, 2005; Aase, Meyer, & Sagvolden, 2006; Douglas, 1999; Houghton, Douglas, West, Whiting, Wall, Langsford et al., 1999; Porrino, Rapoport, Behar, Sceery, Ismond, & Bunney, 1983). This has been ignored in the ADHD research field almost entirely until recently, when it was proposed as an aetiologically important characteristic requiring systematic analysis (Castellanos & Tannock, 2002). Behavioural and performance fluctuations are displayed over seconds, minutes, hours or days.

2.4 Diagnostic criteria

In the DSM-IV (American Psychiatric Association, 2000) diagnosis is made according to core symptom lists describing either inattention or overactivity-impulsiveness. There is no single test for the diagnosis of ADHD, but the diagnosis is based largely on careful elicitation and integration of reports by teachers and parents of past and current development and behaviour (Arnsten, 2001; Hudziak, Heath, Madden, Reich, Bucholz, Slutske et al., 1998; Leung, Luk, Ho, Taylor, Mak, & Bacon-Shone, 1996). Physical examination should be done to make sure that no other illness is present that may be the cause of the symptoms (Barkley & Murphy, 2006).

Parental checklists, and accounts from teachers, are valuable in order to make sure that are indeed the presenting problems (Taylor et al., 2004). The DSM-IV (American

Psychiatric Association, 2000) allows either symptoms of hyperactivity-impulsiveness or inattention to be sufficient grounds for diagnosis (Eiraldi , Power, & Nezu, 1997; Leung et al., 1996). Therefore, developmentally inappropriate inattention, impulsiveness, and motor restlessness make up the diagnosis of Attention- Deficit /Hyperactivity Disorder (Shue & Douglas, 1992). According to the DSM-IV (American Psychiatric Association, 2000) it is possible to have ADHD without being inattentive (Sagvolden & Sergeant, 1998). For a child to be diagnosed as having ADHD, the symptoms must be to a degree that is developmentally deviant or leads to impairment across many situations (Shue et al., 1992). The symptoms must also have developed by 7 years of age (Barkley, 2006).

2.4.1 Diagnostic criteria for ADHD

Swanson et al. (1998) state that perplexing series of changes in labels and definitions produced confusion since the 1960s, when criteria for childhood psychiatric disorders were first included in diagnostic manuals. Differences arose between countries about the diagnosis and subsequently about the national differences in the epidemiology of these disorders. The DSM-IV criteria for Attention-Deficit/Hyperactivity Disorder are as follows:

2.4.1.1 DSM-IV-TR-Criteria

A Either (1) or (2)

1. Six (or more) of the following symptoms of inattention have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Inattention

- Often fails to give close attention to details or makes careless mistakes in schoolwork, or other activities
- Often has difficulty sustaining attention in tasks or play activities
- Often does not seem to listen when spoken to directly

- Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the work place (not due to oppositional behaviour or failure to understand instructions)
- Often has difficulty organizing tasks and activities
- Often avoids, or dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)
- Often loses things necessary for tasks or activities (for example, toys school assignments, pencils, books or tools)
- Is often easily distracted by extraneous stimuli
- Is often forgetful in daily activities

2. Six (or more) of the following symptoms of hyperactivity-impulsiveness have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity

- Often fidgets with hands or feet or squirms in seat.
- Often leaves seat in classroom or in other situations in which remaining seated is expected.
- 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).
- Often has difficulty playing or engaging in leisure activities quietly.
- Is often "on the go" or often acts as if " driven by a motor".
- Often talks excessively.

Impulsiveness

- Often blurts out answers before the question is completed.
- Often has difficulty waiting turn.
- Often interrupts or intrudes on others (for example, buts into conversations or games).

B. Some hyperactive-impulsive or inattentive symptoms that cause impairment were present before the age of seven years.

C. Some impairment from the symptoms is present in two or more settings (for example, at school 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 a pervasive developmental disorder, schizophrenia, or other psychotic disorder and are not better accounted for by another mental disorder (for example, Mood Disorder, Anxiety Disorder, Dissociative Disorder, or Personality Disorder (American Psychiatric Association, 2000).

The ICD-10 provides more restrictive criteria for ADHD, since symptoms must be present in all three dimensions (Inattention, Hyperactivity, and Impulsiveness), it requires that full criteria for the disorder must be met in each of two or more settings in which the child is observed and the diagnosis is excluded in the presence of Anxiety/Depression Disorders (World Health Organization, 1993).

2.4.2 ADHD Subtypes

Most individuals have symptoms of both inattention and hyperactivity-impulsiveness; there are some individuals in whom one or the other pattern is predominant. Therefore, for a current diagnosis the appropriate subtype should be indicated based on the predominant symptoms pattern for the past six months.

Attention-Deficit/Hyperactivity Disorder, combined type (ADHD-C) should be used if six (or more) symptoms of inattention and six (or more) symptoms of hyperactivity-impulsiveness have persisted for at least 6 months.

Attention-Deficit/Hyperactivity Disorder, predominantly inattentive type (ADHD-PI) should be used if six (or more) symptoms of inattention (but fewer than six symptoms of hyperactivity-impulsiveness) have persisted for at least 6 months.

Attention-Deficit/Hyperactivity Disorder, predominantly Hyperactive-Impulsive type (ADHD-HI) should be used if six (or more) symptoms of hyperactivity-impulsiveness (but fewer than six symptoms of inattention) have persisted for at least 6 months. Inattention may often still be a significant clinical feature in such cases (American Psychiatric Association, 2000).

2.5 Prevalence

According to Barkley (1998) true prevalence of Attention Deficit/Hyperactivity Disorder cannot be accurately determined, because it cannot be strictly defined and precisely measured. The prevalence of ADHD is approximately 3 to 5% of the childhood population. This number depends on how ADHD is defined and measured, the population studied and the degree of agreement between parents and teachers. According to a study conducted by Meyer et al., (2004) in the Limpopo Province of South Africa it was established that out of the total sample of 6094, 5.5% met the criteria for ADHD. A closer look at subtypes yield that ADHD-PI was the most frequently reported condition, with prevalence of 3.2%, followed by ADHD-C at 1.3% and ADHD-HI at 0.9%.

Report rates also vary substantially in different geographical areas and across countries (American Academy of Pediatrics, 2000). ADHD is not a culture-bound syndrome, as it is present in various cultures (Alarcon, Wester, Meyer, Foulks & Rulz 1999; Leung et al., 1996; Meyer & Aase 2003; Meyer 1998; Taylor 1998). (See Table 2.1).

In the general population, 9.2% (5.8% to 13.6%) of males and 2.9% (1.9% to 4.5% of females are found to have behaviour consistent with ADHD (American Academy of Pediatrics, 2000).

Table 2.1 Prevalences obtained in school/community populations of various cultures using DSM-IV criteria

Country	Prevalence %	Age group	Study
Australia	6.8	6 -17	Graetz et al., (2001)
Brazil	18.0	6 - 8	Guardiola et al., (2000)
Brazil	5.8	12 - 14	Rohde et al., (1999)
China	5.3	6 - 11	Liu et al., (2000)
Columbia	16.0	4 - 17	Pineda et al., (1999)
Iceland	5.7	6 - 8	Magnusson et al., (1999)
Nigeria	8.0	6 - 12	Ofovwe et al., (2005)
South Africa	5.4	6 - 15	Meyer et al., (2004)
United Arab Emirates	14.9	6 - 12	Bu-Haroon et al., (1999)

2. 6 Gender Differences

Sex appears to play a significant role in determining prevalence of ADHD within a population. On average, male children are between 2.5 and 5.6 times more likely than female children to be diagnosed as ADHD within epidemiological samples, with the average being roughly 3:1. According to Tannock (2004) gender ratios also vary substantially as a function of age (child, adolescent, and adult) and whether the sample was derived from a clinic or the community. The disorder is more common in boys than in girls, but during adolescence and young adulthood relatively more females are affected. Male to female ratios vary in clinic-referred samples, ranging from 9:1 to 6:1 and

approximately 3:1 in population based studies (Swanson et al., 1998). Meyer et al., (2004) conducted a study on primary school children of all cultures of the Limpopo Province of South Africa. They found prevalence rates of 5.5% in the total primary school population with prevalence among boys being 6.9% and among girls 3.9%.

Gershon (2002) and Rietveld, Hudziak, Bartels, van Beijsterveldt, Boomsma (2004) state that in epidemiological and community samples, girls are rated as less impaired than boys in core DSM-IV symptoms of inattention and hyperactivity/impulsivity by both parents and teachers. According to Cantwell (1996) and Swanson (2003) females with ADHD are often rated as more impaired in inattentiveness than males with ADHD and also seen to have the tendency to be diagnosed with ADHD later, with less cognitive problems and fewer symptoms of ODD, CD or aggressive/impulsive behaviour.

Tannock (2004) argue that girls are found to be at slightly or equivalent greater risk of substance abuse as compared with males with ADHD. Girls with ADHD exhibited higher rates of verbal aggression which include teasing, taunting, name calling and they are more likely to show more maladaptive attributional styles for negative events, more dissatisfaction with their relationships with teachers, and more external locus of control (Ruchkin, Loeber, Koposov, Schwab-Stone & Sukhodolsky, 2007).

Heptinstall and Taylor (2002) maintain that boys are generally more frequently afflicted with neurodevelopmental disorders than girls. Girls appear to be more mature than boys at all developmental stages. Boys with ADHD engage in more rule-breaking and display more externalizing behaviours, including ADHD symptoms. Heptinstall and Taylor (2002) argue that across all cultures boys tend to have more externalising problems and girls more internalising problems.

2.7. Cultural differences

Fabrega (1979) German, (1987) IIechukwu (1991) and Meyer (2005) state that the influence of culture on behaviour disturbance has a long history in psychiatry. Meyer et al., (2004) conducted a study among the indigenous children in the Limpopo Province of South Africa. Focusing on six different cultural groups, their study aimed at investigating the psychometric properties of the Disruptive Behaviour Disorders rating scale. The findings were that ADHD is more common in males and further that symptoms may vary with age.

According to Gingerich, Turnock, Litfin and Rosen (1998) culture influences the very same areas that are central to mental health, such as behavioural expectations and tolerance, language, emotion, attention, attachment, traumatic experiences, conduct, personality, motivation, setting limits and other aspects of parenting and child rearing. Gingerich et al. (1998) argue that cultural context plays an important role not only in structuring the environment in which an ADHD individual functions, but also in the way such an individual is understood and treated. Gingerich et al. (1998) argue that people prone to hyperactivity may be "suffering from a culturally induced stimulus overload."

2.8 Aetiology of ADHD

The real cause of ADHD is unknown but there are factors that appear to be likely associated with the symptoms comprising this disorder. Several research studies proposed numerous causes of ADHD, but these causes lack evidence, especially the social or dietary causes, even though the public may believe that they are the chief causes of ADHD. Attention-Deficit/Hyperactivity Disorder is known to be related to a direct effect on brain development or functioning. The real pathways by which these factors lead to ADHD are yet unknown.

2.8.1 Genetic factors

Research studies on the comparison of monozygotic to dizygotic twins for symptoms of hyperactivity and inattention show that if one twin is diagnosed as having ADHD, there is a likelihood of the other monozygotic twin to have the disorder as compared to dizygotic twins (Sonuga-Barke, 1996; Stevenson, 1992). Family adoption and twin studies provide convincing evidence for a genetic component in ADHD (Samudra & Cantwell, 1999).

The genetic basis of ADHD might be rather complicated. Evidence for a genetic basis for ADHD includes greater concordance in monozygotic than in dizygotic twins (Thapar, Holmes, Poulton & Harreington, 1999). Biological parents of children with ADHD have a higher risk for ADHD than adoptive parents (Biederman 2005; Castellanos, & Tannock 2002). According to Sprich (2000) the adoptive relatives of adopted ADHD probands had rates of ADHD and other associated disorders that were lower than those observed in the biological relatives of non- adopted ADHD probands and similar to those found in relatives of control probands. Parents of children with ADHD are more likely to have a history of numerous problems such as antisocial behaviour, alcoholism and learning disabilities (Accardo, 1999), as well as depression and marital problems (Heptinstall et al., 2002).

According to Pliszka, Carlson and Swanson (1999) stimulant medication has pronounced effects on the catecholamine systems, norepinephrine and dopamine. Genes that govern the norepinephrine and dopamine systems were a logical place to start (Pliszka, 2003). Most genetic research has been concerned with the dopaminergic genes (Swanson, Flodman, Kennedy, Spence, Moyzis & Schuck et al., 2000). The dopamine transporter gene DAT1 was associated with ADHD in several studies (Cook, 1995).

The DRD4 (repeater gene) was most reliably found in samples of children with ADHD (Faraone, Biederman, Chen, Millberger, Warburton & Tsuang, 1995; Swanson et

al., 2000). Specifically, it was the 7-repeat form of this gene that was found to be over-represented in children with ADHD (LaHoste, 1996). Barkley (2006) states that this gene was previously associated with the personal trait of high novelty-seeking behaviour.

The gene's impact on postsynaptic sensitivity is primarily found in frontal and prefrontal cortical regions and it is believed to be associated with executive functions and attention (Barkley, 2004). Swanson et al. (2000) give a summary of several studies investigating dopamine genes and indicate that all three published studies of the DAT1 gene reported an association with ADHD, while four of the five studies reported an association of the DRD4 gene with ADHD.

Five dopamine receptor subtypes, coded by five different genes, have been identified and grouped into 2 families; D1-type, consisting of subtypes D1 and D5; and the D2-type, consisting of subtypes D2, D3 and D4. The D1 and D2 receptor subtypes are present in high concentrations in the accumbens and caudate/putamen complex, the D3 and D4 receptors are sparse compared to the other receptors, but are localised in the meso-cortico-limbic pathway, while the D5 receptors are limited to the hippocampus, hypothalamus and cortex (Solanto, 1998). According to Sagvolden and Sergeant (1998) ADHD seems to have genetic components associated with genes coding for receptors in the dopamine D2 family and membrane dopamine transporter (DAT) proteins.

2.8.2 Environmental factors

Environmental factors are also known to cause ADHD. These include lead contamination, foetal exposure to alcohol and benzodiazepines, as well as maternal smoking during pregnancy, pre-eclamptic toxaemia, brain diseases and injuries, low foetal heart rate during labour and small head circumference at birth (Biederman, 2005; Taylor, 1998; Taylor et al., 2004).

Maternal smoking during pregnancy can lead to ADHD because nicotinic receptors modulate dopaminergic activity and doparminergic disruption is involved in the

pathophysiology of ADHD (Biederman, 1991; Biederman, 2005). It also has impact on the expression of attention problems, other externalising problems and academic problems.

2.8.3 Neuroanatomy

ADHD seems to be mainly associated with reduced metabolism and volume of the right cortex and right subcortical structures, smaller total cerebral volume, smaller cerebellum, as well as reduced corpus callosum (Oades, 1998; Solanto, 1998). The corpus callosum is the primary connecting structure for the cerebral hemispheres and smaller anterior corpus callosal areas are consistent with involvement of prefrontal cortical regions in ADHD (Paule, 2000).

Studies using Positron Emission Tomography (PET) have found lower cerebral blood flow and metabolic rates in the frontal lobe areas of children with ADHD than in controls (Sadock & Sadock 2003). Neuroimaging studies indicate anomalies in brain morphology in ADHD in those brain regions that subserve time perception as well as executive functioning (Berquin, 1998).

According to Taylor et al. (2004) over the past 25 years, theories about the biological basis of ADHD and/or Hyperkinetic Disorder (HKD) have suggested neuroanatomical location deficits is in the frontal basal ganglia and neurochemical disorder which involves dopamine pathways, which result in impaired neuropsychological functioning. Filipek (1997) maintains that children with ADHD have smaller brain volumes in anterior superior regions (that is, posterior prefrontal, motor association and midanterior cingulated) and those abnormalities indicate the neuroanatomical networks of executive control and alerting.

2.9. Neurochemistry

Most theories emphasise the role of the neurotransmitters dopamine and norepinephrine in the deficits underlying ADHD. Arnsten (2001) modified the

noradrenergic theory of ADHD. Theories proposed that different abnormalities may exist in two noradrenergic regions: underactivity in a cortical region, dorsa-lateral prefrontal) resulting in primary memory deficits and overactivity in a subcortical region (Locus coeruleus), resulting in overarousal. Solanto (1998) suggested the possibility of relative overactivity both in the dopaminergic and the noradrenergic systems.

The behaviour of children with ADHD and normal children is differently affected by reinforcement contingencies (Johansen et al., 2002). According to Sagvolden and Sergeant (1998) the key feature of ADHD, deficient sustained attention, overactivity, and impulsiveness, may all be due to altered reinforcement mechanisms and a shorter delay- of – reinforcement gradient.

2.10 Theoretical viewpoints of underlying deficits

2.10.1 Sonuga- Barke's dual pathway model

Sonuga-Barke (2002) in conjunction with the National Institute for Mental Health (NIHM), compared the two main opposing views of the deficits underlying ADHD. This study suggests that the poor inhibitory control and delay aversion put forth by these two viewpoints are independent, co-existing characteristics of ADHD (combined type). These findings were used to propose a dual pathway model of ADHD that recognizes two distinct subtypes of the disorder.

One subtype is the result of dysregulation of thought and actions resulting from poor inhibition control associated with the meso-cortical branch of the dopamine system projecting to the cortical centres (prefrontal cortex). The other subtype is a motivational style characterised by altered reward processes associated with the meso-limbic branch (nucleus accumbens). This model describes ADHD as a developmental outcome of two distinct psychological or developmental processes. This model predicts that the dysregulation of thought and action pathway ADHD will be context independent, associated with relatively severed generalised cognitive dysregulation, be categorical in

nature and be less strongly associated with genetic factors, while the motivational ADHD is characterized by attempts to avoid delay (Sonuga-Barke, 2003).

2.10.2 Barkley's' Theory and Executive Functions

Barkley (1998) argues that poor behavioural inhibition is the core deficit of ADHD and the inhibitory deficits are responsible for secondary deficiencies in other executive functions. This impairs the development of executive functions necessary for self-regulation of behaviour, cognition, and emotion, leading to deficits in four types of executive functions. These are (a) Working Memory, (b) Self-Regulation of effect (c) Internalisation of speech and (d) Reconstruction behavioural analysis and synthesizing information in order to solve social and cognitive problems.

2.10.3 Sergeant's Cognitive Energetic Theory

According to Sergeant (1999) the Cognitive-Energetic model draws attention to the fact that ADHD has effects at the three levels: Cognitive mechanisms such as response output, energetic mechanisms such as activation, effort and control systems of Executive Functions. Oosterlaan (1998) states that disruptive disorders have common deficiencies in Executive Function control systems, and may be possibly differential either at an energetic level or at specific elementary cognitive stages. The Cognitive Energetic model does not suggest single executive function deficits in ADHD. It suggests that disorders such as Higher Function Autism will have communalities with ADHD in terms of inhibition of responding. Conversely, it assumes that a range of executive functions can, in principle, differentiate the syndromes (Barkley, 2004).

Oosterlaan (1998) and Smith (1999) argue that differences between ADHD, and ODD/CD should be sought in terms of reward mechanisms influencing inhibitory control. In specifying the differences between ADHD and Higher Function Autism, the cognitive-energetic model predicts that the executive function deficits found will be dependent upon both task parameters and the processing of the child.

2.10.4 Sagvolden's Theory

Sagvolden and co-workers (2005) suggest that ADHD symptoms are caused by a dysfunctioning dopamine system that impairs signal transmission. A dysfunctioning meso-cortico-limbic dopamine branch will produce altered reinforcement and extinction processes, that on a behavioural level give rise to deficient sustained attention, hyperactivity, behavioural variability, motor and cognitive impulsiveness (Johansen et al., 2002; Sagvolden 1999).

A dysfunctional nigro-striatal dopamine branch will manifest into poor motor control, longer reaction time, poor response timing, abnormal control of eye saccades, poor handwriting and poor correlation of the activity of different body parts (Johansen et al., 2002; Sagvolden et al., 2005). Thus, findings previously attributed to response disinhibition due to frontal- lobe dysfunction may rather be due to impaired motor control associated with dopamine dysfunctioning of the neostriatum. Douglas, (1999); Sagvolden (1996); Sagvolden and Sergeant, (1998) and Sonuga-Barke (1992) argued that the key features of ADHD may all be due to altered reinforcement mechanisms and a shorter delay of reinforcement gradient.

2.11 Neuropsychology of ADHD

Numerous studies indicate that ADHD is associated with deficits on a variety of neuropsychological measures which come from different psychological models of ADHD (Sergeant, 2000). Children with ADHD are impaired in various Executive Function domains (Barkley et al., 1992; Pennington, 1996; Sergeant, Geurts & Oosterlaan, 2002).

According to Castellanos and Tannock (2002) response variability across a variety of tasks is one of the most consistent findings associated with ADHD, particularly when motor decision or effortful response organization is required.

In a study done by Banaschewski, Brandeis, Heinrich, Albrecht, Brunner and Rothenberger (2003) measures of domains with less of an executive component, such as processing speed, rapid naming, fine and gross motor skills, timing functions, early and automatic information processing stages, are impaired as well. Deficient response inhibition may be a marker for a genetic susceptibility to ADHD.

2.12 Neurobiological Theory

The scientific belief that Attention Deficit/Hyperactivity Disorder is best regarded as a biogenetic neuropsychiatric disorder receives support from the large and growing literature on the genetics and neurobiology of ADHD as cited earlier. Indeed, twin, family and adoption studies suggest a large genetic, but relatively small (mainly non-shared) environmental component, which seems to remain relatively constant across levels of symptom severity (Rietveld et al., 2004).

Molecular genetic studies implicate a number of potentially (neurobiologically) functional susceptibility genes in the pathophysiology of the condition especially those coding for the structure of dopamine receptors e.g. DRD4 and transporters e.g. DAT1; (DiMaio 2003). Genetic effects in the case of ADHD are necessarily expressed within, enabled by, and in some cases doubtlessly mediated and or moderated by particular biological and social environments that are presently not well mapped.

Sonuga-Barke (2002) argues that several potential neuropsychological endophenotypes for ADHD have been described, including a specific abnormality in reward-related circuitry that leads to shortened delay gradients and delay aversion, deficits in temporal processing that result in high intra-subject inter-trial variability (Smith, Taylor, Rogers, Newman & Rubia, 2002) deficits in working memory (Bedard, 2004; Rhodes, 2004) and non-working visual memory (Rhodes, 2004) impaired stop-signal inhibition (Schachar, Tannock, Marriott, & Logan 1995) and attentional set-shifting (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2002).

The heritability and co-segregation within families remain unexplored for most endophenotypes. Only attentional set shifting and stop signal inhibitions have been demonstrated to be familial and more frequent in non-affected family members (Nigg, Balskey, Huang-Pollock, & Rappley, 2002). While several of these endophenotypes have been demonstrated to be relatively sensitive markers for ADHD, their specificity to ADHD is less clear (Banaschewski, 2005). This is one of the reasons for neuropsychological tests being relatively ineffective tools in the diagnosis of DSM or ICD defined ADHD.

Several of the proposed endophenotypes appear to be common to several neurodevelopmental disorders. For example, working memory has also been proposed as an endophenotype for schizophrenia (Gtesman & Gould, 2003) and stop signal inhibition has been implicated in schizophrenia, language disorders, conduct disorder and autism. Indeed, the neuropsychological and neurophysiological similarities and dissimilarities between ADHD and other commonly comorbid conditions, such as conduct disorder (Banaschewski et al., 2003; Schachar, et al., 1995; Oosterlaan, & Sergeant, 1998) and Specific Learning Difficulties (Tannock, Martinussen, & Frijters 2000) remain contentious and unresolved.

Despite widespread recognition of ADHD as a developmental neuropsychiatric condition, very few casual explanations have seriously considered the two-way, interactions between pre-existing abnormal functioning and biological, cognitive, emotional, motor and social developmental processes, and their contribution to the expression of the behavioural phenotype (Schilling & Nigg, 2004). The potential importance of this concept to casual modelling for ADHD can be illustrated by considering the role played by working memory deficits in the development of ADHD.

According to Castellanos and Tannock (2002) working memory deficits, although relatively understudied, have been considered by many to be the core cognitive risk factors

for ADHD requiring accommodation within a casual model of ADHD deficits in timing (Toplak, Rucklidge, Hetherington, John & Tannock, 2003) and non-working visual memory (Rhodes, Coghill & Matthews, 2004) which, while not dependent on working memory performance, may themselves impact on the development of working memory, have been recently identified.

They argue that if the usual development of accurate working memory performance is contingent upon the development of cerebellar timing functioning and spatial recognition memory, then impaired development of either of these abilities may impact on the development of spatial working memory functioning.

Jonkman (2004) states that the possibility that very basic sensory and perceptual processes may be impaired in ADHD, which, over the course of development, may manifest subsequently as impaired performance on various tasks and interpreted as 'impairments in Executive Function remains relatively unexplored.

2.13 Diagnosis and Assessment

Assessment should comprise a clinical interview with the parents, and separately with the child; obtaining kindergarten, preschool or school information; testing intelligence, achievement, attention and impulsiveness as indicated; making behavioural observations during clinical examinations and testing; and physical evaluation. The child must be seen on more than one occasion; ADHD symptoms must be evaluated carefully against what is expected at that developmental level, the assessment needs to be adequate enough to find any alternative explanation of the symptoms that may be present and any significant comorbidity (Barkley, 2006).

2.13.1 Clinical Interview

The clinical interview can consist of open-ended questions, focused questions about specific behaviours, and standardised interviews, questionnaires and rating scales (American Academy of Pediatrics 2000).

2.13.2 The child interview

The child's self-report is helpful, especially if the child is six years or older, but more for general adjustment and comorbidity than for the presence or absence of diagnostic symptoms (Taylor et al., 2004). It should therefore be focused on functioning in the family, the school and the peer group; general evaluation of psychopathology (especially emotional problems and self-esteem) and the children's attitude to and coping with their disorder. Self-report rating scales may be useful, as a supplement to the interview, especially for detecting emotional problems in children aged nine years or more.

2.13.3. The parent interview

General evaluation should clarify presenting complaints, make a systematic evaluation of psychopathological symptoms, and describe how problems developed (Taylor et al., 2004). The developmental history is important and should include previous professional reports.

One needs to reach an adequate account of affected family members (relevant to a genetic aetiology), pregnancy and birth history (foetal growth, toxaemia, bleeding or severe infections in pregnancy, maternal diabetes or epilepsy, other maternal illnesses or traumas, poor nutritional state of the mother, use of medication, nicotine, alcohol or drugs, gestational age, birth complications, birth weights, neonatal complications), early developmental history (milestones for psychomotor development, language, attachment, sleep and feeding problems, growth, and early temperament); medical history, especially tics and epilepsy medication (especially anticonvulsants, antihistamines, sympathomimetics, steroids) and (if adolescent) history of psychosis.

The assessment also needs to be sufficiently detailed to address family functioning and family problems for example, financial problems, problems of other family members, parental conflicts), coping styles of the parents, expressed warmth and hostility, social network and other resources. Specific questioning should include the behaviours that comprise the ICD-10 and DSM-IV diagnoses, any situational variation in them, their times of onset and development, and their presence in other family members together with that of related problems (such as behavioural and learning problems, emotional problems, tics, Conduct Disorder, and alcoholism (European Child & Adolescent Psychiatry, 2004). Parent rating scales are useful as a supplement to the interviews and not as a replacement (Barkley, 2006).

2.13. 4. The teacher interview

It is important to obtain information from the teacher about behaviour and behaviour problems in the classroom, developmental and social functioning and situational variation in behaviour and symptoms indicating comorbid or differential diagnoses. This can occur by telephone and may help to further clarify the nature of the child's problems. Many ADHD children have problems with academic performance and classroom behaviour, and this information is important. Taylor (1998) states that this can also include standardised questionnaires and rating scales.

2.13. 5 Medical interview

The purpose of the medical interview is its focus on the differential diagnosis of ADHD from other medical conditions, particularly those that may be treatable. It is also necessary to determine whether the child's conduct or learning problems are related to the emergence of a seizure disorder (Barkley, 1998). Another purpose of the medical interview is to thoroughly evaluate any co-existing conditions that may require medical management, and also to determine whether physical conditions exist that are contradictions for treatment with medications (Barkley, 2004).

2.13.6 Physical examination

According to Barkley (2004), in the course of physical examination, height, weight and head circumference require measurement and comparison to standardized graphs and hearing, vision and blood pressure should be screened. The formal neurological examination often includes testing of cranial nerves, gross and fine motor coordination, eye movement, finger sequencing, rapid alternating movements, impersistence, sky kinesia and motor overflow as well as testing for choreiform movements and tandem gait tasks. It is also used to look for signs of previous central nervous system injury or a progressive neurological condition, abnormalities of muscle tone and a difference in strength, tone, or deep tendon reflex response between the two sides of the body (Barkley, 2004; Barkley, 2006).

2.13.7 Laboratory Tests

A number of studies have used a variety of physical, physiological and psychological measures to assess potential differences between ADHD and other clinical or control group of children. Although some of these have demonstrated differences, such as cerebral blood flow to the striatum or diminished orienting galvanic skin responses in ADHD children, none of these laboratory measures are of value in the diagnostic process as yet (Barkley, 2006).

Parents, teachers, or even other mental health professionals are sometimes misled by reports of such findings or by the conclusion that ADHD is a biologically based disorder, and frequently ask for medical tests to be done on their children to confirm the diagnosis.

2.13.8 Screening (rating scales)

The Disruptive Behaviour Disorders screening questionnaire, which is based on the DSM-IV, was developed by Pillow, Pelham, Hoza, Molina and Stultz (1998). The DBD questionnaire is used for screening children for the Disruptive Behaviour Disorders,

(ADHD, ODD and CD. It consists of 42 Items with specific items for ADHD, ODD and CD and also categorizes the children according to the Inattentive type, Hyperactive–Impulsive type and the combined type (Pelham, Gnagy, Greenslade, & Milich, 1992).

This questionnaire must be filled in by the teacher or the parent or both to rate the child according to the specific items of ADHD, CD, and ODD. The child is rated according to four ratings "not at all" "pretty much" and "very much." At least six or more of the specific items of the inattentive type that persisted for six months or more will be enough for a child to be identified as having ADHD-Inattentive type and six or more specific symptoms of hyperactive– impulsive type, to make 12, will be enough for the child to be identified as having ADHD Combined type (Pelham et al., 1992; Pillow et al., 1998).

The scale has been translated from the original US English into Xitsonga, Tshivenda, Sepedi (North-Sotho), Setswana and Afrikaans. Internal consistency measures and norms for these language groups have been established (Meyer et al., 2004).

2.14. Psychological tests

Psychological tests are useful, in that they can provide additional information and aid in exploring comorbid conditions from various sources and not just on the result of tests (American Academy of Pediatrics, 2000; Sagvolden & Sergeant, 1998). Barkley (1998) states that psychological testing can help the clinician to address the three fundamental questions underlying the evaluation of ADHD:

1. Is the diagnosis justified?

2. If the diagnosis is not justified, are there alternative explanations that better account for the symptoms?

3. If the diagnosis is justified, are there comorbid conditions that should be justified and treated?

Certain tests has been proved to distinguish between children with ADHD and normal control groups which are: cognitive tests, general neuropsychological batteries, and individual neuropsychological tests.

2.15 Treatment of ADHD

Treatment strategies for ADHD should be designed to address the behavioural, cognitive, family and social problems characteristics of ADHD (Coffey, 1997). Multimodal treatment is recommended (Meyer & Aase, 2003; Taylor, 1998) and should involve both pharmacological and psychosocial interventions (Swanson et al., 1998; Taylor, 1998).

2.15.1 Pharmacological Intervention

A sufficient number of studies of adequate design have provided strong evidence that short acting stimulants are effective in reducing core ADHD symptoms (Barkley, 1998; Gadow, Nolan, Sverd, Sprafkin & Schwartz, 2002). Stimulants are recommended as "first line" treatment for the core symptoms of ADHD in children and young people.

Psychostimulant drugs which include medications such as amphetamine (Dexedrine®), methylphenidate (Ratalin®, Concerta®) and dl-amphetamine (Adderall®) have been proved significantly to improve the core symptoms of inattentiveness, impulsiveness and hyperactivity in ADHD (Elia, Ambrosini, & Rapoport, 1999; Gillberg, Melander, von Knorring, Janola, Themlund & Hagglof et al., 1997; Greenhill, Halperin, & Abikoff, 1999). The improvement in impulsive and overactive behaviour is more pronounced than in inattentiveness (Swanson 2003; Swanson, & Castellanos, 1998). These drugs release and inhibit the reuptake of catecholamines, mainly dopamine in the central nervous system. The clinical effects peak at about 1 hour after each dose and dissipate in about 4 hours, therefore the drugs are usually given two or three times per day. They produce beneficial results on tests of cognitive performance, academic productivity, oppositionality, and social interactions with parents and peers (Schachar et al., 1995; Tannock, Ickowicz & Schachar, 1995).

Atomoxetine (Strattera®) is increasingly used as a non-stimulant agent that has been found to increase dopamine and norepinephrine in the prefrontal cortex. It has also been found to improve ODD symptoms and unlike with methylphenidate, atomoxetine's advantages include its minimal risk for substance abuse (Newcorn, Spencer, Biederman, Milton & Michelson, 2005). For this, it is regarded as a better option, especially for children with comorbid conduct problems. Atomoxetine is continuing to gain more preference as it has been shown to be safe and effective; moreover it also seems to work in adult populations as well (Prasad, 2005).

2.15.2 Psychosocial Intervention

According to Meyer and Aase (2003) behavioural therapy programmes for children with disruptive behaviour disorders have been increasingly well developed during the past two decades. These programmes aim to teach parents and teachers behaviour management skills based on sound behavioural learning principles, particularly the systematic use of reinforcement contingencies. Among the most comprehensively described are the parent and teacher training programmes (Barkley, 1997; Barkley, 1998).

The goal of the programmes are (1) to improve parental and teacher management skills and competence in dealing with child behaviour problems (2) to increase parental and teacher knowledge of the causes of childhood defiant behaviour, (3) to improve child compliance with commands, directives, and rules, given by parents and teachers and (4) to increase family harmony through the 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 appropriate child behaviour and general reliance on principle guided behaviour (Barkley et al., 1992; Barkley, 2006). Early intervention by health care professionals is essential in children with ADHD who are at risk for future delinquency, substance abuse and other risk taking behaviour (Meyer & Aase, 2003).

2.15.3 School Intervention

Academic interventions are frequently warranted for children with ADHD. Counsellors may not have primary responsibility for developing and implementing academic interventions but will want to be sure that these interventions are included in the comprehensive intervention plan. The first step is to provide the teacher with the knowledge and training necessary to provide the individualized attention that is usually necessary to best help the ADHD child in school (Biederman et al., 1996).

According to Biederman et al., (1996) and Greene (1996) alterations to the classroom environment such as changes in the pace, presentation, or level of instruction to make the classroom environment a better match for the needs of the student along with implementation of behaviour support programmes are usually helpful. Regular contacts between the counsellor and school personnel over a relatively long period of time provide maximum cooperation and success in working together (Pelham et al., 1992). The counsellor should provide sufficient social reinforcement for the teacher, particularly in the beginning stages of intervention, after which the child's improved performances will help reinforce teacher effort.

2.16 Outcome / Prognosis

According to Biederman and Faraone (2004) the symptoms of hyperactivity and impulsiveness tend to decline rapidly at an earlier age than those of inattention. Based on these findings, that ADHD symptoms continue into adolescence and adulthood, and that it is a risk factor for later psychiatric disorders and social dysfunction, it is clear that a better understanding of the disorder, leading to improved and timely interventions is essential (Sagvolden & Sergeant,1998).

Cantwell and Baker (1991) state that if the impairing symptoms of ADHD are not treated they may continue into adolescence and adult life and further predispose a child to

psychiatric and social pathology in later life. The adolescent with ADHD is at risk for academic failure, low self-esteem, poor peer relationships, parental conflict, delinquency, smoking and substance abuse, as well as worse automobile driving records in adolescence (Mannuzza, Klein, Bessler, Malloy & LaPadula, 1993; Weiss & Hechtman, 1993).

Chapter 3

ADHD: DIFFERENTIONAL DIAGNOSIS, MIMICKING DISORDERS AND COMORBIDITY

3.1 Introduction

ADHD should be viewed as a diagnosis of exclusions. Buttross (2000) asserts that there are no laboratory tests that are diagnostic of ADHD, and there are no physical features that are helpful in the diagnosis. A thorough evaluation must first exclude health problems, learning disorders, behavioural disorders, psychosocial stressors and age-appropriate overactivity. The symptoms of ADHD should occur in all aspects of life.

3.2 Differential diagnosis

According to DSM-IV-TR (American Psychiatric Association, 2000) the following conditions should be considered before the diagnosis of Attention-Deficit/Hyperactivity Disorder is made:

A temperamental constellation consisting of a high activity level and short attention span, but in the normal range of expectation for a child's age, should be considered first.

1. Anxiety disorder: Anxiety Disorder may be manifested by overactivity and easily distractibility.

2. Bipolar disorder: ADHD must also be distinguished from mania, although they share many core features such as excessive verbalizations, motor hyperactivity and high levels of distractibility and irritability.

3. Obsessive-Compulsive Disorder (OCD) is characterized by recurrent unwanted obsessions and/or compulsions that interfere significantly with a sufferer's ability to

function normally (American Psychiatric Association, 2000). Tynan and Jefferson (2006) maintain that most children with OCD are inattentive and often hyperactive in an attempt to maintain focus and control.

4. Adjustment Disorder (AD) is a relatively common disorder in childhood in which emotional or behavioural symptoms develop in response to a stressor, such as the death of close family members, a move, or a recent parental divorce that has occurred within 3 months of the onset of the stressor (American Psychiatric Association, 2000). Inattention, poor sleep, or behavioural difficulties from exposure to the stressors may develop. According to Buttross (2000) the resultant effects of an adjustment disorder may be conduct problems, problems with emotional functioning or poor academic performance.

5. Autistic Spectrum Disorder (ASD) is a pervasive developmental disorder, as are Asperger's Syndrome, Rett's Syndrome and Childhood Disintegrative Disorder (American Psychiatric Association, 2000). Children with autism share many symptoms with children with ADHD but may also show problems less associated with ADHD, like in theory of mind (thought studies are contradictory) and in weak central coherence (Booth, Charlton, Hughes & Happe, 2003).

Few studies have compared autism and ADHD directly. Ozonoff and Jensen (1999) found a double dissociation between both disorders. Children with autism showed difficulties in planning and cognitive flexibility, but not in inhibition, whereas children with ADHD showed the opposite pattern (Ozonoff & Jensen, 1999) .

6. Asperger's Syndrome: Essential features of this disorder include a severe impairment in social interaction, as manifested by poor use of non-verbal behaviour, interests and activities (American Psychiatric Association, 2000). There are no delays in cognitive, language or motor development. The disturbances cause significant deficiencies in social and academic functioning. Because of their poor social interaction and lack of

social reciprocity, they are often viewed as inattentive and they function poorly in the classroom (American Psychiatric Association, 2000).

7. Tourette's syndrome (TS) is a neuropsychiatric disorder characterized by the presence of multiple motor tics plus one or more vocal (phonic) tics (American Psychiatric Association, 2000). Tourette's syndrome is often associated with a broad spectrum of psychopathological and behavioural disorders (Kurlan, Como, Miller, Palumbo, Deeley & Anderson et al., 2002; Wodrich, Benjamin & Lachar, 1997). Children with Tourette's syndrome have difficulty in school and poor self-esteem, they are more likely to have symptoms of ADHD (Kurlan, et al., 2002).

8. Mental Retardation is a significant sub-average general intellectual functioning existing concurrently with deficits in adaptive behaviour and manifested during the developmental period (American Psychiatric Association, 2000). It can be translated as having an IQ below average (<70), causing problems in communicating with others and learning the skills necessary for daily living and work.

9. Childhood Depression is defined as an individuals exhibiting a depressed mood most of the day, every day (American Psychiatric Association, 2000). In children and adolescents the mood may be irritable rather than depressive in appearance. Often there is a markedly diminished interest in pleasurable activities. There may be a significant weight loss or weight gain.

However, in children a failure to make expected weight gain may also be symptoms of depression, sleep problems such as insomnia or hypersomnia may also be present. The depressed mood and fatigue from lack of sleep can be misinterpreted as uninterested or lack of motivation. Psychomotor agitation or retardation, which can be presented as hyperactivity or inattention, may also be apparent.

10. Attachment Disorder Some children who have had massive and prolonged disruption of attachment relationships in early childhood show a rather characteristic course in which an initial period of indiscriminate clinging to adults is followed during school years, by a pattern of inappropriately outgoing and inattentive activity, with an unreserved contact with strangers and often a lack of deep and trusting relationships (Taylor et al., 2004).

11. Learning Disorder is characterised by academic functioning that is substantially below that expected, given the person's chronological age, measured intelligence and age appropriate education (American Psychiatric Association, 2000). Learning disabilities usually fall into the category of language-based disorders of learning, impaired mathematic performance, dysgraphia and pragmatic language disorders related to language use in the social context.

Learning Disorder can occur in reading, mathematics, written expression or language, and can be either receptive or expressive in nature. Early signs of learning disorder might include delayed speech, difficulty following commands, fine motor delays, poor memory skills, clumsiness, trouble in differentiating left from right, inattention and a dislike of learning to read or write (Buttross, 2000).

3.3 Mimicking disorders of ADHD

3.3.1 Sensory deficits

A child's development, attention span and social interaction can be markedly influenced by a sensory impairment. The earlier the onset and the greater the severity of the impairment, the more significant the impact on the child. Vision and hearing screenings are imperative in any child who has a history of inattention or hyperactivity, often vision or hearing are not recognized early because the child does not realize what his peers can see or hear (Buttross, 2000).

3.3.2 Chronic illness

Success in school for a child depends on the child's academic abilities, emotional well-being, social interaction, school attendance and adequate school performance. Chronic medical conditions can potentially affect all of these areas. According to Buttross (2000) chronic medical problems occur in up to 20% of school aged children in the United States. Dingle (1997) also argues that school-related difficulties can develop in as many as 40% of these children.

Nass (1997) maintains that migraine headaches, absence seizures, hypothyroidism or hyperthyroidism, lead intoxication, asthma, haematological disorders, childhood cancer, and insulin-dependent diabetes mellitus are all illness that can interfere with a child's classroom performance. According to Buttross (2000) academic problems may be direct effects of the illness or results from the therapy that the child has received.

3.3.3 Substance abuse

Substance abuse is an extensive use of illicit substances with negative consequences. It is when a drug enters the brain and spinal cord, where it acts as a depressant. The DSM-IV-TR identifies substance abuse as one or more of the following occurring repeatedly in a 12-month period: recurrent substance use resulting in a failure to fulfill major roles obligations at school, or home; recurrent substance abuse in situations in which it is physically hazardous; or continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance (American Psychiatric Association, 2000).

Risk factors that are likely to increase the likelihood of substance abuse are depression, low self-esteem and lack of connectedness with family members. (Centre for Disease Control and Prevention, 2002). According to Buttross (2000), substance abuse and substance use can affect a child's academic outcome through numerous modalities.

Substance abuse disorders are frequently linked with disruptive behaviour, mood and anxiety disorders, risk taking, aggression and suicidal behaviour. Also, an abuse pattern can encourage "active" behavioural changes such as inhibition, lethargy, hyperactivity, agitation, somnolence, hyper vigilance and changes in cognition (CASA, 2002).

3.3.4 Sleep disorders

Sleep disorders are characterized by a disturbance in the amount, quality, and timing of sleep manifested by poor sleep settling, night-time waking, or excessive daytime sleepiness (Buttross, 2000). In non-clinical populations, 20% to 30% of children have been documented to have sleep-related problems that are regarded as significant to their families. 34% of children with sleep problems have an increased rate of behavioural and emotional symptoms and psychiatric diagnoses, including anxiety disorders, ADHD, depression and Conduct Disorder (Dahl, 1996).

Corkum, Tannock, Moldofsky, Hogg-Johson and Humphries (2001) yielded inconsistent findings regarding objective measures of sleep in ADHD. There was either an absence of any discernible differences in sleep variables among ADHD subjects and control, or significant changes emerged in sleep onset latency, in the sleep efficiency, or in the characteristics of rapid eye movement sleep (cyclical movements of closed eyes during sleep; rapid eye movement (REM) and non-rapid eye movement (NREM) sleep in children with ADHD (O'Brien, Ivanenko, Crabtree, Holbrook, Bruner & Klaus et al., 2003).

3.4. Other mimicking disorders

3.4.1 Malnutrition, abuse and low socio economic status

Beal (1997) states that a clearly recognized aetiology of academic difficulties includes psychosocial malfunctioning in the family. The presence of stress in a child's home predicts poorer outcome for the child's academic performance. During the period of

time when the family experiences a divorce, illness of a parent, relocation, or a death of a close family member, tension is high (Buttross, 2000).

Children often suffer academically because of anxiety, lack of organization and stress that is going on in the home. Sexual, physical or emotional abuse or physical or emotional neglect likely causes children to function poorly because of the preoccupation with issues going on at home or the fearfulness of what is going to happen to them next. A chaotic home, which does not afford a child the opportunity to appropriately prepare for school, can cause the child to have symptoms that mimic the disorganized, forgetful behaviour of child with ADHD (Buttross, 2000).

3.4.2 Behavioural disorders

Parent-child relation problems are observed when there is an abnormal or impaired interaction between the parent and the child (American Psychiatric Association, 2000). The problem areas typically are in communication, overprotection and discipline. When the history is taken, it is apparent that the child's problems occur in the home. Typically adults outside the home do not have the difficulty that the parent experiences with the child at home. Counselling the parent to teach appropriate parenting skills are necessary to alleviate the symptoms of ADHD.

3.5 Comorbid disorders

Vella, Aragona and Alliani (2000) define comorbidity as two or more diseases with distinct aetio-pathogenesis and this disorder coexists in presence of the other disorders. It may occur as a result of overlapping symptomatology, one disorder manifesting itself as an earlier form of the other or from shared risk factors (Volk, Neuman & Todd, 2005). Beyond the myriad cognitive, academic, developmental, and medical risks that exist in children with ADHD a high probability of them having comorbid psychiatric disorders also exists (Barkley, 2006).

Individuals diagnosed with ADHD are often found to have a number of other disorders. In community-derived samples, up to 44% of ADHD children had at least one other disorder and 43% had at least two or more additional disorders. In clinically diagnosed ADHD children, it was found that 87% had at least one other disorder, and 67% had at least two or more (Kadesjo & Gillberg, 2001). Jensen et al. (2001) found that only 32% of children with the combined type of ADHD had ADHD alone.

This chapter will focus on the following disorders that are co-morbid with ADHD: Oppositional Defiant Disorder (ODD), Conduct Disorder (CD), Learning Disorder (LD), Anxiety Disorder (AD), Mood Disorders (MD), Autistic Spectrum Disorders (ASD), Obsessive-Compulsive Disorders (OCD) and Tic Disorders (Tourette's Disorder).

3.5.1 ODD and CD with ADHD

According to Acosta, Arcos-Burgos and Muenke (2004) externalizing disorders such as ODD and CD are the most common comorbid conditions associated with ADHD. Studies of clinic referred ADHD children found that 54-67% will meet the criteria for ODD, while 20–50% of these children may have CD (Barkley, 2006; Lahey, McBurnett, & Loeber, 2000). ADHD, ODD and CD are grouped together under the label Disruptive Behaviour Disorders in the DSM-IV (American Psychiatric Association, 2000).

There is a high comorbidity between Attention-Deficit/Hyperactivity Disorder and ODD. Most of the children diagnosed as having ADHD were also reported to have ODD. These children have frequent temper outbursts, argue with adults, often deliberately annoy others, blame others for their own mistakes, and often refuse to obey adults requests or rules. These symptoms are found to be developmentally inappropriate (American Psychiatric Association, 2000).

ODD is mostly associated with ADHD-HI. ADHD is most likely a developmental precursor to ODD/CD (Barkley, 2006). Symptoms of hyperactive-impulsive behaviour (but not inattention) do predict later ODD symptoms, Burns and Walsh, (2002) and the combination of the two increases the stability of ODD from the preschool to the school age period (Lavigne, Cicchetti, Gibbons, Binns, Larsen & DeVito, 2001; Speltz, McClellan, DeKlyen & Jones, 1999).

ADHD may even cause or at least contribute to risk for ODD alone. August et al. (1999) suggest that ODD by itself in samples with ADHD is not a precursor to later CD and may not be especially stable over later development. Barkley (2006) argues that ODD alone declines significantly with age, while CD increases with age. Maughan, Rowe, Messer, Goodman and Meltzer (2004) maintain that it is only the combination of ODD with CD that is likely to explain the persistence of ODD into adolescence.

3.5.2 Anxiety and Mood Disorder with ADHD

The overlap of Anxiety Disorders with ADHD was found to range from 10-40% in clinical referred children, averaging about 25% (Barkley, 2002; Barkley, 2006). Jensen et al., (2001) found that Anxiety Disorder was as common as ODD in their sample of children with combined subtype ADHD. This will be discussed more in details in Chapter 4.

Evidence of the co-occurrence of Mood Disorders, Major Depression and Dysthymia with ADHD is now fairly substantial and most studies place the association between 20 and 30% (Faraone, Biederman, Wozniak, Mundy, Mennin, & O'Donnell, 1997; Jensen, & Cantwell, 1997; Spencer, Wilens, Biederman, Wozniak, & Harding-Crawford, 2000). The overlap between ODD, Mood and Anxiety Disorders is also increasingly documented.

Researchers have shown high rates of ODD in children diagnosed with severe major depression and bipolar disorder (Angold, Costello & Erkanli, 1999; Biederman et al., 1996; Geller & Luby, 1997; Wozniak & Biederman, 1996; Wozniak, Biederman, Kiely, Ablon, Faraone & Mundy et al., 1995). Greene, Biederman, Zerwas, Monuteaux, Goring and Faraone (2002) confirm that in one study, nearly 70% of children diagnosed with severe major depression and 85% of children diagnosed with bipolar disorder were also diagnosed with ODD.

Meaningful rates of Anxiety Disorders have been found in youths with ODD (Greene et al., 2002). Researchers have shown that approximately 30% of youth with Tourette's Disorder (TD) have a concurrent mood disorder, a similar rate of comorbidity has been found between TD and Obsessive-Compulsive Disorder (Budman, Bruun, Park, Lesser and Olson, 2000). Greene et al. (2002) state that over 60% of youth diagnosed with ODD had a comorbid Anxiety Disorder, and that 45% of youth diagnosed with Anxiety Disorder had comorbid ODD. The overlap between ODD and obsessiveness may be particularly compelling (Garland and Weiss, 1996).

3.5.3 Language Disorder and Oppositional Defiant Disorder

Language development is also crucial to the evolution of problem solving, emotion regulation, frustration tolerance and adaptability. There is a demonstrated association between ODD and language impairment. Greene et al. (2002) have shown that over 20% of youths diagnosed with ODD have a comorbid language processing disorder, and that 55% of youth with language processing disorders are further diagnosed with ODD. Thus, it is useful to explore the potential mechanisms by which language processing delays might give rise to adult-child incompatibility and a child's oppositional behaviour (Greene et al., 2002).

According to Budman et al., (2000) cognitive skills such as labelling, categorizing, communicating feelings, needs, identifying and selecting corresponding behavioural strategies are strongly mediated by language. Language permits children to obtain verbal feedback about the appropriateness of the behavioural strategies they select, thereby facilitating the capacity to think about and reflect on previous and future actions.

3.5.4 Learning Disorder and ADHD

Comorbid rates for Learning Disorders and ADHD have also been studied (Willcutt & Pennington, 2000). Willcutt and Pennington (2000) investigated the association between reading disability (RD), internalizing and externalizing psychopathology in a large sample of twins selected on RD. They found that RD was not significantly associated with symptoms of aggression, delinquency, ODD or CD, after controlling for a significant relationship between RD and ADHD.

The association between RD and externalizing psychopathology was stronger for boys, while a significant relationship between RD and internalizing symptoms was largely restricted to girls. Levy, Hay, McLaughlin, Wood and Waldman (1996) reported a strong association between ADHD, reading and speech problems they also reported that there were more symptoms in males than females. According to Cantwell and Baker (1991) children with impairments of speech and or language often have comorbid ADHD.

Mehl-Medronna (2002) argue that children with ADHD who have communication impairment or whose reading, writing and arithmetic abilities are compromised are at significant risk for academic underachievement and for problems in social interaction and fragile self-esteem.

3.5.5 Obsessive-Compulsive Disorder and ADHD

Children and adolescents with Obsessive-Compulsive Disorder (OCD) have been found to have high rates of comorbidity with ADHD (Hanna, Yuwiler & Coates, 1995; March & Leonard, 1996), ranging from 10% to 33%.

3.5.6 Sleep Disorder and ADHD

Dagan, Zeevi-Luria, Sever, Hallis and Yovel (1997) contend that sleep patterns of ADHD children have different architecture, which can explain their tiredness and low energy during the day. Although these children do fall asleep, they are observed to sleep for a few hours, hence they become tired most of the time. They suffer from aggressive behaviour and their self-esteem is low (Slomkowski, Klein & Mannuzza, 1995).

Studies suggest that children with ADHD have a higher likelihood of sleep problems than normal children. As many as 56% of ADHD children have problems falling asleep as compared to 23% of normal children. Up to 39% of ADHD children show problems of frequent night time awakening. Resistance to going to bed and fewer total hours of sleep seem to be a major problem with many children with ADHD (Zeevi-Luria, Hallis, Yovell, Sadeh & Dolev, 1997).

3.6. Conclusion

It has been outlined so far that ADHD exists along with other developmental disorders of childhood. However, it becomes important that clinicians also consider these comorbid disorders which also exist in the midst of ADHD. Again, it is essential that during assessment those other comorbid conditions be paid attention to, for effective intervention especially, in the school and home environment.

Chapter 4

COMORBIDITY OF ADHD: EXTERNALISING DISORDERS

4.1 Introduction

Disruptive Behaviour Disorders include two persistent constellations of disruptive symptoms categorized as Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD), which result in impaired social or academic function in a child. According to Sadock and Sadock (2003) some defiance and refusal to comply with adult requests is developmentally appropriate and marks growth in all children, yet children with these disorders are themselves impaired by the frequency and severity of their disruptive behaviours.

CD is a psychiatric disorder of childhood and adolescence that is characterized by a persistent disregard for societal norms and rules, as manifested by aggression towards people or animals, destruction of property, theft or persistent lying, and other serious violation of rules such as truancy and running away from home (American Psychiatric Association, 2000).

ODD is a psychiatric disorder of childhood and adolescence that is characterized by a persistent pattern of negativistic, hostile or defiant behaviours. Hallmark behaviours of this disorder include frequent arguments with adults, disregard of rules, refusal to comply with the request of adults, loss of temper, vindictive or spiteful acts and displays of anger or resentment (American Psychiatric Association, 2000).

CD encompasses a more serious disregard for societal norms than ODD. In both diagnoses, the behaviours must occur more frequently than expected given the child or adolescent's age or developmental level, and must cause significant impairment in social, academic or occupational functioning (American Psychiatric Association, 2000).

4.2 Oppositional Defiant Disorder

The essential features of ODD are a recurrent pattern of negativistic, defiant, disobedient and hostile behaviour toward authority figures (American Psychiatric Association, 2000; Oosterlaan, Geurts, Knol, & Sergeant, 2005). Greene, Ablon and Goring (2003) maintain that behaviour associated with ODD includes temper outbursts (sometimes referred to as rage attacks); persistent stubbornness, resistance to directions, unwillingness to compromise, give in, or negotiate with adults or peers, deliberate or persistent testing of the limits and verbal (and minor physical) aggression.

These behaviours are almost always present in the home and with individuals the child knows well, and often occur simultaneously with low self-esteem, mood liability, low frustration, tolerance and swearing (American Psychiatric Association, 2000). Children with ODD frequently argue with adults and become easily annoyed by others leading to a state of anger and resentment (Sadock & Sadock, 2003). They have difficulty in the classroom and with peer relationships but generally do not resort to physical aggression or significant disruptive behaviour. ODD is frequently the antecedent to CD.

4.2.1 DSM-IV diagnostic criteria 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:

- Often loses temper
- Often argues with adults
- Often actively defies or refuses to comply with adults requests or rules
- Often deliberately annoys people
- Often blames others for his or her mistakes or misbehaviour

- Is often touchy or easily annoyed by others
- Is often angry and resentful
- Is often spiteful or vindictive

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

C. The behaviours do not occur exclusively during the course of a psychotic or mood disorder.

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

4.2.2 Epidemiology of ODD

Oppositional Defiant Disorder often emerges and gradually worsens in the preschool years in the home setting, the full syndrome usually becomes apparent before the age of 8 (Buitelaar, Montgomery & Zwieten-Boot, 2003). The disorder is more prevalent in boys than in girls before puberty, and sex ratio appears to be equal after puberty (American Psychiatric Association, 2000). There are no distinct family patterns, but many parents of children with the disorder are themselves overly concerned with issues of power, control and authority (Sadock & Sadock, 2003).

4.2.3 Characteristics contributing to ODD behaviour

According to Greene et al. (2003) psychiatric disorders that are commonly co-morbid with ODD may set the stage for compromised skills in the domains of emotion regulation, problem solving, frustration, tolerance and adaptation. For example, ADHD is a diagnosis often applied to children compromised in the skills of self-regulation, deficiencies in higher order problem solving, and adjusting behaviour to fit shifting

environmental demands (Greene et al., 2003; Wozniak & Biederman, 1996) and the overlap and developmental continuity between ADHD and ODD is well established.

4.2.4 Aetiology of ODD

Johnston and Mash (2001) in a study of examining interaction patterns among families with children having ADHD, have found parents to be more directive, commanding and negative than parents of children without ADHD. Dysfunctional parenting may partly be a reaction to the difficulties of raising a child with ADHD, but it may also serve as an aetiological role in the emergence of comorbid disruptive behaviour disorders among children with ADHD (Johnston & Mash, 2001).

Parental depression has been linked to disruptive behaviour in children, perhaps via common familial vulnerabilities (Biederman, Newcorn & Sprich, 1991) through an association with parental antisocial behaviours or due to the difficulty of raising children with a disruptive behaviour disorder. Parenting practices form a second set of family risk factors (Rathouz & Judice, 2004).

Children with oppositional problems often have families characterized by coercive interaction styles, inconsistent discipline, lack of parental involvement and lack of positive and warm interactions between parent and child (Fletcher, Fischer, Barkley & Smallish, 1996). Recent studies with ADHD children suggest that certain kinds of dysfunctional parenting, include maternal lack of responsiveness (Johnston & Mash, 2001; Johnston, Murray, Hinshaw, William & Hoza, 2002), warmth and positive involvement, overly negative discipline (Kashdan, Jacob, Pelham, Lang, Hoza, Blumenthal & Gnagy, 2004), lax and inconsistent parenting, and lack of cohesion among family members are related to comorbid oppositional or conduct problems rather than ADHD per se (Lindahl, 1998).

Negative parenting practices also predict persistence of comorbid ODD rather than ADHD (August, Realmuto, Joyce & Hektner, 1999). Coy, Speltz, Deklyen and Jones

(2001) maintain that children with ODD were twice as likely as controls to generate aggressive solutions to problems. In a study conducted by van Goozen, Cohen-Kettenis, Snoek, Matthys, Swaab-Barneveld and van Engeland (2004) of testing Executive Functioning in children with ODD, without ADHD and normal controls (NC), they found that the ODD/ADHD group was worse than the NC in set shifting, and also both ODD groups performed worse on a response perseveration task. They concluded that ODD/ADHD children have problems in regulating their behaviour under motivational inhibitory conditions. Once they are stimulated by the possibility of a reward they become less sensitive to the possibility of punishment (van Goozen et al., 2004).

4.2.5 Intervention of Oppositional Defiant Disorder

Parent Management Training, a modality of cognitive-behavioral therapy (CBT) intended to modify the child's behaviour through alteration of the parent's way of dealing with the child has proved effective for ODD (Souza, Pinheiro, Denardin, Mattos & Rohde 2004). Chow, Mikulis, Zipursky, Scutt, Weksberg and Bassett (1999) maintain that cognitive therapies have recently come more into evidence with response rates as high as 74%. Probably the appropriate choice of therapy depends on the psychological characteristics of the patient (Greene et al., 2003). Kazdin (2000) has demonstrated that CBT can even improve family functioning and marital satisfaction.

4.3 Prognosis for Oppositional Defiant Disorders

The prognosis is relatively poor; CD and ODD are associated with late psychiatric disorder, antisocial personality disorder and substance abuse disorder (Buitelaar et al., 2003). Buitelaar et al. (2003) state that CD is also associated with criminal behaviour, increased risk of marital break-ups, early pregnancy, poor employment record and deaths related to violent behaviour.

4.4 Conduct Disorder

Du, Li, Wang, Jiang, Livesley, and Jang et al. (2006) argue that CD is characterized by a recurring and persistent pattern of tantrums, destructiveness, lying, stealing, truancy and fighting, or of overt aggressive and hostile acts towards other people. Geller and Proschan (1996) maintain that CD is also associated with several psychosocial factors such as low socioeconomic level, harsh, punitive parenting family discord and lack of appropriate parental supervision and social competence.

The DSM-IV-TR specifies that truancy from school must begin before 13 years of age to be considered a symptom of CD. The disorder can be diagnosed in a person older than 18 years only if the criteria for antisocial personality disorder are not met (American Psychiatric Association, 2000).

The DSM-IV-TR describes a mild level of the disorder as showing few if any conduct problems in excess to those needed to make the diagnosis and conduct problems that cause only minor harm to others (for example lying, truancy and breaking parental rules). A classification of "moderate" is applied when the number of conduct problems and effects on others are intermediate between "mild" and "severe." The "severe" classification is justified when many conduct problems exist which are in excess of those required for diagnosis, or the conduct problems cause considerable harm to others or property (e g. rape, assault, mugging and breaking) (Buitelaar et al., 2003). Children with CD often exhibit low intellectual functioning and low academic achievement from the outset of their school years (American Psychiatric Association, 2000).

4.4.1 Subtypes of Conduct Disorder

Hedren and Mullen (2004) state that several approaches have been used in the classification of CD. In the DSM-IV-TR two diagnostic subtypes a Childhood-Onset Type and an Adolescent-Onset Type are recognized (American Psychiatric Association, 2000).

According to Hedren and Mullen (2004) the value of this subtyping is clearly related to the prognostic significance of age at onset, with early onset being more ominous. Buitelaar et al., (2003) argue that the onset of Conduct Disorder may occur as early as the age of 5 to 6 but, is common in late-childhood or adolescence.

The Childhood-Onset Type of the disorder is defined by the presence of at least one criterion characteristic of CD before the age of 10 and is most prevalent in males Physical aggression is frequently displayed towards others, peer relationships are typically disturbed and the criteria for ODD are often met during early childhood (American Psychiatric Association, 2000). Biederman et al. (1991) and Lewis (2004) hypothesize that individuals with childhood-onset CD appear to be more likely to develop adult antisocial personality disorder than young people with onset of CD in adolescence.

The second type is Adolescent-Onset Type which is defined by the absence of CD prior to the age 10 (American Psychiatric Association, 2000). Compared to individuals with the childhood-onset type, they are less likely to display aggressive behaviours. They tend to have more normal peer relationships, and they are less likely to have persistent CD or to develop adult antisocial personality disorder.

4.4.2 Symptoms and diagnosis of Conduct Disorder

The American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV) (American Psychiatric Association, 2000) describes CD as an ongoing pattern of behaviours that clearly violate the rights of others or disregard the accepted rules of the home, school, or community. The behaviours must occur more frequently than is typically observed in individuals of comparable age and developmental level and must cause significant impairment in social, academic, or occupational functioning. The onset age is important when considering a diagnosis of CD or ODD. The age of onset for ODD is much lower than that for CD. Loeber et al. (1991)

argue that most youth diagnosed with CD have a history of ODD, but not all ODD cases will progress to Conduct Disorder.

When ODD develops into CD, the behaviours initially seen may include fighting, bullying, lying and vandalism. Later CD behaviours may include school vandalism, running away, truancy, shoplifting, house breaking, rape, aggravated assault and homicide (American Psychiatric Association, 2000). The World Health Organization (1993) describes an overall category of CD characterized by a repetitive and persistent pattern of behaviour in which, either the basic rights of others or major violations of age appropriate social expectations that last for at least 6 months are violated.

4.4. 3 DSM-IV–TR diagnostic criteria for Conduct Disorder

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

Aggression to people and animals

- Often bullies, threatens, or intimidates others
- Often initiates physical fights
- Has used a weapon that can cause serious physical harm to others (e.g., a bat, brick, broken bottle, knife, gun)
- Has been physically cruel to people
- Has been physically cruel to animals
- Has stolen while confronting a victim (e.g., mugging, purse snatching, extortion, armed robbery)

- Has forced someone into sexual activity

Destruction of Property

- Has deliberately engaged in fire setting with the intention of causing serious damage
- Has deliberately destroyed other's property (other than by fire setting)

Deceitfulness or Theft

- Has broken into someone else's house, building or car
- Often lies to obtain goods or favours or to avoid obligations (i.e., "cons" others)
- Has stolen items of non-trivial value without confronting a victim (e.g., shoplifting, but without breaking and entering property

Serious Violation of Rules

- Often stays out at night despite parental prohibitions beginning before the age of 13.
- 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)
- Is often truant from school, beginning before the age of 13

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

C. If the individual's age is 18 years or older, criteria for antisocial personality disorder are not met (American Psychiatric Association, 2000).

4.4.4 Gender Differences and Prevalence of Conduct Disorder

The prevalence of CD is difficult to estimate because of the different definitions that have been used and the variations that occur in different age groups and between the sexes (Hedren & Mullen, 2004). In the DSM-IV the prevalence of CD is estimated to approximately 9% for males and 2% for females younger than 18 years (American Psychiatric Association, 1994). In DSM-IV-TR the prevalence is estimated to be between 1% and 16% (American Psychiatric Association, 2000).

In a longitudinal cohort study of children in New Zealand, the prevalence rates of CD in males and females at the age of 15 years were very similar (7.2%) for males and (7.4%) for females (Monuteaux, Fitzmaurice, Blacker, Buka & Biederman, 2004). The prevalence rates for ODD and CD in the Limpopo Province of South Africa are also in line with those of Western countries (Meyer 2005; Meyer, 1998).

In a study conducted by Earls (1994) it was found that males are 3 to 4 times more likely to manifest CD than females. Boys relative to girls peer ecologies are characterized by higher levels of competition, aggression, rough and tumble play and domination, and by lower levels of cooperation, supportive verbal exchange, mutual accommodation and conflict avoidance (Macoby, 1998).

Synder (2004) maintains that these data imply that a gender-differentiated approach may be needed to accurately identify the peer processes mediating the linkage of deficits in Executive Functions to risk for early onset and persisting conduct problems. There is evidence that boys, on the average, experience higher levels of impulsiveness-inattention than girls (Moffitt 2001; Waschbusch, 2002). According to Moffitt (2003) early gender differences in self-regulation, along with the quality of peer relationships, have been reported to account for 50-56% of the variance in gender differences in conduct problems and antisocial behaviour. Moffitt (2001) strongly contends that deficits in self-regulation

and poor peer relations (along with temperamental traits of under-control and weak constraints) have three key features (a) they predict antisocial behaviour in both sexes, but (b) males are more likely to experience them, and as a result (c) these factors explain the sex differences.

Gender also appears to be a factor in the age of onset. While boys and girls tend to go through the same sequence of behaviour problems in the progression toward CD, these misbehaviours have a later onset in girls than in boys (Wenar & Kerig, 2000). Developmental factors also come into play in other gender- differentiated ways. For example, sex maturation is a powerful predictor of antisocial behaviour for girls (Wenar & Kerig, 2000).

4.4.5 Aetiology of Conduct Disorder

There has been much speculation about the cause of CD. CD has been linked to brain damage, genetic vulnerability, school failure, traumatic life experiences, physical and sexual abuse during childhood (Loeber, Burke, Lahey, Winters & Zera, 2000; Rothenberger , Banaschewski, Heinrich, Moll, Schmidt, & van Klooster, 2000; Schulenberg & Soundy, 2000; Toupin, Dery, Pauze, Mercier & Fortin, 2000).

4.4.5.1 Genetic Factors

Simonoff, Pickles, Chadwick, Gringras, Wood, and Higgins et al., (2006) state that one important risk factor in CD is genetics. Genetic risk factors have been associated with antisocial behaviour (Raine, 1993) and CD (Wamboldt & Wamboldt, 2000). Edelbrock, Rende, Plomin and Thompsom, (1995) and Simonoff, Pickles, Meyer, Silberg and Maes (1998) argue that genetic risk factors may have a differential influence on covert and overt CD symptoms. Although there are no criteria with which to define overt and covert CD "cases," several studies have empirically examined the genetic influence on these symptomatic subtypes using forensic categories.

Monuteaux et al., (2004) postulate that adoption studies evaluating criminal behaviour found that criminal convictions in biological parents were significantly associated with violent crimes. A family study found that parents of early onset, aggressive CD children had more antisocial behaviours than the parents of late–onset, less aggressive CD children (Lahey, Loeber, Quay, Applegate, Shaffer, & Waldman et al., 1998).

4.4.5.2 Adoption Studies

In an adoption study of 197 males and females (Cadoret, Yates, Troughton, Woodworth & Stewart, 1995) antisocial personality disorder in the biological parents was significantly associated with dimensional measures of aggression and DSM-III Conduct Disorder symptoms in the adopted offspring. In a study of 181 twin pairs aged 7 to 15, Edelbrock et al. (1995) found significant genetic effects on a dimensional measure of aggressive behaviour, but not for a measure of non- violent delinquent behaviour.

4.4.5.3 Molecular Studies

A recent molecular genetic study found an association between the tryptophan hydroxylase gene, which codes for an enzyme involved in serotonin biosynthesis, and measures of aggression and anger (Manuck, Flory, Ferrell, Dent, Mann & Muldoon, 1999). Another study conducted by (Manuck, Flory, Ferrell, Mann & Muldoon, 2000) found an association between the gene for Monoamine Oxidase-A (MAO-A) and aggression.

4.4.5.4 Prenatal Toxin Exposures

Wakschlag, Loeber, Green, Gordon and Leventhal (1997) demonstrate that maternal smoking during pregnancy is an established risk for adverse birth, health, and developmental outcomes for children. According to Maughan et al. (2004) a growing body of evidence has also highlighted an association between prenatal smoking and aggressive, antisocial behaviour in offspring. Prediction of antisocial outcomes has been reported in both clinical and epidemiological samples (Woodward & Horwood 1998), in offspring

ranging in age from preschool to adulthood (Brennan, Grekin, and Mednick, 1999) and on measures as diverse as behaviour rating scales, diagnostic interviews and crime records (Hicks, Krueger, Jacono, McGue, & Patrck, 2004).

Wakschlag, Pickett, Cook, Benowitz and Leventhal (2002) have documented dose response relationships with prenatal smoking that appear specific to antisocial outcomes and that are possibly specific to males. Wakschlag et al. (2002) suggest that there is a possibility of an aetiologic role for prenatal smoking in the development of antisocial behaviour operating through influences on early brain development. Prenatal smoking is more common among young, less well educated mothers (Matthews 2001), among women in adverse social circumstances, (Curtins, Ventura, Matthews & Park 1998) and among those who are depressed (Maughan et al., 2004).

Bardone (1998) states that all of these factors are a known risk for the development of conduct problems in children. Smoking is more common among women with antisocial traits, (Bardone, Moffitt, Caspi, Dickson, Stanton & Silva 1998) and smoking during pregnancy may be specifically a trait (Wakschlag et al., 2003). Antisocial behaviour in parents increases the risk of antisocial behaviour in children through multiple routes, both genetic (through the transmission of heritable traits) and environmental (through effects on the prenatal environment and through later risks of child and partner abuse, (Crosby, Stall, Paul & Barrett 1998), harsh and coercive discipline, comorbid drugs and alcohol problems (Margolin & Gordish, 2000).

Maughan et al., (2004) maintain that prenatal smoking may be a proxy measure indexing genetic risk for antisocial behaviour. Antisocial behaviour is partly heritable, (Rhee & Waldman 2002) as is women's smoking initiation and nicotine dependence, (Kendler, Neale, Sullivan, Corey, Gardner & Prescott 1999), raising the possibility that children who

are exposed to smoking in the utero would be at risk for antisocial behaviour regardless of whether their mothers smoked during pregnancy or not.

4.4.5.5 Parental psychological factors

According to Pfiffner, McBurnett, Rathouz and Judice (2005) it is known that a child's risk of developing CD is increased in the event of parent psychopathology. Parental substance abuse, especially in fathers, is predictive of Conduct Disorder in children (Wenar & Kerig, 2000). Maternal depression has also been linked to child conduct problems, as well as a number of other kinds of maladjustment (Sadock & Sadock, 2003).

Depressed mothers have been shown to direct a higher number of commands and criticisms towards their children, who in turn respond with increased non-compliance and deviant behaviour (Webster-Stratton & Hammond, 1999). Depressed and irritable mothers indirectly cause behaviour problems in their children through inconsistent limit setting, emotional unavailability and reinforcement of inappropriate behaviours through negative attention (Webster-Stratton & Dahl, 1999).

4.4.5.6 Familial contribution: Divorce, Marital Distress and Violence

Inter-parental conflicts surrounding divorce have been associated with the development of CD (Sadock & Sadock, 2003). It has been noted that although some single parents and their children become chronically depressed and report increased stress levels after separation, others do relatively well. Poor modeling of impulse control and the chronic lack of having their own needs met leads to a less well-developed sense of empathy (Sadock & Sadock, 2003).

Wenar and Kerig (2000) argue that boys growing up in single-parent households are also at risk. Vaden-Kiernan, Ialongo, Pearson and Kellam (1995) found that, once family income, neighborhood, and earlier aggressive behaviour were taken into account, boys with a father or father-figure in the home were less likely to be rated as aggressive

than boys in mother-only families. Wenar and Kerig (2000) argue that family stress also increases the likelihood of CD. Children who develop behaviour problems are more likely to come from families that have experienced more negative life events, daily hassles, unemployment, financial hardship, moves and other disruptions (Wenar & Kerig, 2000). According to MacMashon (1997) the family members of disruptive children have few sources of social support and engage in chronic conflict with others in the community.

4.4.5.7 Parental inconsistency

It is not only the severity of parental discipline but also a pattern of parental inconsistency (an inconsistent mix of harshness and laxness) that is related to antisocial acting out (Wenar, & Kerig, 2000). Laxness may be evidenced in a number of ways, lack of supervision, parents being unconcerned with the children's whereabouts and absence of rules concerning where the children can go and whom they can be with. Bor and Sanders (2004) identified parental coercive behaviour (such as hitting, shouting and scolding) as one of the most important risk factors for future psychopathology, including the emergence of antisocial behaviour. Maughan et al. (2004) state that coercive parenting also increases the risk for less severe adult mental health problems with intimate and social relationships.

4.5 Treatment of Conduct Disorder

A number of interventions have been identified which are useful in reducing the prevalence and incidence of Conduct Disorder (Sadock & Sadock, 2003). Interventions consist of prevention and treatment, although these should not be considered as separate entities. Prevention and treatment for CD primarily focuses on skill development, not only for the child but also for others involved with the child, including the family and school environments (American Psychiatric Association, 2000).

Kaplan, Simms, and Busner (1994) state that in the USA conventional antipsychotic agents have been the most commonly prescribed drugs for children and adolescents with aggressive behaviour with a target symptom of aggression. Positive controlled trials in children with CD have been reported for haloperidol, lithium (Adams, Small, Kafantaris, Silva, Shell, Perry, & Overall 1995; Malone, Delancy, Lucbbert, Cater & Campbell 2000), methylphenidate (Klein, Abikoff, Klaas, Ganeles, Seese & Pollack, 1997) and risperidone (Buitelaar 2001; Buiterlaar, 2003; Van der Gaag, Kettenis & Mclman, 2001).

4.5.1 Child Training

Child training involves the teaching of new skills to facilitate the child's growth, development and adaptive functioning. Kazdin (2000) indicate that as a means of preventing child CD there is a need for skill development in the area of child competence. Competence refers to the ability for the child to negotiate the course of development including effective interactions with others, successful completion of developmental tasks, contacts with the environment and use of approaches that increase adaptive functioning (Kazdin, 2000).

4.5.2 Family Intervention

A child's family system has an important role in the prevention and treatment of Conduct Disorder. The child needs to be considered as a component of a system, rather than as a single entity (Sadock & Sadock, 2003). Research supports the notion that parents of Conduct -Disordered children have underlying deficits in certain fundamental parenting skills (Webster-Stratton & Hammond, 1999). The development of effective parenting skills has been considered as the primary mechanism for change in child Conduct Disorder, through the reduction of the severity, duration and manifestation of the disorder (American Psychiatric Association, 2000).

Various training programs have been developed for the treatment of CD. The first one is parent management training (PMT) which focuses on altering the interactions between parent and child so that prosocial rather than coercive behaviour is reinforced (Wenar & Kerig, 2000). The second one is social problem-solving skills training. The focus is in intervention on helping children to develop interpersonal problem-solving skills such as generating alternative solutions to problems, anticipating the consequences of their behaviour and planning and approaching problems in a step by step fashion (Wenar & Kerig, 2000).

4.5.3 Day Treatment

Day treatment programs have shown promise for treating youth who cannot be treated successfully on an outpatient basis (Grizenko, 1997; Kolko, Bukstein & Barron, 1999). One study found that youth diagnosed with CD or ODD who were involved in a multi-modal day treatment programmes utilizing a combination of pharmacological interventions, various forms of individual and group therapy, and family therapy maintained the benefits of treatment over a five-year period (Grizenko, 1997). A second study found that a partial hospitalization program, which included methylpenidate in combination with behaviour therapy, resulted in a decrease in oppositional behaviour and an increase in positive social behaviour (Kolko, 1999).

4.5.4 Psychopharmacology

Julien (2001) states that pharmacological agents achieve their therapeutic effects on CD related behaviours by attaching to receptors and blocking the action of an endogenous transmitter (e.g., dopamine, norepinephrine, and serotonin), which is referred to as antagonistic. Monoamine oxidase (MAO) inhibitors are a type of antidepressants that achieves therapeutic effect by the agonist action of enhancing the availability of releasable neurotransmitters, particularly serotonin and norepineprhine (Julien, 2001).

The ultimate goal of either antagonist or agonist pharmacological agents is to prolong the synaptic action of specific neurotransmitters or restore normal levels of transmitter-receptor responsiveness (Julien, 2001). Several studies have examined the use of noradrenergic agents for these conditions. They found that Bupropion, a mixed dopaminergic/noradrenergic agonist, has been shown to be effective in treating children with ADHD and adults with comorbid aggressive symptoms (Newcorn, Spencer, Biederman, Milton and Michelson, 2005; Prince, Wilens, Biederman, Spencer & Woxniack, 1996).

4.6 Conclusion

CD and ODD carry substantial social risks to children and their families and communities, and need to be treated at the bio-psychosocial levels (Mpofu 2002). Conduct and oppositional problems are complex behaviours, the manifestation and causes of which vary according to the developmental stage. These behaviours are determined by a multitude of factors. Predisposing child characteristics include male gender (at least for early onset conduct problems), temperamental difficulties, early behavioural problems, lack of social skills and some characteristic cognitive distortions. A variety of other interactional and contextual factors appear to play a mediating role, increasing or decreasing the risk of those who are already vulnerable.

Chapter 5

COMORBIDITY OF ADHD: INTERNALISING DISORDERS

5.1 Introduction

The presence of comorbid externalising disorders (ODD and CD) within children with ADHD has been well established for several decades (see Chapter 4). Only in the past 15 years has it become apparent that internalising disorders (anxiety and depressive disorders) also commonly co-occur with ADHD (Jensen et al., 2001). The comorbidity between ADHD and internalising symptoms (anxiety and mood disorders) is not as strong as between ADHD and externalising disorders (ODD and CD) but it exists and it is also important (Barkley, 2006; Taylor, 1998). Studies show that 25 – 30% of children with ADHD have comorbid anxiety or mood disorders (Barkley, 2006; Biederman, 2005; Pliszka , 1999; Swanson et al., 1998; Taylor et al., 2004).

5.2 Anxiety Disorders

Anxiety Disorders are among the most common of childhood psychiatric disorders, which may be associated with low self-esteem, substance abuse, depression, social isolation, inadequate social skills and academic difficulties (Yorbik, Birmaher, Axelson, Williams & Ryan, 2004). Although most children have, through their childhood, fears and worries that can be labelled as anxiety, Anxiety Disorders occur when such worries or fears impede the child's daily activities or functioning (Biederman, 1991). Anxiety Disorders are one of the most common mental health problems that children encounter (National Institute of Mental Health, 2003).

5.2.1 Types of anxiety disorders in childhood

Huberty (2002) argues that Separation Anxiety Disorder (SAD) is the only Anxiety Disorder that specifically applies to children.

The main categories of Anxiety Disorder from the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) (American Psychiatric Association, 2000) are described below.

5.2.1.1 Separation Anxiety Disorder

Separation Anxiety Disorder (SAD) is characterized by the child's excessive distress when separated from persons to whom there is a strong attachment and by the avoidance of situations that require separation (Huberty, 2002; National Institute of Mental Health, 2003). According to Winder, Johnson and Berrin (2002) this is the only disorder specifically ascribed to children. Symptoms of Separation Anxiety Disorder include: apprehension about harm occurring to loved ones, reluctance to go to school or away from home, nightmares involving separation, inability to be alone, or repeated somatic complaints (American Psychiatric Association, 2000).

SAD is found in 2 to 3.5% of the general population and in 10% of the clinical population (Wenar & Kerig, 2000). Young children with SAD may display disruptive, oppositional behaviours in addition to avoidance behaviour that can cause significant interference in child and family functioning and in normal social development. For example, children may refuse to sleep in their own rooms, refuse to attend school, may throw tantrums when presented with situations that might involve separation and may outrightly refuse to comply with parents commands (Tonge, 1994).

5.2.1.2 Generalized Anxiety Disorder

Generalized Anxiety Disorder is defined as exaggerated or uncontrollable anxiety or worry about events. It is characterized by self-consciousness, excessive worry about future events (e.g. going to see a doctor), or about past events (e.g. something a person said) and anxiety about performance and competence (American Psychiatric Association, 2000). Children with General Anxiety Disorder (GAD) worry a great deal about things such as future events, peer relationships, social acceptability, competency and pleasing others. They are described by their parents as always worrying and overly conscientious. They predict catastrophic outcomes for future events, and underestimate their ability to cope with unfavourable situations (Bernstein & Layne, 2004).

5.2.1.3 Obsessive-Compulsive Disorder

Obsessive-Compulsive Disorder (OCD) is a heterogeneous chronic and disabling disorder belonging to the Anxiety Disorders (The European Medicines Agency Evaluation of Medicines for Human Use (EMEA, 2005). According to the DSM-IV-TR definition (American Psychiatric Association, 2000) the essential features of OCD are recurrent obsessions and or compulsions that are severe and time consuming (more than one hour a day) or cause marked distress or significantly interfere with the person's normal routine, occupational functioning, usual social activities or relationships.

Obsessions are defined as recurrent and persistent thoughts, impulse or images that are experienced as intrusive and inappropriate and cause marked anxiety or distress (Sadock & Sadock, 2003). The thoughts, impulse or images are not simply excessive worries about real-life problems, they are recognized by the patient as a product of his or her own mind (e.g. fear for contamination and symmetry obsession) (The European Medicines Agency Evaluation of Medicines for Human Use (EMEA , 2005).

Compulsions are defined as repetitive behaviours (e. g. hand washing, ordering, hoarding and checking) or mental acts (e.g. praying, counting and repeating words silently) that the child feels driven to perform in response to an obsession or according to rules that must be applied rigidly (The European Medicines Agency Evaluation of Medicines for Human Use (EMEA, 2005).

The clinical presentation of OCD in children is generally similar to that in adults (Freeman, Garcia, Swedo, Rapoport, Fucci, & Leonard, 2004). The most commonly reported ritual in children is excessive cleaning (hand washing, showering, bathing or tooth brushing), which is experienced by 85% of the patients, followed by repeating rituals, such as going in and out of doors, getting up and down from chairs, restating phrases, rereading (51%) and checking behaviours, such as making sure that doors and windows are locked, that appliances are turned off, or that homework is done right (46 %) (Freeman et al., 2004).

5.2.1.4 Post-Traumatic Stress Disorder (PTSD)

The DSM-IV describes a traumatic event as a threat to the physical integrity of self or others or as a serious injury (American Psychiatric Association, 2000; Sadock & Sadock, 2003). There are three categories of behaviours that define the disorder:

(a) Persistent re-experiencing of the event, such as intrusive, distressing recollections of the event or, in young children, repetitive play in which aspects of the trauma are expressed.

(b) Persistent avoidance of stimuli associated with the trauma, or numbing of general responsiveness such as efforts to avoid thoughts, or activities and people that arouse recollections of the trauma, or markedly diminished interest and participation in activities.

(c) Persistent symptoms of increased arousal such as irritability, outbursts of anger, difficulty in concentrating and hyper vigilance (Sadock & Sadock, 2003).

Childhood PTSD is a complex disorder with many different symptom presentations. It is therefore, not easy to establish a diagnosis. Once established PTDS in children is often both chronic and debilitating, although the course of PTDS in a given child can be highly variable. Factors responsible for the variability in the course of the disorder include the nature of the stressor, pre-existing psychopathology, quality of attachment, coping and resiliency strengths, poverty and family support (Donnelly, March & Amaya-Jackson, 2004).

5.2.1.5 Panic disorder

Panic disorder is characterised by recurrent, unexpected panic attacks, which are discrete periods of intense fear or discomfort, accompanied by specific somatic symptoms and associated with characteristic sequelae such as fear and worry (American Psychiatric Association, 2000). Panic disorder with or without agoraphobia usually begins in adolescence or early adult life but may develop at any age.

The symptoms and course, in children with panic disorder appear to be very similar to those observed in adults with panic disorder (American Psychiatric Association, 2000). The most commonly reported symptoms are trembling, dizziness or faintness, pounding heart, nausea, shortness of breath and sweating. An important and common feature in children and adolescents is comorbid separation anxiety disorder (Black, Garcia, Freeman, Karitani & Leonard, 2004).

5.2.1.6 Specific Phobia

Specific phobias are marked and persistent fears that are excessive or unreasonable, cued by the presence or anticipation of a specific object or situation that provokes immediate anxiety (American Psychiatric Association, 2000). The anxiety

response may be accompanied by a variety of somatic symptoms or, in children, may be expressed by crying, tantrums, freezing or clinging (Black et al., 2004).

Many children have fears and anxieties, but they usually decline with age, and the specific focus of the fears changes (Gullone, 2000). Preschool children are typically afraid of strangers, the dark, animals or imaginary creatures. Children of elementary school age are more likely to be afraid of animals, darkness, threats to their own safety or thunder and lightning. Older children are more concerned with health, social and school fears. If the fears persist into older ages or if there is significant and persistent distress or functional impairment, clinical evaluation is indicated (Black et al., 2004).

5.2.2 Prevalence and Prognosis of Anxiety Disorders

Wenar and Kerig (2000) state that the estimations for prevalence of anxiety disorders in population ranges from 1% to 21%. Parks (2001) argues that childhood anxiety disorders are formally believed to be less prevalent at clinical level of impairment. The prevalence is believed to be unrelated to adulthood anxiety disorders and therefore, capable of being outgrown (Parks, 2001).

The relation between childhood anxiety disorder and adult impairment is widely accepted. 54% of adults with panic disorder had an anxiety disorder as children (Wenar & Kerig, 2000).

5.2.3 Comorbidity of Anxiety Disorder with ADHD

Findings from the Collaborative Multisite Multimodal Treatment Study of children with ADHD indicate that ADHD children with comorbid anxiety disorders have treatment responses that are qualitatively different from those of children without comorbid anxiety (MTA-Cooperative Group, 1999). The children with comorbid anxiety (33.5% of the sample) show an enhanced response to behaviour therapy on outcome measures of ADHD and internalising symptoms, relatively to non-anxious children with

ADHD. They also show a better response to combined treatment methods (medication and behaviour therapy) than to medication alone, which is not the case in the non-anxious ADHD group, who responded equally to medication and combined treatment (MTA-Cooperative Group, 1999).

Children diagnosed with ADHD often relate several troubles with parents, teachers and friends (Souza, Pinheiro, & Mattos, 2005). These difficulties can lead to low self-esteem, feelings of incapacity and inadequacy which may be increased by the existence of a comorbid Anxious Disorder (Souza et al., 2005; Tannock, Ickowicz & Schachar, 1995).

ADHD children with internalizing symptoms, attending special education classrooms, are more prone to have low self-esteem. When neuropsychological tests are used, ADHD children with comorbid anxiety usually portray worse performance in tasks that involve working memory and effortful mental processing and these difficulties precede poor academic achievements (Bussing , Zima & Perwien, 2000; Souza et al., 2005). For the purpose of this study, only symptoms of General Anxiety Disorder (GAD) were assessed.

5.2.4 Aetiology of Anxiety Disorders

5.2.4.1 Family Factors

Bogela, Brechman and Toussaint (2006) reported a large overlap between anxiety disorders in family members. Children of parents with anxiety disorders have an elevated rate of anxiety disorders and they also experience an increases incidence of anxiety disorders compared to the general population (Beidel & Turner, 1997; Biederman, Rosenbaum, Bolduc, Faraone & Hirsfchfield, 1991; Merikangas, 1998).

Bogels et al. (2006) maintain that parental anxiety is generally seen as a risk factor for childhood anxiety disorders. Family factors are likely contributors to the development of anxiety disorders from inherited anxiety disposition within families and may explain the large overlap of anxiety disorders within families (Bogels et al., 2006).

5.2.4.2 Attachment

One of the potential aetiological factors for child anxiety disorders is insecure attachment of the child to his or her caregivers (Bogels et al., 2006). The attachment theory states that children have an evolutionary bias to behave in ways that enhances proximity to their caregivers. The sensitivity of the caregiver's response determines the security of the child in the relationship. Insecure early attachment experiences lead individuals to form internal working models that, though adaptive to the primary caregiver interfere with other relationships (Bogles et al., 2006).

5.2.4.3 Family structure and family functioning

Bogels et al. (2006) assert that there is little doubt that parental conflict has negative consequences on children. Emery (1998) has suggested four processes through which marital turmoil may contribute to childhood disorder (1) children model ineffective conflict resolution styles (e.g. withdrawal and anxiety) (2) parents practice inconsistent disciplinary actions which may through an experience of uncontrollability lead to anxiety (3) parental conflict disrupts parental bonds with their children and (4) parental conflict serves as a general stressors to the child's environment, threatening the child's sense of security.

In a study conducted by Cummings, Goeke-Morey and Papp (2003) insight was given into what aspects of marital conflict are related to child anxiety on a trait versus a state level. Their research suggests that children's state of anxiety levels can be explained by parental aggression towards each other during the conflict, whereas children's states seem to be a function of parent's fear, sadness and lack of problem solving during the conflict.

5.3 Mood disorders

Mood Disorders are common psychiatric illnesses that represent major causes of disability and mortality worldwide (Marvel & Paradiso, 2004). Andreasen and Black (1995)

maintain that it is estimated that 8% to 20% of the population will experience a depressive episode at some point in their lives. According to Marvel and Paradiso (2004) Mood Disorders are characterized by conspicuous disturbances in emotional disposition (that is extreme lows [depression] or highs [mania]. The lack of inability to enjoy what once was pleasurable (anhedonia) is also a primary symptom and may occur during major depression in place of a depressed mood (Marvel & Paradiso, 2004).

In the past, childhood mood disorders were under-diagnosed and misdiagnosed, but affective disorders in pre pubertal children are now receiving much more attention but continue to present difficult diagnostic problems (Weller, Weller & Danielyan, 2004).

5.3.1 Categories of mood disorders

5.3.1.1 Major Depressive Disorder

According to DSM-IV-R Major Depressive episode is a change from previous functioning over a 2 week period that includes a depressed mood or a loss of interest or pleasure (American Psychiatric Association, 2000). In addition, at least 4 of the following symptoms must be present: difficulty in sleeping or sleeping too much; loss of appetite or eating too much, difficulty concentrating or making decisions, feelings of being slowed down or agitated, feeling worthless or having low self-esteem, or having thoughts of suicide or deaths.

Although the essential features of major depression are similar in children, adolescents, and adults, there are noticeable differences in symptomatology. Somatic complaints, psychomotor agitation, and mood-congruent hallucinations are considered to be more prevalent among pubertal children. Among adolescents, antisocial behaviour, substance use, restlessness, grouchiness, aggression, withdrawal, problems with family and school, feelings of wanting to leave home or of not being understood and approved of are more frequent (Weller et al., 2004). Many children with childhood depression have atypical

features that can make it difficult to diagnose the depression (Ulloa, Birmaher, Axelson, Williams, Brent, Ryan et al., 2000).

5.3.1.2 Bipolar disorder

Little is yet known about childhood bipolar disorder. Adult criteria are usually applied to the diagnosis of mania in children, but with some modifications for differences in age and developmental stage. However, mania in childhood is still frequently misdiagnosed because of its atypical clinical picture (Weller et al., 2004). A clear differentiation between episodes of mania depression is often lacking (Carlson, 1995).

In contrast to manic adults, manic children are seldom characterised by euphoric mood, instead, the mania in young children usually presents as irritability, with prolonged and aggressive temper outbursts, worsening of disruptive behaviour, moodiness, difficulty sleeping, impulsiveness, hyperactivity, inability to concentrate, explosive anger followed by guilt, depression and poor school performance (Weller et al., 2004).

There is a big symptom overlap of childhood mania with ADHD, resulting in diagnostic confusion. Because distractibility, impulsiveness, and hyperactivity can be present in both ADHD and bipolar disorder, the differential diagnosis may be difficult (Carlson, 1995). Further, mania and ADHD can co-exist, which adds to the confusion (Biederman et al., 1996).

ADHD however, differs from bipolar disorder in the age of onset. ADHD typically manifests itself in the preschool years while in BPD the age of onset is usually later (Biederman et al., 1996). Also, the overactivity of a manic child is goal-directed, whereas that of a child with ADHD is often disorganised and haphazard. ADHD is also not associated with psychosis, flight of ideas, euphoria, or grandiosity; children with ADHD usually display low self-esteem (Weller et al., 2004).

5.3.2 Comorbidity of Mood Disorders and ADHD

The combination of ADHD and a depressive disorder could represent a subtype of ADHD, with both shared and specific features related to aetiology, outcome and clinical presentation (Jensen, 2003). Alternatively, as Angold, Costello and Erkanli (1999) have suggested that the association between depression and ADHD may be epiphenomenal, that is, attributed to the relationship that both disorders have with anxiety or conduct disorders.

In the few studies that have directly examined the characteristics of comorbid ADHD and depression (Biederman et al., 1996; Connors, Edwards, Fletcher, Baird, Barkley & Steingard 2003) found more severe ADHD symptoms and remarkably high rates of aggression among children with ADHD and depression. Despite the apparently high rates of aggression displayed by children with both ADHD and depression, both community and clinic samples have failed to find depression to be differentially represented among the respective inattentive and combined subtypes of ADHD (Crystal, Ostrander, Chen and August 2001; Power, Costigan, Eiraldi & Left 2004).

ADHD and Oppositional Defiant Disorders (ODD) are highly comorbid with MD. Recent comorbidity studies of MD and ADHD indicate that ADHD is frequently associated with MD, dysthymic disorder (DD), and bipolar disorder (BP) (Biederman, Mick & Faraone, 1998; Masi, Perugi, Toni, Millepiedi, Mucci & Bertini et al., 2006; Pliszka, 1998). In many cases, "internalizing" and "externalizing" disorders are not entirely separate but closely related (Biederman et al., 1998). For the purpose of this study only the symptoms of Major Depressive Disorder were assessed.

5.3.3 Aetiology of Mood Disorders

Parental psychopathology is the strongest predictor of the subsequent onset of child and adolescent depression and mania, although parental mania is much more specific

in predicting offspring mania (Faraone et al., 1997). Kessler (2001) maintains that effects of parental psychopathology could be due to genetic influences, environmental influences, or a combination. Kessler, Davis and Kendler (1997) argue that parental psychopathology is often part of a complex cluster of risk factors that include family violence, neglect, abuse and other types of childhood adversity.

Other premorbid correlates of depression include numerous pre and perinatal factors, developmental conditions, and physical illness, particularly childhood–onset diabetes, migraine and asthma (Breslau, Chilcoat, Johnson, Andreski & Lucia, 2000). In a study of the relationship between a range of pre-and perinatal influences and subsequent risk of psychopathology in a community-based sample of adolescents, Allen, Lewinson and Seeley (1998) found that major depression in late adolescence was associated with not being breastfed and maternal emotional problems during the pregnancy, mediated by the effects of maternal depression, maternal-child conflict and physical symptoms in the child.

5.4 Conclusion

Internalising disorders (anxiety and mood disorders) have often been associated with ADHD in children. They may as a result affect the child's overall degree of impairment, the course of the disorder and the treatment plan.

Chapter 6

PROBLEM STATEMENT AND HYPOTHESES

6.1 Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a highly prevalent, clinically heterogeneous disorder that exerts an enormous burden on society in terms of financial cost, stress to families, and adverse academic and vocational outcomes (Biederman, 2005; Biederman & Faraone, 2004; Harpin, 2005). ADHD is a multifactorial disorder with complex aetiology and strong genetic underpinnings (Faraone et al., 2005). The inattention component of ADHD is manifested as daydreaming, distractibility and difficulty in focusing on a single task for a prolonged period, whereas the hyperactive component is expressed as fidgeting, excessive talking and restlessness (Biederman, 2005).

The symptoms of ADHD predispose to accidents, create strain in interpersonal relationships, and disrupt the environment through interruptions and inappropriate behaviour (Barkley, 2006; Biederman, 2005). It is notable that the more overt symptoms of Hyperactivity/Impulsiveness tend to wane early in life, whereas the more covert symptoms of inattention tend to persist over time (Biederman et al., 1996). The areas of impairment associated with childhood ADHD include academic and social dysfunction and skill deficits (Barkley, 2006; Biederman & Faraone, 2005; Taylor et al., 2004). ADHD is a basic, neuro-behavioural disorder, not a cultural phenomenon (Aase et al., 2006; Meyer, 2005).

ADHD is often comorbid with externalising disorders (ODD and CD) and/or internalising disorders (anxiety and mood disorders). Only fairly recently, comorbidity in ADHD has come to the forefront as one of the most important aspects of the disorder, and it is commonly agreed that when ADHD is associated with the presence of one or

more disorders, and these problems are often at least as important as ADHD in contributing to the longer term outcome in the individual child (Gillberg & Gillberg, 2004).

6.2 The problem: Comorbidity of ADHD, with ODD, CD, Anxiety and Mood Disorders

6.2.1 Problem Statement

As much as ~60% of all children with ADHD in the general population meet the criteria for at least one other diagnosis (Kadesjo and Gillberg, 2001), which means that young children with ADHD are usually handicapped by several different types of psychiatric/developmental problems.

The externalising disorders, ODD and CD are the most frequently reported co-existing disorders in ADHD (Cunningham & Boyle, 2002; Gillberg et al., 2004; Jensen, 2001; Pliszka, 2000). Children with comorbid ODD and CD are at an increased risk to develop problems with antisocial behaviour than children without externalising disorders (Barkley, 2006; Pliszka, 1999) and this may even lead to antisocial personality disorder (Mannuzza, Klein, Abikoff & Moulton, 2004).

ADHD with comorbid anxiety and depression may lead to low self-esteem, feelings of incapacity and inadequacy (Souza et al., 2005; Tannock et al., 1995). Children with comorbid anxiety also perform worse on cognitive and neuropsychological tasks (Tannock et al., 1995), usually the tasks that involve working memory and effortful mental processing are severely affected and these difficulties precede poor academic achievements (Bussing et al., 2000; Souza et al., 2005). ADHD children with comorbid internalising disorders also need different treatment methods than those with non-comorbid ADHD (Jensen, 2001).

Evidence from clinical, school and community samples documented that ADHD is highly comorbid with externalising (ODD and CD) and internalising (anxiety and mood) disorders (Biederman et al., 2005). This high level of psychiatric comorbidity within ADHD has been clearly recognised in the guidelines of the American Academy of Pediatrics, (2000) and the European clinical guidelines (Taylor et al., 2004). The question can therefore be asked: Is this also the case in indigenous South African children?

6.3 Aims of the study

The aim of the investigation was twofold:

1. To establish a relationship between the core systems of ADHD (Inattention and Hyperactivity/Impulsiveness) and the externalising disorders, ODD and CD, and the internalising disorders anxiety and depression.
2. To establish differences in comorbid symptoms (ODD, CD, anxiety and depression) between children with ADHD and a non-ADHD control group as a function of gender and subtype.

6.4 Hypotheses

6.4.1 Research hypothesis 1:

There is a relationship between the core symptoms of ADHD (inattentiveness and hyperactivity/impulsiveness) and those of the externalising disorders (ODD and CD) and the internalising disorders (anxiety and depression).

6.4.1.1 Specific hypothesis derived from research hypothesis 1.

Null hypothesis 1.1

There is no relationship between inattention symptoms and symptoms of ODD.

Null hypothesis 1.2

There is no relationship between inattention symptoms and symptoms of CD.

Null hypothesis 1.3

There is no relationship between inattention symptoms and symptoms of anxiety.

Null hypothesis 1.4

There is no relationship between inattention symptoms and symptoms of depression.

Null hypothesis 1.5

There is no relationship between hyperactivity/impulsiveness symptoms and symptoms of ODD.

Null hypothesis 1.6

There is no relationship between hyperactivity/impulsiveness symptoms and symptoms of CD.

Null hypothesis 1.7

There is no relationship between hyperactivity/impulsiveness symptoms and symptoms of anxiety.

Null hypothesis 1.8

There is no relationship between hyperactivity/impulsiveness symptoms and symptoms of depression.

6.4.2 Research hypothesis 2:

There will be differences in the symptoms of externalising disorders (ODD and CD) and internalising disorders (anxiety and depression) between a group of children with ADHD and a control group without ADHD symptoms. The children with ADHD will exhibit more symptoms of ODD, CD, anxiety and depression than the control group. There will be differences in symptomatology among the subtypes and between the genders.

6.4.2.1 Specific hypotheses derived from Research hypothesis 2:

Null hypothesis 2.1

There is no difference in symptoms of ODD between children with ADHD and a non-ADHD control group.

Null hypothesis 2.2

There is no difference in symptoms of CD between children with ADHD and a non-ADHD control group.

Null hypothesis 2.3

There is no difference in symptoms of anxiety between children with ADHD and a non-ADHD control group.

Null hypothesis 2.4

There isno difference in symptoms of depression between children with ADHD and a non-ADHD control group.

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

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Chapter 7

RESEARCH METHODOLOGY

7.1 Introduction

ADHD has a strong association with Externalising Disorders (conduct problems and antisocial disorders) as well as Internalsing Disorders (Anxiety and Depression). Children who experience co-occurring conditions are likely to experience greater symptom severity and persistence, and more negative outcomes, than children without any comorbidity and should be identified at an early age in order to benefit from intervention.

The purpose of this study was (1) to establish a relationship between the core symptoms of ADHD (Inattention and Hyperactivity/Impulsiveness) and the externalising disorders, ODD and CD, and the internalising disorders anxiety and depression and (2) to determine whether there are differences in the symptoms of externalising disorders (ODD and CD) and internalizing disorders (anxiety and depression) between a group with ADHD symptoms and a non-ADHD control group analysed as a function of subtype and gender.

This chapter focuses on the research design applied, sampling, measurement instruments used, procedures employed and the methods of analysis.

7.2. Research Design

A correlation and comparative study were undertaken. First, the scores on the DBD rating scale (Inattention and Hyperactivity/Impulsiveness) were correlated with the ODD and CD scores obtained from the DBD rating scale and with the scores on the Anxiety and Depression scales of the "Terry" picture questionnaire, to establish a relationship between the symptoms of ADHD and the externalizing and internalizing comorbid disorders.

Secondly, the children classified as ADHD (all three subtypes) (n=50) were compared for symptoms of ODD, CD, anxiety and depression with a comparison group without ADHD symptoms as a possible function of gender and subtype.

This is a quantitative study with a quasi-experimental research design, as the subjects cannot be randomly assigned to the conditions of the independent variable because they already exhibit the variable.

7.3 Sample

The sample for this study was drawn from Tsonga speaking primary school children aged 6-12 who were screened for ADHD using the Disruptive Behaviour Disorders Rating Scale (DBD). Children were recruited from rural, semi-rural and township areas of Hlanganani and Kgapane in the Vhembe District. The control group consisting of children without ADHD was matched with the experimental group for age, sex and socio-economic status (SES).

Figure 7.1 gives an overview of the gender distribution of the sample

Figure 7.1 Distribution of gender groups:

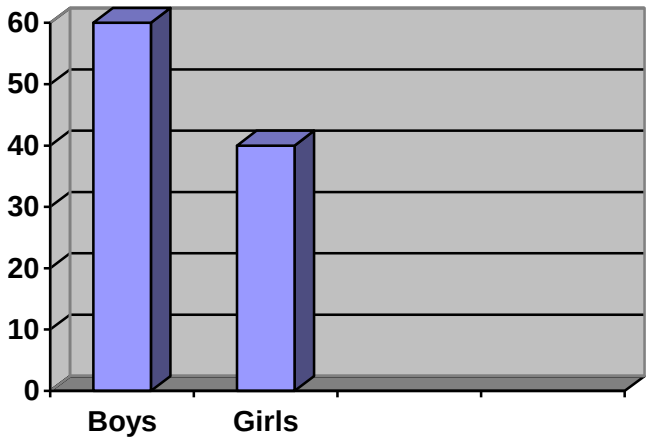

Figure 7.1 show that 40% of the sample consists of girls and 60% boys.

The distribution of the subtypes is illustrated in Figure 7.2.

Figure 7.2 Distribution of subtypes:

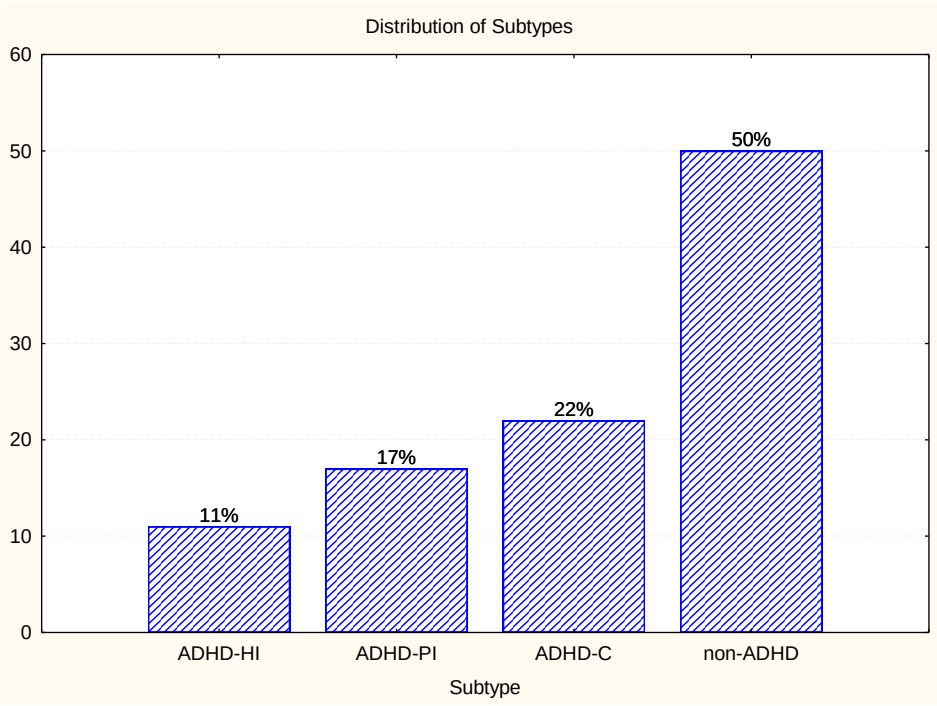

Figure 7.2 shows that 11% of the sample consist of ADHD-HI, 18% ADHD-PI subtype, 18% ADHD-C subtype and 47% non-ADHD control group.

Table 7.1 shows the mean age, as well as the scores on the Hyperactive/Impulsiveness and Inattention scales of the DBD rating Scales for gender groups and subtypes.

Table 7.1 the demographic characteristics in Age and DBD scores on the Hyperactive/Impulsive and Inattention scales

Gender	Subtype	N	Age (months) Mean	H/I score Mean	Inat. score Mean
Boys	ADHD-HI	6	124.40 ± 29.02	17.40 ± 0.55	12.00 ± 4.00
	ADHD-P1	7	120.43± 22.99	8.29 ± 5.9	22.86 ± 3.91
	ADHD-C	13	103.69 ± 15.62	22.85 ± 2.82	24.54 ± 3.91
	Non-ADHD	26	122.41± 25.16	6.22 ± 4.08	6.59 ± 5.22
Girls	ADHD-HI	5	130.00 ± 31.05	19.00 ± 2.45	10.50 ± 3.94
	ADHD-PI	11	116.36 ± 24.94	9.36 ± 6.55	20.18 ± 4.31
	ADHD-C	5	105.80 ± 23.16	19.00 ± 2.92	22.40 ± 3.91
	Non-ADHD	21	121.45 ± 25.20	3.65 ± 3.31	5.00 ± 5.47

The difference in age between the ADHD groups and the non-ADHD controls was statistically not significant (p = 0,17).

The difference in scores between the ADHD groups and the non-ADHD controls on both the Hyperactive/Impulsive and Inattention scales of the DBD rating scale was statistically significant (p = 0,00 in both instances).

7.4 Measurement Instruments

The following measurement instruments were used in the study:

7.4.1 DBD Rating Scale

The (DBD) rating scale developed by Pelham et al., (1998) was used for screening the children to form two groups: with ADHD symptoms and non-ADHD controls. This scale has been translated into the official languages of the Limpopo Province (Tsonga, Venda, Tswana, Northern Sotho, English and Afrikaans) and norms were established for those populations groups (Meyer et al., 2004).

The DBD assesses the presence and the degree of ADHD-related symptoms (inattention and hyperactivity/impulsiveness, Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) as formulated in the DSM-IV. The DBD consists of 42 items based on the DSM-IV diagnostic criteria (American Psychiatric Association, 2000) of which 18 measure ADHD-related symptoms. Teachers were asked to rate the behaviour on a four point scale consisting of the following options: not at all (0); just a little (1); pretty much (2); and very much (3). The total score is added up for ADHD-related symptoms, ODD and CD and compared to the cut-off point of the 95th percentile, which has previously been identified as clinically significant (Barkley, 1997; Barkley and Murphy, 2006). This group was matched for gender and age by children whose scores fell below the 85th percentile and formed the control group.

7.4.2 ODD and CD

The ODD and CD scales from the DBD rating scale (Meyer et al., 2004; Pelham et al., 1992; Pillow et al., 1998) as reported by the teachers was used to establish the symptoms of ODD and CD.

7.4.3 Anxiety and Depression Scale

The Anxiety and Depression Scales of the "Terry" picture questionnaire were used to establish the symptoms of Anxiety and Depression (Valla et al., 2000). The Terry is a cartoon-like questionnaire designed to evaluate the mental disorders of children aged 6-12 years. It is based on DSM-IV criteria (American Psychiatric Association, 2000). It takes into account the cognitive limitations of children. The "Terry" Picture Questionnaire has 96 pages comprising two notebooks of 49 and 47 pages respectively.

The first notebook also contains four introductory pages designed to familiarize the child with material and characters, and indicate what he or she is expected to do. It covers seven most frequent mental disorders in children aged 6-11 years: simple phobia (10 drawings), separation anxiety (13 drawings), over-anxiety (11 drawings), major depression

(19 drawings), hyperactivity with attention deficit (16 drawings), ODD (13 drawings) and CD (12 drawings). Despite its graphic elements, the "Terry" is not a projective test because it illustrates specific situations that leave little room for interruption (Valla et al., 2000).

7.5 Procedure

Written permission to conduct research among primary school children was obtained from the Department of Education. A letter obtained from the Ethics Committee of the University of Limpopo outlining the aim and the purpose of the study was presented to the respective school principals who in turn presented the research project to the teachers. Written consent was obtained from the parents.

Teachers were asked to complete the Disruptive Behaviour Disorders checklist (DBD). The ADHD and non-ADHD control groups were selected. A biographical questionnaire was completed. Children on ADHD medication were requested not to take any for at least 24 hours prior to assessment. Children with a history of neurological trauma, psychosis or other severe psychiatric disorders were not included in this study.

The children were tested during school time as arranged with the school principal and class teachers. The tests were administered by two student researchers fluent in the children's language (Tsonga) and with experience in the administration of the neuropsychological tests. The researcher and one assistant conducted the testing. The researcher is registered with the Health Professions Council of South Africa as a Psychometrist and has experience in the administration of psychometric tests and the assistant is registered with the Health Professions Council of South Africa as an intern clinical psychologist.

7.6 Method of data analysis

The computer programmes SPSS-13 (SPSS 2007) and STATISITCA 7 (StatSoft Inc, 2007) were employed. Pearson's product-moment correlation was used to investigate the relationship between ADHD scores on the DBD rating scale and CD and ODD scores

on the DBD scale and the Anxiety and Depression scores on the "Terry" picture questionnaire. Multiple Analysis of Variance (MANOVA) was used to investigate possible group differences (the groups were divided according to subtypes and gender) in raw scores. Post-hoc (Bonferroni) tests were used to establish where the differences occurred.

Chapter 8

RESULTS

8.1 Introduction

The aim of this study was (1) to establish a relationship between the core symptoms of ADHD (Inattention and Hyperactivity/Impulsiveness) and the externalising disorders, ODD and CD, and the internalising disorders anxiety and depression and (2) to establish differences in comorbid symptoms (ODD, CD, anxiety, and depression) between children with ADHD and a non-ADHD control group as a function of subtype and gender.

8.2 Results of the study.

The results of the study are presented in the following format:

1. Descriptive Statistics for the externalizing and internalizing disorders.

Correlation Study:

2. Correlation coefficients

3. Scatterplots of the results

Comparison study:

4. Graphic representations of results

5. MANOVA results

6. Results of the *post-hoc* analysis (Bonferroni correction)

8.2.1. Descriptive statistics

Table 8.1 depicts the scores obtained on the DBD rating scales for the externalizing and internalizing disorders.

Table 8.1: Scores obtained for ODD, CD, Anxiety and Depression

Gender	Subtype	N	CD Mean	ODD Mean	Anxiety Mean	MDD Mean
Boys	ADHD-HI	8	9.80 ± 3.70	10.20 ± 3.56	12.40 ± 2.51	5.80 ± 3.35
	ADHD-PI	7	6.43 ± 5.16	13.43 ± 4.79	14.43 ± 2.57	10.43 ± 4.99
	ADHD-C	15	6.48 ± 5.16	18.15 ± 4.79	9.69 ± 4.01	6.23 ± 2.74
	non-ADHD	30	2.56 ± 2.36	4.48 ±3.67	8.93 ± 4.09	4.63 ± 2.66
Girls	ADHD-HI	3	12.33 ± 10.63	12.83± 8.11	10.33 ± 2.58	5.00 ± 3.22
	ADHD-PI	10	3.27 ± 2.45	7.09 ± 4.30	13.91 ± 3.53	13.09 ± 3.75
	ADHD-C	7	8.40 ± 4.39	12.00 ± 5.61	12.40 ± 4.22	7.00 ± 4.53
	Non-ADHD	20	1.80 ± 1.28	2.80 ± 3.11	8.20 ±4.4	4.25 ± 2.59
All Groups		100				

8.2.2 Correlation results

Table 8.2 represents the correlation (Pearsons product moment) between ADHD scores of the DBD scales and symptoms of ODD and CD, as reported on the DBD rating scale and the symptoms of Anxiety and Depression as reported on the 'Terry' picture questionnaire.

Table 8.2 Correlations between the scores of the DBD scale and comorbid disorders

	CD	ODD	Anxiety	Depression
Hyperactivity/Impulsiveness	0.69*	0.76*	0.11	0.06
Inattention	0.48*	0.74*	0.38*	0.43*

*p<0.05

There were statistically significant relationships ($p < 0.05$) between Hyperactivity/ Impulsiveness and CD and ODD symptoms. The relationships could be described as moderate to strong in the case of CD ($r = 0.69$) and strong ($r = 0.76$) in the case of ODD.

There were no statistically significant correlations between the symptoms of Hyperactivity/Impulsiveness and the symptoms of Anxiety and Depression. The relationships obtained were very weak (Anxiety, r = 0.11 and Depression, r = 0.06).

There were statistically significant relationships ($p<0.05$) between Inattention symptoms and the symptoms of CD, ODD, Anxiety and Depression. The relationship between Inattention symptoms and ODD symptoms was moderately high (r = 0,74), while the relationships between Inattention symptoms and symptoms of CD (r = 0.48), Anxiety (0.38) and Depression (0.43) were moderate to weak.

Figure 8.1 illustrates the relationship between the scores on the Hyperactivity/ Impulsiveness and CD scales of the DBD questionnaire

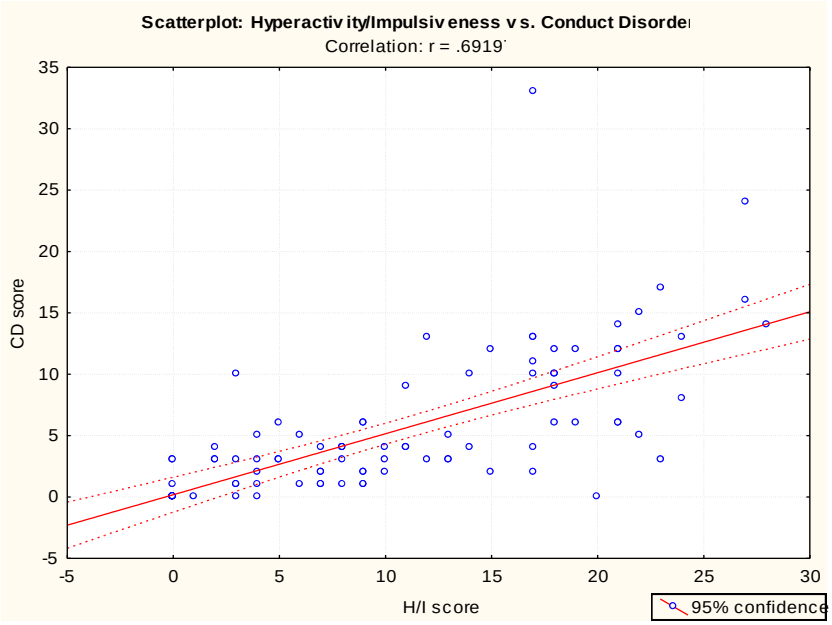

Figure 8.1: The slope of the graph in the scatterplot depicted in Figure 8.2 shows a moderate to strong positive correlation between the symptoms of Hyperactivity/Impulsiveness and CD.

Figure 8.2 illustrates the correlation between Hyperactivity/Impulsiveness and Oppositional Defiant Disorder.

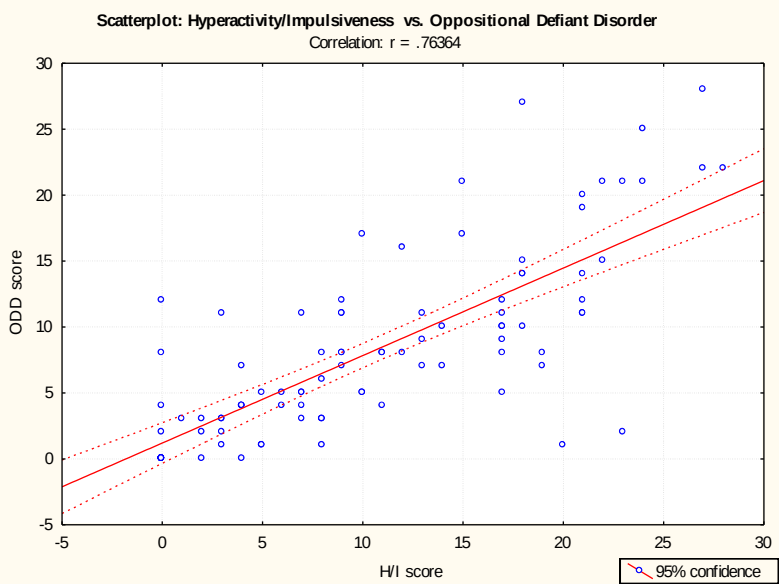

Figure 8.2: The graph shows an increase in slope, which indicates a strong positive relationship between the symptoms of Hyperactivity/ Impulsiveness and the symptoms of ODD as reported on the DBD rating scale.

Figure 8.3 illustrates the correlation between Hyperactivity/Impulsiveness (scores on the DBD) and Anxiety Disorder (scores on the 'Terry' questionnaire).

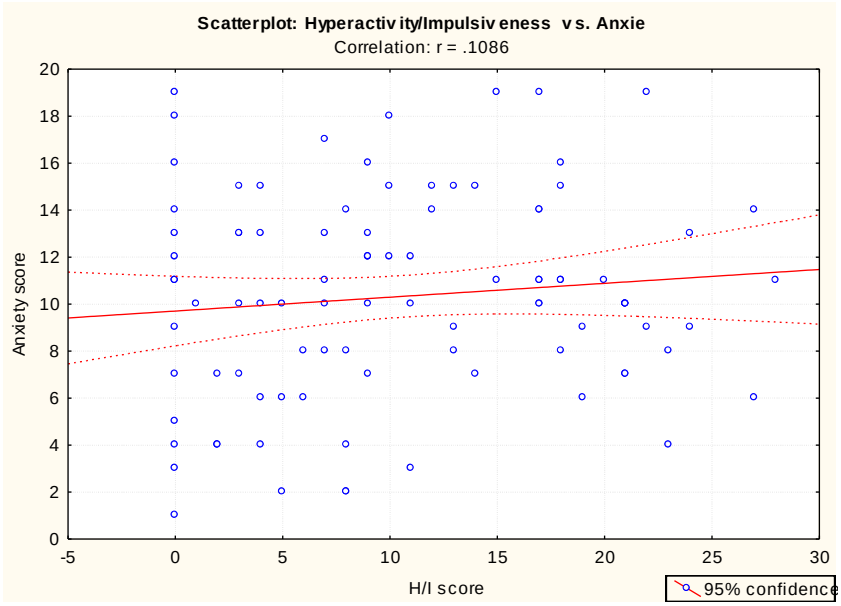

Figure 8.3: The slope of the graph shows a very slight relationship between the symptoms of Hyperactivity/Impulsiveness and the symptoms of Anxiety. This relationship was not statistically significant.

Figure 8.4 illustrates the results of the correlation between the symptoms of Hyperactivity/Impulsiveness as reported on the DBD rating scale and the symptoms of MDD as reported on the "Terry" picture questionnaire.

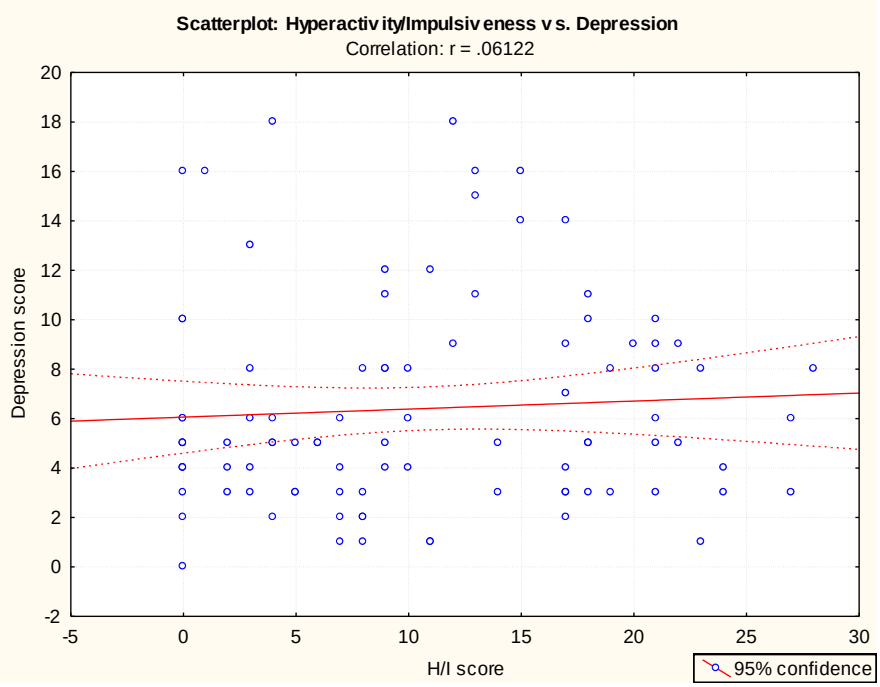

Figure 8.4: The slope of the graph shows a very weak correlation (statistically not significant) between the symptoms of Hyperactivity/Impulsiveness and MDD.

Figure 8.5 illustrates the correlation between the symptoms of Inattention and the symptoms of Conduct Disorder as reported on the DBD rating scale.

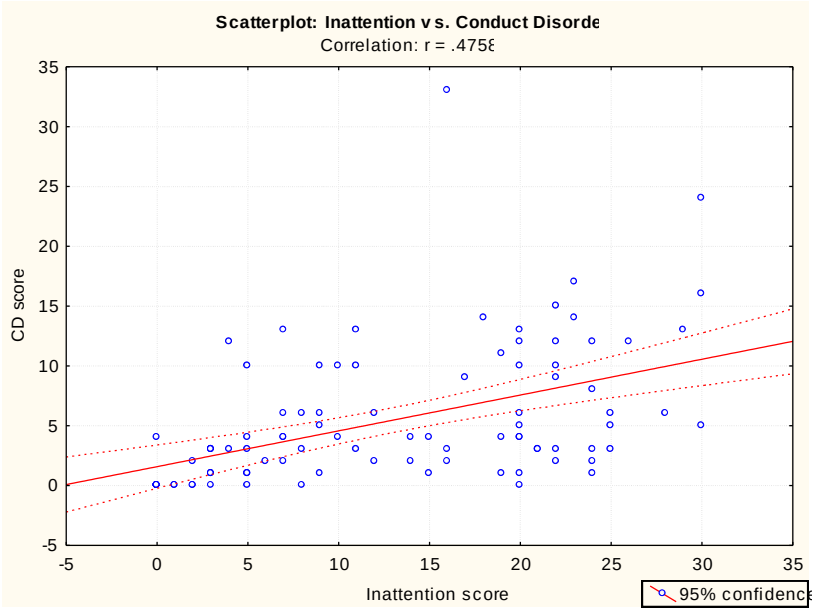

Figure 8.5: The slope of the graph shows a moderate positive correlation between the symptoms of Inattention and CD symptoms. The relationship is statistically significant.

Figure 8.6 illustrates the correlation between the symptoms of Inattention and the symptoms of Oppositional Defiant Disorder as reported by the teachers on the DBD rating scale.

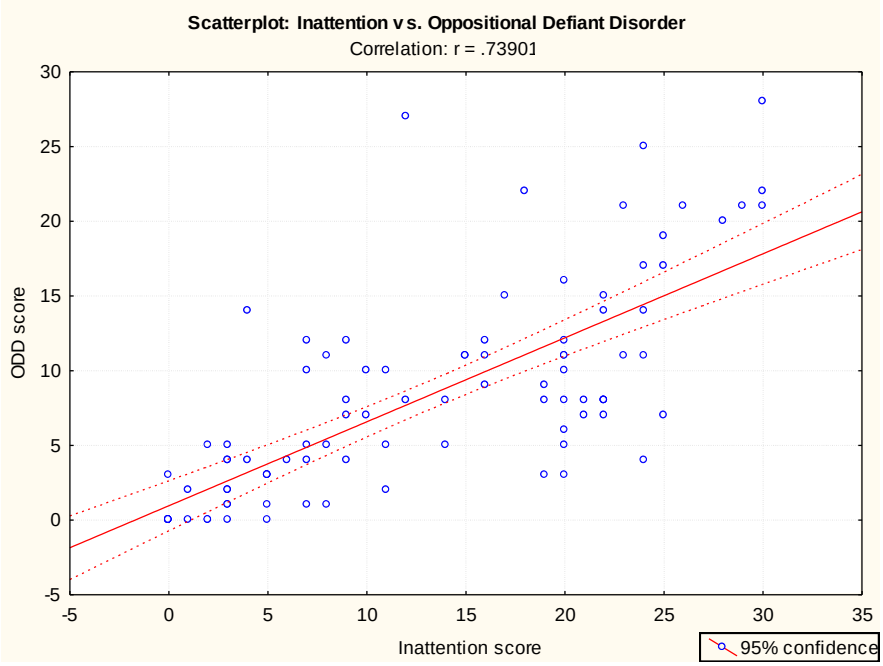

Figure 8.6: shows a moderate to strong increase in slope which shows a positive correlation between Inattention and ODD symptoms.

Figure 8.7 illustrates the correlation between symptoms of Inattention as reported on the DBD rating scale and Anxiety symptoms as reported on the 'Terry' picture questionnaire.

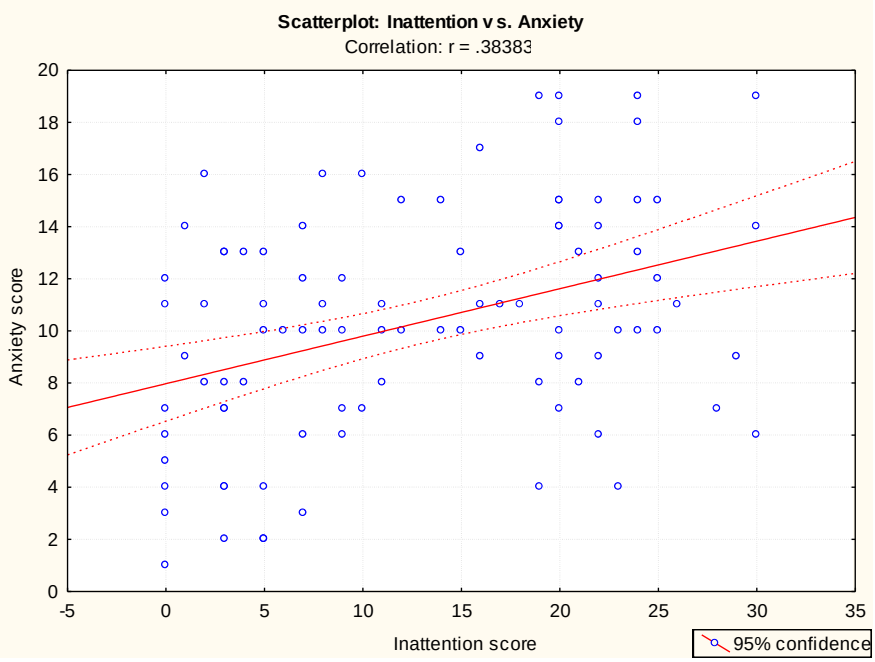

Figure 8.7: The slope of the graph shows a positive, although weak, correlation between the symptoms of Inattention and Anxiety symptoms. However, the relationship was statistically significant.

Figure 8.8 shows the scatter plot of the relationship between symptoms of Inattention as reported on the DBD rating scale and the symptoms of Major Depression Disorder as reported by the children on the 'Terry' picture questionnaire.

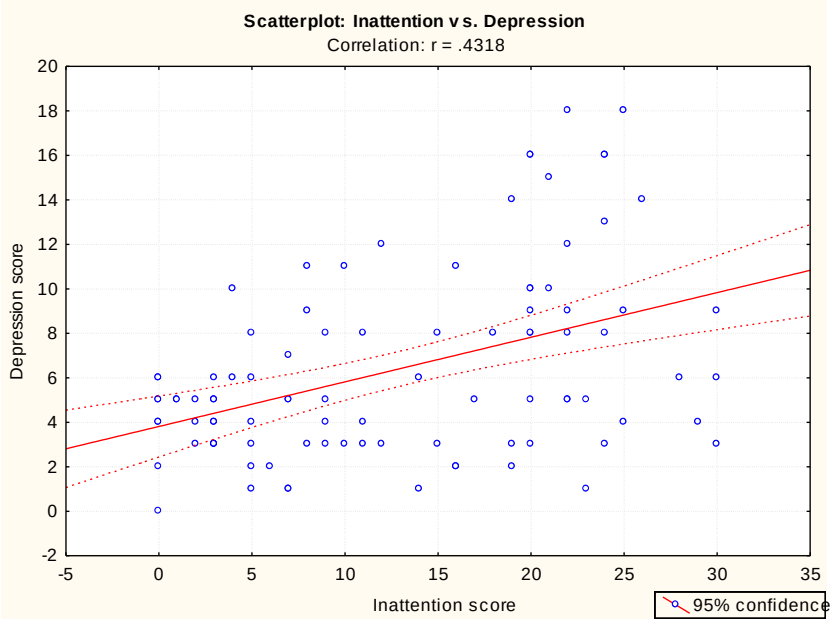

Figure 8.8: The slope of the graph shows an increase which shows that there is a moderate relationship between the symptoms of Inattention and symptoms of MDD.

8.3.2 Comparison study

The second phase of the study tried to establish differences in symptomatology of comorbid (both externalizing and internalizing) disorders between the three subtypes of ADHD (ADHD-HI, ADHD-PI, and ADHD-C) and a non-ADHD control group. The results are given below.

The tabulated descriptive statistics of the results of the symptoms of ODD and CD, (externalizing disorders) and anxiety and depression (internalizing disorders) are given in Table 8.1

Graphic representation of the results of Externalising Disorders:

Figure 8.9. illustrates the plot of the means for CD and ODD scores on the DBD rating scale for the subtypes and gender groups:

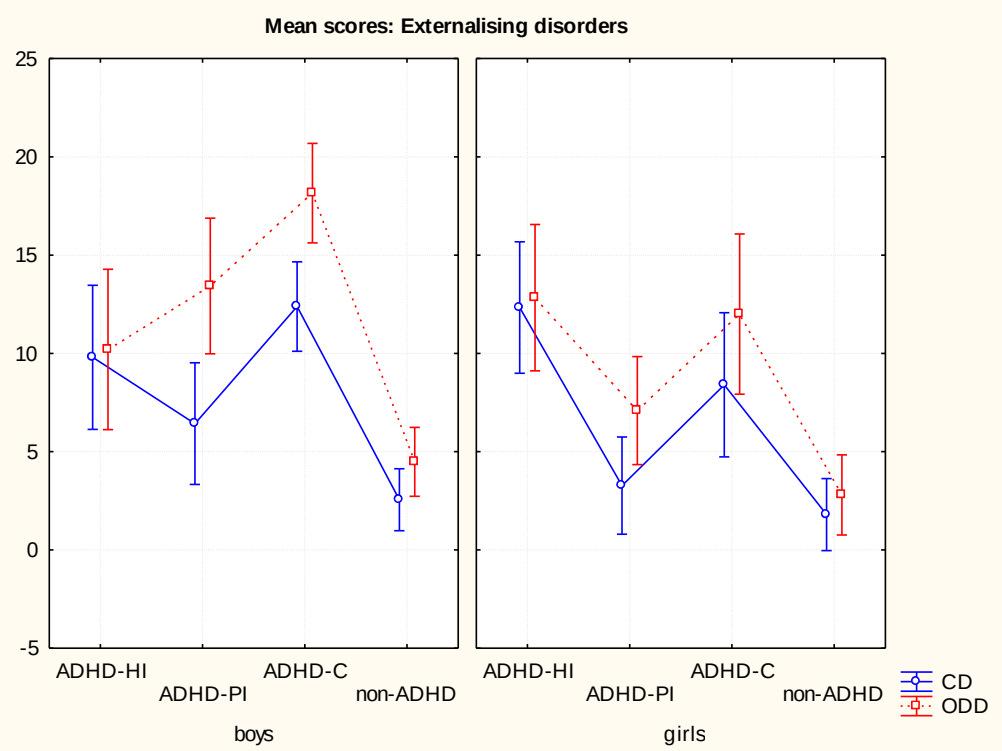

Figure 8.9: shows that for boys the non-ADHD group has the lowest score for both ODD and CD symptoms, while for ODD the ADHD-PI subtype had a higher score than the ADHD-HI subtype. On CD symptoms the ADHD-HI scored higher than the ADHD-PI subtype. The ADHD-C subtype had the highest symptomatology for both the symptoms of ODD and CD. In the case of the girls, the symptoms for ODD and CD followed the

same pattern: most symptoms were encountered in the ADHD-HI subtype, followed by the ADHD-C, ADHD-PI and non-ADHD groups.

8.3.3 Internalising Disorders:

Figure 8.10 illustrates the plot of means for Anxiety and Depression scores on the "Terry" picture questionnaire, for the gender groups and subtypes of ADHD.

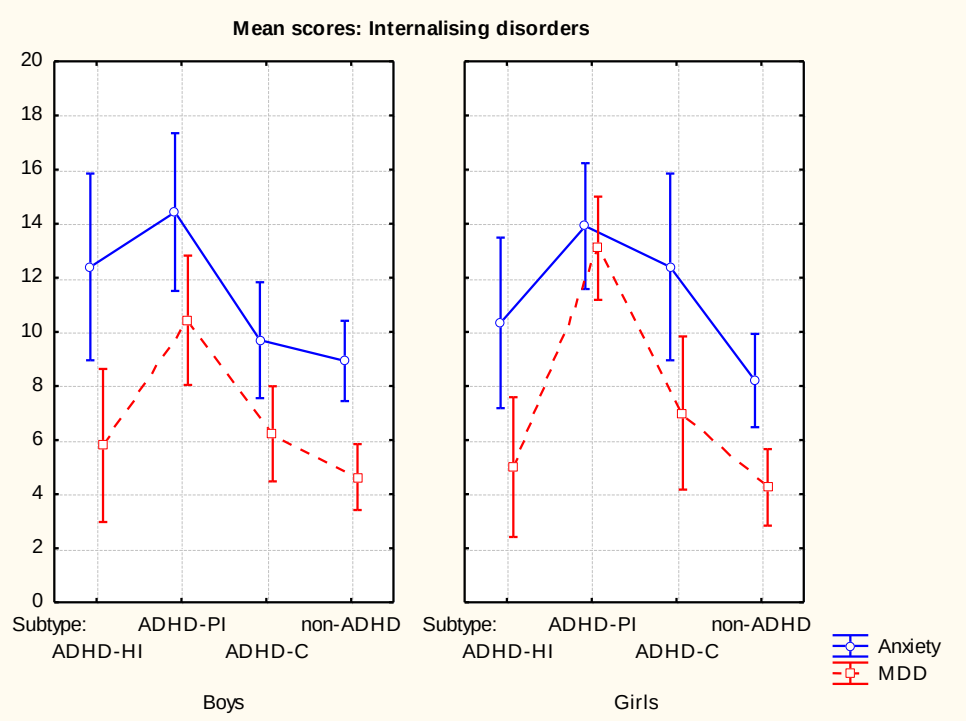

Figure 8.10: shows that for both boys and girls the most symptoms for anxiety and depression were observed in the ADHD-PI subtype, followed by the ADHD-C, ADHD-HI, and non-ADHD groups.

MANOVA Analysis:

Table 8.3 reflects the results of the MANOVA for Externalising Disorder scores on the DBD rating scale for the ADHD subtypes and non- ADHD control group.

Table 8.3 Results of the MANOVA: Externalising Disorders

	Test	F	DF	P
Gender	Wilks	3.5995	2, 85	0.03*
Subtype	Wilks	18.1103	6, 170	0.00*
Gender x Subtype	Wilks	1.8917	6, 170	0.08

*p<0.05

There were significant effects of gender (p = 0.03) and subtype (p = 0.00), therefore the gender groups were analysed separately.

Post-hoc test (Bonferroni) was used to establish where the differences occurred for the Externalising Disorders. The results of the *post-hoc* (Bonferroni) test performed on the scores of the CD scale of the DBD rating scale as a function of gender, is depicted in Table 8.4.

Table 8.4: Results of the Post-hoc analysis (Bonferroni) test for CD symptoms

Gender	**Comparison**	**Significance (p-level)**
Boys	ADHD-HI v. non-ADHD	0.01*
	ADHD-PI v. non-ADHD	n/s
	ADHD-C v. non-ADHD	0.00**
Girls	ADHD-HI v. non-ADHD	0.00**
	ADHD-PI v. non-ADHD	n/s
	ADHD-C v. non-ADHD	0.05*

*p≤0.05; **p<0.001

The analysis showed statistically significant differences in CD symptomatology between the ADHD-HI and ADHD-C subtypes when compared to the non-ADHD control group, with the ADHD groups showing more severe symptomatology. This was both the case for the boys (p = 00,1) for the ADHD-HI subtype and (p = 0.00) for the

ADHD-C subtype and the girls (p = 0.00) for the ADHD-HI subtype and (p = 0.05) for the ADHD-C subtype.

There were no statistically significant differences in CD symptoms when the ADHD-PI subtype was compared to the non-ADHD controls. This was the case for both genders.

Table 8.5 shows the results of the post-hoc (Bonferroni) test on the ODD symptoms when the three subtypes are compared with the non-ADHD controls.

Table 8.5 Results of the post-hoc analysis (Bonferroni) tests: ODD symptoms

Gender	Comparison	Significance (p-level)
Boys	ADHD-HI v. non-ADHD	n/s
	ADHD-PI v. non-ADHD	0.00**
	ADHD-C v. non- ADHD	0.00**
Girls	ADHD-HI v. non-ADHD	0.00**
	ADHD-PI v. non-ADHD	n/s
	ADHD-C v. non- ADHD	0.00**

**$p \leq 0.001$

The analysis showed no significant differences in ODD symptomatology between ADHD-HI subtype and the non-ADHD control group for the boys, while for the girls there was a statistically significant difference (p = 0.00). In the case of the ADHD-PI groups the analysis showed the opposite: There was a significant difference (p = 0.00) in ODD symptoms when the ADHD-PI boys were compared with the non-ADHD group, with the ADHD groups having more symptoms of ODD. The comparison between the ADHD-PI girls and non-ADHD girls did not show a significant difference in ODD symptomatology. When the ADHD-C subtype was compared with the non-ADHD

controls, both gender groups showed statistically more ODD symptoms than their non-ADHD comparisons (p = 0.00).

Table 8.6 represents the results of the MANOVA for Anxiety and Depression scores on the "Terry" picture questionnaire

Table 8.6 MANOVA results for gender and ADHD subtypes

	Test	**F**	**DF**	**P**
Gender	Wilks	0.3321	2, 85	0.72
Subtype	Wilks	10.9725	6, 170	0.00*
Gender & Subtype	Wilks	1.0139	6, 170	0.42

*p < 0.01

There were no effects of gender, neither main nor interacting; therefore the gender groups were not analyzed separately. The difference for internalizing disorder symptoms between the subtypes was statistically significant (p = 0.00).

Table 8.7. represents the *post-hoc* (Bonferroni) test for symptoms of anxiety disorder for the ADHD groups, according to subtypes.

Table 8.7 Post-hoc (Bonferroni) test for Anxiety Disorder

Comparison	**Significance (p-level)**
ADHD-HI v. non-ADHD	n/s
ADHD-PI v. non-ADHD	0.00*
ADHD-C v. non-ADHD	n/s

*****p **<** 0.01

The *post-hoc* analysis did show that there were statistically significant differences in symptoms of Anxiety Disorder between the ADHD-PI and non-ADHD groups only (p = 0.00), with the ADHD-PI subtype exhibiting more anxiety symptoms.

Table 8.8 indicates the results of the *post-hoc* (Bonferroni) test performed on the symptoms of Major Depression Disorder.

Table 8.8 Post-hoc (Bonferroni) test for Depression Disorder

Comparison	Significance (p-level)
ADHD-HI v. non- ADHD	n/s
ADHD-PI v. non-ADHD	0.00*
ADHD-C v. non- ADHD	n/s

*$p < 0.01$

Like in the case of anxiety symptoms, the *post-hoc* analysis showed that there were statistical significant differences in symptoms of MDD only between the ADHD-PI subtype and the non-ADHD controls ($p = 0.00$). The ADHD-PI subtype had significantly more symptoms of MDD.

8.4 Hypothesis testing

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

8.4.1 Research hypothesis 1:

Null hypothesis 1.1 must be rejected, as there was a statistically significant correlation between the symptoms of Inattention and symptoms of ODD as reported by the teachers on the DBD ratings scale.

Null hypothesis 1.2 must be rejected as there was a statistically significant correlation between the symptoms of Inattention and symptoms of CD as reported by the teachers on the DBD ratings scale.

Null hypothesis 1.3 must rejected, as there was a statistically significant correlation between the symptoms of Inattention as reported by the teachers on the DBD rating scale

and symptoms of General Anxiety Disorder as reported by the children on the 'Terry' picture questionnaire.

Null hypothesis 1.4 must be rejected, as there was a statistically significant correlation between the symptoms of Inattention as reported by the teachers on the DBD rating scale and symptoms of Major Depressive Disorder as reported by the children on the 'Terry' picture questionnaire.

Null hypothesis 1.5 must be rejected, as there was a statistically significant correlation between the symptoms of Hyperactivity/Impulsiveness and symptoms of ODD as reported by the teachers on the DBD ratings scale.

Null hypothesis 1.6 must be rejected, as there was a statistically significant correlation between the symptoms of Hyperactivity/Impulsiveness and symptoms of CD as reported by the teachers on the DBD ratings scale.

Null hypothesis 1.7 must be accepted, as there were no statistically significant correlation between the symptoms of Hyperactivity/Impulsiveness and the symptoms of General Anxiety Disorder as reported by the children on the "Terry" picture questionnaire.

Null hypothesis 1.8 must be accepted, as there was no statistically significant correlation between the symptoms of Hyperactivity/Impulsiveness and the symptoms of Major Depressive Disorder as reported by the children on the "Terry" picture questionnaire.

8.4.2 Research hypothesis 2

The specific null hypothesis can be rejected or accepted as follows:

Null hypothesis 2.1 can only be partially rejected as there were significantly more symptoms of ODD symptomatology in the ADHD-PI boys, the ADHD-HI girls and the ADHD-C for both genders only. No significant more symptoms for ODD were found in the ADHD-HI boys and ADHD-PI girls, when compared to non-ADHD controls.

Null hypothesis 2.2 can only be partially rejected as there were significantly more symptoms of the ADHD-HI and ADHD-C subtypes only when compared to non-ADHD controls. There were no significantly more CD symptomatology when the ADHD-PI group was compared with the non-ADHD controls. This was the case for both genders.

Null hypothesis 2.3 can be partially accepted as only the ADHD-PI group showed significantly more symptoms of anxiety than the non-ADHD controls. No significantly more symptoms of anxiety were found when the other two ADHD groups were compared with their non-ADHD comparisons.

Null hypothesis 2.4 can be partially accepted as only the ADHD-PI group showed significantly more symptoms of anxiety than the non-ADHD controls. No significantly more symptoms of depression were found when the other two ADHD groups were compared with their non-ADHD comparisons.

A discussion of the obtained results is given in the next chapter.

Chapter 9

DISCUSSION OF RESULTS

9.1 Introduction

Research studies have shown that Attention-Deficit/Hyperactivity Disorder (ADHD) frequently co-occurs with other childhood psychiatric disorders (Biederman et al., 1998; Faraone et al., 2001; Volk et al., 2005). Acosta et al. (2004) state that externalising disorders, such as Conduct Disorder (CD) and Oppositional Defiant Disorder (ODD) are most frequent, with an estimated 20% of children diagnosed with ADHD also having CD and 30% - 15% having ODD. Acosta et al. (2004) further argue that children with ADHD also exhibit Mood and Anxiety Disorders. Comorbidity may occur as a result of overlapping symptomatology, one disorder manifesting itself as an earlier form of the other, or from shared risk factors (Volk et al., 2005).

The main aim of the present project was twofold:

1. To establish a relationship between the core symptoms of ADHD (Inattention and Hyperactivity/Impulsiveness) and the externalising disorders, ODD and CD, and the internalising disorders, Anxiety and Depression.
2. To determine whether there are differences in the symptoms of externalising disorders (ODD and CD) and internalizing disorders (anxiety and depression) between a group of children with ADHD symptoms and a non-ADHD control group as a function of subtypes and gender.

9.2 Results of the correlation study

The first phase of this investigation was to establish a relationship between the core symptoms of ADHD (Inattention and Hyperactivity/Impulsiveness) and the externalising disorders, ODD and CD, and the internalizing disorders Anxiety and Depression. To investigate whether the symptoms of ADHD (Hyperactivity/Impulsiveness and Inattention) are related to the externalising disorders ODD and CD and to the Internalising Disorders Anxiety and Depression, the scores were correlated, using Pearson's product-moment.

The obtained correlations and their significance are as follows (Table 9.1):

Table 9.1 ***Relationships between ADHD symptoms and Externalising and Internalising Disorders***

	Externalising		**Internalising**	
	CD	**ODD**	**Anxiety**	**Depression**
Hyperactivity/ Impulsiveness	r = 0.69 p = 0.00	r = 0.76 p = 0.00	r = 0.11 n/s	r = 0.06 n/s
Inattention	r = 0.48 p = 0.00	r =0.74 p = 0.00	r = 0.38 p = 0.00	r = 0.43 p = 0.00

The obtained results of the relationship between the symptoms of ADHD (Inattention and Hyperactivity/Impulsiveness) and Externalising Disorders (ODD and CD) and Internalising Disorders (Anxiety and Depression) can therefore be summarized as follows (Table 9.2):

Table 9.2 ***Relationship between the symptoms of ADHD and Comorbid Disorders (Externalising and Internalising)***

	Inattention	**Hyp/Imp**
ODD	Strong	Strong
CD	Moderate	Moderate to strong
Anxiety	Weak	No relationship
Depression	Weak to moderate	No relationship

9.2.1 Discussion of the relationships

9.2.1.1 Externalising Disorders

9.2.1.1.1 ODD

Inattention and ODD

There was a strong relationship between the symptoms of Inattention and the symptoms of ODD as reported by the teachers on the DBD rating scale. This can be explained by the fact that ODD usually is comorbid with ADHD (Barkley, 2006; Hinshaw & Lee, 2003). It is well established that children with ADHD display a greater degree of difficulties with oppositional behaviour than typical children (Loney & Milich, 1982; Pliszka & Carlson, 1999).

Studies suggest that 55% of children with ADHD will meet full diagnostic criteria for ODD either alone or with CD (Barkley, 2006; Wilens, Biederman, Brown, Tanguay, Monuteaux & Blake et al., 2002), and as most children with ADHD have symptoms of inattention, the relationship between these symptoms and those of ODD can be explained. Sagvolden et al., (2005) state that ODD develops from ADHD, while other researchers regard it as a separate entity (Banaschewski et al., 2003; Faraone et al., 1998). ODD is mainly associated with ADHD-HI and ADHD-C. The latter subtype, of course, displays the diagnostic criteria for Inattention, which may explain the relationship. However, according to Burns and Walsh (2002) symptoms of hyperactive-impulsive behaviour (but

not inattention) do predict later ODD symptoms. However, Nigg et al., (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.

Hyperactivity/Impulsiveness and ODD

There was also a strong relationship between the symptoms of Hyperactivity and the symptoms of ODD. This outcome was expected as oppositional behaviour is associated with ADHD-HI and ADHD-C in many studies (Barkley, DuPaul & McMurray, 1990; Barkley, 2006). Symptoms of Hyperactive-Impulsive behaviour (but not inattention) do predict later ODD symptoms (Burns & Walsh, 2002; Crystal et al., 2001; Willcutt, Pennintgton, Chhabildas, Friedman & Alexander, 1999) and the combination of the two increases the stability of ODD from preschool to the school age period (Lavigne et al., 2001; Speltz et al., 1999). August et al., (1999) and Barkley (2006) state that ODD by itself in samples with ADHD is not a precursor to later CD and may not be especially stable over later development. According to Gadow and Nolan (2002) ADHD and ODD are distinct disorders, when they coexist, the features are largely addictive rather than unique to the comorbid group.

9.2.1.1.2 CD

Inattention and CD

There was a moderate relationship between the symptoms of Inattention and the symptoms of CD. This can explained by the fact that Inattentive type of ADHD is infrequently associated with the disruptive behaviour disorders of CD (Barkley, 2006; Pliszka et al., 1999). Wenar and Kerig (2000) reported that children with the Inattentive type of ADHD are at less risk than children with the Hyperactive-Impulsive type for delinquency, school suspension or expulsion.

According to Lahey et al. (2000) the combination of ADHD and CD is associated with an earlier age of onset for CD and more persistent and serious conduct problems. ADHD with CD is a more severe subtype of ADHD in which the outcomes are often worse than those seen in ADHD alone (Barkley et al., 2004). Unless signs of early aggressiveness or other CD features are present, children with ADHD do not seem to be more prone to developing CD or to greater antisocial activities in later life, even if they have ODD (Barkley et al., 2004; Lynam, 1998).

Hyperactivity/Impulsiveness and CD

There was a moderate to strong relationship between the symptoms of Hyperactivity/Impulsiveness and the symptoms of CD. Children with comorbid ADHD and CD appear to have higher levels of Impulsiveness than children with only ADHD (Lynam, 1998). Newcorn, Halperin, Jensen, Abikoff, Arnold and Cantwell et al. (2001) state that they also have more impulsive behaviour than hyperactive behaviour.

This implies that the presence of comorbid CD in ADHD augurs for a more severe form of ADHD (Barkley, 2006). Most investigators express the belief that ADHD-HI and ADHD-C are precursors of CD (American Academy of Pediatrics, 2000; Kutcher, Aman, Brooks, Buitelaar & van Fegert et al., 2004; Taylor et al., 2004), another possibility is that the ADHD and CD combination represents a unique disorder or subtype of ADHD (Banaschewski et al., 2003).

9.2.2 Internalising Disorders

9.2.1.2.1 Anxiety

Inattention and anxiety

There was a weak relationship between the symptoms of Inattention and the symptoms of anxiety. This outcome was expected as anxiety symptoms have been associated with ADHD-PI in many studies (Barkley, 2006; Edelbrock , Costello & Kessler, 1984; Lahey et al., 1984; Lahey, Schaughency, Hynd, Carlson & Nieves, 1987). It is

estimated that 25% of children with ADHD will also have a coexisting anxiety disorder (American Psychiatric Association, 2000; Goldman, Genel, Bezman & Slanetz, 1998; Pliszka, 1998). Children with anxiety disorders have extreme feelings of fear, worry or panic and often seem agitated. According to Spencer, Biederman, and Wilens, (1999) other behavioural features include tantrums, attention seeking, overdependence and rituals.

According to the DSM-IV-TR (American Psychiatric Association, 2000) preliminary studies suggest that these coexisting conditions are more frequent in children with the predominantly inattentive and combined subtypes of ADHD. The MTA project which studied 489 clinic, referred children with ADHD-C found that those with associated Anxiety Disorders demonstrated significantly greater levels of Inattention than Impulsiveness relative to those children not having an Anxiety Disorder (March, Swanson, Arnold, Hoza, Conners & Hinshaw et al., 2000).

Hyperactivity/Impulsiveness and anxiety

There was no relationship between the symptoms of Hyperactivity/Impulsiveness and the symptoms of Anxiety. This can be explained by the fact that the nature of anxiety symptoms among children with the comorbid conditions does not appear to differ from those seen in children who have only an Anxiety Disorder (Tannock et al., 2000), though few studies exist on this issue. March et al. (2000) seem to suggest that anxiety in ADHD-C may be more closely related to ODD and generally disruptive behaviour than to fearfulness. According to Barkley (2006) the presence of anxiety with ADHD in some studies does seem to alter the expression of ADHD.

Anxiety was associated with a significantly reduced level of impulsiveness below that seen in children with ADHD but without anxiety, in some studies, though the latter remained more impulsive than non-disabled children (Pliszka, 1998; Pliszka, 1992; Tannock et al., 2000). Tannock et al. (2000) reported that other researchers have not found

this to be the case or have not even found the opposite. Consistent with the notion that anxiety decreases impulsiveness, were the findings of the above mentioned MTA project that ADHD children with associated Anxiety Disorders demonstrated significantly greater levels of Inattention than Impulsiveness relative to those children with ADHD not having an Anxiety Disorder (March et al., 2000).

9.2.1.2.2 Depression

Inattention and depression

There was a weak to moderate relationship between the symptoms of Inattention and the symptoms of Depression. This can be confirmed by the fact that a review of literature on the comorbidity of Major Depressive Disorder (MDD) and ADHD cases found a range between 15% and 75% of ADHD associated with depression (Spencer et al., 2000). Most studies reported rates of 9-32% of ADHD comorbid with MDD. Depression is strongly associated with ODD/CD and with anxiety, raising the possibility that the presence of one of these latter disorders is what mediates the relationship between ADHD and MDD (Biederman et al., 1991).

This was also suggested in the Fischer, Barkley, Smallish, and Fletcher (2002) follow-up study, where lifetime CD predicted occurrence of MDD. Angold et al. (1999) provided evidence, where the association of ADHD with depression was greatly reduced when the investigators controlled for comorbidity of ADHD with ODD/CD. With anxiety, depression may be a phenomenon that arises because of the association of ADHD with ODD/CD and ADHD with Anxiety (Angold et al., 1999). Barkley (2006) states that in the absence of these other two types of disorders, ADHD may not have an association with depression. The comorbidity of depression with ADHD is often associated with a poorer outcome than disorder alone (Spencer et al., 2000).

Hyperactivity/Impulsiveness and depression

There was no relationship between the symptoms of Hyperactivity/Impulsiveness and the symptoms of Depression. Blackman, Ostrander and Herman (2005) reported that the relationship between ADHD and depression cannot be attributed to the shared association that both disorders have with anxiety or conduct symptoms. Crystal et al. (2001) hypothesize that the high level of depression displayed by children with ADHD does not seem to be differentiated when comparisons are made between the inattentive and combined subtypes of ADHD.

According to Blackman et al. (2005), children with ADHD and comorbid depression have similar levels of inattention and hyperactivity-impulsivity when compared to their non-depressed ADHD counterparts. ADHD in combination with high levels of depression is associated with a number of negative outcomes, including unusually high rates of suicide, aggression and psychiatric hospitalization (Biederman et al., 1996; Treuting & Hinshaw, 2001; Ostrander, Crystal & August, 2006). Costello et al. (2006) state that ADHD is first manifested very early in development before age 7; in contrast, depression is one of the latest developing disorders of childhood. A study by Mannuzza et al. (1993) found that 23% of their hyperkinetic children had a lifetime diagnosis of depression in adulthood.

9.3 Results of the comparison study

The results of the comparison study between the ADHD subtypes and a non-ADHD control group for Externalising Disorders are summarised in Table 9.3. As there was a main effect of gender, the gender groups were analysed separately.

Table 9.3 ***Summary of results for the comparison of externalising symptoms***

Gender	Comparison	ODD p	CD p
Boys	ADHD-HI v. non-ADHD	n/s	0.01*
	ADHD-PI v. non-ADHD	0.00**	n/s
	ADHD-C v. non-ADHD	0.00**	0.00**
Girls	ADHD-HI v. non-ADHD	0.00**	0.00**
	ADHD-PI v. non-ADHD	n/s	n/s
	ADHD-C v. non-ADHD	0.00**	0.05*

* $p \leq 0.05$; **$p < 0.001$

The results for the Internalising Disorders are depicted in Table 9.4. There was no effect of gender, neither main nor interacting, therefore the gender groups were not analysed separately.

Table 9.4 ***Comparison of symptoms of Internalising Disorders between the ADHD and control groups***

Comparison	Anxiety p	Depression p
ADHD-HI v. non-ADHD	n/s	n/s
ADHD-PI v. non- ADHD	0.00*	0.00*
ADHD-C v. non-ADHD	n/s	n/s

*$p < 0.001$

9.3.2 Discussion of the results

9.3.2.1 Externalising Disorder: ODD

There were significant differences in the symptoms of ODD when the ADHD subtypes were compared with the non-ADHD controls. The ADHD-PI and ADHD-C boys had significantly more ODD symptoms than the non-ADHD control boys. However, there were no significant differences in ODD symptomatology between the ADHD-HI

boys and the non-ADHD controls. When the girls were compared, the ADHD-HI and ADHD-C subtypes showed significantly more symptoms of ODD while there were no significantly more symptoms of ODD when the ADHD-PI girls were compared with the non-ADHD controls. According to the literature (Barkley 2006; Biederman & Faraone, 2005; Taylor et al., 2004) ADHD-HI can be positively linked to ODD. Therefore, the obtained results in the ADHD boys were not expected, as ADHD-HI is a developmental precursor to ODD/CD (Barkley, 2006; Burke, Loeber, Lahey & Rathouz, 2005).

Symptoms of hyperactive/impulsive behaviour (but not Inattention) do predict later ODD symptoms (Burns & Walsh 2002), and the combination of the two increases the stability of ODD from preschool to the school age period (Lavigne et al., 2001; Speltz et al., 1999). Barkley (2006) postulated that ADHD may even cause or at least contribute to the risk for ODD alone, but other studies suggest that ODD by itself in samples with ADHD is not a precursor to later CD and may not be especially stable over later development (August et al., 1999).

Barkley (2006) asserts that ODD alone declines significantly with age, while CD increases with age. It is only the combination of ODD with CD that is likely to explain the persistence of ODD into adolescence (Maughan et al., 2004). Gadow and Nolan (2002) hypothesize that ADHD and ODD are distinct disorders, when they coexist; the features are largely additive rather than unique to the comorbid group. Therefore, the early onset and persistence of CD symptoms, which often co-occur with ODD symptoms, are the hallmark of the unique group with comorbid ADHD and ODD/ CD (Barkley, 2006).

The fact that the ADHD-HI group had less ODD symptoms than the ADHD-C group can be interpreted as follows; the combined type is a more severe form of ADHD, and that the children classified as such are more at risk for developing the Aggressive Behaviour Disorders. The findings of the current study indicate that boys with ADHD-PI

have more symptoms of ODD than the non-ADHD control groups. As Inattention is closely related to hyperactivity, being two dimensions of the same disorder, it is not unexpected that children with ODD, like the ADHD-HI and CD groups, also should have some symptoms of Inattention, although not to the same degree as the ADHD-PI and ADHD-C subtypes (Holmes, Payton, Barrett, Harrington, McGuffin & Owen et al., 2002; Tremblay & Schultz, 2000). This finding was unexpected, since Inattention is not usually positively linked to Aggressive Behaviour Disorders (Hinshaw & Lee, 2003; Lahey et al., 1999; Pliszka, 1999).

In most research findings ODD symptoms are not associated with Inattention (Hinshaw & Lee, 2003; Loeber et al., 2000). There were significant differences between the ADHD-HI boys and ADHD-PI boys on the ODD scale with the ADHD-PI subtype having more symptoms of ODD. These findings are surprising, as the Aggressive Behaviour Disorders are usually associated with Hyperactivity and Impulsiveness, and not with Inattention (Hinshaw & Lee, 2003; Loeber et al., 2000). However, the girls followed the expected pattern, with the ADHD-HI girls showing more ODD symptoms than the non-ADHD controls.

The ADHD-C subtype for both genders had significantly more symptoms of ODD than the non-ADHD control group. The results of the current study suggested that ADHD-C children have a greater variety of problems and more significant behavioural and social problems. Therefore, they may have more comorbid psychiatric disorders as a result. Crystal et al. (2001) and Willcutt et al., (1999) postulated that children with the ADHD-C subtype manifest more oppositional and aggressive symptoms, a greater likelihood of having ODD and CD, and more peer rejection than children with ADHD-PI . The results of the current study for both boys and girls are in accordance with research findings by McBurnett, Pfiffner and Frick (2001), who found that children with ADHD-C are

described as more noisy, disruptive, messy, irresponsible and immature; in contrast, children with ADHD-PI are characterized as more day-dreaming, hypoactive, passive, apathetic, lethargic, confused, withdrawn and sluggish. Children with ADHD-C are at significantly greater risk for ODD and CD, academic placement in programs for behaviourally disturbed children, school suspensions and psychotherapeutic interventions than are children with ADHD-PI (Barkley, 2004).

CD:

There were significantly more symptoms of CD in the ADHD-HI and ADHD-C subtypes for both genders when they were compared with their non-ADHD controls. However, significantly less CD symptoms were found when the ADHD-PI group was compared with their non-ADHD comparisons.

The study showed that children with ADHD-HI have more symptoms of CD than the non-ADHD control group. This result is consistent with those of other researchers (Blair, Colledge & Mitchell, 2001; Moeller, Barratt, Dougherty, Schmitz & Swann, 2001) which further suggest that impulsiveness may act as an underlying mechanism for the development of ADHD and CD, and their comorbidity.

A study by Blair et al. (2001) reported that the relationship between ADHD and psychopathology was primarily due to the association between the impulsiveness-conduct problems component of CD and the Hyperactivity-Impulsiveness component of ADHD. The current results are also supported by the findings of McGee and Williams (1999). Willcutt et al., (1999) argue that CD-type problems are specifically related to hyperactivity, more so than to Inattention. This was confirmed by Hinshaw and Lee (2003) Lahey et al., (1999) and Volk et al. (2005).

Hinshaw (1987) concluded that the hyperactivity component of ADHD and the impulsive-conduct problems of CD are indeed unique but correlated processes. One

explanation for the covariation of symptoms of hyperactivity and impulsive-conduct problems of CD is that they share an underlying mechanism (Patterson, DeGarmo & Knutson 2000) which has been purported to be impulsiveness (Moeller and Dougherty, 2001). Therefore, the results suggest that impulsiveness is a risk factor for both ADHD and core features of CD. Furr, Dougherty, Marsh and Williams (2007) and Mathias, Stanford, Marsh, Frick, Moeller and Swanson (2007) hypothesize that if impulsiveness does act as an underlying mechanism of both ADHD and CD, consideration of which particular aspects of impulsiveness account for the relationship between ADHD and CD is needed.

Impulsiveness is a multidimensional construct (Dougherty, Bjork, Harper, Mash, Moeller & Mathias, 2003; Patterson, Littman & Bricker, 1967) composed of behavioural processes that are response initiation, response inhibition, and consequence sensitivity, that are distinct in terms of operationalisation, neuroanatomical substrate, responsivity to treatment and role in clinical syndrome (Dougherty et al., 2003).

The ADHD-PI subtype did not differ significantly from the non-ADHD control group. This was the case for both genders. This supports the findings that Conduct Disorder is less associated with inattention than with hyperactivity and impulsiveness (Holmes , Slaughter, & Kashani, 2001; Johansen et al., 2002).

The ADHD-C subtype had more symptoms of CD when compared to the non-ADHD control group. The significantly more symptoms of CD of the ADHD-C subtype showed that this group is more severely affected and they are at a greater risk to develop symptoms of Conduct Disorder. Tervo, Azuma, Fogas, and Fiechtner (2002) postulated that children with ADHD-C, but not those with ADHD-HI are significantly more impaired on measures of Executive Functions and motor deficits. In addition, Barkley (1997) asserts that Executive Function deficits are central to the conceptions of ADHD-C. The study conducted by Nigg et al., (2002) showed that ADHD-C is also associated with

low IQ when compared with the non-ADHD control group. The current results support the findings by Hodgens, Cole and Boldizar, (2000) that children with the ADHD-C subtype are more prone to fighting and arguing, whereas children of the ADHD-PI subtype are more shy. It is clear from these findings that when there are more symptoms of hyperactivity/impulsiveness present, the higher the score on the CD scale (Connor et al., 2003; Holmes et al., 2001; Rey, Sawyer & Prior, 2005).

Gender differences

ODD appears to be more prevalent in males than females before puberty in the general population (American Psychiatric Association, 2000; Swanson, 2003; Whitaker, Van Rossem, Feldman, Schonfeld, Pinto-Martin & Tore et al., 1997). There is an increased risk of behavioural problems in both genders but this becomes evident at an earlier age in boys than in girls (Avery, Fletcher & MacDonald, 1999). No gender differences were determined for CD. The findings are that across all cultures boys tend to have more externalising problems and girls more internalising problems (Crijnen, Achenbach & Verhulst, 1997; Heptinstall & Taylor, 2002).

The results of the current study are in accordance with research findings that also found that girls lower levels of impulsiveness and inattention may not have been sufficient to engender consistent coercive and counter-coercive peer processes requisite to systematic training in aggression and defiance (Snyder, Prichard, Schrepferman, Patrick & Stoolmiller, 2004). According to Snyder et al., (2004) impulsiveness and inattention simply have a different impact on girls primarily in same-gender peer groups in which cooperation, accommodation and coordinated social exchange are highly valued.

Girls who are unable to successfully instigate and negotiate such skilful exchanges because of impulsiveness and inattention may be disliked and ostracized, but in indirect and less blatantly coercive ways than among boys (Grotpeter & Crick, 1996). Although

girls are clearly engaged in direct verbal and physical aggression in the peer group, it is less frequent and less clearly linked to impulsiveness and inattention (Huang-Pollock & Nigg, 2003) than for boys. There is strong evidence from the meta-analytic study that was done by Gaub and Carlson (1997) that ADHD in males and females is associated with a number of functional impairments and psychiatric morbidity across childhood, adolescence and adulthood.

There are however, specific gender-related differences in the clinical manifestation of ADHD and related problems which vary as a function of referral source (community versus clinical sample). In community samples, girls are rated as less impaired than boys in core DSM-IV symptoms of inattention and hyperactivity/impulsivity by both parents and teachers (Gershon, 2002; Rasmussen, Neuman, Heath, Levy, Hay & Todd, 2002; Rietveld et al., 2004).

By contrast, in clinically-referred samples, females with ADHD are often rated as more impaired in attentiveness than males with ADHD with an increased likelihood of having the DSM-IV predominantly inattentive subtype (Biederman, Mick, Faraone, Braaten, Doyle & Spencer et al., 2002; Gershon, 2002). From the meta-analysis study of Gaub and Carlson (1997) it was found that girls with ADHD were less likely to demonstrate externalizing symptoms (i.e. aggression, defiance and conduct problems).

Levy et al. (2005) postulated that ADHD was more prevalent among males for all three subtypes, with the male-female ratios for the combined type being approximately twice that of the hyperactive/impulsive and inattentive subtypes. Graetz, Sawyer, Hazell, Arney and Baghurst (2001) found the male-female ratio for the hyperactive/ impulsive (1.7:1) and combined (4.6:1) subtypes to be somewhat lower and higher, respectively, than in previous community-based studies. Boys have been shown to be at an increased risk of DSM-III ADHD (Faraone et al., 1995) but only in families exhibiting antisocial disorders.

Faraone et al., (1995) postulate that gender differences might provide clues to the genetic heterogeneity of ADHD. A comparison of gender differences in observed classroom behavior of 403 boys and 99 girls with ADHD (Abikoff et al., 2002) showed that boys with ADHD engaged in more rule-breaking and externalizing impulsive behaviours (disruptive behaviour disorders) than did girls with ADHD. Gaub and Carlson (1997) in their meta-analysis of gender differences in ADHD, found that non-referred girls with ADHD displayed lower levels of inattention, internalizing behaviour and peer aggression than boys with ADHD, while clinic-referred samples displayed similar levels of impairment and comorbidity.

Gershon (2002) has pointed out that identification of ADHD females has proven difficult, as fewer females than males are evaluated in ADHD clinics. He points out that while epidemiological samples estimate gender differences at 3:1, clinical samples range closer to 9:1. This compares with the findings of Graetz et al., (2001) of the male-female ratio for hyperactive/impulsive (1.7:1) and combined subtypes (4.6:1) in an Australian sample. Gershon (2002), indicates that girls manifested significantly fewer externalizing problems but significantly more internalizing problems than boys with ADHD; the latter in contrast with the findings of Gaub and Carlson (1997).

Levy, Hay and Bennett (2005) postulate that girls with ADHD performed worse on Full Scale IQ and Verbal IQ. Teachers rated girls with ADHD as less inattentive and having fewer externalizing problems than boys with ADHD. Clinic-referred samples tende to manifest more severe symptoms than community samples. Gaub and Carlson (1997) also found a possible gender bias in rating scales, with a tendency to rate all boys higher, but despite this, there were large gender differences in the manifestation of ADHD symptoms and correlates.

9.3.2.2 Internalising disorders

This study investigated the quantity of internalizing psychological symptoms, namely general anxiety and major depression as reported by ADHD children in comparison to the non-ADHD cohort.

The ADHD-PI children of both gender groups reported significantly more symptoms of anxiety, while the ADHD-HI and ADHD-C groups did not have significantly more anxiety symptoms than the non-ADHD controls. This finding is consistent with most research findings (Barkley, 2006; Baldwin & Dadds, 2007; Biederman et al., 1996c; Crystal et al., 2001; Levy, 2004; Pliszka et al., 1999; Schatz & Rostain, 2006). Although most authors also link comorbid anxiety to female gender (Bauermeister et al., 2007; DuPaul et al., 1994; Gadow et al., 2002; Newcorn et al., 2001; Pliszka et al., 1999; Tannock, 2004), the present study did not find a difference in anxiety symptoms between the genders.

According to Brown (2000) and Pliszka et al. (1999) anxiety may inhibit impulsiveness while worsening inattention, explaining the present study findings of anxiety linked to the ADHD-PI subtype. Pliszka et al., (1999) showed that children with ADHD and anxiety are less likely to display off-task and hyperactive behaviour and have longer reaction times than pure ADHD children. There may be a separate subtype of ADHD with comorbid anxiety that is pathogenically different from other strains of ADHD and this subtype may have decreased impulsiveness but increases in attentional measures (Jensen and Cantwell, 1997).

The presence of comorbid ADHD and anxiety is associated with more attentional problems, school fears, mood disorders and lower levels of social competence compared to children who had ADHD only (Bowen, Chavira, Bailey, Stein, & Stein 2008). Some

authors maintain that there is a subtype of ADHD called Sluggish Cognitive Tempo (SCT) subtype (Carlson & Mann, 2002; Schatz & Rostain, 2006). When children with ADHD-PI were divided into two groups: those with high SCT and those with low SGT and these groups were compared, the group with SCT showed higher levels of anxiety and depression (Carlson and Mann, 2002). Hartman, Willcutt, Rhee and Pennington, (2004) also found that the SCT subtype had less hyperactivity but more inattention and anxiety than either the ADHD-C or ADHD-HI subtype.

During the past 30 years, many explanations for the phenomenology of ADHD have been forwarded (Schatz & Rostain, 2006). Some of these theories also explain comorbid anxiety:

1. Slowed information processing, particularly during inhibition of response to rewards leads to failure to conform with social and academic norms, leading to subsequent anxiety (Schachar et al., 1995).
2. Dysregulation between posterior scanning and anterior executive attentional system may lead to anxiety through overactivity of the posterior system and may explain peripheral noradrenergic dysfunction in ADHD (Posner and Peterson, 1990).
3. The inhibition during Executive Functions is deficient which may cause poor performance, leading to anxiety (Barkley, 1997).
4. Poor regulation of both attention and affect may lead to increased attention to noxious stimuli and decreased regulation of accompanying affect (Brown, 2000).
5. Dysfunctional response to rewards and fear/anxiety because of accumbens-striatum-orbitifrontal circuits lead to socially inappropriate behaviour and as a result anxiety and increased nonspecific arousal states (Quay, 1998).

6. Effort, arousal, and activation are deficient in ADHD and non-specific anxiety may partially lead to the disruptive arousal and activation seen in ADHD (Sergeant, 2000).

7. There is impaired physiology of the nucleus accumbens, leading to dysfunction in the mesolimbic, mesocortical, and nigrostriatal circuits which causes poor regulation of anxiety states, poor gating of stimuli leading to anxiety, and deficient harnessing of attention away from anxiety-provoking stimuli (Levy, 2004).

Besides these theories or models, other investigators explain ADHD comorbid with anxiety in a somewhat different way:

Baldwin and Dadds (2007) state that the inattention component of ADHD and anxiety symptoms are covarying phenomena that are linked with an irritable temperament and disruptive behaviour. ADHD with comorbid anxiety is also associated with maternal and family characteristics that are different from both controls and non-anxious ADHD children. The presence of ADHD along with comorbid anxiety disorder is associated with a family environment that is usually insular, dependent, and discouraging of autonomy. Comorbid anxiety can also be associated with relatively high rates of maternal alcohol and substance abuse (Kepley & Ostrander, 2007).

Depression

Only the ADHD-PI group displayed statistically more significant symptoms of depression in comparison with their non-ADHD counterparts. No differences in symptoms of depression were found between the ADHD-HI and ADHD-C subtypes when compared to the non-ADHD controls. Like in the case of anxiety, it seems that symptoms of depression are linked to the inattention component of ADHD. This observation coincides with similar research by Hurtig, Ebeling, Taanila, Miettunen, Smalley

and McGough et al. (2007) who found that inattentive symptoms, MDD comorbidity, and a family history of attention problems (fathers specifically) contribute to the risk of persistent ADHD.

Other studies also did find an association between inattention and MDD. Volk et al., (2005) found that the largest percentages of subjects meeting criteria for DSM-IV depression were concentrated in the ADHD-C and ADHD-PI subtypes. This was confirmed by Power et al. (2004) in their study. However, contrary to the findings of this study, the results indicated that children with ADHD-C and ADHD-PI had similar levels of depression (Crystal et al., 2001; Power et al., 2004; Volk et al., 2005). A relationship between depression and inattention was also postulated by others (Blackman et al., 2005; Johansen et al., 2002). The present study's results confirm that inattention explains the total influence of depression, suggesting that the relatively high levels of depression in the depressed ADHD group can be accounted for solely by the inattentive symptoms of ADHD (Blackman et al., 2005).

Although not specifically mentioning ADHD subtypes, many studies link ADHD to MDD (Acosta et al., 2004; Angold et al., 1999; Barkley, 2006; Blackman et al., 2005; Connor et al., 2003; Jensen, 2003; Keiley, Lofthouse, Bates, Dodge & Pettit, 2003; MTA-Cooperative Group, 1999; Pliszka et al., 1999; Treuting & Hinshaw, 2001). This suggests a clear comorbidity of ADHD with depression. However, depression is also strongly associated with ODD/CD (Barkley 2006; Biederman et al., 2008; Blackman et al., 2005; Ostrander & Herman, 2006) and with anxiety (Barkley, 2006; Blackman et al., 2005; Fischer et al., 2002). Youths with ADHD comorbid with MDD are at increased risk for suicide, due to their impulsiveness (Biederman et al., 1991; Cho, Kim, Choi, Kim, Shin, & Lee et al., 2007; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Plattner, Kraemer, Williams, Bauer & Kindler et al., 2007). Children with ADHD and depression also display

more impairment in social and academic functioning compared to children with only ADHD (Biederman et al., 1993; Blackman et al., 2005; Cabiya-Morales, Padilla, Sayers-Montalvo, Pedrosa, Perez-Pedrogo, & Manzano-Mojica, 2007; Jensen & Cantwell, 1997; Karustis, Power, Rescorla, Eiraldi & Gallagher, 2000).

Although the present study did not find any gender differences in the symptomatology between boys and girls with ADHD, most studies show that ADHD status is associated with depressive symptoms in boys but not in girls (Biederman et al., 2002; Bauermeister, Shrout, Chavez, Rubio-Stipec, Ramirez and Padilla et al., 2007; Ruchkin, Lorberg, Koposov, Schwab-Stone & Sukhodolsky, 2007; Tannock, 2004). However, others found that girls manifested significantly fewer externalizing problems but significantly more internalizing (both depression and anxiety) problems than boys (Gershon, 2002).

ADHD first manifests very early (before the age of 7); in contrast, depression is one of the latest developing disorders of childhood and rarely precedes any other comorbid disorder (Costello et al., 2006; Rohde et al., 1991). Therefore, ADHD with comorbid depression would have been probably more prevalent in an older sample as longitudinal studies have found ADHD individuals to have an increased risk of depression as development progresses (Biederman et al., 2006).

As in the case of comorbid anxiety, there are many theories about the nature of the relationship between ADHD and depression:

1. The combination of ADHD and a depressive disorder could represent a subtype of ADHD, with both shared and specific features related to aetiology, outcome and clinical presentation (Barkley 2006; Jensen, 2003; Keiley et al., 2003; Spencer et al., 2000).

2. Some authors have suggested that the association between depression and ADHD may be epiphenomenal, that is, attributed to the relationship that both disorders have with anxiety or Conduct Disorder (Angold et al., 1999).

3. Children with ADHD would be at greater risk for depression because of their poor overall social competence therefore a child's own self-appraisals of social competence are expected to mediate the relationship between depression and others' social appraisals (Hoffman, Cole, Martin, Tram & Seroczynski, 2000; Ostrander et al., 2006).

4. A hypofunctioning monoaminergic system is thought to underlie the clinical pictures of ADHD and depression. The neurocognitive deficits of depression include memory loss, poor concentration and rumination. ADHD is characterized by inattention, impulsiveness and hyperactivity. Both syndromes effectively respond to raising serotonin and dopamine levels, respectively. It is hypothesized that the core symptoms can be attributed to an imbalance between the neuromodulatory effects of monoamines and acetylcholine (Vakalopoulos, 2007).

9.4 Integration of results

Both the correlation and comparison studies did indicate that the ADHD group showed increased externalizing and internalizing psychological symptoms in comparison to their non-ADHD counterparts. The externalizing disorders (ODD and CD) were linked mostly to hyperactive/impulsive symptoms than to symptoms of inattention. However, it was found that symptoms of inattention were also indicated in ODD.

Most studies cited confirmed the findings of the present study, namely that the ODD component was specifically associated with hyperactivity/impulsiveness. The

association between inattention and ODD however, was only linked infrequently to ODD symptoms (Mathias et al., 2007), and mostly in cases where internalising disorders were also present (Blackman et al., 2005; Ostrander et al., 2006). The only finding on differences in symptomatology between the genders was that in the case of boys not hyperactivity/impulsiveness, but inattention was associated with ODD symptoms, which is difficult to explain, while in the case of girls the results were in line with the most observed results: both ODD and CD were associated with the hyperactive/impulsive component. An explanation might be that according to most investigators, boys with ADHD have a higher incidence of depression, in other words, members of the sample might be comorbid for both externalising and internalising disorders, hence the association with inattention.

The findings that internalising disorders were linked to the inattention component of ADHD were in line with most studies cited. The present study did not find gender differences in symptomatology for both anxiety and depression, which contradicts most studies cited. Usually girls with ADHD have more symptoms of comorbid anxiety, while boys have more comorbid depression (Tannock, 2004).

9.5 Psychological symptoms

Internalising and externalizing psychological symptoms have implications for all levels of functioning.

Internalising symptoms: symptoms of depression during childhood probably have adverse implications for social, emotional, cognitive and behaviour development. On a cognitive level, school performance of depressed children is affected by a combination of difficulty in concentration, slowed thinking, lack of interest and motivation, fatigue, sleepiness and preoccupations.

Anxiety disorder: it is apparent that anxiety occurs with any human experience, so it is not surprising that anxiety symptoms also occur in children**.** As the child develops,

there are many personal challenges that they must learn to deal with. However, some react differently to social, academic and familial challenges than others. Anxiety is a common symptom in many psychological disorders. An actual anxiety disorder can occur when anxiety is intense, prolonged, and more disruptive than normal. Anxiety disorders are among the most frequently diagnosed disorders in children (Yorbik, Birmaher, Axelson, Williams & Ryan, 2004).

Externalising disorders: The presence of ADHD in childhood is a major risk factor for the development of aggressive and antisocial behaviour (Taylor, Chadwick, Heptinstall and Danckaerts 1996). ADHD has been postulated to arise from a deficit in executive functioning (EF) (Barkley 1997; Pennington 1996 & Pennington 1996). EF encompasses meta-cognitive processes that enable efficient planning, execution, verification and regulation of goal-directed behavior (Oosterlaan, Scheres & Sergeant 2005).

The frontal cortex and its subcortical connections have been suggested to serve as the major neurological underpinnings for EF (Eslinger 1996; Lezak 1995; Pennington 1996). ODD/CD is seen as a possible precursor for antisocial behavior in adults (Lynam, 1998). CD has been linked to brain damage, genetic vulnerability, school failure, traumatic life experiences, and physical and sexual abuse during childhood (Schulenberg and Soundy 2000).

Aggressive children may display cognitive and behavioural social skills deficits when they interact with peers. For example, Webstratton and Lindsay (1999) found that children with conduct problems also showed more negative attributions, fewer prosocial problem-solving strategies, and a significant delay in their social skills during play interactions with friends, as compared to those without conduct problems. In a study, Gadow and Nolan (2002) found that preschoolers with ODD and ADHD had the highest scores for difficulties with peers and developmental deficits.

9.6 Limitations of the study

One of the limitations of this study is that the sample consisted of 100 participants, (50 children with ADHD and 50 non-ADHD controls). Generalisations from results obtained in this sample, particularly in the age groups, would be more reliable if a bigger sample had been used. The sample consisted of children attending rural schools and of the Tsonga culture only.

The "Terry" picture questionnaire needs to be standardized for local populations. Therefore, cultural and tribal factors should be taken into consideration as they may influence the results of the study.

The "Terry" picture questionnaire was administered to participants only for measurement of internalizing symptoms. Though children are sometimes considered as better reporters of internal distress (Chavira, Stein, Bailey & Stein, 2004), making use of multiple reporters may reflect a more holistic clinical impression of emotional functioning.

The socioeconomic status was not established which may have influenced the results of the study. Only the teacher rating scale was used, because many children did not live with their parents. They live with their caregivers and guardians. Moreover, parents are difficult to contact as many people in rural areas lack postal addresses and telephones. Some of the parents are illiterate.

9.7 Clinical Implications

ADHD is a chronic disorder of childhood that progresses into adult ADHD in a significant number of children. The clinical presentation of ADHD easily overlaps with other disorders of childhood. ADHD can also co-occur with other psychiatric conditions of childhood in a significant number of children. Therefore, for the clinician tasked with children's mental health, a thorough psychiatric history followed by both medical and neurological assessments should precede a diagnosis of ADHD in a child (Shokane, Rataemane & Rataemane, 2004).

It is important to have more assessment tools that measure anxiety and depression in order to predict internalized disorders: this will be essential in assisting clinicians to link internalising disorders that is, anxiety and depression with ADHD symptoms.

9.8 Suggestion for further Research

From the study, it was clear that certain factors, when considered, would improve the findings of this study in future research. Future research should be extended to a larger sample of many different ethnic groups from both rural and urban areas. More research should be conducted to determine whether the effect of comorbid ADHD, ODD, CD, anxiety, and depression would produce a difference on measures of executive and motor functions.

For future studies, it is further recommended that parents should help the teachers to fill in the DBD scale for the fairness of the diagnosis. The comparison of the DBD scored by the teachers, the caregivers, guardians and the parents would possibly provide more reliable information about the child (American Academy of Pediatrics, 2000). In addition to this, a full clinical examination by a psychologist should be carried out in order to arrive at an accurate diagnosis.

9.9 Conclusion

In conclusion, the study showed that there is a relationship between the symptoms of ADHD (hyperactivity/impulsiveness and inattention) and the externalizing disorders, ODD and CD. Only the Inattentive component of ADHD showed a relationship with internalizing disorders (anxiety and depression).

The comparison study showed that the predominantly hyperactive/impulsive and the combined subtypes displayed the most symptoms of externalizing disorders, while the predominantly inattentive subtype had significantly more symptoms of internalizing

disorders. Gender differences were observed in the symptoms of externalizing disorders, but not in the symptomatology of internalizing disorders.

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Appendix A: Letter to the school principals

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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

Appendix B: Letter to the parents

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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:

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 matiro 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)				

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				

APPENDIX E: EXAMPLE OF THE "TERRY" PICTURE QUESTIONNAIRE